Sorghum Root and Stalk Rots

A Critical Review

International Crops Research Institute for the Semi-Arid Tropics

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Proceedings of the Consultative Group Discussion on Research Needs and Strategies for Control of Sorghum Root and Stalk Rot Diseases

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Publication Editor

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Foreword

Despite several decades of research, diseases remain a major constraint to sorghum production throughout the world. That was the reason ICRISAT and Texas A&M University cosponsored the first international workshop on sorghum diseases, hosted by ICRISAT at Hyderabad, India, in December 1978. It was also the reason for a second major international effort exactly 5 years later, by INTSORMIL and ICRISAT, to gain more understand ing for better control of these diseases. This took the form of a highly specialized consultants' group meeting to make recommendations on the research needs and strategies for control of sorghum root and stalk rots.

Root and stalk rots are a group of diseases that reduce crop stands in the emergence and seedling stages, or most commonly cause stalk lodging in the postf lowering and grain-filling stages of plant development. The improved high-yield-potential varieties and hybrids under good management tend to be particularly susceptible to lodging i nduced by root and stalk rot. Although good progress has been made against other diseases of sorghum, research for control of root and stalk rots has been painfully slow. This is due to the complexity of the diseases themselves and the paucity of intensive interdisciplinary research on them.

ICRISAT and INTSORMIL are committed to supporting and conducting research that will provide the technology necessary for farmers to improve sorghum yields and help meet world food needs. Their joint sponsorship of the Consultative Group Discussion on Research Needs and Strategies for Control of Sorghum Root and Stalk Rot Diseases and the publication of these proceedings are important steps toward this objective. The contents of the background papers, the discussions, and the recommendations contained in this book represent the combined experience and knowledge of 27 scientists in the disciplines of breeding, physiology, and pathology. We feel certain that if national, international, and other sorghum improvement programs follow the various strategies and recommendations, significant progress will be made in this field.

ICRISAT expresses special appreciation to the scientists who participated in this meeting, to INTSORMIL for its help in funding the conference, and to the Rockefeller Foundation for hosting the meeting at its Study and Conference Center at Bellagio, Italy.

L.D. Swindale
Director General, ICRISAT

Inauguration

Opening Address and Objectives of the Meeting

L.K. Mughogho*

It is with great pleasure that I welcome you all to this Consultative Group Discussion on Research Needs and Strategies for Control of Sorghum Root and Stalk Rot Diseases. Our meeting here has been made possible by the encouragement and financial support of ICRISAT and INTSORMIL. We are most grateful to both institutions. We are also grateful to the Rockefeller Foundation for the use of their excellent conference facilities at this villa and the generous hospitality accorded to us. I would also like to thank the organizing committee at ICRISAT Center and Dr. R.A. Frederiksen of Texas A&M University, USA, for everything they did to make this meeting a reality.

It may appear strange that we are meeting to discuss sorghum diseases in a country not commonly known for sorghum production. Although this is not the right season to see sorghum, an examination of the FAO production figures for 1981 shows that Italy produced 84000 tons of sorghum grain on 15000 ha at a yield of 5600 kg/ha. This yield was eight times higher than the average of 700 kg/ha in the developing countries of Africa and Asia, where the crop is a major cerealfood crop. Diseases, including root and stalk rots, are partly responsible for this yield gap.

The idea to hold this discussion group meeting originated from a review in September 1980 of the ICRISAT sorghum charcoal rot research project. The discussions at that review emphasized the need for a multidisciplinary research thrust involving pathologists, physiologists, and breeders for effective control of the disease. As a starting point, we felt that a meeting of scientists familiar with the problem should be held to make recommendations for future research. During the planning stage other root and stalk rot diseases were included for discussion in order to provide a comprehensive and overall picture of the role and importance of various root and stalk rot pathogens.

Although a number of pathogens are implicated as causal agents of root and/or stalk rots in sorghum, little is known about (a) the etiology and epidemiology of the diseases they cause, (b) the plant physiological and environmental factors that predispose sorghum to infection and favor disease development, (c) host resistance and techniques to identify and utilize host resistance in breeding projects, and (d)

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the effect of various crop management practices on disease incidence. Effective control measures have not yet been devised, largely because of lack of or incomplete knowledge and understanding of the diseases concerned.

This meeting has three objectives: (a) to assess present knowledge and research activity, (b) to determine where gaps in knowledge exist, and (c) to plan and make recommendations for future research and strategies for effective control of root and stalk rot diseases of sorghum. The participants here include pathologists, physiologists/nutritionists, biochemists, and breeders with valuable experience in sorghum research. These disciplines represent the four major areas in which joint research is required that would eventually lead to the understanding and control of these diseases. We also have participants with research experience on similar diseases in maize and legumes.

Finally, I would like to emphasize that from this consultative group discussion we expect positive, realistic, and practical recommendations that can be taken up by national, regional, international, and other sorghum improvement programs for the eventual benefit of the sorghum grower. The wide experience and expertise assembled here will no doubt achieve this objective. We look forward to fruitful discussions and a pleasant stay at Bellagio.

Welcome from ICRISAT

Curtis R. Jackson*

You have my sincere apologies and personal regrets that I cannot be with you for this important conference. I have asked John Scheuring, our principal breeder in Mali, to read this short message for me on behalf of ICRISAT.

Sorghum is ICRISAT's most promising grain crop at this time, and we have 18 principal scientists working on this crop in Asia, Africa, and Latin America.

The traditional rainfed sorghum crops in the semi-arid tropics have yields ranging from 200 to 1000 kg/ha. But in good years we can easily obtain on-station yields exceeding 3.5 t/ha using F_1 hybrids or exotic inbreds with so-called high yield potential. Yet, as a general rule, the higher the yield potential under optimal conditions the greater susceptibility to stalk rot under stress conditions. In Mali we have seen CSH-5 yield over 4t/ha. We have also seen it fall 100% to charcoal rot. We've picked out excellent ICRISAT varieties one year only to watch them buckle under stalk rot the next year.

One might argue that even grain from lodged plants can still be harvested. In fact we've salvaged yields exceeding 2t/ha from completely lodged fields. But the grain is virtually worthless as food. Food grain, unlike feed grain, must have the quality to assure long term-on-farm storage, processing with minimal bran loss and acceptable food taste, texture, and keeping quality.

It is not by accident that local tropical sorghum cultivars are photoperiod-sensitive, with flowering usually at 75 to 100 days. Dry matter accumulation comes slowly in tropical latitudes compared to temperate latitudes. During the rainy season, night temperatures are relatively high and daylengths are 1 to 3 hours shorter than those in temperate latitudes. The higher night-time temperatures and shorter days translate into limited source size and accentuated source stress in high-yield-potential varieties.

In brief, the lessons we learn about stalk rots in temperate latitudes certainly can take us far in understanding stalk rots in the tropics. But let us take care to recognize the distinct differences of the plant environment and the grain uses between temperate and tropical sorghums. We may very well find that resistance mechanisms and critical source-sink thresholds are entirely different for temperate and tropical sorghums.

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Let us remember that the clientele of INTSORMIL and ICRISAT are farmers in the semi-arid tropics and that when it comes to sorghums growing in the tropics, particularly below 20° latitudes, most of us—breeders, physiologists, and pathologists alike — are only at the beginning of description, much less understanding.

There is no guru (master) among us. So let us be prudent and brief in our comments and let us carefully consider the comments of others.

Hopefully this collaborative exchange of ideas will lay the groundwork for solid collaborative research between scientists in temperate and tropical areas.

But collaborative research or no collaborative research—we must address the stalk rot question if we are to raise sorghum yield levels in the tropics.

We are pleased to have you here as our guests and guests of our near relative, INTSORMIL I hope you will have a productive and rewarding meeting. Please give us your best thoughts and actions.

Thank you.

Welcome from INTSORMIL

Earl R. Leng*

INTSORMIL is pleased to be a cosponsor of this important group discussion. Our stated mission is to conduct collaborative research on topics likely to be significant for enhancing sorghum and millet production in the developing world. There is no better way to insure collaboration than to bring together, as this meeting is doing, most of the world's experts on a particular problem.

Stalk rots remain one of the major detrimental influences on production of large-stemmed cereals such as sorghum, maize, and to some extent pearl millet. Many years ago, researchers were confident that problems with stalk rot would be quickly solved, largely by selection for genetic resistance. This hope has proved to be an illusion. Stalk rots of various types are with us almost everywhere, and solutions appear to be less near than we thought some thirty years ago.

For this reason, a group discussion such as this is really needed, since from it we should have a chance to focus research better, and maybe to make faster progress. Some may feel that stalk rots are more of a problem for developed than for developing countries, since they present particular difficulties for machine harvesting. But, probably no one in this group needs to be told what severe yield losses can result from stalk rot attacks, no matter how the crop is harvested. Therefore, it is clear that you will be dealing with a problem of major concern to the developing world.

To repeat, INTSORMIL is happy to have the opportunity to cosponsor this conference and to participate in its deliberations. We really hope that out of it will grow more productive research on the problem, which in turn will lead to improved sorghum production and a better life for sorghum-using peoples of the world.

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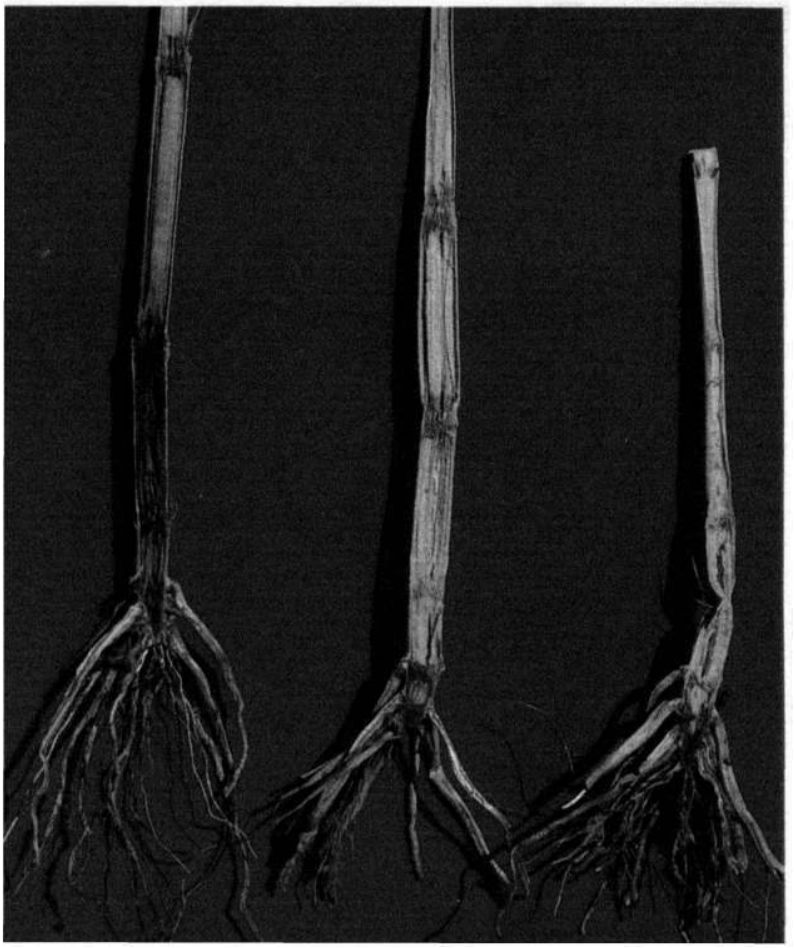
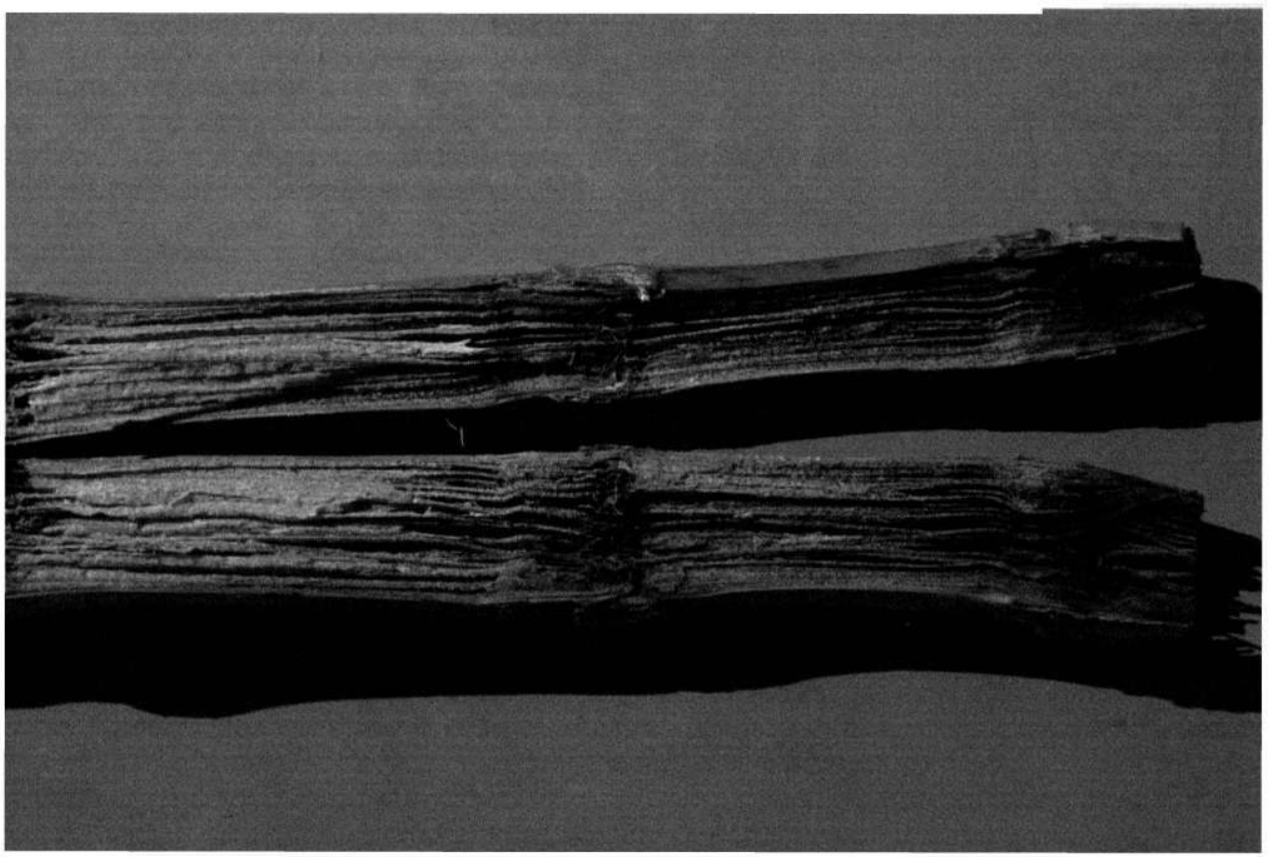
Basic Disease Problems



Charcoal Rot of Sorghum



Charcoal Rot of Sorghum

L.K. Mughogho and S. Pande*

Summary

Charcoal rot of sorghum caused by the fungus Macrophomina phaseolina *is a root and stalk rot disease of great destructive potential in most sorghum-growing regions. Improved, high-yielding cultivars under good management tend to be very susceptible to the disease. M.* phaseolina *is a common soilborne, nonaggressive, and plurivorous pathogen that attacks plants whose vigor has been reduced by unfavorable growing conditions. Drought stress is the primary factor that predisposes sorghum to charcoal rot. In diseased roots and stalks,* M. phaseolina *is often associated with other fungi, suggesting that the disease is of complex etiology. Control by fungicides, cultural practices, and host resistance are briefly discussed, and priority areas for future research are listed.*

Charcoal rot, caused by *Macrophomina phaseolina* (Tassi) Goid., is the most common and probably also the most important root and stalk rot disease of sorghum. Reviews by Tarr (1962), Dhingra and Sinclair (1977, 1978), and Sinclair (these proceedings) provide comprehensive information on the biology of *M. phaseolina* and the epidemiology and control of the diseases it causes in many plant species. Several papers in these proceedings (Sessions III, IV, and V) discuss in detail the physiological and environmental factors that influence charcoal rot and its control by fungicides, cultural practices, and host resistance. In this review, emphasis will therefore be on those aspects of the pathogen and disease that have or may have important implications in disease control and management.

Occurrence and Geographical Distribution

Charcoal rot is a worldwide disease: it has been reported from all the ecologically diverse areas of sorghum culture in the tropics, subtropics, and temperate regions (Tarr 1962, ICRISAT 1980). When inoculum is present, the occurrence of charcoal rot in a particular area is greatly influenced, like most plant diseases, by environmental conditions. It may be widespread in some years and localized or even absent in others. In India the disease occurs on sorghums growing in both red (Alfisol) and black (Vertisol) soils. In general the worldwide distribution of the disease would indicate its occurrence on many different soil types.

Symptoms

A variety of symptoms are associated with charcoal rot. These include root rot, soft stalks, lodging of plants, premature drying of stalks, and poorly developed panicles with small inferior-quality grain (Hsi 1956, Uppal et al. 1936),

The most striking and usually first indication of the disease is lodging of plants as they approach maturity. Lodging is due to the weakened condition of the stalk, caused by the disintegration of the pith and cortex by the pathogen, leaving the lignified fibrovascular bundles suspended as separate

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strands in the hollow stalk; hence "hollow stalk of sorghum" as the disease was first named by Uppal et al. (1936). The vascular bundles are profusely covered with tiny black sclerotia of the pathogen, which give the charcoal appearance to the affected area. Thus the name "charcoal rot" describes the appearance of the disease inside infected roots and stalks.

Sometimes charcoal rot symptoms are not easily noticeable. Harris (1962) reported that in Nigeria the disease escaped attention because symptoms were inconspicuous. Affected plants looked healthy but had much thinner stalks than normal and had very small panicles.

M. phaseolina may also infect seedlings, causing seedling blight or damping-off symptoms under moist and high temperature conditions (Uppal et al. 1936). There is also one report of the pathogen causing leafspot symptoms in sorghum (Raut and Bhombe 1972). Very little is known about these two phases of the disease.

Economic Importance

In order to determine the research needs and strategies for control of charcoal rot, a realistic definition of the problem with reference to crop losses is required. The literature contains many reports on the destruction of sorghum crops by charcoal rot, but sound and reliable quantitative data on yield losses are not given. Uppal et al. (1936) determined that the disease was of "sufficient economic importance" on postrainy-season crops in Maharashtra State, India. Harris (1962) reported that in Kano, Nigeria, charcoal rot caused "considerable loss in yield." In nearby Cameroon S.B. King and D. Barry (Major Cereals in African Project, Samaru, Nigeria, 1970; unpublished report of a trip to Cameroon and Chad) saw severe symptoms of charcoal rot in farmers' fields and estimated yield losses of over 50%. Similarly "serious losses" in several states in the USA were reported, but no quantitative data on crop loss were given (Leukel et al. 1951).

In spite of the lack of data on field crop losses, the destructive potential of charcoal rot in susceptible cultivars is unquestionable. Four types of crop losses may be recognized: (1) loss in grain yield and quality due to stunted plants, smaller stalks than normal, and premature drying; (2) poor crop stands due to seedling blight; (3) complete loss of yield in lodged plants where mechanical harvesting of grain is practiced, and where harvesting is manual, destruction of lodged plants by termites or other animal pests before the grain or fodder is collected; (4) loss in quality and quantity of fodder due to infection and destruction of the stalk.

Under experimental conditions we have obtained 100% lodging and grain yield losses of 23 to 64% in CSH-6 hybrid at three locations in India and one in Sudan (Table 1). In these experiments natural charcoal rot infection of plants was induced by subjecting them to drought by withdrawing irrigation at different growth stages; grain yield was determined from both lodged and standing plants. Although drought alone must have contributed to some yield reduction, the combined effects of drought and charcoal rot that caused plants to lodge must have greatly increased the level of yield

Table 1. Lodging and yield of charcoal-rot-infected CSH-6 sorghum under four moisture stress treatments at four locations in 1981.

Moisture stress treatments	Locations, lodging (%), and plot yield (kg/18 m^2)							
	ICRISAT Center (India)		Dharwar (India)		IMandyal (India)		Wad Medani (Sudan)	
	Lodging (%)	Yield	Lodging (%)	Yield	Lodging (%)	Yield	Lodging (%)	Yield
irrigation to grain maturity	8	2.2	7	3.3	1	3.0	3	2.1
Irrigation to 50% anthesis	42	2.2	86	2.5	2	2.0	5	1.9
Irrigation to boots swollen	46	1.8	100	2.1	36	1.7	56	1.9
Irrigation to ligule visible	55	1.6	100	1.7	47	1.1	73	1.6
SE±	3.54	0.08	1.92	0.10	3.51	0.17	3.13	0.10
Loss in yield (%) [a]	27		48		64		23	

a. $\frac{\text{Irrigation to grain maturity - irrigation to ligule visible}}{\text{Irrigation to grain maturity}} \times 100$

loss. At Dharwar a 35% reduction in 1000-grain weight was recorded when this technique was used. Similarly Anahosur and Patil (1983) reported 15-55% loss in grain weight in their experiments conducted at Dharwar. These data on grain yield losses clearly show the economic importance of the disease when it occurs as plants approach maturity. However, there is still need for more data, particularly on the various types of losses described above from surveys in farmers' fields.

Improved, high-yielding cultivars tend to be ultra-susceptible to charcoal rot. Improved varieties and hybrids that revolutionized sorghum production in India in the 1970s (Rao 1982) have proved very susceptible to the disease, with 100% lodging in severe cases (Nagarajan et al. 1970, Anahosur and Rao 1977, Avadhani and Ramesh 1979). In West Africa high-yielding exotic cultivars tend to be very susceptible to charcoal rot (J.F. Scheuring, ICRISAT/Mali Program, personal communication, Feb 1983). The susceptibility of improved cultivars to charcoal rot poses a serious problem for sorghum improvement programs worldwide, and a solution must be found that would enable farmers to.benefit from the use of improved cultivars.

Causal Organism

The causal organism of charcoal is a common soilborne fungus often known by its imperfect state *Macrophomina phaseolina* (Tassi) Goid. (Domsch et al. 1980). The perfect state is called *Sclerotium bataticola* Taub. Eight synonyms that may be encountered in the literature are: *Macrophomina phaseoli* (Maubl.) Ashby, *Macrophomina Philippines* Petr., *Macrophomina crochori* Sawada, *Macrophomina cajani* Syd. & Butl., *Macrophomina sesami* Sawada, *Rhizoctonia bataticola* (Taub.) Butl., *Rhizoctonia lamellifera* Small, and *Dothiorella cajani* Syd. & Butl. (Holliday and Punithalingam 1970).

Association with Other Fungi

In diseased roots and stalks with conspicuous signs of charcoal rot, fungal isolations usually reveal the association of *M. phaseolina* with other fungi. In Texas, USA, both *M. phaseolina* and *Fusarium moniliforme* were obtained in cultures of diseased stalks (Tullis 1951). Similar observations were made in Georgia, USA (Luttrell 1950), and in India(ICRISAT1983). In Argentina, where *F.moniliforme* was the predominant fungus isolated from lodged plants, 40% of the isolations were *M. phaseolina.* Other fungi isolated included unidentified *Fusarium* spp, *Rhizoctonia solani, Helminthosporium sativum, and Nigrospora sphaerica* (Frezzi and Teyssandier 1980). Similarly, in New South Wales, Australia, systematic surveys to assess the relative importance of fungi associated with root and stalk rots revealed that, although *F. moniliforme* was predominant, *M. phaseolina* and *N. sphaerica* were regularly isolated simultaneously from diseased roots and stalks (Trimboli and Burgess 1982).

Data cited above show clearly that in most cases of charcoal rot, *M. phaseolina* is not the sole cause of the disease under natural field conditions, but acts in combination with other pathogens to produce it. In other words, what is visually identified as charcoal rot is a sign of one fungus among several in a disease of complex etiology. Wadsworth and Sieglinger (1950) suggested that the several fungi associated with stalk rots attack in some orderly sequence, with *M. phaseolina* being the last and most conspicuous of the sequence. Leukel et al. (1951) also suggested that root and stalk invasion by *M. phaseolina* is preceded by *F. moniliforme. P.* Mayers (Department of Primary Industries, Queensland, Australia; personal communication, Aug 1983) has suggested that temperature influences the dominance of a particular pathogen in the disease complex. *F. moniliforme* is the dominant fungus under low soil temperatures, whereas *M. phaseolina* predominates at high soil temperatures. The pathological significance of the involvement of several fungi in causing root and stalk rot is not known and must be investigated.

Host Range and Physiological Specialization

M. phaseolina is a plurivorous pathogen of over 75 different plant families and about 400 plant species (Dhingra and Sinclair 1977). Among these are important food crops, such as cereals (maize, sorghum, and finger millet), legumes (cowpea, groundnut, soybean, pigeonpea, and chickpea), vegetables (cabbage, tomato, and pumpkin), and fruits (apple, pear, orange, and banana). As its wide host range suggests, *M. phaseolina* is a highly variable pathogen in both its pathogenicity and myco-

logical characteristics. Some isolates of the pathogen are host specific (Hildebrand et al. 1945), while others can attack a wide range of hosts (Holiday and Punithalingam 1970). Physiological races has been reported for isolates of some crops such as jute (Ahmed and Ahmed 1969), and variability in cultural characteristics and pathogenicity of isolates from different parts of the same plant has been reported in soybean (Dhingra and Sinclair 1973).

Pathogen variation and physiological specialization are important factors that require consideration in disease control programs using host resistance, In the case of charcoal rot of sorghum, it would be useful to know (a) if sorghum is susceptible to isolates of the pathogen from other plant species and (b) whether physiological races exist among sorghum isolates of the pathogen. Unfortunately such information is not available in the literature.

Biology and Epidemiology

Most of our knowledge of the biology of *M. phaseolina* is derived from results of research with isolates from crops other than sorghum. It is assumed that the general biology of sorghum isolates is similar to that of isolates from other crops, although the pathosystem may be different. As stated in our introduction, only those aspects of the biology that influence the pathosystem will be reviewed.

Source and Survival of Inoculum

M. phaseolina is a root-inhabiting fungus (Garrett 1956), with little or no saprophytic growth in either soil or dead host cells of infected plants (Norton 1953, Edmunds 1964). In the absence of host plants, it survives or overseasons predominantly as small black sclerotia in diseased root and stem debris or in soil after decay of the plant material in which they were formed (Smith 1969a, Bhattacharya and Samaddar 1976). Thus the primary source of inoculum is sclerotia in the soil. Cook et al. (1973) reported that after 16 months in soil, 23% of sclerotia from sorghum stalks germinated. Sclerotia from other plant hosts are known to survive for several years (Dhingra and Sinclair 1977).

Populations of sclerotia in a maize field ranged from zero to more than 1000/g of soil (Papavizas and Klag 1975). This great variation in inoculum density in soil is one of the factors responsible for the highly variable incidence of charcoal rot in the field. According to Meyer et al. (1973), inoculum density increased in soil with continuous cropping of a susceptible crop of soybeans. This has implications in disease management strategies, which will be discussed later.

Root Penetration and the Effects of Drought Stress on Host Colonization and Disease Development

The process and mechanisms by which *M. phaseolina* penetrates roots and colonizes sorghum roots and stalks are not clearly known or understood. It is assumed from the work of Smith (1969b) with pine and Bhattacharya and Samaddar (1976) with jute that sorghum root exudates stimulate the germination of sclerotia in the soil. What happens next is still being debated. Reports in the literature can be summarized into two views. The first view is that mycelia from germinating sclerotia penetrate rootlets at any time, but no further growth or colonization takes place until the plants are drought stressed, when the pathogen grows extensively and colonizes roots and stalks (Norton 1958). In the second view, exemplified by the work of Odvody and Dunkle (1979), root penetration does not occur until plants are drought stressed. Whatever the truth is with regard to time of penetration, it is clear from the literature that colonization of root and stalk tissue and charcoal rot development occur only when plants are drought stressed during the grain-filling stage (Edmunds 1964, Edmunds and Voigt 1966, Odvody and Dunkle 1979).

Drought causes harmful physiological or metabolic changes in the plant. It reduces plant vigor; plants so affected are predisposed to attack by nonaggressive pathogens such as *M. phaseolina* (Schoeneweiss 1978). From a review of stalk rot problems in maize and sorghum and the associated environmental factors, Dodd (1977, 1980) developed a "photosynthetic stress-translocation balance" concept to explain the predisposition of sorghum to charcoal rot. According to this hypothesis:

a. sorghum plants are predisposed to charcoal rot as the root cells senesce because of a reduction of carbohydrates to maintain metabolic functions, including resistance;

b. the availability of carbohydrate to the root tissue is influenced by the environmental stresses affecting photosynthesis and by competition for carbohydrate by the developing grain;

c. if the combination of photosynthetic stress and translocation balance reduces carbohydrate to root tissue, root cells and also those of the lower part of the stalk senesce and lose resistance to the charcoal rot pathogen;

d. the charcoal rot pathogen invades and destroys root tissue, and subsequently rots the stalk, reducing its strength. This frequently results in lodging.

Although many environmental factors reduce photosynthesis, and hence assimilate (photosynthate) supply, drought stress at grain filling is the primary factor that triggers events that eventually lead to charcoal rot disease and plant lodging.

Dodd's hypothesis implies that the interaction of drought stress and pathogens causes stalk rots and lodging. Direct evidence for this has been provided by P. Mayers (Department of Primary Industries, Queensland, Australia; personal communication, Aug 1983), who reported as follows:

> In field experiments Fusarium stalk rot (*F. moniliforme*) and subsequent lodging developed when plant moisture stress and high inoculum density interacted. Minimal and severe moisture stress were obtained by using irrigation and rain excluding shelters. In the presence of inoculum, stress accentuated stalk rot 13.5 fold. Natural and very low levels of Fusarium inoculum were achieved by soil fumigation with dazomet. Fumigation decreased stalk rot from 59.3% to 1.3% in the most susceptible hybrid. Mean stalk rot percentage was below 2.8% in non-stressed plots irrespective of inoculum level and was below 1.7% on fumigated plots irrespective of stress level. Extensive stalk rot developed only in non-fumigated, moisture stressed plots.

Henzell and Gillieron (1973) and Chamberlin (1978), on the other hand, hold the view that plant lodging under drought stress is a purely physiological problem. Drought stress reduces assimilate supply to the lower part of the stalk for maintenance respiration. This results in senescence, disintegration of pith cells, and hence lodging. These two views on the causes of lodging are fully discussed by Henzell et al. (these proceedings). It is acknowledged that drought stress alone can cause lodging without assistance from pathogens where inoculum is absent. However, where pathogens are present, drought-stressed plants are invariably invaded by them, and this leads to increased damage of plants. It is possible that low or intermediate levels of drought stress may be tolerated by the plant except when combined with the pathogen. There is an obvious need for further research to clarify these issues.

Cultural Practices and Charcoal Rot

Nitrogen fertilization and plant densities have been reported to influence charcoal rot. In India the high levels of nitrogen fertilization needed to maximize the yield potential of improved cultivars increase the severity of charcoal rot (Avadhani et al. 1979, Mote and Ramshe 1980). Patil et al. (1982) reported cultivar differences in the effect of plant density on charcoal rot. While charcoal rot incidence was significantly higher in the hybrid CSH-8R at 180 000 plants/ha than at 45000 plants/ha, no differences were detected in the varieties SPV 86, SPV 265, and M 35-1. In a factorial experiment using line-source irrigation, we obtained highly significant positive correlations between drought stress, plant density, and nitrogen level (Table 2). It appears that high plant density increases plant competition for available soil moisture and that this competition increases with drought. The effect of nitrogen in increasing charcoal rot is probably due to its indirect effect on the ratio of root-to-shoot growth. Nitrogen promotes luxuriant shoot growth, and root development suffers. Under drought stress, the lack of a sufficient root system reduces the ability of a plant to obtain moisture, while at the same time its water requirement is increased by the luxuriant growth (Ayers 1978).

Systems of crop management that reduce pathogen inoculum and increase conservation of soil water decrease the incidence of charcoal rot. Sorghum grown under minimum tillage (ecofallow) in a winter wheat-sorghum-fallow rotation had 11% stalk rot, compared to 39% in conventional tillage (Doupnik and Boosalis 1975).

Sorghum grown in a mixed crop situation has also been reported to suffer less charcoal rot damage than sole crop sorghum (Khume et al. 1980),

Table 2. Percent lodging in CSH-6 sorghum at three levels of nitrogen and three plant populations subjected to ten different moisture stress levels with line source irrigation at ICRISAT Center.

Plant density and moisture stress	Percent lodging			
	Nitrogen levels[a]			
	N_1	N_2	N_3	Mean
D_1[b]	7.57	3.83	6.38	5.93
Stress-1[c]	0.00	1.98	5.24	2.41
Stress-2	1.08	1.09	3.26	1.81
Stress-3	0.36	0.84	2.44	1.21
Stress-4	1.57	1.39	2.09	1.68
Stress-5	0.69	0.64	0.76	0.70
Stress-6	0.00	0.69	0.00	0.23
Stress-7	9.83	7.09	8.76	8.56
Stress-8	21.52	11.64	14.18	15.78
Stress-9	14.35	6.97	11.03	35.35
Stress-10	26.30	5.94	16.00	16.08
D2	13.91	18.40	18.76	17.02
Stress-1	1.01	5.88	3.00	3.30
Stress-2	0.55	1.42	7.58	3.18
Stress-3	2.93	3.03	9.85	5.27
Stress-4	2.25	6.73	3.24	4.07
Stress-5	4.43	12.69	7.97	8.36
Stress-6	7.63	11.24	6.78	8.55
Stress-7	13.33	23.59	18.21	18.38
Stress-8	30.13	37.78	30.64	32.85
Stress-9	37.77	36.43	45.73	39.98
Stress-10	39.03	45.19	54.76	46.33
D3	29.40	37.00	36.17	34.19
Stress-1	1.95	4.54	7.34	4.61
Stress-2	4.83	6.12	10.73	7.23
Stress-3	4.43	8.26	10.35	7.68
Stress-4	9.35	9.48	13.12	10.65
Stress-5	17.68	31.72	28.83	26.08
Stress-6	24.37	49.76	38.12	37.42
Stress-7	43.34	63.07	49.34	51.92
Stress-8	51.62	58.45	55.16	55.08
Stress-9	65.14	69.97	71.77	68.96
Stress-10	71.30	68.62	76.92	72.28
Mean	16.96	19.74	20.44	19.05
SE (±) Density	2.73	2.35	3.09	1.58
SE (±) Stress	3.05	2.46	3.17	1.68
SE (±) Stress x density	5.29	4.26	5.49	2.91

a. N_1 = 20 kg nitrogen/ha
N_2 = 60 kg nitrogen/ha
N_3 =120 kg nitrogen/ha

b. D_1 = 66675 plants/ha
D_2 = 133350 plants/ha
D_3 =266700 plants/ha

c. Stress-1 = Nearest to line source (minimum moisture stress level).
Stress-10 = Farthest from line source (maximum moisture stress level).

Control

Several approaches have been investigated for charcoal rot control. As these will be fully covered in papers by various authors in these proceedings, only brief discussions of their effectiveness and application will be made in this section.

Fungicides

There are very few reports on the control of charcoal rot of sorghum by fungicides. Rajkule et al. (1979) reported that soil treatment with thiram at sowing did not effectively control the disease, but reduced it by 15%. Brassicol treatment had a similar effect. However, Anahosur et al. (1983) obtained no reduction in disease with Brassicol in field trials.

Soil fumigation treatments are generally successful in controlling *M. phaseolina* attack in other crops, e.g., in forest pine nurseries (Watanabe et al. 1970) and in melons (Krikun et al. 1982). Whether similar treatments would be effective in sorghum fields needs to be investigated. In practical terms, however, the cost and technological knowledge required for their successful use would preclude their adoption in areas where sorghum is a low-cash-value crop grown mostly by small farmers.

Williams and Nickel (these proceedings) provide more information about the prospects for fungicidal control of charcoal rot of sorghum.

Host Resistance

The four essential requirements for the identification and utilization of host resistance to charcoal rot have been discussed by Mughogho (1982). Our concern here will be to review briefly the techniques used to identify resistance, resistance sources, and factors associated with resistance. Comprehensive reviews of breeding for host resistance are available in papers by D.T. Rosenow, A.B. Maunder, and Henzell et al. (these proceedings).

Resistance Screening Technique

A reliable, efficient, and epidemiologically sound resistance screening technique for charcoal rot is yet to be developed. Following are three essential requirements of such a technique: (a) adequate inoculum density of *M. phaseolina* must be uniformly present in a virulent condition in the soil (since entry into plants is through roots) in which test genotypes are to be grown, (b) test genotypes should be subjected to the optimum and graded levels of drought stress at the appropriate growth stage to make them sufficiently predisposed to infection, and (c) a disease scoring scale that takes into account both root and stalk rot should be used.

Methods currently used to screen for resistance to charcoal rot do not adequately meet these conditions. The procedure followed by most investigators is essentially that reported by Edmunds et al. (1964). Sorghum is grown under irrigation in an environment known to be favorable for charcoal rot. Drought stress is induced by withholding irrigation at selected stages of plant maturity, and stalks are inoculated by inserting mycelium- and sclerotia-bearing toothpicks into holes made just above the first node. Amount of lodging, soft stalks, and the spread of the fungus from the point of inoculation up the stem are the three measurements taken in assessing the reaction of genotypes to the disease.

Toothpick inoculation and other methods where inoculum is introduced into the plant through the stalk are unsatisfactory primarily because they do not closely simulate the natural infection process, which begins in the roots and only later goes up the stem. Furthermore, the level of disease development with toothpick inoculation is usually less than that which occurs naturally and is therefore unsatisfactory for assessing resistance that could be useful under natural disease incidence (Edmunds et al. 1964).

At ICRISAT we have successfully induced charcoal rot without artificial inoculation in field-grown, susceptible sorghums by two methods. One method is to sow the crop just before the end of the rainy season so that it grows and matures under progressively less soil moisture. This timing is similar to that of the postrainy-season (rabi) crop in India, which suffers most from charcoal rot. The other method is to grow the crop under irrigation during the dry season and to withdraw irrigation at 50% flowering. In both methods charcoal rot incidence and severity vary according to location, probably due to soil type, level of moisture stress, and the pathogen inoculum potential in the soil (see Tables 1 and 2). Nevertheless, disease development in susceptible genotypes is sufficiently high for useful evaluation of test genotypes.

Anatomical and Physiological Factors Associated with Resistance

Several anatomical and physiological plant characters have been associated with resistance to charcoal rot and suggested as selection criteria in resistance screening programs. Maranville and Clegg (these proceedings) discuss the correlation of "stalk strength" with resistance to charcoal rot. Although much variation exists in the stalk anatomy of genetically diverse sorghum lines (Schertz and Rosenow 1977), there is no experimental evidence yet of this variation being associated with resistance.

Maunder et al. (1971) reported that in a charcoal rot nursery where plants were drought stressed from the boot stage to maturity, "bloomless plants" had 38.4% more disease than those with the waxy bloom on the stalk internodes. They suggested that bloomless plants were more predisposed to charcoal rot than "bloomed plants," Further research is needed to confirm this.

The most promising plant character that is positively correlated with charcoal rot resistance, and is increasingly used as a selection criterion, is nonsenescence. Rosenow (1980) reported significant correlations between nonsenescence, lodging resistance, and charcoal rot resistance in Texas, USA. Selection for charcoal rot resistance is based on the degree of nonsenescence exhibited by plants under drought stress during the late grain development stage. Both Duncan and Rosenow (these proceedings) provide more detailed descriptions of the nonsenescence character and its utilization in selection and breeding for charcoal rot resistance.

In India we also found significant positive correlations between charcoal rot resistance and plant nonsenescence (Table 3). However, multilocational testing for stability of the nonsenescence character showed that lines nonsenescent at one location were not necessarily nonsenescent at another location (Table 4), indicating the location specificity of the character. This would be expected from variations in pathogen inoculum density and in the level of drought stress to which plants are subjected during evaluation at different locations. Stability of nonsenescence would most probably depend on the level of drought stress. Up to a specific level of stress, a genotype would show stability in nonsenescence at several locations, but beyond that it may not. Further research is obviously needed to elucidate this.

Disease Rating Scale

Several disease rating scales have been used to evaluate sorghum lines for resistance to charcoal rot or stalk rots in general. The most commonly used is a 1 -to-5 scale based on the percentage of lodged plants, where 1 = no lodged plants and 5 = over 20% plants lodged (Frezzi and Teyssandier 1980). The main disadvantage of this method of disease evaluation is that it excludes infected plants that have not lodged. It is not uncommon in a charcoal rot nursery to see standing plants that are infected by the disease. Where toothpick inoculation is carried out, a rating scale based on the growth of the pathogen up the stem from the point of inoculation is used (Rosenow 1980). As discussed earlier under "Resistance screening technique," this method of inoculation and evaluation is epidemiologically unsound since infection is through the root system in nature. In the ICRISAT charcoal rot research project we have developed a rating scale that takes into account roof infection, soft stalk of infected plants that do not lodge, and lodged plants. This scale is laborious to use when

Table 3. Correlation coefficients among parameters of charcoal rot disease scores under depleting soil moisture condition at four locations in India (Patancheru, Dharwar, Nandyal, and Madhira).

Disease parameter	Lodging (%)	Soft stalk (%)	Mean no. of nodes crossed	Mean score [a] for root infection	Leaf and plant death [a]
Lodging (%)		0.96**	0.88**	0.57**	0.65**
Soft stalk (%)			0.88**	0.52**	0.60**
Mean no. of nodes crossed				0.47**	0.52**
Mean score for root infection					0.92**
Leaf and plant death					

Correlation coefficient at 5% = 0.288, at 1% = 0.372 (**significant at 1%).

a. Based on three locations (Patancheru, Dharwar and Nandyal).

Table 4. Days to flowering, plant height (m), leaf and plant death, grain weight, percent lodging, percent soft stalk, mean number of nodes crossed, and mean score for root infection of six sorghum genotypes (rated as nonsenescent) at four locations in India during 1981 postrainy season.

Genotype	Location	Days to 50% flowering	Plant height (m)	Leaf[a] and plant death	1000-grain weight (g)	Percent lodging	Percent soft stalk	Mean no. of nodes crossed	Mean[b] score of root infection
IS-108	Patancheru	56	0.85	2.50	29.87	0.00	0.00	0.50	3.00
	Dharwar	47	1.62	4.42	19.94	44.62	37.50	1.17	4.00
	Nandyal	53	1.60	4.50	27.68	40.00	55.00	2.00	4.50
	Madhira	55	1.75	2.27	23.82	10.22	3.55	0.31	2.25
IS-176	Patancheru	70	1.25	4.00	26.48	25.00	40.00	0.70	4.50
	Dharwar	59	1.75	4.55	17.10	43.17	62.50	1.67	4.50
	Nandyal	71	1.19	2.40	34.17	5.00	5.00	0.05	3.00
	Madhira	65	1.35	3.36	25.53	0.00	0.00	0.43	1.50
IS-2954	Patancheru	67	1.10	4.50	25.90	20.00	50.00	1.10	5.00
	Dharwar	60	1.35	3.60	24.81	2.38	5.00	0.15	2.00
	Nandyal	71	1.00	2.60	30.66	0.00	15.00	0.80	1.95
	Madhira	65	1.25	4.00	27.67	30.00	61.85	1.53	4.00
IS-3927	Patancheru	61	0.75	4.30	50.97	55.00	55.00	1.95	5.00
	Dharwar	57	1.12	2.95	34.44	26.25	15.00	0.35	2.00
	Nandyal	60	1.05	3.55	40.66	45.00	50.00	2.80	4.00
	Madhira	59	1.25	2.80	45.74	0.00	13.35	0.33	2.50
IS-10722	Patancheru	65	1.15	3.60	31.88	25.00	25.00	0.55	4.50
	Dharwar	60	1.20	3.22	24.32	22.80	35.00	0.75	2.50
	Nandyal	71	0.95	2.90	46.36	10.00	20.00	0.85	3.25
	Madhira	66	1.40	4.07	19.82	48.75	48.75	2.00	4.00
CSH-6	Patancheru	62	1.15	4.79	26.95	78.75	85.00	2.65	4.73
	Dharwar	62	1.57	4.70	25.62	57.41	72.18	2.32	4.87
	Nandyal	67	1.25	4.08	25.22	100.00	100.00	4.70	5.00
	Madhira	56	1.34	4.91	26.36	83.67	92.36	5.41	4.93
SE for cultivar (±)		2.16	2.05	0.26	2.12	9.21	8.91	0.69	0.46
SE for location (±)		0.46	0.44	0.056	0.45	1.97	1.91	0.15	0.14

a. Nonsenescence ratings based on leaf and plant death scores on 1-5 scale, where 1 = completely green and 5 = dead.
b. Root infection score on 0-5 scale, where 0 = no discoloration and infection; and 5 = more than 50% roots showing infection and discoloration.

large numbers of material are to be evaluated. Nevertheless it is essential that the different phases of the disease are considered in a resistance screening program. Since leaf and plant death (senescence) are positively correlated with charcoal rot infection, a leaf and plant death scoring scale would be most useful for disease evaluation of large numbers of plants.

Resistance Sources

Attempts to find sources of resistance to charcoal rot for breeding programs were started in the USA in the 1940s, In one of the most comprehensive testing programs Hoffmaster and Tullis (1944) screened 232 sorghum lines of diverse genetic background at 4 locations for 4 years. Although they found differences in the susceptibility of these lines to charcoal rot, data showed no stability in the performance of the lines from year to year. They thus concluded, "it is impossible from the data available to recommend certain varieties for localities in which *Macrophomina* dry rot is a limiting factor."

In the ICRISAT charcoal rot research project we have also found inconsistencies in the reaction to the disease of a large number of germplasm lines.

This lack of stability is due, as explained earlier, to different levels of drought stress and hence different levels of predisposition to the disease. However, one line, E 36-1, has consistently shown resistance to lodging at several locations in 3 years of testing. The plants were infected, as shown by fungal isolations from roots and stalks, but the infection was not severe enough to cause lodging (ICRISAT 1982).

In the USA the line New Mexico-31 released by Malm and Hsi (1964) as resistant to charcoal rot has been used extensively in breeding programs. In recent years Rosenow (1980) identified 13 non-senescent lines as good sources of resistance to charcoal rot. The stability of resistance of these lines outside Texas is not known. They should be tested for use in other countries where charcoal rot is a problem.

The need for stable and better sources of resistance is obvious. Most of the large (over 20000 lines) ICRISAT sorghum germplasm collection has not been screened, and it is conceivable that among these (especially among lines from drought-prone areas) are lines resistant to charcoal rot. However, the priority should be to develop a reliable screening technique that can be used to distinguish resistant from susceptible lines under graded levels of drought stress.

Crop Management

The ideal and most effective control strategy for charcoal rot is to prevent drought stress from predisposing plants to infection. In other words, resistance to predisposition would be the best method of control. This can be done by proper management of the soil-plant-water system. Except where sorghum is grown under irrigation, farmers have no control over the variability of rainfall in most sorghum-growing areas. Cultural practices that reduce pathogen inoculum in soil and that increase water availability and use by plants (e.g., plant density, rate of nitrogen fertilization, use of varieties with different rooting characteristics, and crop rotation) have been suggested as possible measures of reducing drought-stress-related diseases (Cook and Papendick 1972). Such measures have been successful in controlling fusarium foot rot of wheat (Cook 1980). Whether similar crop husbandry practices would be effective and practicable for control of charcoal rot awaits investigation.

Drought resistance as an indirect method of charcoal rot control raises the obvious and important question: will genotypes that resist drought also resist charcoal rot? We are unable to answer this question because we have insufficient knowledge of the interactions of drought stress, the charcoal rot pathogen, and the host. The only proposition we can offer is that certain levels of drought stress may be resisted by plants in the absence of the pathogen. Where the pathogen is present, such plants may be infected; the pathogen then destroys roots, which contributes to further drought stress. Therefore breeding for drought resistance alone may not provide the answer to the charcoal rot problem.

Priorities for Future Research

This review will have shown that in spite of its importance, research on charcoal rot has been' largely superficial. Wide gaps still exist in the biology of the pathogen and epidemiology of the disease, and in particular, the process of pathogenesis and how it is influenced by environmental and plant physiological factors. The technical problems of working with a soilborne, root-infecting pathogen are partly responsible for these deficiencies. However, techniques are now available that could profitably be used in charcoal rot research.

Following are some of the areas that need research attention in the future:

1. **Crop loss.** Quantitative crop loss data are needed that distinguish between direct effects of drought and indirect effects through crop predisposition and subsequent damage by charcoal rot. Under what conditions are indirect effects more important than direct effects?

2. **Pathogenesis.** Root rot precedes stalk rot. When, in the growth stage of the plant, and under what conditions are roots penetrated by the pathogen? What conditions favor root and stalk colonization?

3. **Interactions with other pathogens.** Since *M. phaseolina* does not infect plants alone, there is need for basic studies on the interactions among the different pathogens involved. What is the sequence of infection? Is there synergism in host-parasite interaction?

4. **Pathogen variation and physiological specialization.** In view of the wide host range of *M. phaseolina,* it would be useful for control of the disease to know (a) if sorghum is susceptible to pathogen isolates from other hosts, and (b) whether physiological races exist among the sorghum isolates.

5. **Predisposition by drought stress and plant growth stage.** What level of drought stress (plant water potential) is optimum for predisposing plants to infection? Is there a varietal difference in this? Can charcoal rot occur in plants at all growth stages if sufficiently predisposed?

6. **Predisposition and plant water potential.** In screening for resistance, can we actually relate predisposition of plants to actual measurements of plant water potential? Graded levels of soil moisture supply, and hence predisposition, can be provided by the line-source irrigation technique.

7. **Sink.** Improved high-yielding varieties and hybrids tend to be ultrasusceptible to charcoal rot. Is it sink size or other factors that make such cultivars vulnerable to the disease? Can we identify the conditions under which a given size of sink is likely to indirectly predispose plants to infection?

8. **Association of nonsenescence and disease resistance.** Study the physiological basis of nonsenescence, its stability under different environmental conditions, and its relationship to charcoal rot resistance.

9. **Correlation between drought resistance and charcoal rot resistance.** Since drought stress is the primary factor that predisposes plants to charcoal rot, would drought-resistant plants also resist charcoal rot?

10. **Development of a reliable field screening technique.** This is essential for success in breeding for resistance.

Answers to most of the questions raised above would require interdisciplinary and collaborative research efforts between pathologists, physiologists, breeders, and soil scientists. We hope that the proceedings of this meeting will help to bring forth this essential cooperation for the understanding and eventual control of charcoal rot of sorghum.

References

AHMED, N., and AHMED, Q.A. 1969. Physiological specialization in *Macrophomina phaseoli* (Maubl.) Ashby causing root rot of jute, *Corchorus* species. Mycopathologia et Mycologia Applicata 39:129-138.

ANAHOSUR, K.H., and PATIL, S.H. 1983. Assessment of losses in sorghum seed weight due to charcoal rot. Indian Phytopathology 36:85-88.

ANAHOSUR, K.H., PATIL, S.H., and HEGDE, R.K. 1983. Evaluation of PCNB against *Macrophomina phaseolina* (Tassi) Goid. causing charcoal rot of sorghum. Pesticides 17:11-12.

ANAHOSUR, K.H., and RAO, M.V.H. 1977. A note on the epidemic of charcoal rot of sorghum in the regional research station, Dharwar. Sorghum Newsletter 20:22.

AVADHANI, K.K., PATIL, S.S., MALLANAGOUDE, B., and PARVATICAR, S.R. 1979. Nitrogen fertilization and its influence on charcoal rot. Sorghum Newsletter 22:119-120.

AVADHANI, K.K., and RAMESH, K.V. 1979. Charcoal rot incidence in some released and pre-released varieties and hybrids. Sorghum Newsletter 21:37-38.

AYERS, P.G. 1978. Water relations of diseased plants, Pages 1 -60 *in* Water deficits and plant growth (ed. T.T. Kozlowski). New York, New York, USA: Academic Press. 323 pp.

BHATTACHARYA, M., and SAMADDAR, K.R. 1976. Epidemiological studies on jute diseases. Survival of *Macrophomina phaseoli* (Maubl.) Ashby in soil. Plant and Soil 44:27-36.

CHAMBERLIN, R.J. 1978. The physiology of lodging of grain sorghum (*Sorghum bicolor* L Moench). Ph.D. thesis, University of Queensland, Australia.

COOK, G.E., BOOSALIS, M.G., DUNKLE, L.D., and ODVODY, G.N. 1973. Survival of *Macrophomina phaseoli* in corn and sorghum stalk residue. Plant Disease Reporter 57:873-875.

COOK, R.J. 1980. Fusarium foot rot of wheat and its control in the Pacific Northwest. Plant Disease 64:1061 - 1066.

COOK, R.J., and PAPENDICK, R.I. 1972. Influence of water potential of soils and plants on root disease. Annual Review of Phytopathology 10:349-374.

DHINGRA, O.D., and SINCLAIR, J.B. 1973. Location of *Macrophomina phaseoli* on soybean plants related to culture characteristics and virulence. Phytopathology 63:934-936.

DHINGRA, O.D. and SINCLAIR, J.B. 1977. An annotated bibliography of *Macrophomina phaseolina* 1905-1975. Vicosa, Brazil: Imprensia Universitaria, Universidade Federal de Vicosa. 244 pp.

DHINGRA, O.D., and SINCLAIR, J.B. 1978. Biology and pathology of *Macrophomina phaseolina,* Vicosa, Brazil: Imprensia Universitaria, Universidade Federal de Vicosa. 166 pp.

DODD, J.L. 1977. A photosynthetic stress-translocation balance concept of corn stalk rot. Pages 122-130 *In* Proceedings of the 32nd Annual Corn and Sorghum Research Conference (eds. H.D. Loden and D. Wilkinson). Washington, D.C., USA: American Seed Trade Association.

DODD, J.L. 1980. The photosynthetic stress-translocation balance concept of sorghum stalk rots. Pages 300-305 *In* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

DOMSCH, K.H., GAMS, W., and ANDERSON, T.H. 1980. Compendium of soil fungi. 2 vols. London, U.K.: Academic Press. 859 and 405 pp.

DOUPNIK, B., and BOOSALIS, M.G. 1975. Ecofallow reduces stalk rot in grain sorghum. Phytopathology 65:1021-1022.

EDMUNDS, L.K. 1964. Combined relation of plant maturity, temperature, and soil moisture to charcoal stalk rot development in grain sorghum. Phytopathology 54:514-517.

EDMUNDS, L.K., and VOIGT, R.L. 1966. Role of seed production in predisposition of sorghum to charcoal rot. Phytopathology 56:876 (abstract).

EDMUNDS, L.K., VOIGT, R.L., and CARASSO, F.M. 1964. Use of Arizona climate to induce charcoal rot in grain sorghum. Plant Disease Reporter 48:300-302.

FREZZI, M., and TEYSSANDIER, E.E. 1980. Summary and historical review of sorghum diseases in Argentina. Pages 11-15 *in* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, AP. 502 324, India: ICRISAT. 469 PP.

GARRETT, S.D. 1956. Biology of root-infecting fungi. London, U.K.: Cambridge University Press. 293 pp.

HARRIS, E. 1962. Diseases of guineacorn. Samaru Technical Notes 2:1-13.

HENZELL, R.G., and GILLIERON, W. 1973. Effect of partial and complete panicle removal on the rate of death of some *Sorghum bicolor* genotypes under moisture stress. Queensland Journal of Agricultural and Animal Sciences 30:291-299.

HILDEBRAND, A.A., MILLER, J.J., and KOCH, LW. 1945. Some studies on *Macrophomina phaseoli* (Maubl.) Ashby in Ontario. Scientific Agriculture 25:690-706.

HOFFMASTER, D.E., and TULLIS, E.C. 1944. Susceptibility of sorghum varieties to *Macrophomina* dry rot (charcoal rot). Plant Disease Reporter 28:1175-1184.

HOLLIDAY, P., and PUNITHALINGAM, E. 1970. *Macrophomina phaseolina.* No. 275 *in* CMI (Commonwealth Mycological Institute), Descriptions of pathogenic fungi and bacteria. Kew, Surrey, U.K.: CMI.

HSI, D.C.H. 1956. Stalk rots of sorghum in eastern New Mexico. Plant Disease Reporter 40:369-371.

ICRISAT. 1980. Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT. 469 pp.

ICRISAT. 1983. Annual Report 1982. Patancheru, A.P. 502 324, India: ICRISAT.

KHUNE, N.N., SHIWANKAR, S.K., and WANGIKAR, P.D. 1980. Effect of mixed cropping on the incidence of charcoal rot of sorghum. Food Farming and Agriculture 12:292-293.

KRIKUN, J., ORION, D., NACHMIAS, A., and REUVENI, R. 1982. The role of soilborne pathogens under conditions of intensive agriculture. Phytoparasitica 10:247-258.

LEUKEL, R.W., MARTIN, J.H., and LEFEBVRE, C.L. 1951. Sorghum diseases and their control. Farmers' Bulletin No. 1959. Washington, D.C., USA: U.S. Department of Agriculture. 50 pp.

LUTTRELL, E.S. 1950. Grain sorghum diseases in Georgia. Plant Disease Reporter 34:45-52.

MALM, N.R., and HSI, D.C.H. 1964. New Mexico 31: a charcoal rot-resistant grain sorghum line. New Mexico Agricultural Experiment Station Research Report 93. Las Cruces, New Mexico, USA: New Mexico State University.

MAUNDER, A.B., SMITH, D.H., and JUDAH, B.W. 1971. Bloom and bloomless isogenics as related to charcoal rot and diffusive resistance. Sorghum Newsletter 14:20-21.

MEYER, W.A., SINCLAIR, J.B., and KHARE, M.N. 1973. Biology of *Macrophomina phaseoli* in soil studied with selective media. Phytopathology 63:613-620.

MOTE, U.N., and RAMSHE, D.G. 1980. Nitrogen application increases the incidence of charcoal rot in rabi sorghum cultivars. Sorghum Newsletter 23:129.

MUGHOGHO, L.K. 1982. Strategies for sorghum disease control. Pages 273-282 *in* Sorghum in the Eighties: Proceedings of the International Symposium on Sorghum, sponsored by INTSORMIL, ICAR, and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

NAGARAJAN, K., SARASWATHI, V., and RENFRO, B.L. 1970. Incidence of charcoal rot (*Macrophomina phaseoli*) on CSH-1 sorghum. Sorghum Newsletter 13:25.

NORTON, D.C. 1953. Linear growth of *Sclerotium bataticola* through soil. Phytopathology 43:633-636.

NORTON, D.C. 1958. The association of *Pratylenchus hexincisus* with charcoal rot of sorghum. Phytopathology 48:355-358.

ODVODY, G.N., and DUNKLE, L.D. 1979. Charcoal stalk rot of sorghum: effect of environment on host-parasite relations. Phytopathology 69:250-254.

PAPAVIZAS, G.C., and KLAG, N.G. 1975. Isolation and quantitative determination of *Macrophomina phaseolina* from soil. Phytopathology 65:182-187.

PATIL, R.C., DESHAMANE, N.B., and PANDHARE, T.M. 1982. Effect of plant density and row spacing on charcoal rot incidence in four cultivars of sorghum. Sorghum Newsletter 25:110.

RAJKULE, P.N., CHAUHAN, H.L., and DESAI, K.B. 1979. Chemical control of charcoal rot. Sorghum Newsletter 22:120.

RAO, N.G.P. 1982. Transforming traditional sorghums in India. Pages 39-59 *in* Sorghum in the Eighties: Proceedings of the International Symposium on Sorghum, sponsored by INTSORMIL, ICAR, and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

RAUT, J.C., and BHOMBE, B.B. 1972. Leaf spot of sorghum caused by *Rhizoctonia bataticola.* Indian Phytopathology 25:586-587.

ROSENOW, D.T. 1980. Stalk rot resistance breeding in Texas. Pages 306-314 *in* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

SCHERTZ, K.F., and ROSENOW, D.T. 1977. Anatomical variation in stalk internodes of sorghum. Crop Science 17:628-631.

SCHOENEWEISS, D.F. 1978. Water stress as a predisposing factor in plant disease. Pages 61-99 *in* Vol. 5, Water deficits and plant growth (ed. T.T. Kozlowski). New York, New York, USA: Academic Press. 323 pp.

SMITH, W.H. 1969a. Comparison of mycelial and sclerotial inoculum of *Macrophomina phaseoli* in the mortality of pine seedlings under varying soil conditions. Phytopathology 59:379-382.

SMITH, W.H. 1969b. Germination of *Macrophomina phaseoli* sclerotia as affected by *Pinus lambertiana* root exudate. Canadian Journal of Microbiology 15:1387-1391.

TARR, S.A.J. 1962. Diseases of sorghum, sudan grass and broom corn. Kew, Surrey, U.K.: Commonwealth Mycological Institute. 380 pp.

TRIMBOLI, D.S., and BURGESS, L.W. 1982. The fungi associated with stalk and root rot of grain sorghum in New South Wales. Sorghum Newsletter 25:105-106.

TULLIS, E.C. 1951. *Fusarium moniliforme,* the cause of a stalk rot of sorghum in Texas. Phytopathology 41:529-535.

UPPAL, B.N., KOLHATKAR, KG., and PATEL, M.K. 1936. Blight and hollow-stem of sorghum. Indian Journal of Agricultural Science 6:1323-1334.

WADSWORTH, D.F., and SIEGLINGER, J.B. 1950. Charcoal rot of sorghum. Oklahoma Agricultural Experiment Station Bulletin No. B-355. Stillwater, Oklahoma, USA: Oklahoma A&M College and U.S. Department of Agriculture. 7 pp.

WATANABE, T., SMITH, R.S., Jr., and SNYDER, W.C. 1970. Populations of *Macrophomina phaseoli* in soil as affected by fumigation and cropping. Phytopathology 60:1717-1719.

Questions

Maunder:
Should your reference to high-yield cultivars being super susceptible, referring to hybrids, not be better stated as first-cycle hybrids? The breeder has a tendency to place yield ahead of lodging, and this will be more dramatic in initial transition to hybrids. But in the case of U.S. sorghum history, after the first 8-10 years the problem with charcoal rot was greatly reduced.

Pande:
Yes, hybrids under Indian conditions are quite susceptible when planted in the postrainy season.

Schoeneweiss:
You stated that *Macrophomina phaseolina* does not grow in dead plant cells. Are you saying that the fungus does not grow as a saprophyte on organic matter derived from sorghum?

Pande:
Yes.

Vidyabhushanam:
It was stated that lodging is not the only criterion for measuring charcoal rot intensity. Is there any alternate measurement possible to know the level of incidence of the disease? Has any correlation between root and stalk rot infection been established?

Pande:
Lodging is the first apparent symptom of charcoal rot, and to confirm charcoal rot one has to split the plants to see the fungal colonization. Probably the two are necessary to assess the clear picture of charcoal rot.

Vidyabhushanam:
It is established that predisposition to drought stress is essential for charcoal rot. Is it clearly understood what stage and intensity of drought stress is required for the disease to manifest itself?

Pande:
I suppose moisture stress is the most important predisposing factor for charcoal rot infection and development. We do not know exactly at what stage the stress is effective. It seems stress at 50% flowering that continues up to maturity gives good charcoal rot expression.

Fusanum Root and Stalk Disease Complex

Fusarium Root and Stalk Disease Complex

N. Zummo*

Summary

This paper presents a brief review of a fusarium root and stalk disease complex of sorghum. The Fusarium *species involved in this disease complex may occur wherever sorghum is grown worldwide and may also affect maize, millet, rice, and sugarcane. There are indications that certain cultural practices, such as maximum cultivation, high fertility levels, and high plant densities, may increase the prevalence of this disease complex. A better understanding of the symptomology, etiology importance, and means of control is necessary. This is especially important in some of the less developed areas of the world where short, high-yielding sorghums are being introduced to replace local native varieties.*

Fusarium moniliforme, one of the most cosmopolitan of plant pathogens, is found in soils wherever sorghum can be grown. The fungus persists on plant residues that remain in the soil and on its surface. Mycelia, conidia, and—in the perfect state (*Gibberella fujikuroi*)—*ascospores* may be produced on or in plants or the soil at any time during the growing season, and secondary infection of host tissues may occur whenever environmental conditions are favorable for disease development. *F. moniliforme* may also be a serious pathogen on maize, millet, rice, and sugarcane (Bolle 1927, Bolle 1928, Dickson 1956, Bourne 1961, Sheldon 1904, Ullstrup 1936, and Voorhies 1933). *F. moniliforme* affects sorghum plants at all stages of growth and can cause seedling blight, root and stalk rot, pokkah boeng, seed mold, and head blight.

The Fungus

According to Booth (1971), Dickson (1956), Tarr (1962), and Saccas (1954), the *Fusarium* spp associated with root and stalk rots in sorghum include:

1. *Gibberella fujikuroi* (Saw.) Wr.
   [G. *moniliformis* (Sheld.) Wine,]
   F. moniliforme Sheld. Conidial stage.
2. G. *fujikuroi* (Saw.) Wr, var. *subglutinans* Ed.
   F. moniliforme Sheld. var. *subglutinans* Woll. & Reink.
3. G. *zeae* (Schw.) Petch
   [G. *saubinetti* (Mont.) Sacc]
   F. graminearum Schw.
   G. *roseum* f. *cerealis* (Cke.) Snyder & Hansen
   F. roseum f. *cerealis* (Cke.) Snyder & Hansen var. *graminearum*

Other *Fusarium* spp associated with sorghum roots and stalks, but for which pathogenicity is still questionable, include:

F. culmorum (W.G. Sm.) Sacc.
F. equiseti (Cda.) Sacc.
F. oxysporum Schlecht
F. sambucinum Fck.
F. scirpi Lambotte & Fantr.
F. solani (Mart.) Appel et Wr.
F. tricinctum Cda.

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Part of the problem in working with fusarial root and stalk rots is that *F. moniliforme* often does not produce macroconidia in culture. Tullis (1951) attributed stalk rot of sorghum in Texas to *F. moniliforme* but noted that it produced only microconidia. Leonian (1929), in a very comprehensive study of 220 cultures of *Fusarium* spp on various culture media, found that conidial production by *F. moniliforme* and three subspecies was markedly affected by 2 and 3% tartaric acid in the medium. *F. monilh forme* var. *subglutinans* produced no macro- or microconidia at all when 2 or 3% tartaric acid was present in the medium. On malt extract agar at 25°C, one strain of *F. moniliforme* failed to produce macroconidia and four others did so only occasionally.

Bourne (1961) discussed the identity of white and purple strains of *Fusarium* occurring in association with cane stalk rots and pokkah boeng disease in Florida. The fungus was found to be identical with *F. moniliforme* (Sheld.) Snyd. et Hans. Subsequent to publication of these data, the purple strain of *F. moniliforme* was maintained in pure culture for 4 years. After this period the isolate still proved highly pathogenic to cane cuttings and growing stalks. However, this strain completely lost the ability to produce septate macroconidia or chromogenic substances when transferred frequently on nutrient potato dextrose agar at 23°C after 2 years in culture. Wineland (1924) found that certain strains of *F. moniliforme* in culture produced macroconidia in abundance at first, lost this character after a time, and afterwards produced only mycelia and microconidia.

Seedling Blight

Young sorghum plants in the one-to-three leaf stage can be severely affected by *F. moniliforme* during periods of prolonged cloudy humid weather. The first symptoms on these plants are tan-brown-red-purple-black irregular lesions on the leaves. The tips of the leaves wither first, and later the entire leaf dies. Because young sorghum plants are rather delicate and grow slowly, infected plants are often killed if cloudy humid weather persists for an extended period (Zummo 1980). If conditions favorable for plant growth resume, the sorghum plants will apparently overcome the disease. Where the disease is severe and the environment remains unfavorable for rapid seedling growth, the crop may have to be replanted. It may be assumed that a percentage of *Fusarium* infected/infested seed will give rise to infected seedlings or spread the disease in some other manner.

Root Rot

Fusarium root rot on sorghum typically involves the cortical tissues first, then the vascular tissues of the roots. Newly formed roots may exhibit distinct lesions of various sizes and shapes. Root rot is progressive, so older roots are often destroyed, leaving little plant anchorage. When sorghum root rot is extensive, the plants are often easily uprooted.

Stalk Rot

Fusarium stalk rot is usually accompanied by root damage. Under irrigation and heavy nitrogen fertilization, root damage may not result in aboveground changes in plant appearance before the stalks begin to rot. Stalk rot may reduce seed fill, resulting in seed weight losses as high as 60% (Edmunds and Zummo 1975).

Fusarium stalk rot has become increasingly common in recent years as a root/stalk rot disease of sorghum in many areas of West Africa (Saccas 1954, Tarr 1962, Zummo 1980). In the United States, the disease is generally found in the 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 is most injurious during periods of moisture stress, fusarium stalk rot is usually most damaging during cool, wet weather following hot, dry weather.

Trimboli and Burgess (1983) reproduced basal stalk rot and root rot on grain sorghum plants grown in the greenhouse in *Fusarium moniliforme* infested soil at optimal soil moisture until flowering, then subjected the plants to a gradual development of severe moisture stress between flowering and the middough stage, followed by rewetting. Stalk rot did not develop, and root rot was not severe in plants grown to maturity at optimal soil moisture, although many of these plants were infected by *F. monili forme.* Stalk and root rot developed in the majority of stressed plants grown in soil initially uninfested but contaminated by *F. moniliforme* after planting.

Fusarium stalk rot can usually be distinguished from charcoal rot because of the less pronounced pigmentation and disintegration of pith tissues and the slower rate of tissue damage by *Fusarium.* Whereas charcoal rot may destroy a field of sorghum in 2 or 3 days, fusarium stalk rot may take 2 or 3 weeks. The presence of sclerotia in the later stages of disease makes the identification of charcoal rot rather easy.

Fusarium stalk rot may be separated from anthracnose red rot and peduncle breakage incited by *Colletotrichum graminicola* (Ces.) G.W. Wils. because the discoloration in fusarium stalk rot is uniform throughout, whereas the discoloration in anthracnose red rot is interspersed with discrete white areas. In some sorghum varieties, individual anthracnose red rot lesions in the peduncle may be easily identified by their distinct lenticular shape.

Pokkah Boeng

Pokkah boeng, a Javanese term denoting a malformed or distorted top, was originally described on sugarcane in Java (Wakker and Went 1896; Bolle 1927, 1928). Most of the definitive research work on pokkah boeng has been done with sugarcane because that crop is normally grown in a humid tropical environment that is favorable to disease development. In some very humid areas, certain sugarcane varieties cannot be grown because of their susceptibility to the disease (Bolle 1928, North 1932).

The disease is caused by the soilborne fungus *F. moniliforme* var. *subglutinans* Woll. & Reink. (G. *fujikuroi* var. *subglutinans* Edwards), which is found in all sorghum areas where high humidity is prevalent during the growing season. Although the disease may be conspicuous on some sorghum varieties, losses from the disease itself on sorghum are usually small.

Pokkah boeng is characterized by deformed, folded, or discolored leaves near the top of the plant. Sometimes the leaves become so wrinkled that they are unable to unfold, resulting in plants with a ladderlike appearance. In extreme cases, infection may move from the leaves and sheath into the stalks, causing death of the tops. In mild cases, symptoms on individual leaves may resemble those of mosaic (incited by sugarcane mosaic virus or maize dwarf mosaic virus) or yellow leaf blotch (incited by a bacterium). Pokkah boeng can be differentiated from these two diseases because of the characteristically wrinkled teat bases and numerous small, transverse cuts in the leaf *margin.* Sometimes the disease causes *stalks to bend or* twist in the nodes or *intemodes* (*Zummo 1972*). *An* abnormality sometimes associated with pokkah boeng is "knifecut," which consists *of narrow,* uniform, transverse cuts in the rind that give *the* impression that the tissue has been removed with a sharp knife. Because "knifecut" lesions *are* covered by the leaf sheaths in sorghum during the early stages of the disease, they may not be apparent when pokkah boeng leaf symptoms are present. Under conditions of physical stress, such as windstorms, affected stalks may break off along the lesions so that the entire top of the plant is lost. The emerging inflorescence may be attacked within the surrounding leaf sheath, which may result in rotting or barrenness. Portions of inflorescences may be attacked so that the flowers dry up and no grain is produced.

Infection occurs during prolonged wet weather when the incitant fungus grows upward on the outside of sorghum stalks, where it temporarily becomes established in moist areas behind leaf sheaths or in whorls. Metabolites produced by the fungus incite distortions and cause rotting of basal portions of leaves while still in the whorls. Later, when wet weather subsides, rotted portions of the plant and the fungus may dry up, and the plant resumes normal growth. When the upper internodes elongate, the lower leaves distorted in the whorl become apparent. During wet weather the fungus can be observed sporulating on rotting material still attached to the plant.

Coinciding with increased *Fusarium* problems are several cultural practices suspected of contributing to the disease increase: maximum tillage, high nitrogen fertilization, high plant populations, and continuous cropping. Doupnik et al. (1975) and Doupnik and Boosalis (1980) showed that cultivation increased the amount of stalk rot incited by *F, moniliforme.* They found that no-till plots had 72% less stalk rot than conventionally tilled plots.

Gourley et al. (1977) reported that *F. moniliforme* generally invades sorghum seedlings through insect or mechanical injuries, or through roots or stems weakened by other factors so that blight or damping-off of susceptible cultivars occurs. They found that in inoculated plants the length of primary roots was reduced 53%, epicotyl length was reduced 32%, and number of lateral roots was reduced 25% in comparison with uninoculated plants.

Reed et al. (1983), in Nebraska, studied the association of soil fungi with the roots and stalks of sorghum throughout the growing season. They found that *F. moniliforme* was the most prevalent species isolated from stalks and *F. equiseti* was most commonly isolated from roots. Other *Fusarium* spp that were isolated included *F. graminearum, F. tricinctum, F. oxysporum,* and *F. solani. F. moniliforme* was rarely isolated from roots before the flag-leaf stage of plant development, but by anthesis it was recovered from 30% of the samples. *F. oxysporum* end *F. solani* combined were almost as common as *F. equiseti* early in the season, but the frequency of these two species declined to almost zero by the end of the season. In contrast, the isolation frequency of *F. equiseti,* although erratic, remained high throughout the season.

Recommendations for Future Research

1. Probably the most important need in research on the fusarium root and stalk disease complex is to determine the amount of damage being done by it in particular sorghum-growing areas. This assessment should take into consideration varietal reaction to the disease; source of and carryover of inoculum; effect of environment, with particular emphasis on moisture stress; soil type and fertilizer, and cultural practices employed.

2. The use of resistant varieties offers promise for the control of some or all of this *Fusarium* disease complex. However, unlike charcoal rot and anthracnose, good sources of resistance to the entire disease complex have not yet been identified. Some sorghum varieties, especially among the sweet sorghums and some of the tall landraces from Africa, appear to be resistant to certain elements of the *Fusarium* disease complex but may be susceptible to other components of it.

3. Further work is also needed in order to more fully understand the mode of infection, source of and carryover of inoculum, effect of environment, soil fertility, and cultural practices on disease severity, and the role of insects and nematodes on disease spread.

References

BOLLE, P.C. 1927. Een onderzoek naar de oorzaak van pokkahboeng en toprot [An investigation into the cause of pokkahboeng and toprot]. Archief Suikerindustrie Nederlands-Indie III, 35:589-609.

BOLLE, P.C. 1928. Verdere onderzoek ingen over pokkahboeng en toprot [Further investigations in pokkahboeng and toprot]. Archief Suikerindustrie Nederlands-Indie I, 36:116-129.

BOOTH, C. 1971. The genus *Fusarium.* Kew, Surrey, U.K.: Commonwealth Mycological Institute. 237 pp.

BOURNE, B.A. 1961. Fusarium sett or stem rot. Pages 187-202 *in* Vol. I, Sugar-cane diseases of the world (eds. J.P. Martin, E.V. Abbott, and C.G. Hughes). New York, New York, USA: Elsevier Publishing Co.

DICKSON, J.G. 1956. Diseases of field crops. New York, New York, USA: McGraw Hill Book Co. 517 pp.

DOUPNIK, B., Jr., and BOOSALIS, M.G. 1980. Ecofallow—a reduced tillage system—and plant diseases. Plant Disease 64:31 -35.

DOUPNIK, B., Jr., BOOSALIS, M.G., WICKS, G.A., and SMIKA, D. 1975. Ecofallow reduces stalk rot in grain sorghum. Phytopathology 65:1021-1022.

EDMUNDS, L.K., and ZUMMO, N. 1975. Sorghum diseases in the United States and their control. U.S. Department of Agriculture Handbook No. 468. Washington, D.C., USA: U.S. Government Printing Office. 47 pp.

GOURLEY, L.M., ANDREWS, C.H., SINGLETON, L.L, and ARAUJO, L. 1977. Effects of *Fusarium moniliforme* on seedling development of sorghum cultivars. Plant Disease Reporter 61:616-618.

LEONIAN, L.H. 1929. Studies on the variability and dissociation in the genus Fusarium. Phytopathology 19:753-868.

NORTH, D.S. 1932. Pokkah boeng. Bulletin No. 100, Proceedings of the Fourth Congress of the International Society of Sugar Cane Technologists, San Juan, Puerto Rico. New York, New York, USA: Elsevier Publishing Co.

REED, J.E., PARTRIDGE, J.E., and NORDQUIST, P.T. 1983. Fungal colonization of stalks and roots of grain sorghum during the growing season. Plant Disease 67:417-420.

SACCAS, A.M. 1954. Les champignons parasites des sorghos (*Sorghum vufgare*) *et* des penicillaries (*Pennisetum typhoidum*) en Afrique Equatorial Francaise. Agronomie Tropicale Nogent 9:135-173, 263-301, 647-686.

SHELDON, J.L. 1904. A corn mold (*Fusarium moniliforme* n. sp.). Nebraska Agricultural Experiment Station Annual Report (1903)17:23-32.

TARR, S.A.J. 1962. Diseases of sorghum, sudan grass

and broom corn. Kew, Surrey, U.K.: Commonwealth Mycological Institute. 380 pp.

TRIMBOLI, D.S., and BURGESS, L.W. 1983. Reproduction of *Fusarium moniliforme* basal stalk rot and root rot of grain sorghum in the greenhouse. Plant Disease 67:891 - 894.

TULLIS, E.C. 1951. *Fusarium moniliforme,* the cause of a stalk rot of sorghum in Texas. Phytopathology 41:529-535.

ULLSTRUP, A.J. 1936. The occurrence of *Gibberella fujikuroi* var. *subglutinans* in the United States. Phytopathology 26:685-693.

VOORHIES, R.K. 1933. *Gibberella moniliforme* on corn. Phytopathology 23:368-378.

WAKKER, J.H., and WENT, F.A.F.C. 1896. Overzicht van de ziekten van het suikerriet op Java [An overview of sugarcane diseases in Java]. Archief Suikerindustrie Nederlands-Indie IV, pp. 425-435.

WINELAND, G.O.1924. An ascigerous stage and synonymy for *Fusarium moniliforme.* Journal of Agricultural Research 28:909-922.

ZUMMO, N. 1972. External *Fusarium moniliforme var. subglutinans* associated with right angle bending and twisting of sweet sorghum stalks. Phytopathology 62:800 (abstract).

ZUMMO, N. 1980. Fusarium disease complex of sorghum in West Africa. Pages 297-299 *in* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

Questions

Partridge:
In your pokkah boeng greenhouse studies, could *F. moniliforme* var. *subglutinans* be recovered from anywhere on the surface of nondiseased plants or on the "healthy" controls?

Zummo:
It was sometimes isolated from healthy plants—but sporadically.

Partridge:
Realizing the difficulty of preventing *F. moniliforme* var. *subglutinans* from spreading—apparently through the air—to "uninfected" plants, one still is left with 100% correlation of association but no controls.

Pappelis:
Are you saying that *F. moniliforme* in the root cortex is not a disease of the root?

Zummo:
Any plant may harbor organisms—sometimes parasitic on other plants—on the roots. Just because these organisms have been isolated is not proof of parasitism until Koch's postulates have been carried out on them.

Clark:
Pokkah boeng appears in symptomology similar to Ca deficiency. What are your comments relating to this?

Zummo:
I believe that pokkah boeng is a disease incited by *F. moniliforme* var. *subglutinans.* I'm aware of the calcium deficiency symptoms on maize that mimic pokkah boeng but still feel that we are dealing with a parasitic disease, not a deficiency symptom.

Maranville:
Relating to the Ca++ implication, isn't it likely that the organism could cause disruption in Ca++ metabolism, which then appears as the symptom? In essence then, the problem may actually be with Ca++, although triggered via metabolism disruption, rather than a true lack of soil Ca++.

Zummo:
It is possible, but I don't think so.

Pythium Root and Seedling Rots

Pythium Root and Seedling Rots

G.N. Odvody and G. Forbes*

Summary

Pythium *spp cause a root and mesocotyl rot of sorghum seedlings in cold, wet soils and a root rot of mature sorghum in warm, wet soils. Identity of the causal species is incomplete for several reasons, including changing and variable nomenclature, isolate variability, and differences in interpretation of fungal structures in culture.* Pythium *can cause death of seedlings and mature plants, and although disease incidence and severity are influenced by several environmental factors, temperature and moisture are most important. Seed treatments with most fungicides are generally ineffective in controlling seedling disease, but new, specific systemic fungicides need to be evaluated for efficacy against pythium seedling disease and pythium root rot of mature plants. There is host plant resistance against both, but better characterization of the resistance is needed. More knowledge is needed concerning the mechanism of pathogen survival, initial inoculum, initial infection, and the influence of specific host-pathogen-environment interactions.*

In the literature of the past several decades, numerous reports describe identified and unidentified *Pythium* spp occurring on sorghum roots, and most are discussed either in the review by Tarr (1962) or the publication by Pratt and Janke (1980). Most published information concerning *Pythium* as a sorghum pathogen is fraught with confusion because of errors in isolation, inoculation, species identification, and establishment of particular isolates as actual causal agents of observed disease in the field (Pratt and Janke 1980). Although *Pythium arrhenomanes* is a true root pathogen of sorghum, its erroneous, long-term acceptance as the causal agent of milo disease (Elliott et al. 1937) until 1948 (Leukel 1948) has cast further doubt on many earlier reports. The great majority of reports of *Pythium* on sorghum refer to its occurrence on young seedlings in the field, but there are some reports of its occurrence on mature plants (Frederiksen et al. 1973, Pratt and Janke 1980). The reported susceptibility of sorghum seedlings to *Pythium* isolates from other hosts provided no beneficial information, because the results obtained under controlled environmental conditions were never related to disease occurrence in field-grown sorghum (Pratt and Janke 1980). Difficulties in species identification, nomenclatural changes, and disagreement about either the characteristics of species or their synonomy further confuse earlier reports.

Seedling Disease and Damping-off

Poor stand establishment of sorghum may involve interactions of several biotic and abiotic factors, but in the United States environment has often been considered the most important through the direct, detrimental effects of low soil temperature, saturated soil, and soil crusting (Leukel and Martin 1943). The involvement of fungal pathogens in pre- and postemergent damping-off of sorghum was

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first studied extensively by Leukel and Martin (1943), who distinguished between seedborne and soilborne pathogens attacking seeds and seedlings in soil. They tried to determine the effect of pathogens on germination, emergence, and subsequent growth of the seedling. Research by Leukel and Martin (1943) and others demonstrated that fungicide seed treatments reduced preemergent damping-off associated with several seed-rotting fungi (e.g., species of *Aspergillus, Rhizopus, Penicillium,* and *Fusarium*), which rapidly colonize unprotected seed primarily in cold, wet soils. Sometimes *Penicillium* and *Fusarium* spp proceed to parasitize the host tissue and cause postemergent damping-off. However, *Pythium* is the fungus most frequently isolated from diseased seedlings grown in cold, wet soil, and Leukel and Martin (1943) demonstrated that *Pythium* spp isolated from root and mesocotyl lesions of sorghum seedlings were highly virulent to sorghum planted in soils with high moisture and low temperature (15°C). Standard fungicide treatments do not protect against infection by *Pythium* attack, probably because either the site of parasitic attack is distal to the seed or the fungicide (captan or thiram) has no significant effect on *Pythium.*

Symptoms on seedlings are either brown or gray water-soaked roots or root tips, or lesions on roots (Forbes 1983) that become flaccid and necrotic (Edmunds and Zummo 1975). Lesions also occur on the plumule and mesocotyl (Leukel and Martin 1943, Forbes et al. 1983), and the mesocotyl produces somewhat more pigment in response to the pathogen than do the roots (Odvody, unpublished observation, 1983). However, the lesion pigmentation is less than that induced by other soilbome pathogens, e.g., *Fusarium* (Edmunds and Zummo 1975). Plants succumbing to postemergent damping-off usually wilt rapidly and die, but often many stunted plants remain alive despite the loss of most leaves and *Pythium* damage to the roots and mesocotyl (Odvody, unpublished observation, 1983; Edmunds et al. 1970). However, there is usually wide variation in plant height and spacing throughout affected fields, and many plants may be adversely affected throughout subsequent development (Edmunds et al. 1970).

Root Rot of Mature Plants

The occurrence of *Pythium* spp on roots of mature plants was first reported in 1937, when P. *arrhenomanes* was erroneously considered the cause of "milo disease" in Texas and other areas of the United States where this disease occurred (Elliot et al. 1937). Severe root rot in the North Texas High Plains in 1971-72 (Frederiksen et al. 1973) was caused by a species of *Pythium* identified as P. *graminicola* (Pratt and Janke 1980). Symptoms on the large adventitious (or buttress) roots are darkened and blackened roots (Frederiksen et al. 1973) and sunken red-brown to black root lesions, and sometimes at root death the entire lesion or root has a tan color (Pratt and Janke 1980). *Fusarium* spp and other fungi may rapidly follow attack by *Pythium* spp (Odvody, unpublished observation, 1982) and cause greater pigmentation of lesions than *Pythium* alone (Edmunds and Zummo 1975). Once established, these secondary fungi are more easily isolated (Frederiksen et al. 1973) than the primary pathogenic *Pythium.*

In 1972, stalk rots (caused primarily *by Fusarium* spp) followed development of the pythium root rot and were important causes of subsequent stalk lodging (Frederiksen et al. 1973). Isolates of *Pythium* obtained from roots of mature plants were highly virulent on sorghum seedlings (Frederiksen et al. 1973, Pratt and Janke 1980), but the occurrence of similar *Pythium* strains in seedling infections in the field is unknown. On the North Texas High Plains, root infections of sorghum began to increase at the boot stage or later when large numbers of adventitious roots were being produced in irrigated fields (high soil temperature and high soil moisture conditions) (Odvody, unpublished observation, 1983). Pratt and Janke (1980) demonstrated that P. *graminicola* from roots could cause stalk rot when plants were inoculated at maturity, but it is probably not the cause of stalk rot in the field. At one South Texas location, Pratt and Janke (1980) isolated P. *myriotylum* and P. *periplocum* from insect-damaged roots and stalks of mature sorghum. Only the former was pathogenic on sorghum seedlings inoculated in the greenhouse. Because only one site was investigated, the extent of the natural occurrence of P. *myriotylum* on mature sorghum roots is unknown.

Taxonomy of Pathogens

The changes in *Pythium* taxonomy during the past few decades present both problems and benefits in understanding the occurrence of this pathogen on sorghum. Hendrix and Campbell (1973) proposed

that P. *graminicola* and P. *arrhenomanes* form a complex in which isolates have characteristics that constitute a continuum between the two type-species and that other similar complexes exist. This helps clarify some of the older literature and may lend more support for the pathogenic activities of *Pythium* reported in 1937, despite its erroneous identification with milo disease (Elliott et al. 1937). The *Pythium* spp attacking seedlings also remain insufficiently characterized. Forbes (1983) demonstrated that a majority of isolates from infected seedlings grown in a field soil in the greenhouse produced lobulate sporangia but only 50% of those produced oospores in culture. The characteristics of these latter isolates were most similar to P. *arrhenomanes* as described by Drechsler (1936).

Etiology and Infection

Pythium spp pathogenic to sorghum probably survive in soil as oospores (Hendrix and Campbell 1973). Saprophytic growth may be of minor importance because *Pythium* is a poor competitor that apparently colonizes tissue only when other organisms are either absent or relatively inactive due to environmental factors (Hendrix and Campbell 1973). Other data (Hendrix and Campbell 1973) indicate that oospores of *Pythium* spp pathogenic to sorghum germinate in response to host seed and root exudates in wet soil—either directly by producing germ tubes or indirectly by producing zoospores that encyst and then germinate. The pathogen then rapidly penetrates host cells and tissues that lack secondary wall thickenings (Hendrix and Campbell 1973). Although numerous environmental factors influence these germination and infection processes by the pathogen, temperature and moisture (especially in combination) are the two most important ones (Hendrix and Campbell 1973, Leukel and Martin 1943), probably because of their additional effect on the host plant and associated soil microflora. Under cold, wet soil conditions, germination of seed and growth of seedlings are slowed such that emergence is delayed, primary root growth is reduced, and in older seedlings, new roots are slow to establish from the mesocotyl and crown. The transitory root system of emerging seedlings is especially vulnerable to attack in cold, wet soils (Edmunds and Zummo 1975), and the delay in production of the permanent root system from the crown is probably one of the greatest factors involved in postemergent damping-off. We have often observed the primary root system and mesocotyl of 2-week-old seedlings being killed by *Pythium,* but without post-emergent damping-off because a healthy, permanent crown root system was established in the well-drained upper soil layer.

Pythium pathogens isolated from seedlings may have an optimum growth temperature in culture much different from the environment in which they are normally pathogenic (Hendrix and Campbell 1973). Thus, the pathogen's competitive ability in the soil-plant environment may be more important than its ability to grow in a noncompetitive cultural environment. However, the *Pythium* root rot that occurs on mature plants in warm, wet (flood-irrigated) soil of the North Texas High Plains correlates well with the high optimum temperature reported for P. *graminicola* (Waterhouse and Waterston 1964a) and P. *arrhenomanes* (Waterhouse and Waterston 1964b). Infection in these mature plants appeared to occur on roots of all sizes and ages, but initial infections may have occurred earlier in root development (Odvody, unpublished observation, 1983).

For both pythium seedling disease and mature plant root rot, nothing is known about either the levels of inoculum or the increase and secondary spread of the pathogen. In maize, oospores and sporangia of *Pythium are* rapidly formed in infected tissues (Nyvall 1981), and oospores, sporangia, and coenocytic mycelia have been observed in roots of infected sorghum seedlings (Forbes et al. 1983). However, the role of these sporangia and zoospores in the infection of proximal, healthy roots of the same and different plants is not known.

The influence of previous crops and of tillage and cultural practices on *Pythium* diseases in sorghum is not known, but these variables are conjectured to have some effect, especially in relation to initial inoculum and soil moisture and soil temperature (Leukel and Martin 1943, Hendrix and Campbell 1973). The incidence and severity of pythium root rot on mature sorghum may be directly affected by irrigation and irrigation frequency.

Potential Control with Fungicides

Most fungicides applied to sorghum seed are not effective in controlling damping-off caused by *Pythium* (Leukel and Martin 1943), but some new systemic fungicides, like metalaxyl and fosetyl-A1,

may provide the previously lacking protection of tissues distal to the seed. The additional cost of seed treatment must be compared with the potential for pythium seedling diseases in the planting area. There is a continued need for additional fungicides like captan to control the principal seed-rotting fungi, because most of the potentially effective systemic fungicides are specific for Phycomycetes.

Potential Control Through Host Plant Resistance

Host plant resistance to *Pythium* attack in the seedling stage is not well defined, but most germplasm is thought to be susceptible in disease-conducive cold, wet soil. In general, varieties are more susceptible than hybrids, and plants from either old or low-quality seed are more susceptible than those from either young or high-quality seed. Although some cultivars were less affected by *Pythium* caused damping-off in the field (Forbes et al. 1983), the reason for and consistency of the reduced damage is not known. The multiplicity of factors involved with stand establishment in the field necessitates evaluations in controlled environments to determine major factors responsible for increased stand establishment. For a particular genotype, the increased stand in cool, wet soils could be due either to resistance to *Pythium* or to physiological and physical factors like continued germination and growth under these conditions, rapid establishment of a permanent root system, and secondary thickening of cell walls in outer root tissues. Any stress factor that delays establishment of the permanent root system from the coleoptile and upper nodes probably increases the potential for *Pythium* caused seedling damage or damping-off in cool, wet soils. Varieties are likely more susceptible to *Pythium* damage because of their reduced vigor. They are generally slower to germinate, emerge, and establish a permanent root system. Plants from old seed of hybrids (and varieties) are more susceptible than plants from young seed because their reduced vigor is especially apparent in cool, wet soils. Plants from either nontreated or damaged seed are also more susceptible to seedling disease, not only because *Pythium* can attack more readily, but also because other seed-rotting fungi additionally reduce vigor and delay emergence and plant establishment (Leukel and Martin 1943).

The *Pythium* disease syndrome on mature plants is incompletely characterized, but some genotypes (e.g., Tx 2751, Tx 2737) demonstrate more late-season plant death associated with *Pythium* than do others. However, other pathogens and factors may either promote *Pythium* attack or contribute to plant death. Root wounding by nematodes and insects may be a contributing factor (Frederiksen et al. 1973), as might primary and secondary infections by other root pathogens like *Fusarium* and *Periconia.* Additionally, genotypes may differ in their tolerance to similar amounts of root loss caused by *Pythium* (Odvody, unpublished observation, 1983).

Research Needs

1. Better characterization of *Pythium* species or species complexes that are pathogenic to sorghum roots.

2. Determination of geographical and local variation in *Pythium* spp pathogenic to sorghum.

3. Improved understanding of initial inoculum, germination stimuli, type of germination (direct or indirect), type of infective propagules (oospores, sporangia, or zoospores), site of infections), and importance of secondary spread of the pathogen.

4. Improved knowledge of environmental factors necessary for disease development and how they interact with the pathogen, soil microflora, and host to cause disease.

5. Development of meaningful screening techniques to identify resistant genotypes and to determine mechanisms of field resistance or susceptibility.

6. Determination of the effects of cropping sequence, tillage, and cultural practices (especially irrigation in relation to mature-plant root rot) on disease incidence and severity and on increase in soilborne inoculum.

7. Investigation of other edaphic and biotic factors that influence disease incidence and severity.

8. Evaluation of new systemic fungicides for ability to control *Pythium* on seedlings and mature sorghum.

References

DRESCHLER, C. 1936. *Pythium graminicolum* and *P. arrhenomanes.* Phytopathology 26:676-684.

EDMUNDS, L.K., FUTRELL, M.C., and FREDERIKSEN, R.A. 1970. Sorghum diseases. Pages 200-234 *in* Sorghum production and utilization (eds. J.S. Wall and W.M. Ross). Westport, Connecticut, USA: AVI Publishing Co.

EDMUNDS, L.K., and ZUMMO, N. 1975. Sorghum diseases in the United States and their control. U.S. Department of Agriculture Handbook No. 468. Washington, D.C., USA: U.S. Government Printing Office. 47 pp.

ELLIOTT, C., MELCHERS, L.E., LEFEBVRE, C.L., and WAGNER, F.A. 1937. Pythium root rot of milo. Journal of Agricultural Research 54:797-834.

FORBES, G.A. 1983. Development of a technique for screening seedlings of *Sorghum bicolor* (L.) Moench for resistance to seedling disease. M.Sc. thesis, Texas A&M University, College Station, Texas, USA.

FORBES, G.A., COLLINS, S.D., ODVODY, G.N., and FREDERIKSEN, R.A. 1983. Seedling disease in South Texas in 1983. Sorghum Newsletter 26:124-125.

FREDERIKSEN, R.A., ROSENOW, D.T., and TULEEN, D. 1973. Pythium root rot of sorghum on the Texas High Plains 1972. Sorghum Newsletter 16:137-138.

HENDRIX, F.F., Jr., and CAMPBELL, W.A. 1973. Pythiums as plant pathogens. Annual Review of Phytopathology 11:77-97.

LEUKEL, R.W. 1948. *Periconia circinata* and its relation to milo disease. Journal of Agricultural Research 77:201 - 222.

LEUKEL, R.W., and MARTIN, J.H. 1943. Seed rot and seedling blight of sorghum. U.S. Department of Agriculture Technical Bulletin 839. 26 pp.

NYVALL, R.F. 1981. Diseases of com (*Zea mays* L). Pages 55-86 *In* Field crop diseases handbook. Westport, Connecticut, USA: AVI Publishing Co.

PRATT, R.G., and JANKE, G.D. 1980. Pathogenicity of three species of *Pythium* to seedlings and mature plants of grain sorghum. Phytopathology 70:766-771.

TARR, S.A.J. 1962. Diseases of sorghum, sudan grass and broom corn. Kew, Surrey, U.K.: Commonwealth Mycological Institute. 380 pp.

WATERHOUSE, G.M., and WATERSTON, J.M. 1964a. *Pythium graminicola.* No. 38 *in* CMI (Commonwealth Mycological Institute), Descriptions of plant pathogenic fungi and bacteria, Kew, Surrey, U.K.: CMI. 2 pp.

WATERHOUSE, G.M., and WATERSTON, J.M. 1964b. *Pythium arrhenomanes.* No. 39 *in* CMI (Commonwealth Mycological Institute) Descriptions of plant pathogenic fungi and bacteria Kew, Surrey, U.K.: CMI. 2 pp.

Questions

Partridge:
You indicated *Fusarium* follows *Pythium* infection. Is there a predominant *Fusarium* that is isolated from roots following *Pythium?*

Odvody:
We didn't routinely identify these *Fusarium* species except to determine their presence, because *Pythium* was so predominantly isolated.

Partridge:
Do cold-tolerant sorghums tend to be resistant?

Odvody:
Not in the limited number observed in the field, but more research is needed to clarify this important aspect of seedling disease.

Anthracnose Stalk Rot

Anthracnose Stalk Rot

R.A. Frederiksen*

Summary

Anthracnose persists as one of the most destructive diseases of sorghum. The pathogen, Colletotrichum graminicola, *affects the foliage and inflorescence* (*including grain*), *as well as the stalks. Losses of up to* 50% *are not uncommon. The use of host resistance has reduced losses, but shifts in populations of the pathogen in many areas where the disease is prevalent have limited the effectiveness of resistance. Factors reducing inoculum, utilization of blended sources of resistance, and levels of resistance that reduce the spread of the pathogen are recommended as means of reducing disease losses.*

Anthracnose, caused by the fungus *Colletotrichum graminicola* (Cesati) Wilson, is one of the most damaging diseases of sorghum, particularly in warm humid sorghum-growing areas (Tarr 1962). These areas include many regions in the semi-arid tropics and temperate regions with warm humid summers, as well as the humid tropics. The common factor among these sorghum-growing regions with prevalent anthracnose is frequent rainfall, particularly during the later stages of plant growth.

Distribution

Anthracnose has been reported from essentially all of the sorghum-growing regions of the world (Pastor-Corrales and Frederiksen 1980). It is far more important in the more humid regions or during rainy seasons. Anthracnose is the most important sorghum disease in Brazil and is a major threat in most of the Latin American countries (Nakamura 1982, Pastor-Corrales and Frederiksen 1979). In India anthracnose can be very damaging in Uttar Pradesh (L.K. Mughogho, ICRISAT; personal communication, Nov 1983), and it can be widespread in regions of West Africa (N. Zummo, USDA; personal communication).

Symptoms

Three phases of anthracnose are recognized in the evaluation of host resistance or extent of damage (Harris et al. 1964, Harris and Fisher 1973, Lohman et al. 1951). These disease symptoms include a foliar phase, stalk rot, and colonization of the panicle including the grain.

Foliar anthracnose can be recognized by a range of symptoms including an "oval leaf spot," diffuse or patchy foliar colonization, and midrib infection. The range in foliar symptoms may be caused by variation in the pathogen, host resistance (Pastor-Corrales 1980), or physiologic status of the host following infection. Weakened, chlorotic, stressed, or senescent leaves of susceptible cultivars are rapidly colonized by the pathogen.

Infection and colonization of the panicle frequently result in losses in both quality and quantity of grain (Reyes et al. 1969). Differences in the extent of colonization of the rachis appear to be influenced greatly by host genotype, specifically by whether the rachis tissues senesce naturally during maturation or are genetically susceptible. Many sorghums with moderately high levels of foliar resistance readily succumb to colonization in the panicle. Frequently, sorghum grains may be colon-

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ized by the anthracnose fungus (Pastor-Corrales 1980); these grains have concentric rings or stripes of black acervuli from the stylar region.

Anthracnose stalk rot, occasionally referred to as red rot, develops after the other phases and ultimately results in lodging. Symptoms of stalk rot can be diagnosed by their irregularly mottled or marbled pattern of colonization. These symptoms are diagnostic during colonization of tissues of leaf midveins, panicle and rachis branches, and stalks. The marbling may result from either multiple infections or colonization of stalk tissue from a single inoculation site. Le Beau et al. (1951) described these symptoms in detail, noting an absence of mycelium in the pale areas surrounded by pigmented, sparsely colonized areas. Variation in pigment and rate of colonization are related to host-plant color and susceptibility to anthracnose.

The Pathogen

C. graminicola has been recognized as the cause of anthracnose for about seven decades. Other reviewers have adequately covered the earlier confusion as to species identification (see Tarr 1962). Problems arose, however, when Arx(1957) attempted to combine the falcate-spored Colletotrichums (C. *graminicola* and C. *falcatum* Went) within the species *Glomerella tucumanenis* (Speg.) Arx and Muller.

Le Beau (1950) demonstrated a high degree of host specificity when he compared isolates from sorghum, johnsongrass, and sugarcane. He studied 593 isolates from 18 grass species, from geographically separated regions. He concluded that isolates of C. *falcatum* from sugarcane were pathogenic to sugarcane but that C. *graminicola* isolates from sorghum were specific for sorghum. Sutton (1968), using size and shape of appressoria of C. *graminicola* from both maize and sorghum and C. *falcatum* from sugarcane, was able to differentiate three groups of isolates. Huguenin et al. (1982) separated these three groups of falcate-spored *Colletotrichum* spp on the basis of electrophoretic patterns. Politis (1975) also provides evidence for the separate speciation of these fungi with the identification of the perfect stage, *Glomerella graminicola* Politis. He obtained an isolate of C. *graminicola* from infected maize that was pathogenic to maize. Wheeler et al. (1974) found an isolate from maize that attacked both sorghum and maize. Perhaps some isolates of *C. graminicola are* virulent on both grass species, but apparently these isolates are rarely observed.

Physiologic Specialization

C. graminicola is a highly variable species. Evidence for races exists from observations made in the United States and from other regions of the world (Harris and Johnson 1967, Foster and Frederiksen 1979, Pastor-Corrales 1980, Nakamura 1982). These and other reports clearly demonstrate differences in pathogenicity of isolates between and within locations. Gradual erosion of resistance is recognized by the changes in reaction of resistance of Tx2536 and other sorghum lines in Georgia and Puerto Rico. Uniquely different populations of *C. graminicola are* suggested by the differential reaction of sorghum entries in the International Sorghum Anthracnose Virulence Nursery (ISAVN) from Nigeria, Brazil, and the USA (King and Frederiksen 1976). Nakamura (1982) identified five races of C. *graminicola* using five differential cultivars of sorghum. These races were classified from 1983 single spore isolates gathered from a number of diseased plants throughout Brazil. Nakamura's work confirms the supposition of many workers that several physiological forms of C. *graminicola are* present not only within an area, but between locations as well. Workers in Brazil have adopted a set of differential cultivars derived in part from the ISAVN. Other races of C. *graminicola are* probably present in India because all of the sorghums in the ISAVN were susceptible to anthracnose at Pantnagar in north India (L.K. Mughogho, ICRISAT; personal communication). Fortunately, other sorghum cultivars screened at Pantnagar are resistant in India. The significance of these observations is obvious: first the species is dynamic and affected by directional selection pressure by host resistance genes, and secondly, profoundly different races exist in different regions of the world. These facts present challenging problems when using host resistance as the sole measure of control in areas with severe anthracnose.

Anthracnose Stalk Rot and Yield Losses

The extent of damage or loss due to stalk rot is a reflection of: (a) the host's susceptibility to anthrac-

nose, (b) the environment, (c) the aggressiveness of the pathogen, and (d) the physiologic state of the host. In Texas a new high-yielding commercial hybrid introduced in 1965 was abandoned in 1968 because of anthracnose (Reyes et at 1969). In regard to environment, some sorghum hybrids vigorously attacked in Georgia are moderately resistant in Texas. Pastor-Corrales (1980) demonstrated the influence of the environment experimentally by growing identical sorghum entries in a nursery where overhead sprinklers simulated periodic rainfall and in naturally rainfed nurseries. In his trials, sprinkling greatly enhanced disease. In regard to the aggressiveness of the pathogen, the qualitative differences (virulence) of *Colletotrichum* spp have been amply recognized; but as with most other pathogens, efficiency in production of disease (aggressiveness) is also recognized. One may justifiably suspect that isolates from India and Brazil are more aggressive than those from other regions. Finally host cell maturity or senescence is extremely important: Duncan (these proceedings) described "normal" sorghum senescence as sequential from the bottom up. This sequential senescence appears to be a factor in foliar anthracnose. Both physically and physiologically, lower leaves are more subject to colonization by C. *graminicola.* But as leaves senesce from the bottom up, stalks mature from the top down. Anthracnose stalk rot in general follows this route. Panjcles, rachis branches, and heads are very vulnerable to colonization in some sorghum-growing regions but not others (Pastor-Corrales 1980). In Texas, the peduncle is often the first part of the host affected by anthracnose under field conditions, followed by colonization of the rachis branches.

Studies on sequential anthracnose development, under natural or experimental conditions, have rarely been reported. Such studies may be very useful in determining the effect of levels of resistance on anthracnose stalk rot. Harris and Fisher (1973) provided some data on the rate of disease development by their periodic evaluation of anthracnose on 49 commercial hybrids. They obtained negative correlations between disease ratings and yield of hybrids, and these correlations decreased over time. Their data provide evidence of slow development of anthracnose on some hybrids. Analysis of disease progress curves for each cultivar would be of value in interpreting these data.

Arguments in favor of a relationship between host senescence and stalk rot are strongly supported by a number of investigators (Dodd 1980, Katsanos and Pappelis 1969). Bockholt et al. (1971) and L. Reyes (Texas A&M University; unpublished data) observed massive yield loss and lodging because of anthracnose in sorghum at 1 and 2 weeks after the normal harvest date. All plants were affected at the time of harvest. Losses resulted from lower test weight and from lodging. More recently Ferreira and Warren (1982) estimated grain losses caused by anthracnose to reach as high as 88.7% in the highly susceptible cultivar IS-4255; RS-671 had yield losses of 42%, whereas resistant material such as IS-9189 and IS-9569 were essentially free from losses.

Breeding and Genetic Progress

Sources of resistance to anthracnose in sorghum have been reported by many workers over the past decades (Harris and Johnson 1967, Le Beau and Coleman 1950, Rosenow and Frederiksen 1982). These resistant sorghums have been used as parents in breeding programs or as replacements for susceptible cultivars. Coleman and Stokes (1954) determined that separate but linked genes conditioned resistances to stalk rot (LsLs) and foliar anthracnose (LL) in the sweet sorghum cultivar Sart. The appearance of new races of C. *graminicola* attacking some of these originally resistant sorghums suggests that other alleles must be involved. Harris and Johnson (1967) found a positive correlation between head and foliar ratings (r=0.50) and between head and stalk ratings (r=0.03). Nevertheless, Pastor-Corrales (1980) argues that environment plays a major role in the interpretation of the genetics of resistance. In his work the stalk rot phase developed under conditions less favorable for disease than did the foliar phase. Jones (1979) found that resistance to anthracnose was conditioned by one dominant gene for one parent and perhaps as many as three dominant genes for another. Jones also noted that the genetic background of the parents affected the level of disease susceptibility ratings in their progeny. She also noted that environmental factors had a great influence on disease ratings.

The Problem

Where sorghum is grown and anthracnose commonly appears, yield losses are likely to occur,

particularly in seasons in which harvesting is delayed because of wet weather. Host resistance reduces the amount of disease, but in the presence of abundant inoculum and a maturing host, losses may not be avoided. Furthermore, effective host resistance is lost because of the frequent occurrence of new races of the pathogen. Consequently, anthracnose control will depend on management of the host resistance genes, utilization of less vulnerable host phenotypes, manipulation of the host-parasite environment, and reduction in sources of inoculum (i.e., destruction of debris or susceptible collateral weed hosts [weed hosts of C. *graminicola* important in its spread include *Echinochloa colonum, Digitaria sanguinalis,* and *Dactyloctenium aegyptum;* ICRISAT Annual Report 1982]).

To date, genetic management in sorghum has been in response to the genetic changes of the pathogen—a practice that encourages shifts in the pathogen population. When many discrete populations of pathogens are present, one tends to predominate on a genetically uniform host population. Host mixtures, blends, or multilines, however, reduce these tendencies of pathogen uniformity (Wolfe and Barrett 1980). Another type of host resistance may be quantitative (horizontal), permitting a slower rate of disease development. Data from Harris and Fisher (1973) suggest that this type of resistance exists. Reevaluating their data, using disease progress curves, may demonstrate the economic value of moderately resistant cultivars. Reduced rates of resistance or even host mixtures may not be adequate in seasons with extensive rainfall at host maturity.

Chemical control has been economically practical under experimental conditions in Columbia (K.F. Cardwell de Castillo, Instituto Colombiano Agropecuario, La Libertad, Colombia; personal communication, 1982) but would be unacceptable where the crop is grown for food.

It appears to me that stalks on most tall (i.e., 2 m or above) sorghum cultivars escape (are resistant to ?) direct invasion by C. *graminicola.* Taller sorghums, or tall photoperiod-sensitive sorghums, that mature during the dry season escape disease Bergquist (1973) suggested that taller stalks have an advantage in being physically farther from the inoculum in the soil and that the leaf-stripping character of some tall sorghums also gives them an advantage against anthracnose. Bergquist et al. (1974) also described a closed floret trait during anthesis as a possible exclusion mechanism for reducing anthracnose infection in sorghum heads.

The presence of C. *graminicola* as a grain mold and grain-weathering fungus only adds to the total destructiveness of this pathogen.

Recommendations for Future Research

1. Continue to identify sources of resistance and the extent of pathogen variability.
2. Examine the epidemiology of C. *graminicola* to further clarify the spread of the pathogen from one part of the plant to another and from plant to plant.
3. Determine the rate of disease development and spread of the pathogen in various genotypes and phenotypes of sorghum (e.g., the effect of plant height, cultivar mixtures, or plant densities) on the rate of disease development.
4. Determine the method and length of survival of inoculum under natural conditions as a means of predicting the disease on a location basis.

References

ARX, J.A. 1957. Die arten der gattung *Colletotrichum* Corda. Phytopathologische Zeitschrift 29:413-468 (in German).

BERGQUIST, R.R. 1973. *Cotietotrichum graminicola* on *Sorghum bicolor* in Hawaii. Plant Disease Reporter 57:272-275.

BERGQUIST, R.R., ROTAR, P., and MITCHELL, W.C. 1974. Midge and anthracnose head blight resistance in sorghum. Tropical Agriculture 51:431-435.

BOCKHOLT, A.J., TOLER, R.W., and FREDERIKSEN, R.A. 1971. Measuring the effect of disease on grain sorghum performance. Pages 13-16 *in* Proceedings of the Seventh Biennial Grain Sorghum Research and Utilization Conference, sponsored by the Grain Sorghum Producers' Association (GSPA) and Sorghum Improvement Conference of North America, Lubbock, Texas. Available from GSPA, Abernathy, Texas, USA.

COLEMAN, O.H., and STOKES, I.E. 1954. The inheritance of resistance to stalk red rot in sorghum. Agronomy Journal 44:41-43.

DODD, J.L. 1980. The role of plant stresses in development of corn stalk rots. Plant Disease 64:533-537.

FERREIRA, A.S., and WARREN, H.L. 1982. Resistance of

sorghum to *Colletotrichum graminicola.* Plant Disease 44:773-775.

FOSTER, J., and FREDERIKSEN, R.A. 1979. Anthracnose and other sorghum diseases in Brazil. Pages 76-79 *in* Proceedings of the Eleventh Biennial Grain Sorghum Research and Utilization Conference, sponsored by the Grain Sorghum Producers' Association (GSPA) and Sorghum Improvement Conference of North America. Available from GSPA, Abemathy, Texas, USA.

HARRIS, H.B., and FISHER, C.D. 1973. Yield of grain sorghum in relation to anthracnose expression at different developmental stages of host. Pages 44-46 *in* Proceedings of the Eighth Biennial Grain Sorghum Research and Utilization Conference, sponsored by the Grain Sorghum Producers' Association (GSPA) and Sorghum Improvement Conference of North America, Lubbock, Texas. Available from GSPA, Abemathy, Texas, USA.

HARRIS, H.B., JOHNSON, B.J., DOBSON, J.W., Jr., and LUTTRELL, E.S. 1964. Evaluation of anthracnose on grain sorghum. Crop Science 4:460-462.

HARRIS, H.B., and JOHNSON, J.B. 1967. Sorghum anthracnose symptoms, importance and resistance. Pages 48-52 *in* Proceedings of the Fifth Biennial Grain Sorghum Research and Utilization Conference, Grain Sorghum Producers.' Association (GSPA) and Sorghum Improvement Conference of North America, Lubbock, Texas. Available from GSPA, Abemathy, Texas, USA.

HUGUENIN, B., LOUR, D.M., and GEIZER, J.P. 1982. [Comparison between isolates of *Colletotrichum falcatum* and *Colletotrichum graminicola* on the basis of their morphological, physiological and pathogenic characteristics.] Phytopathologische Zeitschrift 105:293-304 (in French).

ICRISAT. 1983. Annual Report 1982. Patancheru, A.P. 502 324, India: ICRISAT.

JONES, E.M. 1979. The inheritance of resistance to *Colletotrichum graminicola* in grain sorghum, *Sorghum bicolor.* Ph.D. thesis, Purdue University, West Lafayette, IN 47907, USA.

KATSANOS, R.A., and PAPPELIS, A.J. 1969. Relationship of living and dead cells to spread of *Colletotrichum graminicola* in sorghum stalk tissue. Phytopathology 59:132-134.

KING, S.B., and FREDERIKSEN, R.A. 1976. Report on the International Sorghum Anthracnose Virulence Nursery. Sorghum Newsletter 19:105-106.

LE BEAU, F.J. 1950. Pathogenicity studies with *Colletotrichum* from different hosts on sorghum and sugarcane. Phytopathology 40:430-438.

LE BEAU, F.J., and COLEMAN, O.H. 1950. The inheritance of resistance in sorghum to leaf anthracnose. Agronomy Journal 42: 33-34.

LE BEAU,F.J., STOKES, I.E., and COLEMAN, O.H. 1951. Anthracnose and red rot of sorghum. U.S. Department of Agriculture Bulletin 1035.

LOHMAN, M.L., STOKES, I.E., and COLEMAN, O.H. 1951. Anthracnose and red rot of sorgo in Mississippi, Plant Disease Reporter 28:76-80.

NAKAMURA, KAZUIOSSE. 1982. Especializacao fisiologica em *Colletotrichum graminicola* (Ces.) Wils. (sensu Arx, 1957) agente causal da anthracnose do sorgho (*Sorghum* spp). Doctoral thesis, Universidade Estadual Paulista, Jaboticabal, Brazil. 143 pp.

PASTOR-CORRALES, M.A. 1980. Variation in pathogenicity of *Colletotrichum graminicola* (Cesati) Wilson and in symptom expression of anthracnose of *Sorghum bicolor* (L.) Moench. Ph.D thesis, Texas A&M University, College Station, Texas, USA. 122 pp.

PASTOR-CORRALES, M.A., and FREDERIKSEN, R.A. 1979. Anthracnose and other sorghum diseases in Brazil. Pages 76-79 *in* Proceedings of the Eleventh Biennial Grain Sorghum Research and Utilization Conference, sponsored by the Grain Sorghum Producers' Association (GSPA) and Sorghum Improvement Conference of North America. Available from GSPA, Abernathy, Texas, USA.

PASTOR-CORRALES, M.A., and FREDERIKSEN, R.A. 1980. Sorghum anthracnose. Pages 289-294 *in* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT. 469 pp.

POLITIS, D.J. 1975. The identity and perfect state of *Colletotrichum graminicola.* Mycologia 67:56-62.

REYES, L, FREDERIKSEN, R.A., and WALKER, H.J. 1969. Anthracnose incidence on grain sorghum in the south Texas coastal bend area in 1968. Pages 8-9 *in* Proceedings of the Sixth Biennial Grain Sorghum Research and Utilization Conference, sponsored by the Grain Sorghum Producers' Association (GSPA) and Sorghum Improvement Conference of North America. Available from GSPA, Abemathy, Texas, USA.

ROSENOW, D.T., and FREDERIKSEN, R.A. 1982. Breeding for disease resistance in sorghum. Pages 447-455 *in* Sorghum in the Eighties: Proceedings of the International Symposium on Sorghum, sponsored by INTSORMIL, ICAR, and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

SUTTON, B.C. 1968. The appressoria of *Colletotrichum graminicola* and C. *falcatum.* Canadian Journal of Botany 46:873-876.

TARR, S.A.J. 1962. Diseases of sorghum, sudan grass and broom corn. Kew, Surrey, U.K.: Commonwealth Mycological Institute. 380 pp.

WHEELER, H., POLITIS, D.J., and PONELEIT, C.G. 1974. Pathogenicity, host range/and distribution of *Colletotrichum graminicola* on corn. Phytopathology 64:293-296.

WOLFE, M.S., and BARRETT, J.A. 1980. Can we lead the pathogen astray? Plant Disease 64:148-155.

Questions

Seetharama:
It is difficult to induce (cause) anthracnose before 6th leaf stage. Is this related to the supply of seed reserves to younger leaves? If so, we can manipulate the food supply to the young leaves and try to cause infection.

Frederiksen:
Dhurrin is higher in sorghum seedlings and has been suggested as the reason sorghum seedlings are more resistant to some pathogens.

Periconia Root Rot

Periconia Root Rot

G.N. Odvody and L.D. Dunkle*

Summary

Milo disease, or periconia root rot, threatened to curtail cultivation of milo and milo derivative sorghums in several USA plains states in the 1920s and 1930s. Resistant germplasm from other sources and from among surviving resistant mutants alleviated the problem by the early 1940s. Periconia circinata *was not identified as the causal agent of milo disease until 1948. Virulent isolates of* P. circinata *produce a host-specific toxin* (*tox*+); *host susceptibility to the pathogen and sensitivity to the toxin are conferred by* a *semidominant allele at the* pc *locus. Most strains of* P. circinata *in the milo disease nursery at Garden City, Kansas, cannot produce toxin* (*are tox*-). *In the Texas and Kansas milo disease nurseries, both tox* and tox- isolates of* P. circinata *were obtained from roots of susceptible cultivars, but only tox- isolates were obtained from resistant cultivars. In the 1970s, root rots associated with* P. circinata *were reported on resistant sorghum cultivars in the United States and Australia. No isolate of* P. circinata *from resistant cultivars at any location has produced a demonstrable toxin active against any cultivar. In the absence of susceptible cultivars,* P. circinata *is apparently either a saprophyte or a low-virulence pathogen with a basic level of aggressiveness and is restricted almost exclusively to sorghum.*

Historical Occurrence of Milo Disease

Milo disease, or periconia root rot, is caused by the imperfect fungus *Periconia circinata* (Mang.) Sacc. The disease was first reported in Texas (Chillicothe) in 1924 and then in 1926 in Kansas (Garden City) (Tarr 1962, Leukel 1948, Elliott et al. 1937). In the 1920s and 1930s, this disease threatened to curtail production of susceptible sorghums in the states of Texas, Kansas, Oklahoma, New Mexico, Nebraska, Arizona, and California (Elliott et al 1937, Tarr 1962). Resistant germplasm was available from other sorghum types and was also selected from among surviving resistant mutants in the field and increased (Melchers and Lowe 1943, Karper 1949, Tarr 1962). By the late 1930s and early 1940s, resistant sorghums had largely alleviated the milo disease problem (Tarr 1962, Quinby and Karper 1949), but the original milo disease nurseries at Chillicothe and Garden City are still being maintained. They have provided a continuing, valuable resource for evaluating resistance of sorghum germplasm and for other research purposes.

Symptoms

A summary of milo disease symptoms from several publications is contained in the book by Tarr (1962). Milo disease was initially described as a root, crown, and shoot rot (Elliott et at. 1932). Sus-

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International Crops Research Institute for the Semi-Arid Tropics. 1984. Sorghum Root and Statk Rots, a Critical Review: Proceedings of the Consultative Group Discussion on Research Needs and Strategies for Control of Sorghum Root and Stalk Rot Diseases, 27 Nov -2 Dec 1983, Bellagio, Italy. Patancheru, AP. 502 324, India: ICR1SAT.

ceptible cultivars, growing in soils with apparently high levels of inoculum, began showing symptoms similar to drought stress within a few weeks of planting. Leaves on these plants wilted, drooped, and became slightly rolled, and older leaves turned yellow with leaf tips and margins becoming desiccated and necrotic. The youngest leaves were usually the last to become discolored and die. Plants were generally stunted, and they often died without producing a head. Under conditions of lower inoculum potential and an environment less favorable for disease expression, susceptible cultivars showed initial symptoms at a later stage of growth, and although plant growth was more vigorous, heads were few and poorly filled. In some soils, plants appeared normal or nearly normal throughout the season, but roots were attacked and crowns were internally reddened. Milo disease was commonly identified by splitting open crowns to reveal this dark-red discolored tissue.

Because leaf symptoms were mediated through root damage, the latter were attacked prior to appearance of foliar symptoms (Tarr 1962, Wagner 1936), Roots of infected seedlings showed a water-soaked, reddish discoloration of the cortical and vascular tissues. As the plant aged and the disease developed, smaller roots were destroyed and larger ones became dark red or brown, especially in the stelar tissue, and finally disease symptoms progressed into the crown.

Pathogen

The causal agent of milo disease proved elusive to initial investigators, who probably isolated saprophytes and other primary and secondary pathogens from the roots of dead and dying plants. In 1937, Elliott et al. provided evidence that *Pythium arrhenomanes* was the pathogen causing milo disease. However, they and subsequent workers (Ezekiel 1938, Melchers 1942, Tarr 1962) determined that P. *arrhenomanes* killed resistant as well as susceptible cultivars in the greenhouse and was unable to reproduce symptoms of the disease in field-grown plants. In 1947, Leukel and Pollack suggested *Periconia circinata* as the causal agent because of its frequent isolation from roots of infected plants. The definitive work of Leukel (1948) firmly established P. *circinata* as the causal agent of milo disease more than 10 years after the advent of resistant sorghums (Quinby and Karper 1949).

P. *circinata* was first described from roots of wheat in France (Mangin 1899). According to Leukel (1948), the mycelium of P. *circinata* from sorghum is slender (2-6 µ), branched, and dirty-white to mouse-gray on potato dextrose agar (PDA), but turns black upon sporulation. The aerial conidiophores, single or in groups of two or three, are dark brown to black, thick-walled, and 6-8 µ x 150-250 y . They are typically curved or circinate near the apex, with a slightly swollen apical cell bearing generally three sporogenous cells that undergo division, to form more sporogenous cells. Spherical conidia are borne on these cells in basipetal succession, sometimes in short chains. The conidia are dark brown to black, 15-27 µ diam, and verrucose-spiny when mature.

Another species, *Periconia macrospinosa,* (Lefebvre et al. 1949) often occurs on diseased or senescent sorghum roots and can be easily mistaken for P. *circinata.* Compared to P. *circinata,* the conidiophores of P. *macrospinosa are* longer and more erect, and the conidia have much larger spines. Lefebvre et al (1949) demonstrated that P. *macrospinosa* was not a pathogen of either sorghum or other grasses.

Leukel (1948) had suggested that a pathogen-produced toxin might be involved in milo disease development, and Scheffer and Pringle (1961) demonstrated the production of a host-specific toxin by P. *circinata* that was active only against susceptible cultivars. All symptoms of milo disease are induced by this toxin in the absence of the pathogen (Scheffer and Pringle 1961). Wolpert and Dunkle (1980) demonstrated that the toxin of P. *circinata* was composed of two toxic low-molecular-weight, acidic compounds containing aspartic acid and one or more residues of a polyamine. The latter is apparently responsible for selective biological activity. Host susceptibility to P. *circinata* and sensitivity to its toxin are conferred by a semidominant allele at the *pc* locus (Schertz and Tai 1969). Homozygously recessive *(pcpc)* plants are resistant; heterozygous plants *(Pcpc)* are intermediate; and homozygous dominant plants *(PcPc)* are fully susceptible (Schertz and Tai 1969). The *Pc* allele of the gene is relatively unstable, and mutations of *Pc* to *pc* occur in one of approximately 8000 gametes (Schertz and Tai 1969). This genetic instability was probably responsible for the frequent appearance of resistant plants among those of susceptible genotypes and contributed to rapid development of isogenic resistant sorghum lines (Quinby and Karper 1949, Schertz and Tai 1969),

Conidia and the Infection Process

Leukel (1948) noted the abundance of conidia produced by P. *circinata,* but he and others (Pringle and Scheffer 1963, Oswald 1951) rarely observed their germination. These studies utilized either mycelial or single conidiophore isolates. Dunkle et al. (1975) demonstrated that conidia of P. *circinata* from culture germinated on PDA at higher rates (50-70%) when subjected to a heat shock (45-48°C for 10 min) and at variable rates when not treated (2-54%). Conidia produced on inoculated and field-infected roots responded in a similar manner (Dunkle et al. 1975, Odvody et al. 1977). Conidia of P. *circinata* adjacent to sorghum roots in distilled water had a higher germination rate (88%) than in either distilled water alone (0%) or on PDA (15%) (Odvody et al. 1977). Conidial germination in concentrated root exudates from root washings was greater (22%) than in distilled water alone (6%) (Odvody et al. 1977). On roots in liquid nutrient culture, conidia germinated at a high frequency, forming conidial germ tubes and appressoria-like structures within 48 hours and small, red, cortical lesions within 3-5 days (Odvody et al. 1977). P. *circinata* was easily re-isolated from cortical lesions incited by any isolate on roots of any cultivar.

Characterization of *Periconia Circinata* in Milo Disease Nurseries

The pathogenic (toxin-producing, tox+) strains of P. *circinata* have been perpetuated in nurseries at Garden City, Kansas, and Chillicothe, Texas, by continuously growing susceptible cultivars (primarily S Colby milo) for more than 50 years. Despite this monoculture, only 13% of the P. *circinata* isolates from the soil in the Garden City nursery were tox+ (Odvody et al. 1977). In this same nursery, only 34% of the P. *circinata* isolates from infected roots of a susceptible cultivar were tox+ (Odvody et al. 1977). Although this demonstrated some selection for tox+ strains (34% vs 13%), the predominant strain in the soil population is apparently unable to produce toxin (is tox-). The predominance of tox- isolates on susceptible cultivars may be explained by growth of these isolates in toxin-affected tissue near lesions caused by the tox+ strain (Odvody et al. 1977). Oswald (1951) also obtained several tox- isolates of P. *circinata* from roots of susceptible cultivars in California.

Only tox- isolates were obtained from roots of resistant cultivars growing in the Garden City nursery (Odvody et al. 1977). If tox+ and tox- strains differed only in toxin-producing ability, then we would expect isolates from resistant cultivars at Garden City to reflect the proportions existing in the soil population (i.e., 13% tox+). Although we did not evaluate soil populations in the Chillicothenursery, we obtained only tox- isolates from resistant cultivars, and the proportion of tox+:tox- isolates from susceptible cultivars was similar to that obtained at Garden City (Odvody and Dunkle, unpublished data, 1981-1982).

In laboratory pathogenicity tests, conidia from tox+ and tox- isolates germinated on roots and incited cortical lesions on roots of both resistant and susceptible cultivars, but extensive vascular lesions and seedling death occurred only on susceptible plants inoculated with tox+ isolates (Odvody et al. 1977). These results, considered together with the absence of tox+ isolates from resistant cultivars, indicate that the increase or even maintenance of tox+ isolates in soil is unlikely without regular presence of susceptible cultivars. Odvody et al. (1977) postulated that prior to the introduction of sorghum, P. *circinata* existed in North America as a soil saprophyte or weak parasite of native plants. Several factors influenced incidence and severity of milo disease in specific years, but disease severity apparently increased with continuous sorghum cropping (Elliott et al. 1937, Quinby and Karper 1949). This suggests that tox+ strains comprised a minute proportion of the soil population of P *circinata* until monoculture of susceptible cultivars selected for and increased the tox+ strain to a threshold level where damage was apparent (Odvody et al. 1977). It is unlikely that the ability of P. *circinata* to produce a pathotoxin was the result of a recent mutational event because milo disease developed over a wide geographical area in a very short time (Odvody et al. 1977). Suggested methods of pathogen dispersal (Quinby and Karper 1949) cannot account for the sporadic and sometimes localized occurrence of milo disease (Odvody et al. 1977).

Periconia Problems in Resistant Sorghums

In the early 1970s, there were several reports of P. *circinata* associated with root rots of cultivars pre-

viously known to be resistant (Rosenow and Frederiksen 1972, Troutman and Voigt 1971, Odvody et al. 1977, Dunkle 1979). Plants of these cultivars had poorly developed heads and root systems, necrotic roots, and root lesions, with P. *circinata* present either before or after incubation in the laboratory (Odvody et al. 1977, Rosenow and Frederiksen 1972, Troutman and Voigt 1971). Previously unreported on sorghum outside the United States, P. *circinata* was reported on sorghum roots of resistant cultivars in Australia (Mayers 1976). However, in Australia and the United States, isolates of P. *circinata* from resistant cultivars have neither produced a demonstrable toxin active against either resistant or known susceptible cultivars nor reproduced the above field disease symptoms on inoculated plants (Odvody et al. 1977; Odvody and Dunkle, unpublished data; Rosenow and Frederiksen 1972; Troutman and Voigt 1971; Burns 1974). However, Odvody et al. (1977) demonstrated that the tox- isolates they tested were all low-virulence pathogens on roots of susceptible and resistant cultivars in the laboratory. P. *circinata* was also observed on roots of apparently healthy sorghum plants with well developed heads and root systems (Odvody et al. 1977).

Pythium graminicola was demonstrated to be the primary cause of the root rot in North Texas (Frederiksen et al. 1973; Pratt and Janke 1980; and Odvody, unpublished data), but P. *circinata* was ubiquitous on (or always isolated from) senescent and dying roots and caused numerous small cortical lesions on buttress roots similar to those produced on seedling roots in the laboratory (Odvody et al. 1977).

The reported periconia root rot problems in Arizona and California are not yet resolved (R.L. Voigt, University of Arizona; personal communication, 1983). Partial control of the disease through treatment of soil with benomyl (Burns 1974) implicated involvement of a fungal pathogen (including P. *circinata*) in the Arizona root rot problem. Many elements of the disease syndrome are similar to typical milo disease, including greater damage in late summer plantings, foliar stress symptoms, and stunting (Burns 1974). Genetic studies in Arizona demonstrated that reaction to the root disease was controlled by a single, semidominant major factor favoring susceptibility (Burns 1974). Dunkle (1979) showed that a shattercane (feral *Sorghum bicolor)* source in Nebraska was heterogenous in reaction to the known *Periconia* toxin and tox+ isolates. Dunkle (1979) suggested that these reactions implicated additional genetic factors beyond the two known alleles (*Pc* and pc). The susceptibility of such standard resistant cultivars as Redlan in Arizona (Troutman and Voigt 1971, Burns 1974) necessarily focused more attention on potential changes in the pathogen (Voigt 1972). However, no toxin production was demonstrated (Troutman and Voigt 1971), and root damage was confined primarily to the cortical tissue (Burns 1974), unlike the distinct red stele so characteristic in roots of plants with milo disease.

Most evidence suggests that P. *circinata* is a low-virulence root pathogen restricted primarily to sorghum. Except for its original description on wheat (Mangin 1899) and the reports by Glynne (1939) and Mayers (1976), P. *circinata* is almost exclusively reported on sorghum, and we have not encountered it as a pathogen or saprophyte on roots of any other crop. Odvody (unpublished data, 1978) isolated from wheat straw in Nebraska a saprophytic species of *Periconia* that had some characteristics similar to P. *circinata,* but its conidiophores were more circinate and the culture morphology was different. This *Periconia* species produced no demonstrable toxins against either susceptible or resistant sorghum cultivars, did not incite cortical lesions, and had other dissimilar characteristics that were distinct from P. *circinata.* All isolates (tox- and tox+) of P. *circinata* incited at least cortical lesions on all sorghum cultivars evaluated (Odvody et al. 1977). The true saprophyte P. *macrospinosa* does not incite lesions, and we have observed it on roots of other crops (e.g., maize).

Conclusions

Despite fragmentary data and unresolved root rot problems, some conclusions can be drawn about P. *circinata* as a pathogen of sorghum. The host-specific toxin of P. *circinata* is not required for initial infection of root tissue and early lesion development seen on both resistant and susceptible cultivars. But toxin is required for P. *circinata* to further colonize and kill extensive areas of root tissue on susceptible cultivars. Similar root system damage on susceptible cultivars in the field results in the stem and foliar symptoms commonly associated with milo disease.

Soil populations of P. *circinata* are probably composed predominantly of tox- strains that are weakly virulent pathogens of sorghum but contain an initially undetectable proportion of tox+ strains

capable of increasing when susceptible cultivars are grown in monoculture.

If other host-specific tox+ strains exist as recurring, minute proportions of the *Periconia* soil population, the past 50 years of growing resistant (*pcpc*) sorghums should have allowed sufficient time for selection and increase of any strains specific to these sorghums. Although we expect a predominance of tox- isolates from resistant plants, the complete absence of tox+ isolates from resistant plants negates the disease involvement of a toxin identical to the one already known. Postulating that we have not yet developed proper production and detection methods for these toxins is to assume unrealistically that there are different nutritional requirements for toxin production and even a radically different toxin. The lack of differential virulence among tox- isolates and their inability to reproduce major field disease symptoms on any sorghum argue not only against toxin involvement but against long-term selection of more virulent tox- strains.

The small, cortical lesions incited by tox- strains of P. *circinata* on sorghum roots in the field may either allow the pathogen an advantage in pioneer colonization of dead or dying roots, or, with stress-induced physiological changes, the pathogen may more easily parasitize root tissue. Such a hypothesis is not inconsistent with the normal pathogenic association where tox+ P. *circinata* grows extensively only in susceptible tissue affected by toxin.

The role of P. *circinata* as a soilborne pathogen and saprophyte of sorghum roots is probably interrelated with several factors of the soil environment, including microflora and other root pathogens.

Suggestions for Research

1. Use of chemicals, soil fumigation, and manipulation of environmental variables in the field and laboratory to determine the actual role of P. *circinata* in the Arizona root rot problem and wherever P. *circinata* is implicated as a sorghum root pathogen.
2. Use of a mixture of isolates of P. *circinata* in pure culture inoculations to facilitate detection of a virulent toxin producer.
3. Evaluation of the *Periconia* population in Arizona soils and in other soils around the world, especially where milo types either originated or are grown.
4. Determination of genetic relationships between the *Pc* locus and disease reaction in Arizona and California utilizing crosses between TX09 (resistant in Arizona), Redlan (susceptible in Arizona), and the isogenic lines of S and R Colby that are susceptible (*PcPc*) and resistant (*pcpc*), respectively, to known tox+ isolates of P. *circinata.*

References

BURNS, M. 1974. Inheritance and etiology of an undescribed root disease in *Sorghum bicolor*(L.) Moench. Ph.D. thesis, University of Arizona, USA. 49 pp.

DUNKLE, L.D. 1979. Heterogenous reaction of shattercane to *Periconia circinata* and its host-specific toxin. Phytopathology 69:260-262.

DUNKLE, L.D., ODVODY, G.N., and JONES, B.A. 1975. Heat activation of conidial germination in *Periconia circinata.* Phytopathology 65:1321-1322.

ELLIOTT, C., MELCHERS, L.E., LEFEBVRE, C.L., and WAGNER, F.A. 1937. Pythium root rot of milo. Journal of Agricultural Research 54:797-834.

ELLIOTT, C., WAGNER, F.A., and MELCHERS, L.E. 1932. Root, crown, and shoot rot of milo. Phytopathology 22:265-267.

EZEKIEL, W.N. 1938. Sorghum root and crown rot. Texas Agricultural Experiment Station Annual Report 15:118-119.

FREDERIKSEN, R.A., ROSENOW, D.T., and TULEEN, D. 1973. Pythium root rot of sorghum on the Texas High Plains 1972. Sorghum Newsletter 16:137-138.

GLYNNE, M.D. 1939. Fungal invasion of the roots of healthy wheat plants. Transactions of the British Mycological Society 23:210.

KARPER, R.E. 1949. Registration of sorghum varieties, V. Agronomy Journal 41.536-540.

LEFEBVRE, C.L., JOHNSON, A.G., and SHERWIN, H.S. 1949. An undescribed species of *Periconia.* Mycologia 41:416-419.

LEUKEL, R.W. 1948. *Periconia circinata* and its relation to milo disease. Journal of Agricultural Research 77:201-222.

LEUKEL, R.W., and POLLACK, F.G. 1947. *Periconia circinata* (Mangin) Sacc. as a possible causal factor in "milo disease." Phytopathology 37:440.

MANGIN, M.L. 1899. Sur le pietin ou maladie du pied du ble. Bulletin de la Societe Mycologique de France 15:210-239,

MAYERS, P.E. 1976. The first recordings of milo disease and *Periconia circinata* on sorghums in Australia. Australian Plant Pathology Society Newsletter 5:59-60.

MELCHERS, L.E. 1942. On the cause of the milo disease. Phytopathology 2:640-641.

MELCHERS, LE., and LOWE, A.E. 1943. The development of sorghums resistant to milo disease. Kansas Agricultural Experiment Station Bulletin 55. 24 pp.

ODVODY, G.N., DUNKLE, L.D., and EDMUNDS, L.K. 1977. Characterization of the *Periconia circinata* population in a milo disease nursery. Phytopathology 67:1485-1489.

OSWALD, J.W. 1951. The relation of *Periconia* to milo root rot in California. Phytopathology 41:28 (abstract).

PRATT, R.G., and JANKE, G.D. 1980. Pathogenicity of three species of *Pythium* to seedlings and mature plants of grain sorghum. Phytopathology 70:766-771.

PRINGLE, R.B., and SCHEFFER, R.P. 1963. Purification of the selective toxin of *Periconia circinata.* Phytopathology 53:785-787.

QUINBY, J.R., and KARPER, R.E. 1949. The effect of milo disease on grain and forage yields of sorghum. Agronomy Journal 41:118-122.

ROSENOW, D.T., and FREDERIKSEN, R.A.1972. Lodging in the Texas high plains. Sorghum Newsletter 15:133-134.

SCHEFFER, R.P., and PRINGLE, R.B. 1961. A selective toxin produced by *Periconia circinata.* Nature 191:912-913.

SCHERTZ, K.F., and TAI, Y.P. 1969. Inheritance of reaction of *Sorghum bicolor* (L) Moench to toxin produced by *Periconia circinata* (Mang.) Sacc. Crop Science 9:621 - 624.

TARR, S.A.J. 1962. Diseases of sorghum, sudan grass and broom corn. Kew, Surrey, U.K.: Commonwealth Mycological Institute. 380 pp.

TROUTMAN, J.L, and VOIGT, R.L. 1971. Root rot problems associated with sorghum in Yuma, Arizona. Pages 22-23 *in* Proceedings of the Seventh Biennial Grain Sorghum Research and Utilization Conference, sponsored by the Grain Sorghum Producers' Association (GSPA) and Sorghum Improvement Conference of North America. Available from GSPA, Abernathy, Texas, USA.

VOIGT, R.L. 1972. A potential new strain of milo disease. Sorghum Newsletter 15:3.

WAGNER, FA. 1936. Reaction of sorghums to the root, crown, and shoot rot of milo. Agronomy Journal 28:643-654.

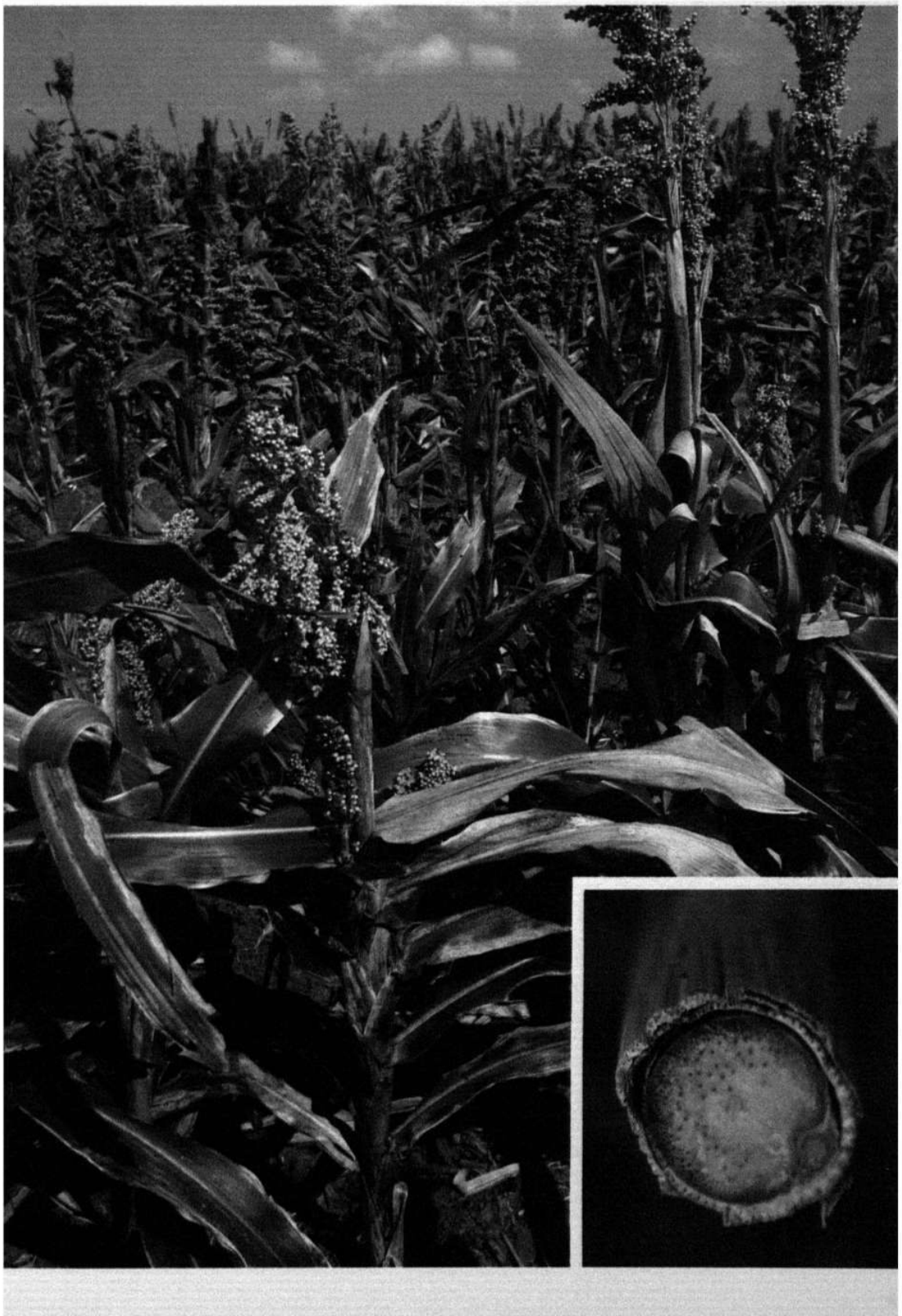
WOLPERT, T.J., and DUNKLE, L.D. 1980. Purification and partial characterization of host-specific toxins produced by *Periconia circinata.* Phytopathology 70:872-876.

Questions

Partridge:
You indicated the toxin produced all symptoms of the disease. You also presented a slide showing lodged plants. My question is: Is there a stalk decomposition or degeneration due to *Periconia* infection or application of its toxin?

Odvody:
I stated that the toxin produced nearly all symptoms of disease, but even this is based on statements of other researchers. To my knowledge researchers have subjected only seedlings to toxin by itself, so I can't directly comment on stalk degeneration. However, plants succumbing to milo disease normally did not lodge in the field.

Acremonium Wilt



Acremonium Wilt

R.A. Frederiksen*

Summary

Acremonium wilt has become an important disease of sorghum in part because of the cultivation of recently developed high-yielding cultivars. The pathogen Acremonium strictum *Gams appears to invade the foliage and colonize vascular tissues. Symptoms include vascular browning and both foliar and stalk wilt. The disease is widespread and probably best controlled by avoiding cultivation of unusually susceptible cultivars.*

Acremonium wilt is one of the more recently described diseases of sorghum (Natural et al. 1982). In Egypt El-Shafey et al. (1979) and *Salama (1979)* have described a wilt of sorghum caused by *Cephalosporium acremonium* Cord. Gams (1971) reduced this species to synonymy with *Acremonium strictum* Gams. In this paper both diseases are presumed to be caused by the same pathogen, and differences between the diseases will be mentioned.

In the USA, acremonium wilt was observed when wilted plants of BTx423, BTx622, and BTx425 developed a vascular wilt at Plainview, Lubbock, and Chillicothe, Texas (Frederiksen et al. 1980). Subsequently, the disease was reported in Argentina (Forbes and Crespo 1982) and Venezuela (Silva et al. 1983), and I observed it in Mexico, Sudan, and Honduras. The disease probably develops in susceptible sorghums wherever the environment favors infection.

Symptoms and Etiology

Symptoms of acremonium wilt involve foliar desiccation and vascular browning of lateral leaf veins. Initially only vascular browning is evident, but as the disease progresses, large areas of wilted tissue develop on an axis of a leaf on either or both sides of the midvein. Vascular plugging continues through the leaf sheath and into vascular bundles of the stalk. Wilted leaves can be distinguished from other pathological or physiological wilt by the vascular browning. In wilted plants free from stalk-rotting organisms, browning of vascular bundles can be followed vertically in the stalk. Infection and colonization from the roots appears to be the exception.

In Egypt, reports suggest that the pathogen is soilborne and colonizes the roots prior to invading vascular tissue (El-Shafey and Refaat 1978). Observations in Texas suggest that infection develops from foliar invasion. Root dipping, soil amending, and hypodermic injection in sorghum whorls with conidia of A *strictum* all cause infection and wilt; however, many cultivars treated in this manner wilt more severely than under natural conditions (Frederiksen et al. 1981). The cultivars Redlan (BTx378), Martin (BTx398), and Wheatland (BTx399) are examples of field-resistant cultivars susceptible to root inoculations. In the Nile delta near Numberia, Egypt, I have observed disease development in the field from foliar infection. According to Salama (1979), wilting occurs commonly in regions of upper Egypt. It may be one of Egypt's most important sorghum diseases. Acremonium wilt is not a stalk rot, because *A. strictum* acts like a true vascular parasite. However, stalk-

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rotting fungi often develop in wilted plants; in this respect *A. strictum* acts as a predisposing agent.

A. *strictum* acts as a saprophyte on dead organic material and is probably a poor colonizer in soils low in organic matter. Sources of primary inoculum and environmental conditions conducive to infection in the field remain unclear. Information on epidemiology may resolve differences between infection patterns in Egypt and elsewhere.

In a recent study (H.J. Kim and J.K. Mitchell, Department of Plant Pathology and Microbiology, Texas A&M University; personal communication, May 1983), blending conidia with steamed soil at planting failed to cause disease, but disease did develop when seedlings were transplanted into infested soil, indicating that root wounding may be necessary for infection. Kim and Mitchell also found differences in pathogenicity between two isolates of A.*strictum* based on symptoms and determined that their isolates infected maize, pearl millet, and oats in greenhouse studies. They were unable to infect wheat and barley with these isolates.

Table 1. Incidence of naturally occurring acremonium wilt among selected sorghum entries at College Station, Texas, in 1980 and Choluteca, Honduras, in 1982.

	% of plants with wilt	
Sorghum entry	College Station	Choluteca
CS-3541 (CSV-4)	38	52
QL-3 (Combine Kafir der.)	2	3
SC-103-12(IS-410 der.)	0	40
SC-170-6-17 (IS-12661 der.)	4	17
SC-326-6 (IS-2816 der.)	11	3
SC-414-12(IS-2508 der.)	18	0
SC-748-5 (IS-3552 der.)	41	35
TAM-428	0	0
Tx-378	5	15
Tx-430	0	10
Tx-623	20	63
Tx-625	61	100
Tx-7078	21	17
77-CS-1 (IS-2930 x IS-3922)	0	25

The Problem

Tropically adapted sorghums derived from IS-12610 are unusually susceptible to acremonium wilt. Elite inbreds such as ATx623 and ATx625 and many hybrids produced with these inbreds are extremely susceptible in Honduras (D. Mechanstock, INTSORMIL Sorghum Breeder, Choluteca; personal communication, 13 Sept 1982). The vulnerability of some of these sorghums raises the issue of the potential risk of growing these agronomically superior inbreds in locations favoring disease development. Reaction of sorghum entries to acremonium wilt in Honduras and Texas are similar (Table 1). Differences in reaction and incidence of disease between these locations may be explained by the variables of host maturity, degree of infection, or observer. Losses on an individual plant basis can be total, but affected plants of susceptible hybrids in one trial produced about half the yield of disease-free controls (Natural et al. 1982).

Another aspect of this problem concerns the geographical distribution of A *strictum.* In a recent visit to Honduras, I observed widespread acremonium wilt in farmers' fields of landrace cultivars. In most of these cultivars, the disease appeared to develop slowly and caused little foliar wilting; however in other fields, presumably sown to other cultivars, *A. strictum* caused severe wilting in up to 30% of the plants. Since plant densities were low, crop loss must have been substantial. It is likely that *A. strictum* was not introduced; rather it was present and not recognized. Similar comments can be made for Sudan and Mexico, where I have observed similar symptoms that have yet to be reported. The global distribution and severity of acremonium wilt awaits further study.

Acremonium wilt can be confused with bacterial streak or symptoms of maize dwarf mosaic because the symptoms overlap. This is particularly true of cultivars with vascular discoloration and limited foliar wilting. For example, Riccelli (1980) reported that QL-3 was susceptible to maize dwarf mosaic virus based on symptoms later determined to have been caused by *A. strictum* (Silva et al. 1983). Most sorghum cultivars appear to be moderately resistant, but further evaluation is necessary. Only with the identification of highly resistant parents and with improved inoculation techniques will it be possible to conduct studies on the inheritance of resistance.

Future Research Needs

1. Clearly, further work is needed to elucidate the etiology of acremonium wilt. Sources of inocu-

lum and inoculum survival, as well as the mode of infection, are poorly understood.

2. The nature of host resistance to the disease needs to be determined. Recognition of the causes of infection may provide some insight into this.

3. Studies should be carried out on the relations between wilt and stalk rotting and lodging; any such interaction may be significant in areas where root infection is important.

4. A reliable large-scale field screening technique must be developed to determine the nature of known field resistance or susceptibility. Susceptible sorghums may represent the exception. Nevertheless highly resistant sorghums remain undescribed.

References

EL-SHAFEY, HA, ABD-EL-RAHIM, M.F., and REFAAT, M.M. 1979. A new *Cephalosporium* wilt of grain sorghum in Egypt. Pages 514-532 *in* Proceedings of the Third Egyptian Phytopathological Congress.

EL-SHAFEY, HA, and REFAAT, M.M. 1978. Studies on the stalk rot diseases of grain sorghum in Egypt. Agricultural Research Review (Ministry of Agriculture, Cairo) 56:71-79.

FORBES, G.A., and CRESPO, L.B. 1982. Marchitamiento en sorgo causado por *Acremonium strictum* Gams. Information Tecnica 89, Estacion Experimental Agropecuaria Manfredi, INTA, Argentina.

FREDERIKSEN, R.A., NATURAL, M., ROSENOW, D.T., MORTON, J.B., and ODVODY, G.N. 1980. Acremonium wilt of sorghum. Sorghum Newsletter 23:134.

FREDERIKSEN, R.A., ROSENOW, D.T., and NATURAL, M. 1981. *Acremonium* wilt of sorghum. Pages 77-79 *in* Proceedings of the 12th Biennial Grain Sorghum Research and Utilization Conference, sponsored by the Grain Sorghum Producers' Association (GSPA) and Sorghum Improvement Conference of North America, Lubbock, Texas. Available from GSPA, Abernathy, Texas, USA.

GAMS, W. 1971. Cephalosporium-Artige Schimmelpilze (Hyphomycetes). Stuttgart, Germany: G. Fisher. 242 pp.

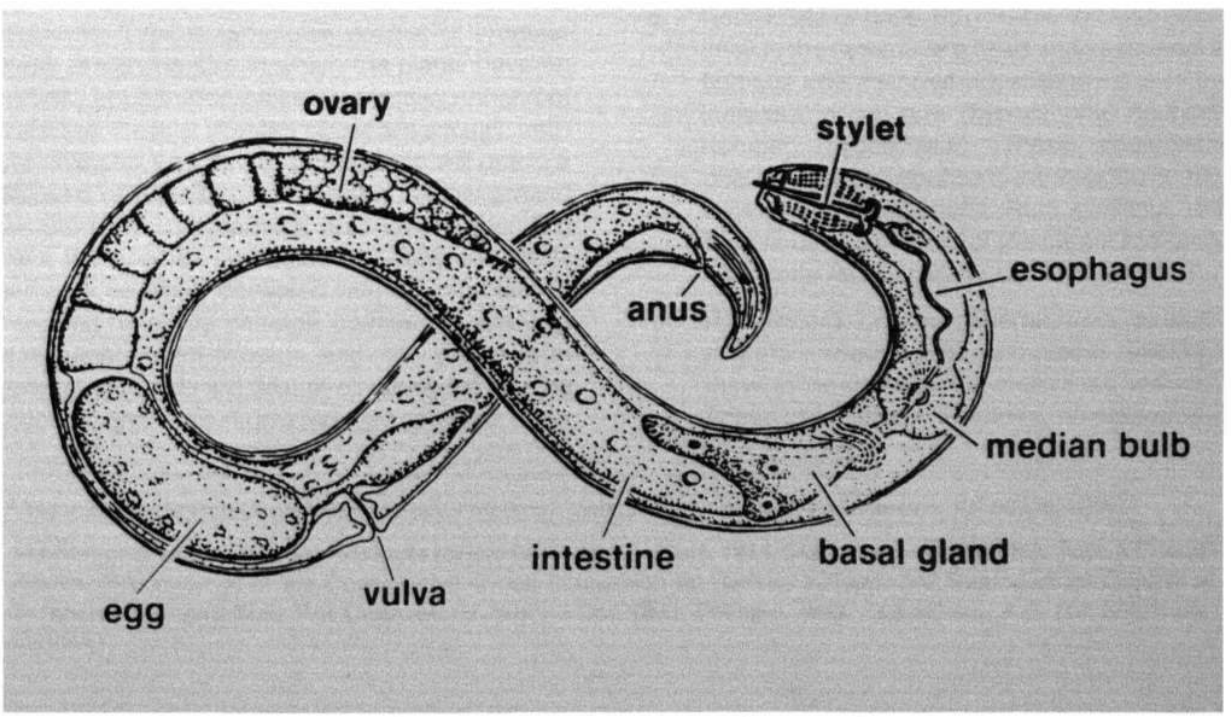
NATURAL, M.P., FREDERIKSEN, R.A., and ROSENOW, D.T. 1982. Acremonium wilt of sorghum. Plant Disease 66:863-865.

RICCELLI, M. 1980. Current strategies and progress in breeding disease-resistant sorghums in Venezuela. Pages 434-453 *in* Sorghum Diseases, a World Review-Proceedings of the International Workshop on Sorghum Diseases, sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, AP. 502 324, India: ICRISAT.

SALAMA, I.S. 1979. Investigations of the major stalk, foliar, and grain diseases of sorghum *(Sorghum bicolor)* including studies on the general nature of resistance. Fourth Annual Report, Field Crops Research Institute, United States Agricultural Research Program, PL 480, Project No. E6-ARS-29, Giza, Egypt.

SILVA, E., SAVINO, A., MENA, T.H., PONS, N. [1983.] *Acremonium striatum* W. Gams en sorgo granifero (*Sorghum bicolor* (L) Moench) en Venezuela. Agronomia Tropical (in press).

Nematodes Affecting Sorghum



Plant-Parasitic Nematodes Affecting Sorghum

L.E. Claflin*

Summary

Plant-parasitic nematodes have been shown to cause yield losses in sorghum. Meloidogyne, Tylenchorhynchus, Belonolaimus, Pratylenchus, Xiphinema, *and* Trichodorus *are important genera in the evaluation of possible nematode damage in sorghum. Nematology research utilizing sorghum as the host crop is very limited. The potential interrelationships of fungi, bacteria, and nematodes as they relate to the stalk rot complex in sorghum have not been researched in depth.*

Plant-parasitic nematodes are often conveniently classified by their feeding behavior. Ectoparasites generally feed on cells near the surface, or they may perforate the cell wall with the stylet and insert the head portion into the cell when feeding. They are generally larger and have a longer stylet than endoparasites. Endoparasites enter the plant, pass through the maturation process, lay eggs, and complete their life process within the plant. Sedentary nematodes enter the root or are attached to plant tissue and remain sessile, whereas migratory nematodes move within the host or between the host and soil.

Nematode damage to field crops is often difficult to ascertain and may closely mimic drought stress, nutrient deficiencies, and other disease and insect problems. Typical symptoms consist of irregular areas of varying size in which the plants have an unthrifty appearance, closely resembling other root maladies. Heavily infested plants are smaller, usually chlorotic, and have a tendency to wilt due to a reduced or unhealthy root system. Below-ground symptoms of root damage may vary, depending upon the specific nematode attacking the roots, and may be easily confused with other problems, including herbicide damage, compacted soil (e.g., plow pans), insect damage, and other diseases. In general, root cells are destroyed during the feeding process, which results in a reduced uptake of water and nutrients. Most nematodes secrete digestive fluids into the tissue while feeding, and a large part of the injury is caused by a reaction of the tissue and digestive fluids, Endoparasites cause substantial damage by their movement through the host tissue.

Nematodes Affecting Sorghum

Table 1 lists those nematode genera that are cosmopolitan and are most likely to be implicated in sorghum yield losses. Symptoms on sorghum roots caused by nematodes would include one or more of the following:

a. Root-knots or galls: Root tissue in close proximity to the nematode's head often assumes a bulbous and distorted appearance due to an increase in cell size (hypertrophy) and cell number (hyperplasia). These anatomical changes are in response to the injected nematode salivary secretions. Root swellings are the principal symptom of the cereal root-knot nematode (*Meloidogyne naasi*) in sorghum.

b. Root lesions: Lesions generally develop when migratory endoparasitic nematodes enter and move in the parenchyma cells of the host root. These cells usually die and cavities develop in

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International Crops Research Institute for the Semi-Arid Tropics. 1984. Sorghum Root and Stalk Rots, a Critical Review: Proceedings of the Consultative Group Discussion on Research Needs and Strategies for Control of Sorghum Root and Stalk Rot Diseases, 27 Nov- 2 Dec 1983, Bellagio, Italy. Patancheru, A.P. 502 324, India: ICRISAT.

Table 1. Plant-parasitic nematode genera that are pathogenic on sorghum.

Nematode	Mode of parasitism	Characteristic symptoms	Hosts other than sorghum
Root-lesion (*Pratylenchus* spp)	Endoparasitic	Decline in plant vigor, necrotic root lesions, association with microorganisms in causing disease complexes, low kernel test weight	Grasses, cereals, cabbage, beet, tomato, legumes, tobacco; over 400 hosts
Root-knot (*Meloidogyne* spp)	Endoparasitic	Decline in plant vigor, stunting, root galls, proliferation of branch roots, reduced stands, delay in flowering	Grasses, cereals, legumes, cotton, tobacco, tomato, potato
Stunt (*Tylenchorhynchus* spp)	Ectoparasitic	Stunting, lack of root development, decline of seedling vigor, root tips may be short and thickened	Grasses, cereals, legumes
Sting (*Belonolaimus* spp)	Ectoparasitic	Decline in plant vigor, stunting, root systems with very limited development, threshold level in maize is 1 sting nematode/ 100 cm^3 soil, generally only detected in sandy soils	Cotton, cereals, legumes, vegetables, tobacco
Dagger (*Xiphenema* spp)	Ectoparasitic	Decline in vigor, poor root development, extensive necrosis of root tissue	Citrus, fruit and shade trees, cereals, grasses, legumes, vegetables

the tissue. If extensive damage has occurred, the cortex tissue may slough away from the endodermis. Small roots may be girdled by injuries, which results in root pruning and reduces the uptake of water and nutrients. Digestive enzymes are secreted during the feeding process, which often results in death of cells. Primary and secondary microorganisms enter the roots through the wounds caused by nematodes.

c. Abnormal or reduced root development: Ectoparasitic nematodes commonly feed on root tissue near the meristematic and cell-elongation regions. Either the injection of enzymes or the stylet entering the cell will cause death of the cell, or that cell will cease to perform its intended function. With extensive feeding, elongation of root tissue is minimal, while the diameter of the root increases, producing the symptom of short, thickened roots. In certain cases (e.g., *Belonolaimus* spp), extensive necrosis develops where the nematode has fed near the meristematic region and destroys the ability of the root to grow and develop.

Sorghum Yield Losses

Estimated loss of grain and forage sorghums in the United States is 6% (Anonymous 1971). Losses vary from locality to locality and from areas within a particular field. Numerous factors are involved, including soil type, rotational sequence with other crops, tillage practices, and application of insecticidal chemicals. In Kansas, insecticide treatments resulted in 43% (1981) and 7% (1982) increases in sorghum yields above the untreated control (Claflin et al. 1983). Carbofuran (Furadan) 4F (1.12 kg a.i./ha band) and fonofos (Dyfonate, 2.24 kg a.i./ha band) were the most effective treatments, increasing yield 56% in 1981 and 15% in 1982. In general, band applications were more effective than in-furrow applications. *Tylenchorhynchus martini* populations increased from 178 (preplant) to 2942/100 cm^3 at physiological maturity (Hafez and Claflin 1982). Sorghum growth in soil infested with *T. nudus* was reduced 10% in fertilized and 56% in nonfertilized plots in South Dakota (Smolik 1977),

The effect of *Quinisulcius acutus* (stunt nematode) on foliar and root weight and height of sorghum plants was enhanced as the nematode

populations were increased (Table 2). *Pratylenchus zeae* and 0. *acutus* were recovered from 61 % of soil samples and 48% of root samples from sorghum fields in Mississippi (Cuarezma-Teran 1983). The economic threshold of 0. *acutus* was in the range of 100 to 1000 nematodes/100 cm^3 soil (Table 2).

Several root-knot species, including *Meloidogyne incognita* (Syn. *M. incognita acrita)* and *Meloidogyne acronea,* have been reported as parasites on sorghum. The cotton root-knot nematode (*M. incognita*) has caused serious losses in sorghum when included in a rotational sequence with cotton (Orr 1967). *M. acronea* has been reported only from South Africa (Coetzee 1956, Coetzee and Botha 1965). Typical field symptoms resulting from *M, incognita* infestations include irregular areas containing chlorotic and stunted plants, delayed flowering, and yield reductions up to one-third. Root tissue may exhibit galls, elongated swellings, and discrete knots or swellings with root proliferation (Orr and Morey 1978). *M. acronea* symptoms are subtle, with inconspicuous galls on roots and limited or no visible effect on plant growth.

The cereal root-knot nematode, *M. naasi,* is a parasite of cereals, grasses, and sugar beet in Wales, Belgium, England, Yugoslavia, Iran, and the United States. Five physiological races of *M. naasi* exist; however, only the Kansas isolate (Race 5) was capable of producing egg masses on sorghum (Michell et al. 1973). Prominent symptoms attributed to *M. naasi* include stunted, chlorotic plants in irregular patterns within sorghum fields (Aytan 1968, Aytan and Dickerson 1969). Root galls may be elongated swellings or discrete knots that are relatively small in comparison to other root-knot galls. Infested roots are often curved in the shape of a hook, horseshoe, or a complete spiral without excessive proliferation of secondary roots, as is common with other *Meloidogyne* spp.

P. zeae, Helicotylenchus spp, and *Tylenchorhynchus crassycaudatus* were commonly detected in soil samples from sorghum fields in Puerto Rico (Ayala and Bee-Rodriguez 1978). *Aphelenchus, Aphelenchoides, Tylenchus, Pseudhalenchus, Trophurus, Neotylenchus, Longidorus, Meloidogyne,* and *Rotylenchulus* were other genera identified in lesser numbers from soil and root samples of sorghum. *P. zeae* was implicated in causing death of 2- to 3-week-old sorghum plants. The plants exhibited a purple color, wilted, and died within several days. Roots of these plants assumed a dark red color and the cortex was generally separated from the endodermis (Ayala and Bee-Rodriguez 1978).

Economic Threshold Levels

Research involving tolerance levels of sorghum to plant-pathogenic nematodes has received very limited attention. Various parameters, including the reproductive potential of the nematode species, host plant genotype, and the effect of environment, must be ascertained before establishing threshold limits. Other complicating factors in establishing levels include the interaction of other nematodes with the target organism, interactions with soil microorganisms, and the susceptibility or tolerance of the particular cultivar of the host. Economic threshold levels of *Q. acutus* appear to range between 100 and 1000 nematodes/100 cm^3 soil (Table 2). In contrast, only one sting nematode per sample (100 cm^3 soil) is an economic threshold level for sorghum in South Carolina (SA Lewis,

Table 2. Effect of various inoculum levels of *Quinisulclus acutus* on sorghum grown under greenhouse conditions. (Source: Cuarezma-Teran 1983.)

Treatments	Plant height (cm)	Top weight (g)		Root weight (g)	
		Fresh	Dry	Fresh	Dry
Control	55.87a*	25.43a	3.36a	8.31a	2.51a
Supernatant	53.93a	24.41 ab	2.93ab	8.01a	2.25a
Sterile Supernatant	52.00a	23.25abc	2.98ab	6.03ab	2.25a
100 nematodes	55.00a	24.22ab	2.97ab	9.02a	2.42a
1000 nematodes	38.37b	17.86bc	2.28b	3.83b	0.83b
5000 nematodes	37.27b	17.27c	2.34b	3.82b	1.04b

*Means followed by the same letter indicate no significant difference ($P<0.05$), according to Duncan's Multiple Range Test.

Associate Professor, Clemson University, Clemson, S.C., USA; personal communication, 1983).

In preliminary tests, two sorghum accessions (B 35-6 and BTx 378) were found to be less favorable for nematode reproduction, although not significantly different (Table 3) (L.E.. Claflin and T.C. Todd; unpublished data, 1983). In several instances, the fresh foliar weight of inoculated plants exceeded those of the controls. As expected, greater differences were observed in root weights.

Disease Complexes Involving Fungi, Bacteria, and Nematodes

The importance of researching various interactions among microorganisms was stated very eloquently by Fawcett (1931):

> Investigation with one microorganism kept pure and free from contamination with any other has been the classical procedure ever since Koch and others perfected the pure-culture methods that facilitate so greatly the separation of microorganisms. Students in our laboratories have been thoroughly inbred with the idea that cultures must be pure for a single organism. This necessary insistence on pure cultures of single organisms has perhaps led unconsciously to a feeling that to allow the use of a mixture in plant-pathological work is extremely unscientific if it is not actually a deadly plant pathological sin. Nature does not work with pure cultures alone but most frequently with associations.

Numerous interactions of nematode, fungal, and bacterial species in disease complexes on various hosts have been reported (Norton 1978). A comprehensive review of nematode-fungal disease complexes was published by Powell (1971). In sorghum, interactions involving *P. zeae* and *Curvularia* spp, *Fusarium moniliforme, Rhizoctonia solani,* and *Macrophomina* spp resulted in suppressed root and top growth (Table 4). Interactions involving *Macrophomina phaseolina* and *Pratylenchus hexincisus* in sorghum plants with adequate moisture showed little significant difference in disease ratings or top weights (Norton 1958). In the drought-stressed plants, the highest disease rating as well as the lowest foliar dry weight occurred when *M. phaseolina* and *P. hexincisus* were mixed. Lodging was observed only when the fungus and nematode were combined.

Future Research Priorities

1. Nematology research utilizing sorghum as the host crop has received limited attention. Information is needed in the following areas:
   a. surveys to ascertain the genera and species of phytoparasitic nematodes present in samples obtained from diverse areas;
   b. determination of the effect of nematodes on yield when sorghum is grown in various soils, under different cultural practices, and in different climates;
   c. determination of threshold limits if nematodes are shown to be a major factor in

Table 3. Effect of stunt (*Tylenchorhynchus martini*) nematodes on various grain sorghum accessions.

	Stunt population* (100 cm^3 soil)		Fresh foliar weight (g)		Fresh root weight (g)		Plant dry weight (g)	
Pedigree†	I‡	C	I	C	I	C	I	C
SC 599-NE	1147a§	0.0	3.07a	2.59a	3.69b	4.79a	2.61 bc	2.95ab
SC 170-6-17	1060a	0.0	2.63ab	2.13a	4.67ab	5.85a	3.04ab	3.00ab
B 35-6	880a	0.0	2.53bc	2.58a	3.57b	4.25a	2.02c	2.45ab
BTx 378	840a	27.0	2.29bc	2.34a	5.71a	5.25a	3.58a	3.24a
Tx 7078	980a	0.0	2.23bc	2.22a	4.70ab	5.69a	2.76abc	3.31a
SC 103-12	1360a	0.0	2.19bc	2.15a	4.77ab	5.98a	2.70abc	3.25ab
T x 430	1360a	0.0	2.00c	2.36a	5.50a	5.82a	3.18ab	3.12ab

*Populations were determined 60 days after inoculation.

†Accessions obtained from D.T. Rosenow, Texas A&M Agricultural Experiment Station, Lubbock, Texas, USA.

‡Initial inoculum consisted of 500 stunt nematodes per pot; I = inoculated, C = control.

§Number8 in a column followed by the same letter are not significantly different ($P < 0.05$), according to Duncan's Multiple Range Test.

Table 4. Growth and root necrosis of sorghum plants inoculated with *Pratylenchus zeae* alone and in combinations with four soil fungi. (Source: Bee-Rodriguez and Ayala 1977.)

Treatments	Foliar dry weight (g)	Root(g)		Necrosis index*
		Fresh weight	Dry weight	
P. zeae-Curvularia spp	25.1 c†	30.50d	2.6a	0.8d
P. zeae-F. moniliforme	27.4abc	32.25cd	3.1a	1.2bcd
P. zeae-R. solani	27.5abc	33.85cd	3.2a	2.2abc
P. zeae-Macrophomina spp	26.9abc	35.60bcd	3.3a	1.0bcd
F. moniliforme	26.4bc	35.65bcd	3.1a	1.8abc
R. solani	27.8abc	37.80bcd	3.0a	1.6bc
P. zeae	26.4bc	39.20abcd	3.9a	3.2a
Macrophomina sp	28.1 abc	42.35abc	3.8a	1.6bc
Curvularia sp	28.9ab	45.65ab	3.7a	2.4ab
Control	29.7a	49.25a	6.0b	0.0d

*Scale of 0 (no symptoms) to 5 (extensive necrosis).

†Means followed by the same letters do not differ significantly *(P* < 0.05), according to Duncan's Multiple Range Test.

sorghum production. Otherwise control recommendations are of limited value.

2. The potential importance of nematodes in interactions with bacterial and fungal incitants in stalk rot complexes of sorghum has not been researched. Information is needed in the following areas:

   a. the potential synergistic relationships between nematodes and other incitants;

   b. the role of nematodes in "breaking" varietal resistance of host plants to fungi (primarily *Fusarium* spp);

   c. the potential role of nematodes in providing portals of entry for other microorganisms;

   d. the role of nematodes in predisposing the host to invasion and extensive lesion development by other microorganisms present in the rhizosphere. A research project of this type might be applicable to understanding the etiology of seedling disease problems.

References

ANONYMOUS. 1971. Estimated crop losses due to plant parasitic nematodes in the United States. Supplement to Journal of Neonatology, Special Publication No. 1. 7 pp.

AYALA, A., and BEE-RODRIGUEZ, D. 1978. Control of phytoparasitic nematodes attacking sorghum in Puerto Rico. Journal of Agriculture of *the* University of Puerto Rico 62:119-132.

AYTAN, S. 1968. Pathogenicity, life cycle and host range of *Meloidogyne naasi* Franklin found on sorghum in Kansas. M.Sc. thesis, Kansas State University, USA. 43 pp.

AYTAN, S., and DICKERSON, O.J. 1969. *Meloidogyne naasi* on sorghum in Kansas. Plant Disease Reporter 53:737.

BEE-RODRIGUEZ, D., and AYALA, A. 1977. Interaction of *Pratylenchus zeae* with four soil fungi on sorghum. Journal of Agriculture of the University of *Puerto* Rico 61:501 - 506.

CLAFLIN, L.E., HAFEZ, S.L., and TODD, T.C. 1983. Effects of insecticides-nematicides on stunt nematode populations and grain sorghum yields. Page 145 *in* Abstracts of Papers, Fourth International Congress of Plant Pathology, 17-24 August, 1983, University of Melbourne, Melbourne, Victoria, Australia. North Melbourne, Victoria, Australia: Rowprint Services.

COETZEE, V. 1956. *Meloidogyne acronea, a* new species of root-knot nematode. Nature 177:899-900.

COETZEE, V., and BOTHA, H.J. 1965. A redescription of *Hypsoperine acronea* (Coetzee, 1956) Sledge & Golden, 1964 (Nematoda: Heteroderidae), with a note on its biology and host specificity. Nematologica 11:480-484.

CUAREZMA-TERAN, J.A. 1983. Etiology of a sorghum disease complex in Mississippi, Ph.D. thesis, Mississippi State University, Mississippi, USA.

FAWCETT, H.S. 1931. The importance of investigations on the effects of known mixtures of microorganisms. Phytopathology 21:545-550.

HAFEZ, S.L., and CLAFLIN, L.E. 1982. Control of plant parasitic nematodes on grain sorghum and yield response. Fungicide and Nematicide Tests (American Phytopathological Society, St.Paul, Minnesota, USA) 37:198.

MICHELL, R.E., MALEK, R.B., TAYLOR, D.P., and EDWARDS, D.I. 1973. Races of barley root-knot nematode, *Meloidogyne naasi:* I. Characterization by host preference. Journal of Nematology 5:41 -44.

NORTON, D.C. 1958. The association of *Pratylenchus hexincisus* with charcoal rot of sorghum. Phytopathology 48:355-358.

NORTON, D.C. 1978. Ecology of plant-parasitic nematodes. New York, New York, USA: John Wiley & Sons. 268 pp.

ORR, C.C. 1967. Observations on cotton root-knot nematode in grain sorghum in West Texas. Plant Disease Reporter 51:29.

ORR, C.C., and MOREY, E.D. 1978. Anatomical response of grain sorghum roots to *Meloidogyne Incognita acrita.* Journal of Nematology 10:48-53.

SMOLIK, J.D. 1977. Effects of *Trichodorus allius* and *Tylenchorhynchus nudus* on growth of sorghum. Plant Disease Reporter 61:855-858.

Spatial and Temporal Succession of Fungal Species in Sorghum Stalks as Affected by Environment

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Summary

Sorghum plants are infected by various fungi, beginning at the seedling stage and continuing until maturity. The principal avenue of infection by those fungi that cause stalk rot of mature plants, primarily Fusarium *spp and* Macrophomina phaseolina *(Tassi) Gold., is through the roots. Parasitism is established early and continues until an external stress is placed upon the plant. The hypothesis is offered that heat and/or drought stress effects a perturbation of the normal biochemical processes of the stalk tissue, resulting in a quasi-defenseless plant This stress period allows the internal parasites to begin pathogenesis, which leads to the phenomenon of stalk rot.*

In order to address the subject of spatial and temporal succession of fungal species, the entire lifespan of the sorghum plant must be considered. Accordingly, it is necessary to give at least cursory consideration to the various physiological stages through which the plant passes during its developmental process. Inasmuch as pathogenesis occurs in the stalk, our discussion of physiological and biochemical processes of the host will be limited to the stalk. The topics of photosynthate accumulation and source-sink relationships will be left to other discussants. This should not be taken as a failure to recognize these interrelationships but rather as an attempt to keep this presentation within the constraints of environmental effects as they affect, or effect, pathogenesis of stalk-inhabiting parasites.

Additionally, this discussion will be limited to stalk rots involving those pathogens that initially exist as parasites early in the life of the plant and become pathogens only later as the grain reaches maturity. We have chosen not to include anthracnose. Its importance has been amply demonstrated (Chowdhury 1936, Bergquist 1973, Dale 1963, Wheeler et al. 1972, Wheeler et al. 1974). It has been excluded because it is pathogenic upon living tissue and apparently its mode of pathogenesis is quite different from the stalk-rotting organisms that are the subject of this discussion (Katsanos and Pappelis 1965, Katsanos and Pappelis 1966). For like reasons we have excluded *Pythium* spp.

Many of the putative stalk rot pathogens are in reality well-adapted parasites during the greater part of the life of the plant and depend on a weakening of the host in order to become pathogens. Therefore with respect to stalk rots the term pathogen should be applied with caution.

From the outset it must be asserted that sorghum is not maize, and—while both are afflicted by a phenomenon known as stalk rot, are members of the grass family, and have a number of common physiological and biochemical processes that are instructive for comparison—it may be an error to consider that the disease is entirely equivalent in both crops. Sorghum, though grown as an annual in the USA and other temperate regions, is primarily a nonsenescing perennial (Duncan et al. 1981),

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white maize is a senescing annual. Accordingly, the physiological and biochemical processes of the two species are not entirely comparable; therefore the modes of pathogenesis of the several stalk rot organisms may or may not be the same in each host.

Seedborne Organisms

Table 1 presents a compilation of various fungal genera that have been identified in or on seed and are usually reported as part of a head mold complex on sorghum. It is particularly informative to consider these organisms from the perspective of their potential as the initial invaders in a multiple parasite disease. Each of them has also been identified as a constituent of the microflora of stalk-rotted tissue. The ubiquity of the genera *Curvularia, Fusarium* (Williams and Rao 1981), and *Phoma* over such a wide geographic span indicates a potential for universality of the stalk rot phenomenon, rather than a special set of circumstances peculiar to each locale. While of these three only the genus *Fusarium* has been shown to play a major role in stalk rot, it must be acknowledged that

Table 1. Geographical distribution of seed-infecting/infesting fungi involved in seedling-, root-, and/or stalk-rots of sorghum. [a]

	Genus						
Location	*Altemaria*	*Colletotrichum*	*Curvularia*	*Fusarium*	*Helmintho-sporium*	*Nigrospora*	*Phoma*
Bangladesh			x	x	x		
Brazil		x		x			
East Africa	x		x	x	x x		
Ethiopia			x				x
India	x x		x	x	x		
Italy	x			x	x x		
Nigeria	x x		x	x	x x x		
Pakistan	x		x	x			
Philippines			x	x			x
Puerto Rico		x	x	x		x x	
Senegal			x	x			
Thailand			x	x			
USA				x			
Venezuela		x		x			

Table 1 references:

Location	Reference	Location	Reference
Bangladesh	Mian and Ahmed 1980	Italy	D'Ercole and Nipoti 1979
Brazil	Minussi and Kimati 1978	Nigeria	Tyagi 1980
East Africa	Doggett 1980	Pakistan	Hamid 1980
Ethiopia	Hulluka and Gebrekidan 1980	Philippines	Dalmacio 1980
India	Tripathi 1975	Puerto Rico	Feliciano et al. 1982
	Bidari et al. 1978	Senegal	Denis and Girard 1980
	Ravindranath 1980	Thailand	Pupipat 1980
	Siddiqui and Khan 1973	USA	Bain 1950
			Castor and Frederiksen 1980
			Claflin 1981
			Pady1943
		Venezuela	Riccelli 1980

a. Table 1 is not intended to be a complete compilation of all known reports of fungi that are potential stalk rotters, but only to give an indication of the geographical universality of the genera.

any organism that invades the juvenile tissue may debilitate the host sufficiently to allow the succession of other organisms. The lack of identification of any given genus in any geographic location may reflect more upon the interest or techniques of the reporting investigator than upon its presence or absence.

Fusarium moniliforme Sheld. establishes itself as an internal seed parasite (Castor and Frederiksen 1980, 1981), and even though such fungi may seem candidates for seed treatment, the omnipresent nature of these organisms in the soil and their abilities to exist as saprophytes negate the potential for this control measure. Therefore the utility of seed treatment as a control measure for these fungi may be of more academic interest than economic value.

Seedling Infection and Potential Inoculum

Infection of seedlings and/or the establishment of seedborne internal parasites as seedling parasites is nearly coincident with seed germination (Tripathi 1975, D'Ercole and Nipoti 1979, Gourley et al. 1977, Bain 1973, Bee-Rodriguez and Ayala 1977). With respect to charcoal rot, the necessity for seedling infection/infestation by *Macrophomina phaseolina* was documented by Odvody and Dunkle (1979), and is assured by the presence of the sclerotia in soil and their ability to survive (Chidambaram and Mathur 1975; Jadhav 1978; Shokes et al. 1977; Bhattacharya and Samaddar 1976; Livingston 1945a, 1945b) more than one cropping season devoid of host material (Cook et al. 1973, Watanabe et al. 1970). Livingston (1945a, 1945b) reported that seedling blight occurred as a result of infection of the primary roots by *M. phaseolina.* In view of the work of Smith (1969a, 1969b), it is probable that root exudates from the sorghum seedlings triggered sclerotial germination. In greenhouse experiments, soil temperatures above 30°C resulted in 74% infection and up to 30% blighted seedlings (Uppal et al. 1936, Livingston 1945a, 1945b). The importance of soil moisture in conjunction with temperature has been addressed by various workers (Shokes et al. 1977, Odvody and Dunkle 1979, Edmunds et al. 1964). If infection occurred prior to the emergence of secondary roots, the plants succumbed. Less severely diseased seedlings were able to establish secondary roots, and under favorable conditions of sufficient soil moisture, were able to grow to mature plants. Under field conditions, the extent of infection, though not catalogued (Livingston 1945a), was believed to be high. The result of this high level of infection was that *M. phaseolina* was probably systemic in a large percentage of the surviving plants, even though no symptoms were obvious until after a period of environmental stress.

Though several pathogenic strains of *M. phaseolina* have been reported (Khan et al. 1976), the significance of various strains and their different modes of pathogenesis have yet to be investigated or experimentally considered in the development of resistant sorghums.

The work of Reed (1982) and Reed et al. (1982, 1983) indicates that, even in fields where *M. phaseolina* does not play an obvious role in stalk rot, few (if any) seedlings are entirely free of other internal parasites. A consolidation of Reed's analysis of isolation data from various stages of plant growth is presented in Tables 2, 3 and 4.

The data in Tables 2 through 4 are the result of 3 years of field experiments and as such have the advantage of allowing one to assess the import of any given species through the season and observe

Table 2. Percentage of isolation of fungal species from sorghum seedlings.[a]

Fungal species	2 wks	4 wks	6 wks
From stalks			
F. moniliforme	0	0	5
F. graminearum	7	6	5
F. "roseum"	0	0	0
F. tricinctum	1	1	1
F. equiseti	8	8	8
F. oxysporum	4	4	4
Alternaria spp	10	10	10
Epicoccum spp	0	0	0
From roots			
F. moniliforme	5	5	10
F. graminearum	7	10	10
F. "roseum"	5	3	5
F. equiseti	15	10	15
F. oxysporum	20	20	20
Alternaria spp	5	20	20
Epicoccum spp	0	0	0

a. Composite of isolation data taken from Reed (1982). Isolations were taken from apparently healthy tissue and were made onto potato dextrose agar. "Stalks" refers to isolations made from the intemodal tissue between the second and third nodes above the soil level. "Roots" refers to crown tissue and major roots.

Table 3. Percentage of isolation of fungal species from sorghum plants between flowering and soft dough stages. [a]

Fungal species	8wks	10 wks	12 wks
From stalks			
F. moniliforme	12	40	50
F. graminearum	5	6	10
F. *"roseum"*	5	8	10
F. *tricinctum*	1	5	9
F. *equiseti*	8	15	10
F oxysporum	4	6	8
Alternaria spp	15	25	20
Epicoccum spp	0	0	12
From roots			
F. *moniliforme*	10	15	20
F. *graminearum*	12	12	20
F. "roseum"	5	8	15
F. equiseti	15	25	20
F. *oxysporum*	20	15	10
Alternaria spp	25	40	60
Trichoderma spp	40	25	15
Epicoccum spp	0	5	50

a. Composite of isolation data taken from Reed (1982). Isolations were taken from apparently healthy tissue and were made onto potato dextrose agar. "Stalks" refers to isolations made from the internodal tissue between the second and third nodes above the soil level. "Roots" refers to crown tissue and major roots.

Table 4. Percentage of isolation of fungal species from sorghum plant between the soft dough and "mature" stages. [a]

Fungal species	14 wks	16 wks	18 Wks
From stalks			
F. moniliforme	50	70	40
F. graminearum	12	12	30
F. "roseum"	12	12	25
F. tricinctum	15	12	15
F. equiseti	12	15	15
F. oxysporum	10	8	6
Alternaria spp	30	25	15
Epicoccum spp	20	25	20
From roots			
F. moniliforme	20	30	40
F. graminearum	25	20	25
F. "roseum"	15	15	15
F. equiseti	15	20	15
F. oxysporum	15	5	5
Alternaria spp	50	50	50
Trichoderma spp	25	25	25
Epicoccum spp	30	30	25

a. Composite of isolation data taken from Reed (1982). Isolations were taken from apparently healthy tissue and were made onto potato dextrose agar. "Stalks" refers to isolations made from the internodal tissue between the second and third nodes above the soil level. "Roots" refers to crown tissue and major roots.

repeated patterns from year to year. In view of the alleged involvement of F. *moniliforme* as a major contributor in stalk rot (Tullis 1951), it is instructive to note that it is not a primary colonizer of seedlings through root infection. Under the conditions of this study the species *F. graminearum, F. equiseti* (Cda.) Sacc, and *Altemaria* spp are probably more important in the early colonization of seedlings than are any other species. For comparison to other work, it should be noted that the study by Reed et al. was conducted in a conservation tillage system involving a wheat-fallow rotation and no cultivation during the crop season. One might expect an increased inoculum potential for *F. graminearum,* due in part to the wheat in the rotation. The lack of tillage during the crop season is important for discussion because the mode of infection by the organisms isolated must be through means other than cultivation root wounding. The results from this type of tillage system relate well to cultural methods that are not dependent on mechanized agriculture and to efforts aimed at establishing conservation tillage practices.

During the seedling stages of growth, nearly all of the increase in dry weight is in leaf and root tissue, with a relatively small increase in the weight of the crown and stalk, and fungi in the stalk are in an environment completely different from that at the plant's maturity when stalk rot is manifested. Beginning at differentiation of the growing point, the first of two major shifts in growth begins. Leaf expansion is completed, and there is a great increase in stalk volume and dry weight. Stalk cells expand and elongate, and cell density decreases very rapidly. The second major change occurs at anthesis when emphasis shifts from the stalk to the head.

With the onset of flowering, a very dramatic shift in the metabolic activities takes place within the nodes of the plant. Within 5 days of flowering the activity of phenylalanine ammonia lyase (PAL) plummets to a neglegible level, and by this time the anabolic glycosidases have been severely reduced in activity (J.E. Partridge, unpublished data, 1982). The activities of the stalk change from rapid growth and expansion to maintenance and photosynthate accumulation, and finally to translo-

cation for grain fill. These metabolic activities and shifts in composition directed towards accumulation of materials into grain render the plant even more vulnerable to pathogenic activities from its internal parasites.

Postflowering to Soft Dough Stages

Table 3 presents data showing that during this time the stalk becomes even more heavily parasitized by an increasing number of fungal species. The most obvious invader is *F. moniliforme,* which is isolated with increasing frequency well into the 12th week. However, other species are also parasitizing the stalk, though apparently at a lesser frequency than *F. moniliforme.* These include *F. graminearum, F. roseum, F. equiseti,* and *Alternaria* spp. The root system (which has largely ceased growth and expansion by this time) continues to be colonized by various species. The increase in colonization of roots by *Alternaria* spp and *Trichoderma* spp, even when care has been taken to select apparently healthy tissue, may be an indication of increasing saprophytic activity on more mature or senescing tissue.

Evidence indicates that the general pattern of colonization of sorghum stalks and roots by fungi is much the same as that reported for maize (Kommedahl et al. 1979, Young and Kucharek 1977, Warren and Kommedahl 1973). The massive colonization of stalk tissue appears to occur concomitantly with the onset of flowering, but roots seem to be inhabited by fungi regardless of the growth stage of the plant. Fungi are more readily isolated from both stalks and roots as the crop matures.

The fungal species colonizing sorghum in southwestern Nebraska are similar to those reported on maize, especially in the central United States. According to Christensen and Wilcoxson (1966), *F. moniliforme* and *F. graminearum* are commonly associated with ear and stalk rot of maize, and *Nigrospora* spp are less common stalk rot pathogens of maize. *Trichoderma* spp and *Altemaria* spp are frequent secondary invaders of rotted maize stalks. Of the *Fusarium* species that were isolated from sorghum in this study, all are reported to be associated with maize, usually as stem or root parasites.

M. phaseolina was noticeably absent from sorghum stalks and roots throughout the course of the study by Reed et al. (1983). Since charcoal stalk rot of sorghum is fairly common in western Nebraska, it is surprising that *M. phaseolina* was not found as a common inhabitant of sorghum stalks and roots. Since only symptomless plants were sampled, the probability of isolating *M. phaseolina* may have been low due to the absence of environmental conditions favorable for its growth. As mentioned earlier, charcoal rot is favored by high soil temperature and low soil moisture. Of the three seasons in our study, two were unusually cool and moist. The 1980 season most closely approximated "normal" weather conditions for western Nebraska, and even that season was not extremely hot or dry. Additionally, conservation tillage, which was practiced on the test plots, tends to conserve soil moisture and reduce fluctuations in soil temperature. This cultural practice may have been effective in reducing temperature and moisture stress in the roots and thereby inhibiting infection and ramification by this parasite (Doupnik and Boosalis 1980; Doupnik et al. 1975a, 1975b; Edmunds et al. 1975).

"Mature" Plants

The difference between maize (the annual) and sorghum (the perennial) should be noted again at this point. The dry weight percent composition of the maize stalk increases as the plant matures, but the stalk of the sorghum tends to retain a relatively consistent dry weight until it is killed by frost. At least that is the trend in Nebraska, where the planting is done in a cool moist spring, and the plant grows and flowers during a hot dry summer and matures during a progressively cooler fall. Figure 1 shows data (S.G. Jensen, unpublished data, 1983) summarizing several dates of harvest and several genotypes over 2 years. Plants did not dry down from the base to the head or vice versa; in fact they showed little tendency to dry down at all. The crown and the peduncle had the highest level of dry matter, while the middle stalk tissue was the most succulent. These trends show that even within the same stalk there is a gradient of microenvironmental conditions influencing the growth and pathogenicity of the various fungi.

It should also be noted that in our growing conditions there are two general types of tissue necrosis frequently observed. One of these leads to a collapse of the tissue in the base of the plant, premature necrosis of the whole plant, and the lodging of

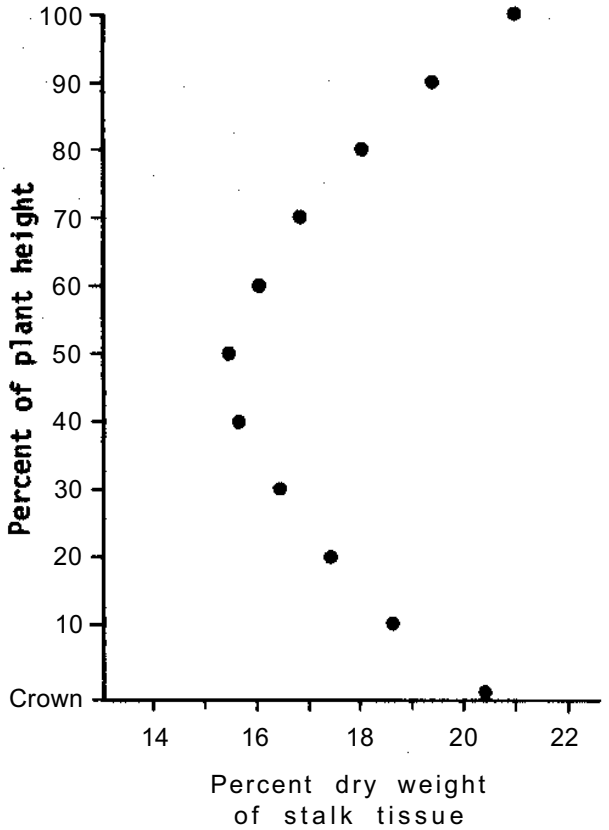

Figure 1. Plant height versus percent dry weight Twenty plants were harvested on each of five dates from soft dough stage to grain maturity. The average percentage of dry weight of the tissue versus the height of the tissue on the plant is presented.

the plant at or near the ground. The second type of necrosis occurs in the peduncle and leads to a premature ripening of the head, with or without breaking of the peduncle. Tissues with either of these conditions have a similar dry weight composition, but it is not known if there is a relationship between that composition and/or the metabolic activities associated with it and the occurrence of rot.

Although it was not tested statistically, a relationship was observed between weather conditions and the number of fungal species found in stalk tissue. The seasons were progressively cooler and wetter from 1980 to 1982, and progressively fewer species were recovered from stalks over the three seasons. Planting was delayed in 1981 and 1982 (Figs. 2a and b), resulting in a shorter growing season and, because of a cool fall, slower crop maturation rate. It was difficult to determine whether the decreases in fungal species were due to climatic conditions, slow maturation rate, lack of stress on the plants, or a combination of all of these factors. The most important observation is that the sequence of infection of stalk tissue by various species was similar during all 3 years, even though the relative abundance of each species differed from year to year. In addition, the sequence could be associated with stages of plant maturity. This suggests that the sequence of infection is a relatively stable and predictable process, associated with physiological and metabolic changes within the stalk as it matures.

As the head approached maturity, *F. moniliforme* was the dominant species isolated from stalk tissue, but it is uncertain whether it predominated by being the most active competitor or if it succeeded simply by default. During 1980 and 1981 an inverse relationship existed between the isolation percentage of *F. moniliforme* from stalks and that of *F. graminearum* and the "roseum" group when they were present: when the incidence of *F. moniliforme* declined, the incidence of these other fungi increased, and the converse was also observed. Populations of *F. equiseti, F. oxysporum* Schlect. emend Snyd. et Hans., and *F. solan*i (Mart.) Appel et Wr. emend Snyd. et Hans appeared to follow a similar but less striking pattern in inverse relation to *F. moniliforme.*

In 1982 *F. graminearum* and the "roseum" group were rarely isolated, while *F. moniliforme* was consistently isolated. In the first 2 years, it appeared that populations of *F. moniliforme* were adversely affected by the composition of stalk tissue as the heads approached maturity, and by the first killing frost. In 1982, though, neither of these trends was observed, possibly because the stalks were still somewhat immature at the first frost date, offering a greater amount of moisture and greater protection from frost damage.

On one hand it might be argued, first, that F. *moniliforme* influenced the incidence of the other *Fusarium* species, as well as having the advantage of being the first to colonize the tissue. Secondly, the decline of F. *moniliforme* could be directly associated with changes in environmental conditions. It is possible that when environmental conditions, such as lower temperatures and greater moisture level, favor the growth of *F. moniliforme,* other species are less able competitors. These other species may be better survivors and may be able to colonize stalks when the population of *F. monili-*

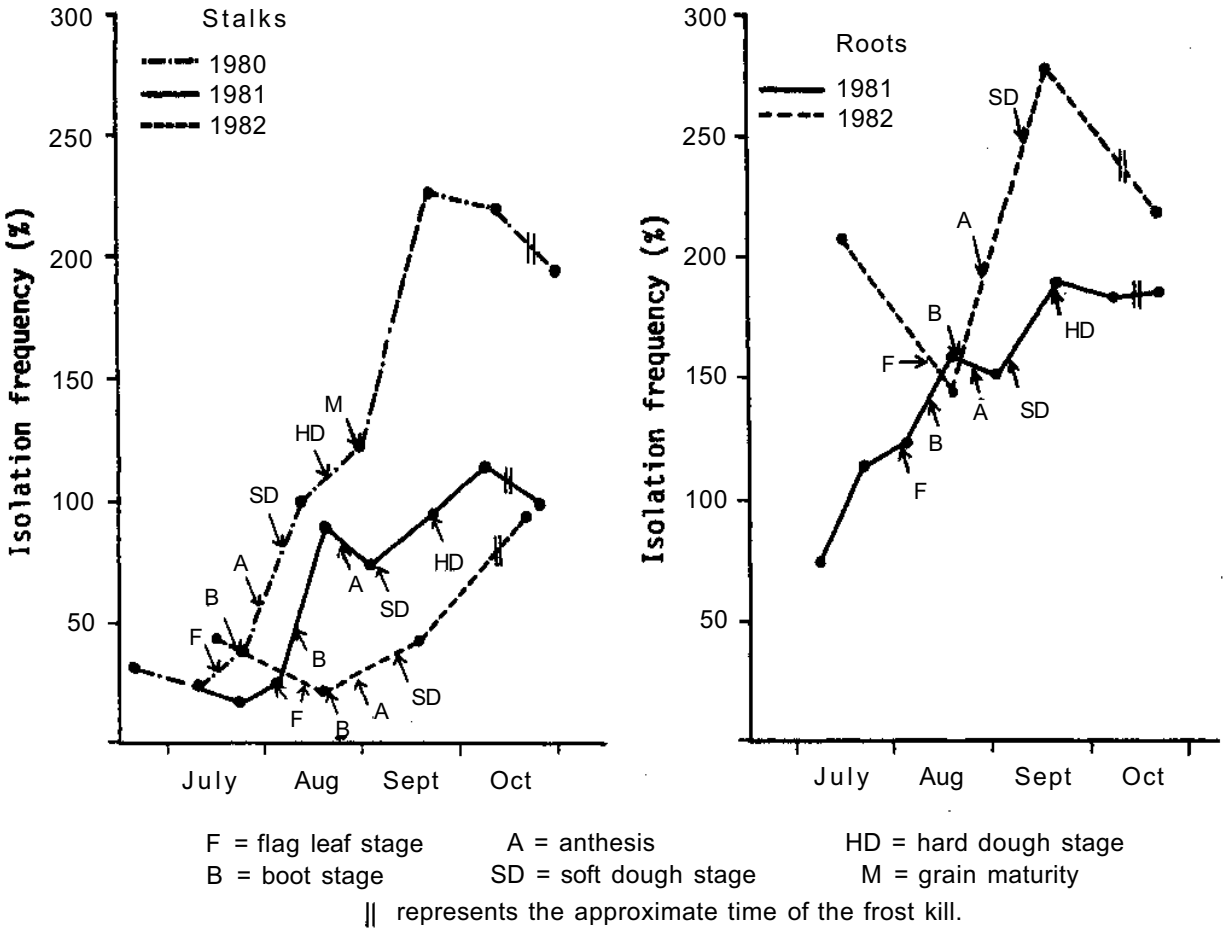

***Figure 2a and b. Isolation frequency of fungal species in stalks and roots of sorghum. Each point represents the number of species isolated expressed as a percentage of the total number of samples collected on that date. Approximate dates of occurrence of plant developmental stages are indicated.* (*Source: Reed 1982.*)**

forme is reduced. However, this same line of reasoning may also be offered to support the converse argument that *F. moniliforme* does not influence the populations of other *Fusarium* species, but instead is influenced by them. It may be argued that when environmental stresses, such as higher temperatures and lower moisture levels, are placed upon the plant, the other *Fusarium* species are more able competitors, and the population of *F. moniliforme* is reduced because of their increased activities. This question could not be resolved in an observationally based study such as Reed's because she noted only the presence or absence of each species. The use of an in vivo assay (Marshall and Partridge 1981, Partridge 1981, Partridge and Marshall 1981) that assesses the abundance and activity of individual species would be desirable to adequately address the significance of each species.

Sorghum roots were colonized by species of *Fusarium, Alternaria,* and *Epicoccum,* all of which are common root inhabitants. Of *the Fusarium* species, *F. equiseti, F. oxysporum,* and *F. solani* colonized roots earlier than did F. *moniliforme, F. graminearum,* and the "roseum" group. This may be indicative of higher initial populations of these species due to their ability to overwinter in soil as chlamydospores. The sequence of infection observed in roots in 1981 was closely paralleled in 1982, which may be due to the similarity of environmental conditions over the two seasons, or it may have occurred regardless of environmental condi-

tions. By observing root colonization over the course of several seasons, it should be possible to determine whether the sequence of infection occurs independently of weather conditions and is dependent on the stage of plant maturity or whether a significant interaction occurs between the two.

The early colonization of roots may indicate that root infection leads to stalk infection, an infection pathway that has often been suggested in the literature (Kommedahl et al. 1979, Young and Kucharek 1977). In our study, an increase in root population of some species appeared to precede an increase in stalk populations. However, in 1981 it was not observed whether roots and stalks of the same plant were colonized by the same species, and in 1982 too few species colonized stalks to afford a comparison. Therefore, it could not be determined if a correlation actually existed. Root colonization is likely to be one of several ways by which stalk colonization occurs.

The statistical analysis of stalk colonization by *F. moniliforme* and *F. roseum* presented by Reed (1982) clearly shows that the incidence of these fungi in stalks increased as the seasons progressed. However, the absence of statistically significant varietal differences in fungal colonization was open to interpretation as to whether the analysis accurately reflected the biological trends that may have been occurring. In Reed's study, the sample sizes were small; therefore intravarietal variation may have masked some intervarietal differences. The fact that some significant varietal differences were observed indicated that significant differences among varieties may occur in more instances than were detected in her study. These differences may become apparent if an experimental design is employed that increases sample size.

The association of stress conditions, senescence, and stalk rot of maize and sorghum is well documented (Edmunds 1964, Edmunds et al. 1964, Odvody and Dunkle 1975, Odvody and Dunkle 1979, Hsi 1961, Patil et al. 1979, Trimboli and Burgess 1983, Wadsworth and Sieglinger 1950), but the interrelationship between host and parasite(s) in the development of the disease is not understood. Research data (Reed 1982, Reed et al. 1983, Bain 1973) demonstrates that fungi are present in stalk and root tissue throughout most of the life of the plant. Therefore, if microbial interactions do occur within plant tissue, they may be occurring very early in the life of the plant. Similarly, interactions between the host and these microorganisms may begin early and continue throughout the host's growth and development. These data are not necessarily in conflict with those of Trimboli and Burgess (1983), even though one can reach the same or different conclusions from Trimboli's data. Trimboli's data were taken primarily from plants infected in the greenhouse by a single organism, while Reed's data are of field origin. The presence of fungi in stalks and roots of sorghum in the absence of any symptoms of stalk rot indicates that fungal colonization of plant tissue does not, in and of itself, lead to development of the disease. The nature of the interactions occurring within plant tissue cannot be determined from this study; however, one may speculate that a balance exists between fungal activity within plant tissue and the ability of the host to withstand such activity. This balance may be shifted by factors adversely affecting the host or by conditions that favor increased fungal activity. The microorganisms may become destructive to stalk tissue, leading to the development of stalk rot.

The Stress Hypothesis

In general, stalk rots are dependent upon stress phenomena for the onset of pathogenesis. Dodd's (1977; 1980a, b,c) hypothesis of photosynthetic stress may explain only a single type of stress placed upon a plant: stress related to reproduction. If applicable, it would seem that photosynthetic stress would probably play a larger role in senescing than in nonsenescing plants. Plants undergo a number of stresses: insect feeding (Frederiksen and Daniels 1970), reproduction (Edmunds and Voigt 1966), and environmental—e.g., heat and drought. These have been examined under controlled conditions in maize and/or sorghum. Since the photosynthetic stress, owing to the availability of solar radiation, is less variable than either temperature or moisture, this discussion cannot be complete without consideration of these two stresses on the basic biochemistry of the sorghum plant.

Plant cells respond to stress with an altered metabolism (Altschuler and Mascarenhas 1982, Bronson and Scheffer 1977, Flores and Galston 1982, Key et al. 1981, Schoeneweiss 1975). One of the responses to heat or drought stress is the synthesis of a new class of proteins (stress proteins). The observation of these changes is not new (Hsiao 1970); however, our understanding of their

role in the cell is (Baszczynski and Walden 1982). When stress occurs, gramineous cells respond by producing a new messenger RNA (Baszczynski et al. 1983). Once produced, this stress mRNA is the predominant messenger that can be translated by the ribosomes. At present it is unclear whether this is the only type of mRNA that can be translated during this period. Neither the mechanism by which the stress mRNA is able to control protein synthesis nor the function of the new proteins is completely understood. Suffice it for this discussion to say that the proteins that are synthesized are probably for the purpose of repairing any damage that may have occurred to the cell during the stress period. Unless the severity of the stress is sufficient to cause cell death, the length of the repair process is proportional to the amount of stress (Baszczynski et al. 1983).

With regard to stalk rot, since only repair proteins are being produced after an environmental stress, the plant is in a very vulnerable situation with respect to its own defense. For the sake of example, if a heat stress of 35°C is placed on a maize plant (or sorghum, for this example), it will begin to produce heat stress proteins within 15 min. It will continue to produce those proteins for as long as the stress is applied. Realistically, in the field, this might be for 6 h or longer. During this stress period the plant will be vulnerable to pathogenic activity. Once the temperature is reduced to 27°C (which in the field may take a number of hours) the heat stress proteins will continue to be synthesized for up to 8 additional hours (Baszczynski and Walden 1982). Therefore the plant is vulnerable to pathogens for a minimum of 14 consecutive hours.

If the environmental heat stress is compounded with a concomitant drought stress, then the effect upon the plant may be extremely severe. Additionally, if the environment is unfavorable during successive days, as is often the case, then in effect those organisms that have established themselves as parasites will have both the opportunity and the competitive advantage to begin pathogenesis. Unfortunately, the environmental conditions described and the resulting pathogenesis are common and annual occurrences in much of the maize- and sorghum-growing areas. Therefore the hypothesis is offered that stress-induced (primarily temperature and moisture) perturbations in the biochemical processes of the plant weaken or curtail its ability to inhibit pathogenesis by its internal parasites for sufficient time that the internal parasites gain the parasitic advantage and stalk rot begins.

Recommendations for Future Research

1. It is necessary to study organisms involved as internal parasites of sorghum during the entire life of the plant in expanded geographical areas. One of the difficulties of stalk rot research is the lack of data collected from single plots over a number of years and published in refereed journals. It is exceedingly difficult to attempt to construct a picture of the role of parasites in stalk rot from fragmentary data. At present, the best one can do is to attempt to draw conclusions based upon one's own data, with all of the accompanying geographical limitations. Obviously, in order to arrive at a universal conclusion about the role of the various parasites, we must have replicated data from a wide-ranging number of geographical sites. We propose that researchers:

   a. publish information based only upon replicated (not single year) data;

   b. identify the genotype of the sorghum plants under investigation;

   c. collect and publish data that pertain to the environmental conditions under which their crop is grown;

   d. conduct research using proper mycological tools; and

   e. encourage, primarily through colleagues or international students, the isolation and description of the internal parasite flora of sorghum plants in countries where sorghum is grown.

2. The tools of plant molecular biology are available and must be applied to the study of stalk rots. In order to understand and ultimately solve the stalk rot mystery, we must gain an understanding of the various interactions of host and parasite(s) as affected by the environment. Considerable data have accumulated to substantiate the fact that an interaction does occur. A challenge for the future is to define what the plant recognizes as stress and to understand both the mechanism for and the

magnitude of the plant's response to the various stresses presented to it. As an example, if heat stress is a factor, one should be able to determine;

a. the temperature and duration necessary to induce altered biochemical activity;

b. the nature of the biochemical changes that occur;

c. the variations that occur between hybrids;

d. the exacerbation of heat stress by drought stress, or vice versa; and

e. a means to apply this knowledge to the selection of hybrids.

3. Research activity on the basic biochemical processes of the sorghum stalk should be increased to provide a basis for understanding the parasitism and pathogenesis of the putative stalk-rotting organisms. Basic questions that should be addressed are:

a. why are stalks apparently relatively free of parasitism until postanthesis;

b. whether there are antifungal compounds normally produced by sorghum plants that inhibit parasites from becoming pathogens; and

c. whether the normal cyanogenic compounds have any role in the stalk rot syndrome.

4. Increased research emphasis should be placed on determining the long-range effects of conservation tillage or other cultural methods on the host-parasite interactions that lead to stalk rots.

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References

ALTSCHULER, M., and MASCARENHAS, J.P. 1982. Heat shock proteins and effects of heat shock in plants. Plant Molecular Biology 1:103-116.

BAIN, D.C. 1950. Fungi recovered from seed of *Sorghum vulgare.* Phytopathology 40:521 -522.

BAIN, D.C. 1973. Association of *Fusarium moniliforme* with infection of sorghum seedlings by *Sclerospora sorghi.* Phytopathology 63:197-198.

BASZCZYNSKI, C.L., and WALDEN, D.B. 1982. Regulation of gene expression in corn (*Zea mays* L) by heat shock. Canadian Journal of Biochemistry 60:569-579.

BASZCZYNSKI, C.L., WALDEN, D.B., and ATKINSON, B.G. 1983. Analysis of the in vitro translation products from RNAs of heat-shocked seedlings. Maize Genetics Cooperation Newsletter (ed. E.H. Coe, University of Missouri, Columbia, Missouri, USA) 57:161-163.

BEE-RODRIGUEZ, D., and AYALA, A. 1977. Interaction of *Pratylenchus zeae* with four soil fungi on sorghum. Journal of Agriculture of the University of Puerto Rico 61:501 - 506.

BERGQUIST, R.R. 1973. *Colletotrichum graminicola* on *Sorghum bicolor* in Hawaii. Plant Disease Reporter 57:272-275.

BHATTACHARYA, M., and SAMADDAR, K.R. 1976. Epidemiological studies on jute diseases. Survival of *Macrophomina phaseoli* (Maubl.) Ashby in soil. Plant Soil 44:27-36.

BIDARI, V.B., SATYANARAYAN H.V., HEGDE, R.K., and PONNAPPA, K.M. 1978. Effect of fungicides against the seed rot and seedling blight of hybrid sorghum CSH-5. Mysore Journal of Agricultural Sciences 12:587-593.

BRONSON, C.R., and SCHEFFER, R.P. 1977. Heat-induced and aging- induced tolerance of sorghum and oat tissues to host selective toxins. Phytopathology 67:1232-1238.

CASTOR, L.L., and FREDERIKSEN, R.A. 1980. *Fusarium* and *Curvularia* grain molds in Texas. Pages 93-102 *in* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored by Texas A&M University (USA) and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

CASTOR, L.L., and FREDERIKSEN, R.A. 1981. Histopathology of *Fusarium moniliforme* infection of sorghum kernels, Phytopathology 71:208.

CHIDAMBARAM, P., and MATHUR, S.B. 1975. Production of pycnidia by *Macrophomina phaseolina.* Transactions of the British Mycological Society 64:165-168.

CHOWDHURY, S.C. 1936. A disease of Zea *mays* caused by *Colletotrichum graminicolum* (Ces.) Wils. Indian Journal of Agricultural Science 6:833-843.

CHRISTENSEN, J.J., and WILCOXSON, R.D. 1966. Stalk rot of corn. Monograph No. 3. St. Paul, Minnesota, USA: American Phytopathological Society, 59 pp.

CLAFLIN, L.E. 1981. Control of seed decay, seedling blights, and covered kernel smut of sorghum with seed treatments. Fungicide and Nematicide Tests (American Phytopathological Society, St. Paul, Minnesota, USA) 36: 161-162.

COOK, G.E., BOOSALIS, M.Q., DUNKLE, L.D., and ODVODY, G.N. 1973. Survival of *Macrophomina phaseoli* in corn and sorghum stalk residue. Plant Disease Reporter 57:873-875.

DALE, J.L. 1963. Com anthracnose. Plant Disease Reporter 47:245-249.

DALMACIO, S.C. 1980. Sorghum diseases in the Philippines. Pages 70-71 *in* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

DENIS, J.C., and GIRARD, J.C. 1980. Factors affecting the development of sorghum grain molds in Senegal. Pages 144-153 *in* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

D'ERCOLE, N., and NIPOTI, P. 1979. Microbiological analysis of sorghum seeds. Informatore Fitopatologico 29:7-12.

DODD, J.L. 1977. A photosynthetic stress-translocation balance concept of com stalk rot. Pages 122-130 *in* Proceedings of the 32nd Annual Corn and Sorghum Research Conference (eds. H.D. Loden and D. Wilkinson). Washington, D.C., USA: American Seed Trade Association.

DODD, J.L. 1980a. The role of plant stresses in development of corn stalk rots. Plant Disease 64:533-537.

DODD, J.L. 1980b. Grain sink size and predisposition of *Zea mays* to stalk rot. Phytopathology 70:534-535.

DODD, J.L. 1980c. The photosynthetic stress-translocation balance concept of sorghum stalk rots. Pages 300-305 *in* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

DOGGETT, H. 1980. Sorghum diseases in East Africa. Pages 33-35 *in* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, AP. 502 324, India: ICRISAT.

DOUPNIK, B., and BOOSALIS, M.G. 1980. Ecofallow—a reduced tillage system—and plant diseases. Plant Disease 64:31-35.

DOUPNIK, B., BOOSALIS, M.G., and WICKS, G.A. 1975a. Wheat-sorghum-ecofallow rotation reduces stalk rot. Proceedings (1974) of the American Phytopathological Society 1:125.

DOUPNIK, B., BOOSALIS, M.G., WICKS, G.A., and SMIKA, D. 1975b. Ecofallow reduces stalk rot in grain sorghum. Phytopathology 65:1021-1022.

DUNCAN, R.R., BOCKHOLT, A.J., and MILLER, F.R. 1981. Descriptive comparison of senescent and nonsenescent sorghum genotypes. Agronomy Journal 73:849-853.

EDMUNDS, L.K. 1964. Combined relation of plant maturity, temperature, and soil moisture to charcoal stalk rot development in grain sorghum. Phytopathology 54:514-517.

EDMUNDS, L.K., BURCHETT, L.A., NORWOOD, C.A., and WITT, M.D. 1975. Effect of minimum tillage on Fusarium populations in soils of dryland sorghum in Kansas. Proceedings (1974) of the American Phytopathological Society 1:102.

EDMUNDS, L.K., and VOIGT, R.L. 1966. Role of seed production in predisposition of sorghum to charcoal rot. Phytopathology 56:876 (abstract).

EDMUNDS, L.K., VOIGT, R.L, and CARASSO, F.M. 1964. Use of Arizona climate to induce charcoal rot in grain sorghum. Plant Disease Reporter 48:300-302.

FELICIANO, C., HEPPERLY, P., and SOTOMAYOR-RIOS, A. 1982. Characterization for sorghum seedborne mycoflora and its effect on 30 sorghum lines under humid tropical conditions in Puerto Rico. Phytopathology 72:169 (abstract),

FLORES, H.E., and GALSTON, A.W. 1982. Polyamines and plant stress: Activation of putrescine biosynthesis by osmotic shock. Science 217:1259-1261.

FREDERIKSEN, R.A., and DANIELS, N.E. 1970. The influence of greenbugs on stalk rots of sorghum. Texas Agricultural Experiment Station Progress Report 2772:1-7, Texas A&M University, College Station, TX 77843, USA.

GOURLEY, L.M., ANDREWS, C.H., SINGLETON, L.L., and ARAUJO, L. 1977. Effects of *Fusarium moniliforme* on seedling development of sorghum cultivars. Plant Disease Reporter 61:616-618.

HAMID, S.J. 1980. Sorghum Diseases in Pakistan. Pages 67-69 *in* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

HSI, D.C.H. 1961. An effective technique for screening sorghum for resistance to charcoal rot. Phytopathology 51:340-341

HSIAO, T.C. 1970. Rapid changes in levels of polyribosomes in *Zea mays* in response to water stress. Plant Physiology 46:281-285.

HULLUKA, M., and GEBREKIDAN, B. 1980. Diseases of sorghum in Ethiopia. Pages 36-39 *in* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored by Texas A&M University (USA) and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

JADHAV, V.T. 1978. Hollow stem blight of sorghum. Food Farming and Agriculture 10:169.

KATSANOS, R.A., and PAPPELIS, A.J. 1965. Seasonal trends in density and cell death in sorghum stalk tissue. Phytopathology 55:97-99.

KATSANOS, R.A., and PAPPELIS, A.J. 1966. Relationship of cell death patterns and spread of *Colletotrichumgraminicota* in sorghum stalk tissue. Phytopathology 56:468-469.

KEY, J.L, LIN, C.Y., and CHEN, Y.M. 1981. Heat shock proteins of higher plants. Proceedings of the National Academy of Science (USA) 78:3526-3530.

KHAN, A.L., FAKIR, G.A., and THIRUMALACHAR, M.J. 1976. Comparative pathogenicity of 2 strains of *Macrophomina phaseolina* from sesame. Bangladesh Journal of Botany 5:77-81.

KOMMEDAHL, T., WINDELS, C.E., and STUCKER, R.E. 1979. Occurrence *of Fusarium* species in roots and stalks of symptomless corn plants during the growing season. Phytopathology 69:961 -966.

LIVINGSTON, J.E. 1945a. Charcoal rot of corn and sorghum. Nebraska Agricultural Experiment Station Bulletin No. 136. Lincoln, NE 68503, USA: University of Nebraska. 32 pp.

LIVINGSTON, J.E. 1945b. Charcoal rot of corn and sorghum in Nebraska. Plant Disease Reporter 34:45-52.

MARSHALL, M.R., and PARTRIDGE, J.E. 1981. Immunochemical identification of *Fusarium* moniliforme ribosomes from diseased corn stalk tissue. Physiological Plant Pathology 19:277-288.

MIAN, M.I.H., and AHMED, A. 1980. Sorghum diseases in Bangladesh. Pages 54-56 *in* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

MINUSSI, E., and KIMATI, H. 1978. Some fungi on sorghum seeds. Reviste do Centro de Ciencias Rurias UFSM 8:307-311.

ODVODY, G.N., and DUNKLE, L.D. 1975. *Helminthosporium sorghicola* on *Sorghum* spp in Nebraska. Proceedings (1974) of the American Phytopathological Society 1:128.

ODVODY, G.N., and DUNKLE, L.D. 1979. Charcoal stalk rot of sorghum: effect of environment on host-parasite relations. Phytopathology 69:250-254.

PADY, S.M. 1943. Diseases of com and sorghum in the Kansas-Nebraska area. Plant Disease Reporter 27:563-564.

PARTRIDGE, J.E. 1981. Electrophoretic comparison of ribosomal proteins of fungal pathogens of corn and sorghum. Phytopathology 71:248 (abstract).

PARTRIDGE, J.E., and MARSHALL, M.R. 1981. Nonhomology of antibodies produced against ribosomal proteins of *Fusarium moniliforme* and *Fusarium roseum* 'graminearum.' Phytopathology 71:24 (abstract).

PATIL, R.C., DESHAMANE, N.B., and PARATBADI, G.S. 1979. Screening of sorghum varieties for reaction to charcoal rot under irrigation. Sorghum Newsletter 22:118-119.

PUPIPAT, U. 1980. Sorghum diseases in Thailand. Pages 72-73 *in* Sorghum Diseases, a World Review: Proceedings of the international Workshop on Sorghum Diseases, sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, A.P. 502324, India: ICRISAT.

RAVINDRANATH, V. 1980. Sorghum diseases in India. Pages 57-66 *in* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

REED, J.E. 1982. Occurrence of fungal species in stalks and roots of sorghum during the growing season. M.Sc. thesis, University of Nebraska, Lincoln, NE 68503, USA.

REED, J.E., PARTRIDGE, J.E., and NORDQUIST, P.T. 1982. Fungal species isolated from roots and stalks of symptomless sorghum plants during the growing season. Phytopathology 71:974 (abstract).

REED, J.E., PARTRIDGE, J.E., and NORDQUIST, P.T. 1983. Fungal colonization of stalks and roots of grain sorghum during the growing season. Plant Disease 67:417-420.

RICCELLI, M. 1980. Current strategies and progress in breeding disease-resistant sorghums in Venezuela. Pages 434-453 *in* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

SCHOENEWEISS, D.F. 1975. Predisposition, stress, and plant disease. Annual Review of Phytopathology 13:193-211.

SHOKES, F.M., LYDA, S.D., and JORDAN, W.R. 1977. Effect of water potential on the growth and survival of *Macrophomina phaseolina,* Phytopathology 67:239-241.

SIDDIQUI, M.R., and KHAN, I.D. 1973. Fungi and factors associated with the development of sorghum ear-molds. Transactions of the Mycological Society (Japan) 14:289-293.

SMITH, W.H. 1969a. Comparison of mycelial and sclerotial inoculum of *Macrophomina phaseoli* in the mortality of pine seedlings under varying soil conditions. Phytopathology 59:379-382.

SMITH, W.H. 1969b. Germination of *Macrophomina phaseoli* sclerotia as affected by *Pinus lambertiana* root exudate. Canadian Journal of Microbiology 15:1387-1391.

TRIMBOLI, D.S., and BURGESS, L.W. 1983. Reproduction of *Fusarium moniliforme* basal stalk rot and root rot of grain sorghum in the greenhouse. Plant Disease 67:891 - 894.

TRIPATHI, R.K. 1975. Head fungi of sorghum phytotoxins and their effects on seed germination. Indian Phytopathology 24:499-501.

TULLIS, E.C. 1951. *Fusarium moniliforme,* the cause of a stalk rot of sorghum in Texas. Phytopathology 41:529-535.

TYAGI, P.D. 1980. Sorghum diseases in Nigeria. Pages 45-52 *in* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

UPPAL, B.N., KOLHATKAR, K.G., and PATEL, M.K. 1936. Blight and hollow stem of sorghum. Indian Journal of Agricultural Science 6:1323-1334.

WADSWORTH, D.F., and SIEGLINGER, J.B. 1950. Charcoal rot of sorghum. Oklahoma Agricultural Experiment Station Bulletin No. B-355. Stillwater, Oklahoma, USA: Oklahoma A&M College and U.S. Department of Agriculture. 7 pp.

WARREN, H.L, and KOMMEDAHL, T. 1973. Prevalence and pathogenicity to corn of *Fusarium* species from crown roots, rhizosphere, residues, and soil. Phytopathology 63:1288-1290.

WATANABE, T., SMITH, R.S., Jr., and SNYDER, W.C. 1970. Populations of *Macrophomina phaseoli* in soil as affected by fumigation and cropping. Phytopathology 60:1717-1719.

WHEELER, H., POLITIS, D.J., and PONELEIT, C.G, 1974. Pathogenicity, host range, and distribution of *Colletotrichum gramlnicola* on corn. Phytopathology 64:293-296.

WHEELER, H., POLITIS, D.J., and WILLIAMS, A.S. 1972. Pathogenicity, host range, and distribution of *Colletotrichum graminicola* on maize. Phytopathology 62:808 (abstract).

WILLIAMS, R.J., and RAO, K.N. 1981. A review of sorghum grain molds. Tropical Pest Management 27:200-211.

YOUNG, T.R., and KUCHAREK, T.A. 1977. Succession of fungal communities in roots and stalk of hybrid field corn grown in Florida. Plant Disease Reporter 61:76-80.

Questions

Williams:
You indicated a belief that *Fusarium graminearum* is more important in stalk rot than F. *moniliforme.* Is this just for your location or do you think it is generally applicable?

Partridge:
The data presented speak specifically to our plots in Western Nebraska; however, Kommedahl and Windels [1979] have made the same conclusion for maize in Minnesota. Nonetheless we would be cautious toward making a statement "in general," though it may be true.

Claflin:
Was each determination a result of hyphal-tip cultures? Who identified the cultures?

Partridge:
Yes, we used hyphal tip cultures. Janet Reed (master's degree candidate) and I identified them, and the identifications were confirmed by Dr. Paul Nelson, Fusarium Research Center, Pennsylvania State University.

Doupnik:
Does it matter which species of *Fusarium* is the actual causal agent of fusarium stalk rot in terms of mechanism of pathogenicity and control measures or developmental genetic resistance?

Partridge:
Yes, our data strongly indicate that one can select for separate resistance to each species, whatever the mechanisms may be.

Sorghum Root and Stalk Rots: Basic Disease Problems

Summary and Synthesis

L.K. Mughogho*

The eight background papers on basic disease problems provide an overall view of the biology of the causal agents and the epidemiology of root and stalk rot diseases. Since the effects of plant physiological and environmental factors and disease control are presented in later sessions, my comments will be confined to crop loss and some aspects of the biology of the causal organisms.

Crop Loss Caused by Root and Stalk Rots

All the authors of the background papers on specific diseases have reported that root and stalk rots cause crop losses. However, the few data available are from experiments conducted at research stations or in glasshouses. There is a dearth of quantitative crop loss data from farmers' fields. Unless sorghum scientists are able to show that root and stalk rots cause unacceptable crop losses in farmers' fields, there is little justification for funds to be spent on research for their control. I would recommend that systematic crop loss surveys in farmers' fields be condugted in areas where root and stalk rots are thought to be economically important.

Causal Organisms and their Distribution

Of a number of organisms often isolated from diseased roots and stalks, the well-known causal agents are the fungi *Macrophomina phaseolina, Fusarium moniliforme, Periconia circinata, Pythium* spp, and *Colletotrichum graminicola.*

M.phaseolina and *F. moniliforme* appear to be widely distributed in sorghum-growing areas. *P. circinata,* formerly thought to be restricted to the USA, has recently been reported from Australia, where it appears to cause little damage to sorghum (Mayers 1976). Perhaps P. *circinata* is more widespread in sorghum-growing areas than has hitherto been reported, and surveys like those conducted by Mayers in Australia would help to determine its distribution.

The identity of the *Pythium* spp, particularly those implicated in root rots, is still incomplete, as is their distribution and importance on a regional or global basis.

Recently, *Acremonium strictum,* a vascular pathogen that causes leaf and stalk death, has been recognized as an important disease in the Americas. The occurrence of this pathogen in other parts of the world needs to be watched since the disease can be very destructive.

Other fungus-incited diseases not included in the presentations but reported by Tarr (1962) include pink root rot (*Pyrenochaeta terrestris*), southern sclerotial rot (*Corticium rolfsii*), and rhizoctonia stalk rot (C. *solani*). Bacteria, particularly *Erwinia* spp, have also been implicated as causal agents of stalk rots in the Philippines (Karganilla and Exconde 1972), in India (Anahosur 1979), Nigeria (King 1973), and the USA (Zummo 1969). Very little is known about the etiology of these diseases. This is an obvious area for future research.

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International Crops Research Institute for the Semi-Arid Tropics. 1984. Sorghum Root and Stalk Rots, a Critical Review: Proceedings of the Consultative Group Discussion on Research Needs and Strategies for Control of Sorghum Root and Stalk Rot Diseases, 27 Nov - 2 Dec 1983, Bellagio, Italy. Patancheru, A.P. 502 324, India; ICRISAT.

Diseases of Complex Etiology

With the exception of anthracnose, acremonium wilt, and pokka boeng where initial infection is through the stem or panicle, all the other diseases considered in the background papers appear to be of complex etiology involving more than one pathogen or pathogen species. This has been clearly brought out in the papers by Zummo, Mughogho and Pande, Partridge et al., and Claflin. The frequent association of the causal agents in isolations from diseased roots and stalks needs investigation to determine the nature of association, i.e., whether the organisms can cause disease singly, in succession, or together, and if synergism occurs. The work of Partridge and coworkers at the University of Nebraska on the temporal and spatial succession of fungi on roots and stalks should be conducted at other sorghum-growing locations worldwide to determine the nature of the association, and also the pathogen species involved at different locations.

The role of nematodes in the root and stalk disease complex has hardly been researched, and the areas of future research suggested by Claflin need attention.

Other Aspects of the Biology of Causal Organisms

Very little is known about pathogen dissemination, survival, and source and form of initial inoculum for most of the root and stalk rot diseases. The possible existence of physiological races, as has been shown for anthracnose, also needs elucidation. This is important in utilization of resistance in disease control.

References

ANAHOSUR, K.H. 1979. Bacterial stalk rot of sorghum in regional research station, Dharwad. Sorghum Newsletter 22:121.

KARGANILLA, A.D., and EXCONDE, O.R. 1972. Bacterial stalk rot of corn and sorghum. Philippine Phytopathologist 8:4 (abstract).

KING, S.B. 1973. Plant pathology annual report (sorghum): Major Cereals in Africa Project. Samaru, Nigeria: Institute of Agricultural Research.

MAYERS, P.E. 1976. The first recording of milo disease and *Periconia circinata* on sorghums in Australia. Australian Plant Pathology Society Newsletter 5:59-60.

TARR, S.A.J. 1962. Diseases of sorghum, sudan grass and broom corn. Kew, Surrey, U.K.; Commonwealth Mycological Institute. 380 pp.

ZUMMO, N. 1969. Bacterial soft rot, a new disease of sweet sorghum. Phytopathology 59:119 (abstract).

Discussion

Etiology/Succession

Doupnik:
Dr. Zummo, is root rot a prerequisite to stalk rot?

Zummo:
When fusarium stalk rot is found, root damage is found with it

Doupnik:
If fusarium stalk rot comes first, are the roots predisposed to infection?

Zummo:
You can have fusarium root rot without stalk rot But you don't see stalk rot without root rot.

Frederiksen:
Fusarium stalk rot can come from rachis and peduncle infection and move down the stalk under wet weather conditions.

Mughogho:
Macrophomina stalk rot is preceded by root rot.

Partridge:
We need to distinguish between root rot and root parasitism.

Pappelis:
Our attention has centered on one pathogen. We should broaden the discussion to include the root rot and stalk rot complex. It is a multiple entry system involving many pathogens. Some may be primary and some may be secondary invaders.

Sinclair:
We have found that *Colletotrichum* and *Macrophomina* cause symptomless, latent infection in soybean. In such plants we can induce symptoms and signs of the pathogen in the laboratory using desiccant herbicides. I suggest that sorghum pathologists should look at multiloci infections that may take place early in the growing season. The expression of symptoms may not occur until stimulated by some factor.

Pappelis:
Another thing to consider is systemic infection by various pathogens.

Sinclair:
We need to distinguish between true systemic infection in the vascular system and multiloci infection.

Schneider:
In the case of *Fusarium moniliforme* in maize and *F. oxysporum* in celery, there are multiple sites of entry in the root system, but only a small proportion of these localized infections then become systemic.

Williams:
What is the role of seedborne inoculum?

Partridge:
In my experience *Fusarium moniliforme* and *F. graminearum* move with the seed; as much as 20% or more of the seed will be infected.

Claflin:
I have observed that tunneling by the Southwestern or European corn [maize] borer results in infection within the damaged internode, i.e., it does not spread beyond the upper or lower node.

Pappelis:
In maize the pith parenchyma is dead, and the living cells of the nodes form a barrier to the spread of the fungus. In the case of sorghum, often this barrier does not exist.

Zummo:
In the case of sugarcane and *Colletotrichum falcatum* there are varieties that allow free flow of conidia and mycelium through the node, and these are considered to be susceptible/Those varieties that prevent the fungus from moving through the node are considered to be resistant.

Pappelis:
If cell death occurs, the fungus will spread following the pattern of cell death.

Clark:
The infection occurs under certain conditions. We need to know more about the conditions and properties of the cells when infection occurs.

Odvody:
In our studies we have never seen the macrophomina stalk rot phase without the root rot phase.

Sinclair:
As far as I know, sorghum pathologists have not presented evidence of the spread of infection from the roots through the crown into the stalk.

Partridge:
I have a student (Janet Reed) whose master's degree thesis contains data on this subject of sequential isolations.

Frederiksen:
Edmunds in 1964 and Odvody in about 1978 have clearly shown that root infection precedes stalk infection.

Partridge:
In our studies using multiple pathogens on maize following toothpick inoculation, we always found sequential movement of the pathogens up and sometimes down, but never skipping nodes,

McBee:
Has anyone related successions to growth stage in sorghum?

Pappelis:
Cell death in the cortical region of the roots can occur within a few days after the seeds are planted or will not occur at all until the plant is stressed. The succession of fungi isolated from roots may depend upon the physiological status of the roots, and this is not measured by isolation techniques.

McBee:
In sorghum there's a drastic change in carbohydrate metabolism and photosynthate partitioning as heading occurs and a deterioration of the root caused by development of the kernel sink. What effect does this have on timing of root infection?

Pappelis:
Translocation patterns have not been presented in any of the papers on sorghum. Hearing no response, gentlemen, should we go back to the subject of succession?

Partridge:
As a general rule, root and stalk pathogens are restricted to the root and crown area on maize until anthesis, after which there is ramification. I feel that sorghum faces the same general pattern, but the data are not as conclusive.

Eastin:
There is a heavy flow of metabolites to the roots prior to anthesis and up to 7 to 10 days thereafter. At the soft-dough stage there is no such flow to the roots in some types of sorghum.

Pappelis:
After anthesis, cells in the stalk die, sucrose increases, and reducing sugars decrease. The other problem with anthesis is that there is no linkage between cell death in the roots and stalk. There is a barrier that prevents the spread of root pathogens into the stalk. After anthesis this barrier no longer exists; i.e., the cells in the root-stalk junction die.

Williams:
Is it possible that drought stress results from impaired water uptake by infected roots?

Rosenow:
If you injure the plant with either a sterile or a *Fusarium* infested toothpick, stalk rot will develop at that site. If the pathogen was there before that time, as indicated by the work of Partridge, why did it not cause stalk rot?

Partridge:
If *Fusarium equiseti* and *F. graminearum* are the primary pathogens of sorghum roots in the field, then there is a decreased spread of *F. moniliforme.* Thus stalk rot caused by *F. moniliforme* may be decreased by the presence of other species of *Fusarium.*

Inoculation/Screening for Resistance

Pappelis:
Fusarium moniliforme can be isolated from stalks, but it appears to be localized in vascular tissues and remains there as though its growth is inhibited. When we use inoculation methods, we place the pathogen in the pith parenchyma, and if the cells are dead, it spreads throughout the tissue. There must be a physiological explanation for this inhibition.

Mughogho:
If what you are saying is correct, screening for resistance to charcoal rot using the toothpick method is not the appropriate method. This supports the point of view we presented in our paper.

Pappelis:
In the case of *Macrophomina,* the fungus will spread through areas of parenchyma cells, and in this way it is not like *Fusarium.*

Sinclair:
In our studies on soybean we have found that several fungi can colonize the seed or the plant at the same time. These fungi have different "ecological niches" and can develop independently of one another, but when we inoculate we upset this relationship.

Pappelis:
The concept is either the fungus grows in dead cells until it comes in contact with living cells, where it stops for physiological reasons; or the pathogen induces senescence and death of the living cell. An example of the first condition is the behavior of *Diplodia maydis,* and the second is that *of Colletotrichum graminicola.*

Frederiksen:
It has been suggested that the toothpick method isn't appropriate for screening sorghum lines for resistance to charcoal rot. However, I haven't found a method superior to it. Perhaps we should discuss other methods, such as Rosenow's method.

Mughogho:
We find it difficult touse the toothpick technique. Our results vary from season to season, and the development of disease depends on the predisposition of the plant to infection. In order to produce consistent results, the plants should be predisposed to infection.

Pappelis:
It's important when using the toothpick method to place the inoculum in the internode rather than the node.

Frederiksen:
The results using the toothpick method for screening for resistance to *Fusarium* are highly correlated with fungal infections in the field. This inoculation procedure provides a uniform method for testing genotypes.

Pappelis:
In maize the time of inoculation was found to be important to obtain the best results in screening for disease resistance. The same studies need to be done in sorghum. The purpose of inoculating is to be sure that there is no disease escape.

Partridge:
We find that we can't obtain typical fusarium stalk rot symptoms in the greenhouse by using one species alone; we must use at least two and sometimes three species of *Fusarium.*

Claflin:
How do you test for stalk rot decay following toothpick inoculation? Do you squeeze the stalk?

Pappelis:
There is a difference between stalk rot and lodging resistance. If you study stalk rot, you must cut the stalk in order to make stalk rot evaluations.

Williams:
One point in favor of the toothpick method as described by Frederiksen is that it eliminates variable inoculum load. Dr. Mughogho, since you didn't use the toothpick method in the field screening experiments described in your paper, what was your field block design?

Mughogho:
Our plot size was 18 meters square in a checkerboard pattern.

Williams:
Since your lines don't flower at the same time, how do you impose the water stress at the same physiological stage?

Mughogho:
We have classified our lines according to flowering stage and grow them as separate plots.

Frederiksen:
Another way to reduce the variability in inoculation studies conducted in the field is to use a highly virulent isolate.

Rosenow:
Dr. Pappelis, have you done pith condition ratings on juicy and pithy sorghum lines and related that to stalk rot?

Pappelis:
Yes, using plasmolysis and deplasmolysis you can identify living and dead cells in these lines. I'm speaking about anthracnose evaluation with pathogens spreading through dead cells. Succulent lines are composed of living cells and pithy lines consist of dead cells.

Mughogho:
We should ask what is the best screening technique for each one of these pathogens, and we also need to know more about the interactions between these pathogens. We would like some suggestions and advice from this meeting about how to handle these problems.

Schneider:
Do you standardize the methods for quantifying the water stress factor in evaluating the breeding lines?

Mughogho:
We have used two methods: (1) the line source irrigation technique, and (2) withdrawal of irrigation in dry areas at the appropriate time.

Schneider:
It seems to me that measurements of plant water status rather than soil water status should be made.

Seetharama:
We do measure leaf temperature and this corresponds well as an indicator of stress levels. However, a single measurement of plant water status does not give a good indication of stalk rot susceptibility.

Frederiksen:
We should look at fungus variability as well as inoculation technique. Clearly these fungi consist of many physiological races. We find variability in our anthracnose isolates. Dr. Partridge, I would like to know more about your *Fusarium* isolation.

Partridge:
We must identify all of the organisms in the stalk and determine which are the pathogens. Our findings suggest that *Fusarium moniliforme* does not cause stalk rot alone and may not be the primary pathogen involved.

Pappelis:
We should bring up other topics. I want to refer to J. Kuc's work [Pages 157-178 *in* Active defense mechanisms in plants (ed. R.K.S. Wood), 1982. Plenum Press, New York and London] where he challenged plants with incompatible pathogens and induced resistance to compatible ones. Does anyone have further information on this subject as it relates to root diseases of sorghum or other crops?

Schneider:
I have had some experience in this area. In work with *Fusarium oxysporum* on celery and *F. moniliforme* on maize, we find that localized root infection by incompatible pathogens protects the root from infection by compatible pathogens and that there is a finite number of infection sites per unit root length.

Partridge:
Is that protection of sites or inhibition of one organism by another?

Schneider:
Apparently it's competition by elimination of substrates.

Partridge:
As the root grows, is the new tissue protected by the previous infections?

Schneider:
No, the new root tip is susceptible and must be continually protected.

Pappelis:
Pathologists seem to ignore the obvious when working on stalk rots. There are no shortcuts. You must inoculate and evaluate disease response to avoid disease escape until the cause and effect "story" is so well understood that a predictive statement can be formulated to apply to all conditions at all locations: the universal, the hypothesis, the law. To that end, I believe we need to establish the following:

1. a disease nursery with the best inoculation methods and rating systems to evaluate varieties and hybrids;
2. drought stress plots in which the best inoculation methods and rating systems are used to evaluate stress effects on varietal and hybrid responses to the pathogen;
3. fumigated control plots and reinfested test plots where we can introduce soil inoculum singly or in mixtures.
4. nematode soil "bins" where fumigated soils can be infested with nematodes and fungal pathogens to test their interactions on root rot production.
5. the adoption of pith condition rating systems for stalks and roots and use of these in the above.

Stalk rot and standability must be studied individually and together in the above conditions. The procedures involve much labor. Nevertheless, it is essential. We must use the best methods we have and improve them until we develop the universal statements relevant to resistance and susceptibility to each of the major pathogens we are now discussing.

Physiological and Environmental Factors in Root and Stalk Rot Diseases

The Role of Edaphic Factors in Disease Development

W.R. Jordan, R.B. Clark, and N. Seetharama*

Summary

This paper presents a brief overview of the roles of abiotic stresses in the modification of processes contributing to the growth and grain yield of sorghum in both the absence and presence of biotic (disease) stresses. Water, temperature, and nutrient stresses promote yield losses through their effects on interception of solar radiation and production of photosynthate. Formation and maintenance of active green leaf area is essential for continued production of photosynthate to maintain carbon and energy flow to both developing grain and plant tissues. Abiotic stresses predispose host tissues to pathogen invasion and promote proliferation and spread of disease in plant tissues, but the mechanism(s) are unknown. The association of charcoal rot with stress during grain filling lends support to the view that carbohydrate mobilization from stalk and root tissues may be intimately associated with host resistance. Further research is needed to define the nature of changes induced by stress that predispose host roots to infection. Since infection and proliferation of the pathogen in host tissues seem to be controlled independently, the changes allowing spread should be studied further. Finally, interactions of abiotic stresses should be studied in a manner that will allow formulation of host-pathogen models necessary to explore possible common bases for disease development and resistance.

Edaphic factors such as water, temperature, and nutrition are universally recognized as important in the development and spread of disease in crop plants. Just as atmospheric turbulence, humidity, and other general features of the aerial climate are important to the epidemiology of disease caused by airborne pathogens, issues such as soil water content and potential, soil temperature, and mineral ion availability are central to our understanding of diseases caused by soilborne pathogens.

Relatively few research reports deal specifically with effects of the environment on root and stalk diseases. In fact, the ICRISAT program appears to be the only major research effort currently dealing with these problems in sorghum. It is our intention not only to review known environmental effects on the development and severity of root and stalk rots in sorghum, but also to provide insights into the effects of specific edaphic factors on the host and pathogen, and their interaction. Because sorghum is a major crop of the semi-arid zones, especially in developing countries, we will concentrate on problems associated with deficiencies in supplies of soil water and mineral nutrients.

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International Crops Research Institute for the Semi-Arid Tropics. 1984. Sorghum Root and Stalk Rots, a Critical Review: Proceedings of the Consultative Group Discussion on Research Needs and Strategies for Control of Sorghum Root and Stalk Rot Diseases, 27 Nov - 2 Dec 1983, Bellagio, Italy. Patancheru, AP. 502 324, India: ICRISAT.

Water in the Soil-Plant-Atmosphere Continuum

Water exists as a continuum from the soil, through the plant, and into the atmosphere. The phase change from liquid water to water vapor within the leaf does not alter this fact. Water moves in such a continuum from regions of high free energy (high water potential) to regions of low free energy (low water potential). Thus, water transpired by a crop flows from moist soil with a relatively high water potential through the plants and into the atmosphere along a water-potential gradient. Typically, soil water potentials (ψ_s) will range between -0.01 and -15 bars, while leaf water potentials (Ψ_L) of mesophytic plants will range between -1 and -30 bars, and atmospheric water potentials will range between -100 and -1000 bars. Just as typically, ψ_s may vary from -100 bars or less at the soil surface to -0.01 bars deeper in the soil profile, while the atmospheric water potential may decrease by 1000 bars or more between dawn and midday. At any instant, ψ_L represents an integration of atmospheric demand and the capacity of the soil to supply water as modulated by the plants' ability to regulate water loss. In this section, we examine soil and plant characteristics that determine rates of water flow through the continuum.

Water Flow in Soils

Over long periods of time, root systems grow to the extremes permitted by physical and chemical constraints, allowing the crop access to water stored deep in the soil profile. However, over short time intervals, it is Water movement through soil rather than root growth that allows uptake of sufficient quantities of water to prevent harmful desiccation. Water flow from the bulk soil to the rhizosphere occurs in response to ψ_s gradients arising from water uptake by roots.

The hydraulic conductivity (H) of a soil is a measure of its capacity to transmit water. A very strong function of ψ_s, H may change from 10 cm day^{-1} for a wet soil to as little as 10^{-8} cm day^{-1} at the lower limit of water availability. Reicosky and Ritchie (1976) found that the rate of water flow through soil did not limit water availability to growing crops of maize and sorghum until water extraction caused H to fall below about 10^{-6} to 10^{-7} cm day^{-1}. When H fell below this value, the roots were no longer able to absorb water at rates sufficient to satisfy the evaporative demand and water stress (low Ψ_L) resulted. Since the experiments were conducted at different locations and on different soils, their estimate for H may be a general result for well-rooted crops. Unfortunately, the relationship between H and soil water content (θv) is unique for each soil (and region of the soil profile); therefore the lower limit of H cannot be translated simply into θv, a quantity more frequently available. For sandy and clay soils, H reached 10^{-6} to 10^{-7} cm day^{-1} at ψ_s of -1 and -8 bars, respectively.

Water Flow in Plants

As in the case of soil, water flow in plants occurs in response to a water potential gradient between sites of water absorption in roots and sites of evaporation in leaves. Flow through plant tissues is most conveniently discussed in terms of the familiar electrical analog: water flow is analogous to the flow of electricity as described by Ohm's Law. Thus, the flow (flux) is directly related to the driving force (ψ gradient in plants) and inversely related to the resistance to flow. Major resistances appear to reside in the radial path of flow between the root surface and the root xylem, and at the stomates. For sorghum, the axial transport of water through healthy roots encounters little resistance (Meyer and Ritchie 1980). Therefore, when soil water is freely available to the crop, high rates of transpiration are maintained with a minimal depression of ψ_L. Values of ψ_L for healthy, well-watered sorghum usually range between -8 and -14 bars during peak transpiration periods, increasing to near zero bars before dawn. In some situations, the predawn value of ψ_L or the ψ_L of leaves covered to prevent water loss may be taken as an estimate of the ψs surrounding the roots.

Pathogens causing root and/or stalk disease may alter the resistance to water flow through tissues in one of at least four ways:

1. If infection occurs through the root cortex and lesions develop in cortical tissues, the intimate root-soil contact may be destroyed. The net effect is to reduce the total root length in contact with soil, thus increasing the resistance in the radial pathway.

2. If the pathogen produces toxins, the permeability of root tissues to water may be altered, or, if the toxins enter the transpiration stream,

stomates may be affected. This aspect of host-pathogen interaction is not well studied, but either increases or decreases in membrane permeability appear possible.

3. Vessel plugging may occur either with tissues of the pathogen (e.g., fungal mycelia) or by induction of tyloses. This mechanism may result in a dramatic increase in axial resistance of the affected root, but if only a few roots are affected the consequences may be minimal, depending upon the soil water supply.

4. Finally, root deterioration reduces the transport capacity, but from a water supply standpoint, sorghum appears to "overproduce" roots, since loss of up to 50% of the root axis appears to have little impact on ψ_L so long as water is freely available to the remaining roots. This latter generalization may not hold true for nutrition, and almost certainly would not be true for crops grown in water-limited environments.

Water Flow in the Integrated Soil-Plant System

Several references were made in preceding sections to the dynamic nature of water potentials, both spatially and temporally, but visualization of the interdependence of soil, atmosphere, and plant is difficult. The question remains as to how soil and plant properties act in concert to regulate the flow of water through the system so the plant can remain relatively stress free. One means of examining this problem is through use of simulation models based on descriptions of flow in soil and plants as presented by Jordan and Miller (1980). An example of a sorghum crop growing in a drying clay soil is illustrated in Figure 1. If we assume that ψ_L remains constant at -15 bars (minimal plant stress) and the average root-length density is 1.0 cm root $(cm^3\ soil)^{-1}$, then a flow rate equivalent to 0.8 mm h^{-1} can be met only if the average ψ_s is above about -1.5 bars. This flow rate is in the range of those experienced in semi-arid field environments. If, on the other hand, flow rates are as low as 0.2 mm h^{-1} (cloudy, humid situation), then these rates could be met from a drier soil at a ψ_s of about -4.5 bars. Although this treatment and example are simplistic, they serve to illustrate the complex interaction of system components under realistic environmental conditions. An understanding of these interactions is central to understanding plant performance in both healthy and diseased conditions.

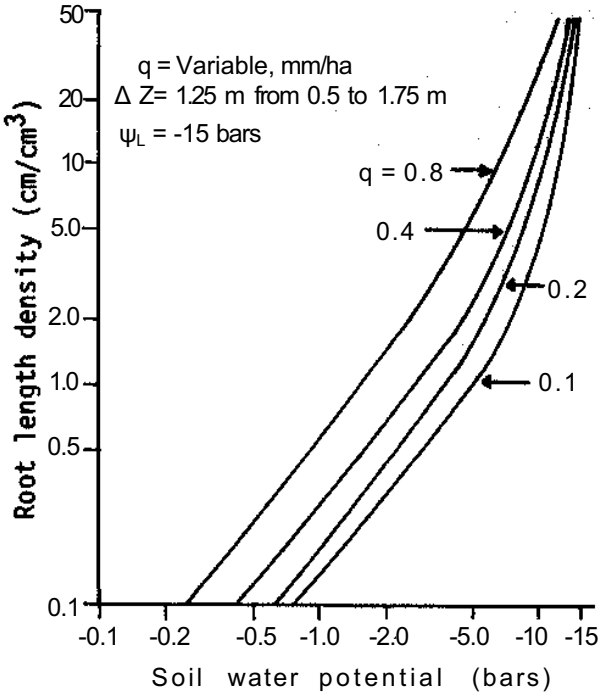

Figure 1. Predicted relations among root length density, soil water potential, and transpiration rate (q) for sorghum plants at a constant leaf water potential of -15 bars. Water uptake is assumed to come from a 1.25-m soil layer (Δ z) between 0.5 m and 1.75 m deep. (Source: Jordan and Miller 1980.)

Soil Water Deficit and Crop Productivity

Sorghum is grown in large areas of the semi-arid tropics because of its ability to produce grain under water-limited conditions. Factors contributing to sorghum's drought resistance have been detailed elsewhere (Jordan and Monk 1980, Jordan et al. 1983, Seetharama et al. 1982, Simpson 1981), but the fact remains that serious yield losses result from moderate to severe soil water deficits (Blum 1970, Garrity et al. 1982). Eastin et al. describe the sensitivities of sorghum to environmental stresses in these proceedings, and hence only a brief discussion of the effects of soil water deficits on development, activity, and duration of various

carbohydrate sources and sinks is presented to examine how root and stalk diseases effect yield reductions. For a more complete treatment of water relations of sorghum, readers are referred to recent reviews by Jordan (1983), Turner and Burch (1983), and Krieg (1983).

So long as cultural and environmental constraints are minimal, total dry matter production appears to be linearly related to the total solar radiation intercepted by a crop during the growing season (Monteith 1977). Light interception depends primarily on the seasonal distribution of the leaf area index (LAI); therefore factors that modify rates of leaf area development and maintenance may also modify the potential for grain yield.

On a whole-plant basis, the leaf area present at any time is a complex function of leaf numbers, leaf sizes, and leaf longevity. Leaf number is fixed within relatively narrow limits by the maturity of the cultivar, and the ultimate number of leaves formed per plant appears to be relatively unaffected by soil water deficits (Kannangara et al. 1983; W.R. Jordan and G.F. Arkin, Blackland Research Center, Temple, Texas, USA, unpublished data, 1982), although rates of leaf appearance are reduced. Since leaf appearance is strongly dependent on cellular expansion, and expansion is inhibited by water deficits (Boyer 1970; Hsiao et al. 1976a, 1976b), the reduction in leaf appearance rates is believed to result primarily from an inhibition of expansion. The net result from soil water deficits that develop progressively during vegetative growth is an overall reduction in leaf area per plant, as illustrated in Figure 2 (Jordan 1983), due primarily to reductions in final leaf sizes. During severe drought, formation of new leaf area may stop completely, giving the appearance that the crop is in a state of suspended animation (growth) while awaiting rainfall.

Reports dealing with the longevity of leaves during periods of drought are both sketchy and contradictory. Much of the confusion arises from failures to consider crop phenology and the rate and severity of water stress when evaluating effects of drought on leaf longevity. Recent results suggest that the longevity of individual leaves is not seriously altered by water deficits that develop gradually over long periods during vegetative growth stages, but rapid development of water deficits may accelerate senescence of lower leaves (Wilson and Allison 1978, Stout et al. 1978, Jordan 1983). However, if water deficits develop after anthesis, leaf senescence may be accelerated due to translocation of carbohydrates and

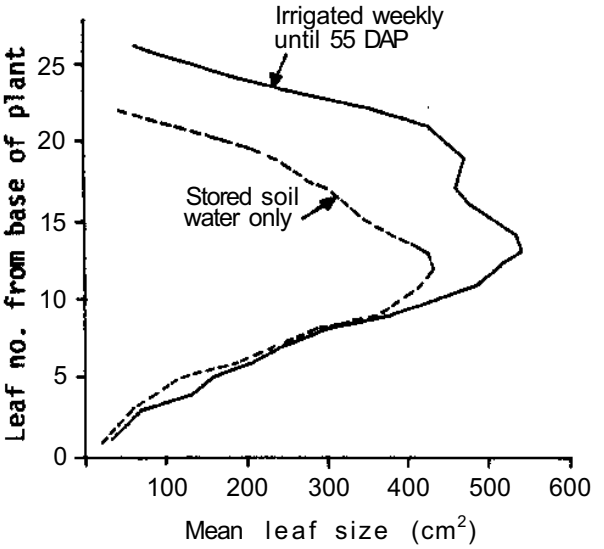

Figure 2. Vertical distribution of leaf area for 100 M sorghum plants grown on stored soil water or irrigated weekly until 55 days after planting (DAP). (Source: J.T. Ritchie, R.G.C. Smith, J.E. Begg, and W.E. Lonkerd, Blackland Research Center, Temple, Texas, USA; unpublished data, 1978.)

nitrogenous compounds to developing grain. This aspect of dry matter redistribution will be enlarged upon in following sections.

The seasonal pattern of dry-matter accumulation in sorghum is illustrated in Figure 3 (Krieg 1983). The period between panicle initiation and flowering, usually referred to as growth stage 2 (GS2), is a time of rapid increases in leaf area and dry matter and is the period when the potential grain number is determined. It is during GS2 that the crop expresses maximum sensitivity to environmental stresses, including water, heat, and light (Eastin et al., these proceedings). The causes underlying yield reductions from water deficit during GS2 are not fully understood, but Fischer's analytical framework of wheat growth and yield under water-limited conditions suggests that a functional balance exists between viable leaf area (source) and potential grain numbers (sink), provided that water deficits develop slowly (Fischer 1979). If this analysis also holds true for sorghum, some sort of feedback regulation between source (leaves) and sink (panicle) is implied. Whether this "communication" between source and sink arises from disruptions in the flow of organic energy and carbon sources from leaves to panicle, or from

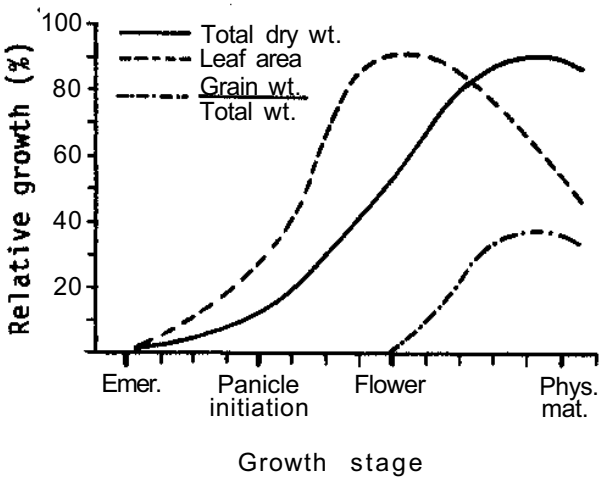

Figure 3. Fractional total dry weight, green leaf area, and harvest index of sorghum grown for grain as a function of growth stage. **(*Source: Krieg 1983.*)**

changes in the hormone balance of the panicle, is unknown (Krieg 1983). Regardless of the basic causes, the net effect of water deficit is expressed soon after anthesis as fewer grains per panicle, as evidenced by small panicles in general or by sterile branches within panicles (head blasting). In extreme cases the entire panicle may be sterile, either because the florets failed to develop or because they aborted.

Actual yields under favorable conditions are limited by source activity: that is, by the photosynthetic capacity of the leaves, stem, and panicle (Krieg 1983). Overall source activity is limited by soil water deficit through its inhibitory effect on total plant dry weight at anthesis, green leaf area remaining after anthesis, and production and translocation of photosynthate during the grain-filling period (GS3). As illustrated in Figure 3, grain filling occurs during a period when green leaf area is decreasing. It is not clear how much dry matter is translocated from senescing sorghum leaves, but in cases of crops well supplied with water, the loss of lower leaves probably has little impact on grain yield. However, if LAI is already low and the loss of leaf area after anthesis is accelerated by drought, serious source limitations may result from an inability to intercept sufficient radiation. In addition, the production of photosynthate may be reduced by stomatal closure during periods of peak evaporative demand when ψ_L is low, further reducing the flow of materials required to maintain grain growth. While some preanthesis assimilate is translocated from stem and leaves to grain under normarconditions, proportionately more may be translocated as photosynthate production rates fall due to drought during GS3. Results with most commercial sorghum hybrids suggest that the harvest index (HI) is maintained at relatively constant values as yields are reduced up to 50% by soil water deficits (Garrity et al. 1982), but that HI falls at extremely low grain yields (Blum 1970).

The total amount of dry matter stored in stem and leaves that is capable of translocation to developing grain is not known, but recent results with senescent and nonsenescent cultivars suggest that genotypic variability for this trait does exist for sorghum. Depletion of stem (and root?) reserves during GS3 may predispose senescent cultivars to infection by soilborne pathogens, especially *Macrophomina phaseolina,* the causal organism of charcoal rot, but a direct causal relationship has not been established. However, it is clear that development of root or stalk diseases that interfere with absorption or transport of water will create internal water deficits, with consequences similar to those described above for soil water deficits.

Disease Development and Soil Water Supply

Root and stalk rot diseases of sorghum often develop most dramatically during GS3, when water is in short supply and soil temperatures are high. Charcoal rot, a serious disease of sorghum and maize, is expressed in this circumstance, but research reports on the causal pathogen and its interaction with the soil environment and host are indeed few. Even considering this paucity of information, the charcoal rot problem appears to be the best studied example of a stalk or root disease causing serious crop losses in, sorghum (Mughogho and Pande, these proceedings). The discussion in the following sections will concentrate on those reports dealing with the effects of soil water deficits on *M. phaseolina* and the development of charcoal rot.

Effects of ψ_s on *M. phaseolina*

Effects of low water potential on the germination of sclerotia and growth of mycelia have apparently been studied in detail only in the laboratory, using

artificial media (Dhingra and Sinclair 1978). Sclerotial germination in culture occurs over a wide range of water potentials and temperatures, including those expected in the field during drought (Odvody and Dunkle 1979, Shokes et al. 1977). The sclerotia appear well adapted for survival for long periods in dry soil, but exposure to wet soil (-0.01 bar) at 30°C for 2 weeks decreased survival in one test (Shokes et al.1977).

Since sclerotia germinate readily in culture over a wide range of conditions, the question of control of germination under favorable soil conditions naturally arises. Some form of nutrient-dependent fungistasis is most often alluded to as a germination control (Ayanru and Green 1974). Odvody and Dunkle (1979) observed higher germination on potato dextrose agar at low osmotic potentials (< -40 bars) than on water agar, but germination was similar at higher osmotic potentials. This suggests that endogenous nutrients should support germination under most conditions. However, they also reported that sclerotia isolated from soil did not germinate when incubated on water agar in the presence of contaminating soil particles, but germinated readily when they were surface-sterilized, suggesting some type of active fungistasis associated with contaminating microorganisms. In other studies, the fungistatic properties of nonsterile soil were reduced or eliminated by additions of nutrients such as amino acids (Ayanru and Green 1974) or additions of root exudate (Smith 1969), suggesting large supplies of appropriate organic compounds can overcome propagule dormancy.

The relationship of low ψ_s to sclerotial germination is not clear, but based on current evidence at least three hypotheses may be proposed. First, low ψs near the soil surface may differentially inhibit the soil microflora, thereby reducing the endogenous or exogenous fungistasis so that germination can occur. A body of evidence suggests that growth and activity of bacteria are inhibited at much higher ψ_s than several fungi (Griffin 1981, Harris 1981). in fact, growth of many fungi, including *M. phaseolina,* appears to be stimulated by reductions in the osmotic potential of culture media by -5 to -20 bars (Shokes et al. 1977, Odvody and Dunkle 1979, Cook and Duniway 1981*).* However, stimulation by equivalent matric potentials in soils is often not observed (Cook and Duniway 1981). Wilson and Griffin (1975) reported that bacterial respiration was negligible at ψ of -20 bars, while total soil respiration was constant to -30 bars, presumably because fungal respiration was unaffected.

A second hypothesis involves the effects of low ψ_s on chemical composition and/or amount of root exudate (Kerr 1964, Cook and Flentje 1967) and the distance these nutrients move from roots. Cook and Duniway (1981) suggest that the availability of nutrients may confine propagule germination to the relatively high water potentials suitable for growth of the host plants, but that low ψs near the surface will not restrict growth or activity of the host if sufficient root length exists in wetter, lower regions. Exudates produced by roots in dry regions may raise the ψ_s in the rhizosphere, but the distance this influence might extend into the soil is not known.

The third hypothesis is related to the growth and senescence of roots induced by low ψ_s. A recent review on the relationships between root growth dynamics and epidemiology of root-invading fungi (Huisman 1982), indicates that the information available on this topic is inadequate. Roots of most species will not grow in soils drier than about -2 bars. Taylor and Klepper (1974) reported the disappearance of roots growing against rhizotron windows when ψs fell below -2 to -3 bars. Presumably these roots had deteriorated and, in doing so, could have provided a nutrient source for the microflora. A recent study with maize (Schneider and Pendery 1983) found enhanced root senescence when plants were exposed to mild water stress, even though symptoms of stress were not visible. The total amount of nutrients that could be supplied by root decomposition is not known because only the fine roots appear to be so affected (W.R. Jordan, personal observations). Root proliferation in surface horizons occurs rapidly and repeatedly as soils are rewet by rainfall or irrigation; therefore the potential amount of nutrients available from this source may be larger than expected.

Water Deficit and Disease Development

The association of soil water deficit with increased severity of stalk and root rots caused by normally weak pathogens (including species of *Fusarium, Gibberella, Diplodia*, and *Macrophomina*) is well established, although the underlying mechanism(s) remain unclear. Recently, Schneider and Pendery (1983) presented evidence that stalk rot of maize at season's end was strongly enhanced by mild stress during earlier growth stages. Mild

stress, insufficient to cause visible symptoms, resulted in pith discoloration or stalk rot in 60.3% of the plants during the pretassel stage of development, 25.3% during postpollination, and 7.7% during grain filling. Even though the stress was mild (ΨL about 2 bars lower than well-watered controls), root senescence was accelerated in uninfested treatments and enhanced infection and root colonization occurred in infested treatments, causing the resistance to water flow to increase about twofold. The causal organism was believed to be *Fusarium moniliforme.*

Similar results were obtained with sorghum growing in pots infested with sclerotia of *M. phaseolina* (Odvody and Dunkle 1979). When water-stressed during the soft dough stage, 83% of the fertile CK60 plants developed charcoal rot symptoms, while male-sterile plants were symptomless. Since both fertile and male-sterile plants had high rates of root infection, male-sterile plants apparently possessed some mechanism to retard spread of the infection. Also, since nonstressed plants growing in infested soil remained relatively infection free, stress appeared to promote initial infection of host roots.

Studies at ICRISAT provide evidence that charcoal rot development is related to the severity as well as the timing of soil water deficits. Using a line-source sprinkler system to establish a gradient in soil water deficit, Seetharama et al. (unpublished data, 1983) examined the relationship between the amount of water applied during GS3 (distance from the line source) and the fraction of plants developing soft stalks following toothpick inoculation (Fig. 4). A linear response between distance from the line source and disease development is clearly illustrated for both years, supporting the view that disease severity and drought severity are coupled during the period when the sink demand from developing grain' is large. Grain yield decreased linearly with distance from the line source in both years. Additional observations provided evidence that the rate at which disease spread from the point of inoculation increased with time after flowering, as well as with stress severity, supporting earlier findings by Edmunds (1964) and Livingston (1945).

While the concept of edaphic factors in "preconditioning'' or "predisposition" of host plants to disease is certainly not new (Schoeneweiss 1978; Yarwood 1976; Dodd 1980a, 1980b), underlying mechanisms remain largely unknown. Recently, Dodd (1980a, 1980b) proposed an explanation of predisposition based on the carbohydrate status of

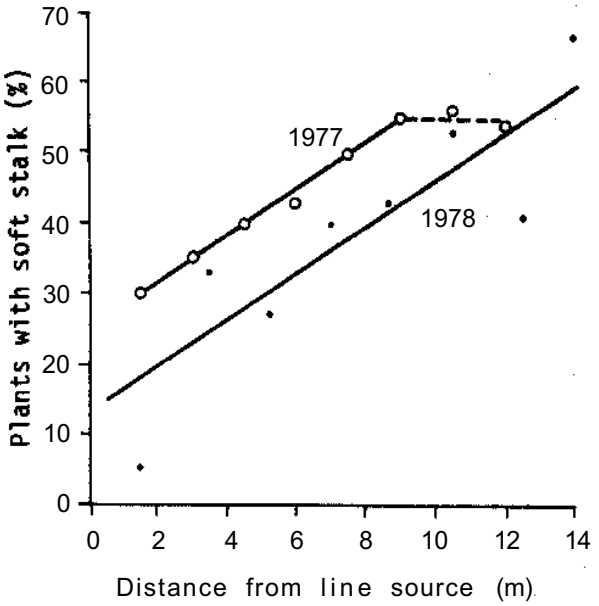

Figure 4. Relation between charcoal rot development (plants with soft stalk) and soil water supply after anthesis (distance from line source) for CSH-6 sorghum grown at Hyderabad, India, in 1977 and 1978. (Source: Seetharama et al., ICRISAT; unpublished data, 1983.)

root tissues and the influence of soil water deficits on deposits and withdrawals from the sink. In this concept, carbohydrate depletion in root tissues weakens the cellular defense mechanisms, allowing the invasion and spread of disease. The effects of soil water deficit on production and redistribution of photosynthate were discussed in earlier sections, and Dodd's concept is supported by work of Edmunds and Voigt (1966), Edmunds et al. (1964, 1965) and Odvody and Dunkle (1979). However, although no alternative explanations of this predisposition phenomenon enjoy wide acceptance, it seems unlikely that any concept based on simple changes in carbohydrate status of roots or stalks will hold up to critical examination. McBee discusses the question of stem reserves in greater detail in these proceedings.

Disease Development and Temperature

Throughout the sorghum-growing regions, high temperatures normally accompany droughts, but

seldom have the effects of high temperature per se been separated from those due to water deficits. A C_4 species, sorghum is adapted to hot, high-radiation regimes, but these same conditions are often cited as facilitating disease incidence and development. In the following sections we explore the effects of high temperatures on the host and pathogen and speculate on the role of this stress on host-pathogen interaction.

Sorghum Response to High Temperature

The effects of both super- and supraoptimal temperatures on sorghum have been recently reviewed (Peacock 1982), with the conclusion that consequences of high temperatures are most serious when they coincide with the critical growth stages of the crop. Thus, germination and emergence are viewed as critical to obtaining an adequate plant population, development and maintenance of leaf area as critical to photosynthate production, and panicle development and growth as critical to yield potential. Since root and stalk rots are normally associated with late-season stresses, we will consider only those effects observed during GS2 and GS3.

The importance of leaf-area development and maintenance has already been discussed with respect to water. Numerous reports document the fact that the general effects of moderate increases in temperature are reflected in faster growth rates in general, as evidenced by earlier maturity. This fact is incorporated in several plant growth models in which process rates are governed by heat-unit accumulation rates. The question of optimum temperature or leaf-area development remains unresolved because much of the growth data collected in controlled environments is not directly applicable to crops grown in a field environment. Data from ICRISAT (Peacock 1982) suggest that leaf extension rates are greatest at an air temperature of about 34°C and that final leaf number and leaf area increase as temperatures increase from 25/20°C (day T/night T) to 35/25°C. Although Quinby et al. (1973) reported genetic variation in leaf growth in relation to air temperature, little use seems to have been made of this information and littfe effort appears to be directed toward identification of genotypes capable of growth maintenance at high temperatures. Escalada and Plucknett (1975) reported enhanced tillering as temperatures were increased from 23.9/15.5°C to 32.2/23.9°C, so long as daylengths also increased, suggesting a link between tiller development and total photosynthate supply.

The effects of temperature on yield and yield components have been studied at several locations, with the conclusion that grain numbers per panicle are not reduced by growth at temperatures as high as 35/25°C. However, yield is markedly reduced by these high temperatures due to a reduction in weight per grain. Excessively high temperatures during panicle development often result in head blasting or localized abortion within the panicle, but these effects have not been well documented, nor have the effects of temperature been separated from those due to dehydration.

Leaf firing occurs in the field in response to hot, dry conditions, and variability in both the extent and pattern of firing appears to be under genetic control. Peacock (1979) reported firing in hybrid RS-610 when leaf temperatures exceeded 43°C, but at least some germplasm will tolerate leaf temperatures as high as 55°C (Peacock 1982). Leaf firing is one component used in the Texas Agricultural Experiment Station breeding program as a selection criterion for drought tolerance (Rosenow et al. 1983).

The causes underlying heat-induced firing are not known, but other evidence also suggests that genetic variability exists for heat tolerance. In one test, grain yields of M35-1 conversion hybrids growing under conditions of heat stress in Nebraska were correlated with an estimate of heat tolerance based on electrolyte leakage from damaged leaf cells (Sullivan and Ross 1979). Other reports document the existence of substantial genotypic variability for heat tolerance based on this method (Sullivan 1972, Jordan and Sullivan 1982). Genetic variability in the ability to maintain high photosynthetic rates at temperatures between 40° and 43°C also exists (Norcio 1976), but present evidence suggests that photosynthetic rates would be greatly reduced in the range of 44° to 48°C, well below the temperature causing firing in some Indian cultivars.

Temperature and Host-Pathogen Interaction

Even though hot, dry conditions enhance charcoal rot on susceptible sorghum cultivars, there is little evidence that heat stress per se plays a role in

disease incidence or development At least one causal organism seems well adapted to the high temperatures that exist near the surface of dry soils. Mycelia of *M. phaseolina* are capable of growth to at least 40°C in culture (Odvody and Dunkle 1979), as are many other soilbome fungi. Although unexplained, the growth optimum of the fungus shifts to lower water potentials at higher temperatures, suggesting a unique form of adaptation to the high T-low ψ_s conditions expected near the surface as the soil dries.

Bell (1982) recently proposed a model showing how temperature may differentially affect the rate of pathogen colonization and active host resistance to differences in relative resistance. His Model A, illustrated in Figure 5, is cited as an example applicable to charcoal rot caused by *M. phaseolina.* In this case, host resistance reaches its maximum at temperatures near or slightly below those optimum for growth, but lower than temperatures for maximum rates of pathogen colonization. Since relative resistance is the ratio of the two rates, increases in temperature result in a decline in relative resistance. While these general predictions appear superficially plausible, there is no direct experimental evidence to support this hypothesis.

Disease Development and Mineral Nutrition

Specific Element Effects on Disease

Each mineral element essential to plant growth has been implicated in disease incidence or severity, as have many not considered essential. The body of literature dealing with interactions between mineral elements and plant disease in general is extensive (e.g., reviews by Graham 1983, and Huber 1978,1980), but few reports deal specifically with sorghum. Reported effects of specific elements on some of the organisms associated with root and stalk rots are summarized in Table 1 .

Macronutrients (N, P, K, Ca, Mg, and S) generally have no effects on disease resistance at supraoptimal levels, but usually have their effects only at low or deficient levels. On the other hand, the micronutrients (Cu, B, Mn, Fe, and Zn) have pro-

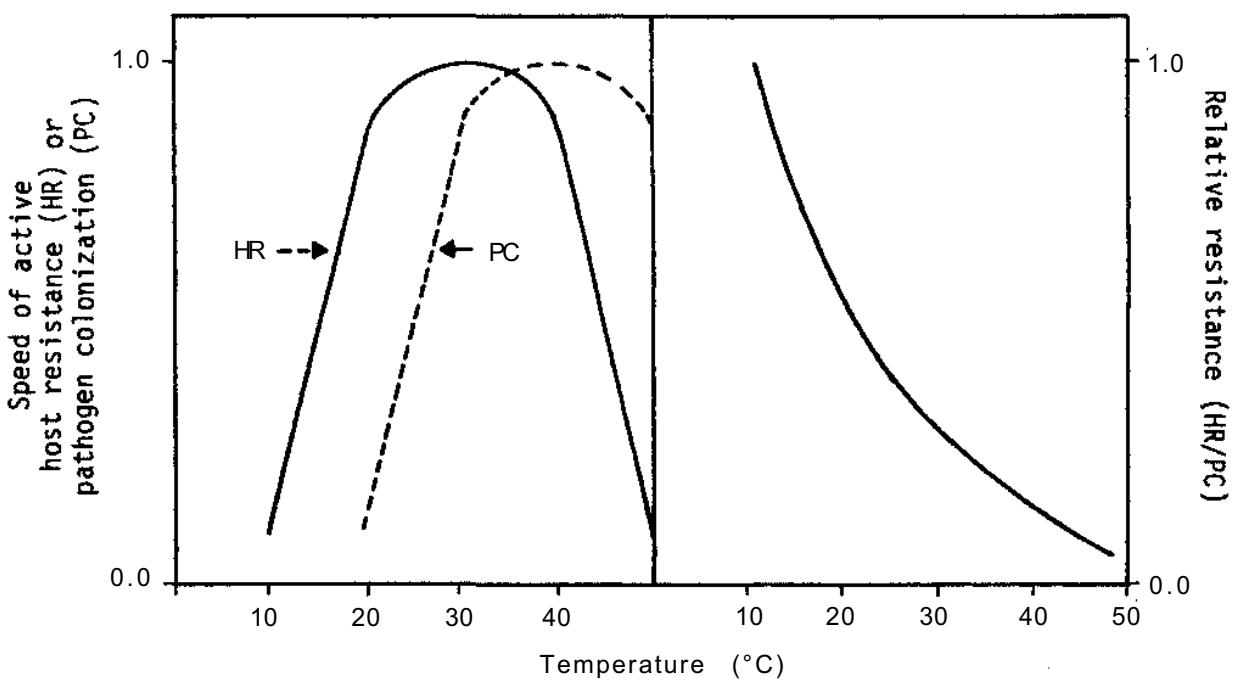

***Figure* 5. *Illustrations of how temperature affects the relative resistance of a host based on its effects on the speed of pathogen colonization and speed of active host resistance. The resultant pattern of decrease in relative resistance at increasing temperatures fits observations of charcoal rot of sorghum caused by* Macrophomina phaseolina (*Adapted from Bell 1982.*)**

Table 1. Summary of reported interactions of mineral elements and disease for some root/stalk rot pathogens. (Source: Huber 1980.)

	Mineral element												
Pathogen	NH4	N03	P	L	Ca	Mg	S	Na	Mn	Fe	Zn	B	Cu
Diplodia zeae	D[a]	I	I	D									
Fusarium colmorum									D	D	D		D
Fusarium moniliforme	I	D		D									
Fusarium nivale				D			D						
Fusarium roseum	I	D			D								
Gibberella zeae	D	±	±	D									
Ophiobolus graminis	D	±	D	D	I		D						
Phythium arrhenomanes	D	±	D										
Rhizoctonia solani	I	D	D	I	D	D	D	D	D				

a. Incidence of disease decreased (D). increased (I), or dependent on hosts or environmental conditions (+).

nounced effects on disease resistance at supraoptimal levels as well as at low or deficient levels. These responses are probably because the macronutrients are involved in compositional, structural, conformational, and osmotic functions in plants, and micronutrients usually function as catalysts, cofactors, and inhibitors. Increasing the supply of an element in deficient or low supply generally increases the resistance of plants to pathogens.

Many factors, interactions, and responses are involved in mineral element relationships to disease resistance. The effects of mineral elements on plant yield may involve not only the plant requirements for a specific element, but also the ways in which the element may change the host's defense mechanism against disease. Mineral elements may also have direct toxic effects on invading pathogens. Lignin and phenol synthesis seem to be more affected by certain elements (N, Cu, B, and Mn) than by others; phytoalexin synthesis also appears to be affected by certain other elements (Zn, Fe, and Ni); certain biochemical pathways for disease defense may also require specific elements (Si, Li, and Ni); competition between host and pathogen may occur with certain of the elements (Fe); and interactions and toxicities appear with almost all elements. The mechanisms for mineral-element defenses in disease resistance are multiple, and the function of each element in metabolism or plant disease resistance processes must be understood separately. So little is understood about the function of mineral elements in disease resistance that similarities of element functions and disease resistance may be coincidental.

Mycorrhizae and Mineral Element Uptake

Mycorrhizal fungi play important roles in assuring sufficient and constant supplies of nutrients to host plants under all conditions, but their importance may be magnified during drought. The importance of mycorrhizae in enhancing uptake of mineral elements became evident only in recent years (Tinker 1980, Tinker and Gildon 1983). Increased uptake of N by plants infected with ectotrophic mycorrhizae (ECM) has been suggested, but proof has not been conclusive. ECM fungi have been found to enhance Zn and K uptake, and their mycelia translocate Ca. On the other hand, vesicular-arbuscular fungi (VAM) have been found to enhance P, Zn, Cu, K, Si, and S uptake by host plants, and also to translocate Ca. VAM fungi are highly involved with enhancing P uptake by plants, especially under conditions of low P. The relationships between VAM fungi and P have been the subject of numerous investigations since their discovery.

Improvement of host plant nutrition by mycorrhizal infection should occur whenever the uptake rate of the specific element by the host root is restricted by transport mechanisms of the element in soils (diffusion and mass flow) below that required for optimum plant growth allowed by the environment, if mycorrhizae can absorb and transfer that particular element. Yield improvements are difficult to predict, but growth responses have been large in some cases. For example, the amount of soluble P fertilizer required to give the same growth response as VAM infection for several plants was around 100 kg P/ha, and as high as 500 kg P/ha for a citrus crop (Menge et al. 1978).

Since micronutrients such as Cu, Zn, and Mn have very low soil mobility, mycorrhizae-enhanced plant uptake has been observed. The mechanisms for uptake and transfer of micronutrients within the mycelia are not fully known, but could be associated closely with P compound complexes such as polyphosphate.

The beneficial effects of VAM on apparent drought resistance of plants (Maronek et al. 1981) may result from two sources. Hyphae from VAM-infected roots extend some distance into the soil mass, effectively increasing the root length density and thereby reducing the distance water must flow through soil (Allen 1982, Gerdemann 1970, Safir et al. 1972). These root extensions could become important in maintaining high water uptake rates as ψ_s and H fall due to evapotranspiration. Equally important, however, may be the continued growth of roots made possible by the enhanced uptake of P described above (Sieverding 1981). Continued or stimulated growth of root axes places larger root areas in contact with unexplored, wetter soil, thereby delaying the onset of stress. Since mineral nutrients added as fertilizers are usually concentrated in the upper 15 cm of the soil profile, mycorrhizal-enhanced uptake of mineral elements from soil too dry to support root growth may be very important to crop health and productivity, and deserves more extensive study.

Influence of Water- and Nutrient-Stress Interactions on Disease Development

Deficiencies of both water and nutrients, especially N, are the rule rather than the exception in many sorghum-producing regions in developing countries. Even in highly productive dryland systems, water availability has a strong influence on the uptake efficiency and recovery of added nutrients and may influence management decisions dealing with the amount and timing of fertilizer applications.

The total and seasonal nutrient requirements of a sorghum crop have been presented in detail (Lane and Walker 1961, Vanderlip 1972) and will not be repeated here except to point out that large quantities of N, P, and K are required during the relatively brief GS3. For example, at maturity, grain contains 67% of the plant's total N, 76% of its P, and 26% of its K. Much of the grains' total requirement can be supplied by uptake from soil, so long as the surface remains moist or is frequently wetted. However, as the surface dries, root activity is forced to deeper strata that are normally low in nutrients, and grain demands are met by remobilizing elements stored in leaves, roots, and stalks. Under drought conditions it is not known whether senescence or firing of lower leaves is triggered by translocation of carbohydrates or mobile nutrients such as N.

High grain yields require high plant populations and large inputs of N, but these conditions also increase disease severity. Sachan et al. (ICRISAT, unpublished data, 1983) found a strong interaction between grain yield, N fertility, and stalk rot for sorghum hybrid CSH-6 subjected to water stress after anthesis with the line source system (Fig. 6). High N fertility resulted in both higher grain yield and greater incidence of stalk rot. The range of water supplies that resulted in a linear increase in grain yield also resulted in a linear decrease in disease incidence for both fertility levels.

Conclusions and Research Needs

Edaphic factors such as water availability, temperature, and mineral nutrient supply have been shown to have a large influence on both infection and disease development by normally weak pathogens causing root and stalk rots. Fungi are among the members of the soil microflora most resistant to low soil water potentials, making them ideally suited to the ecosystem of the near-surface soil horizon. Even mild water stresses trigger changes in host-root resistance, allowing infection at an earlier stage of growth than expected based on symptom expression. Disease spread in host tissues is associated with an increasing demand for carbohydrates and nutrients by the developing grain. Since soil water deficits during grain filling may reduce photosynthate production, greater disease incidence observed under these conditions suggests a causal relationship between the carbohydrate status of roots and stems and disease severity. The generality of this association has not been examined in detail. High supplies of N fertilizer also promote disease, but are required for high grain yields.

The line-source sprinkler system is an important tool for screening for resistance to both drought and disease. It provides a relatively simple means to establish a dependable, multilevel stress condi-

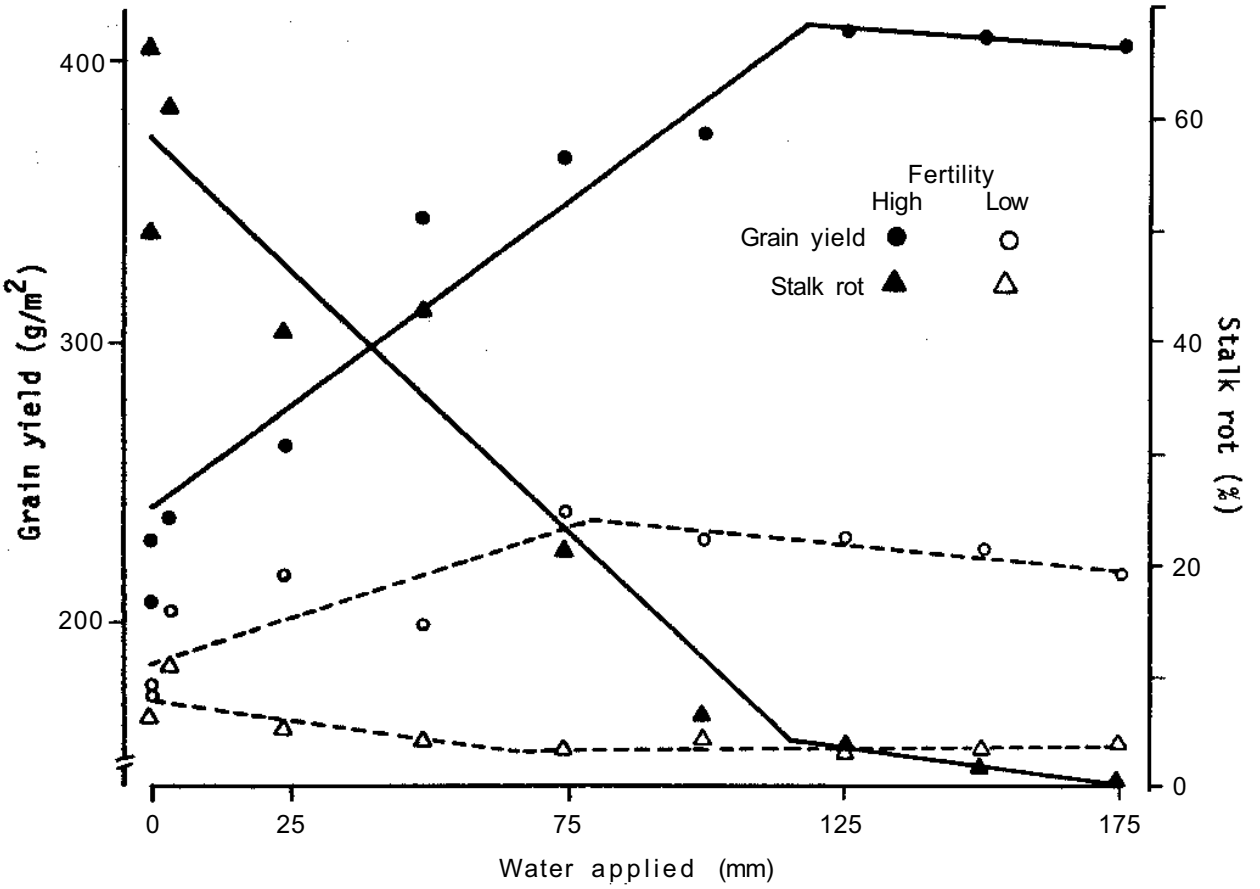

***Figure* 6. *Influence of nitrogen fertility on the interaction among grain yield, charcoal rot incidence, and water applied after anthesis. Low fertility plots received 20 kg N ha^{-1}, while high fertility plots received 100 kg N ha^{-1}.* (*Source: Sachan et al., ICRISAT, unpublished data, 1983.*)**

tion in the field and overcomes many of the experimental limitations in studies of soilborne pathogens. Results from the line source system also suggest strategies of water management that may be effective in disease delay or prevention. Maintenance of high plant water potentials, at least during grain fill, are effective in prevention of yield losses due to disease and may be achieved by a variety of cultural and water management techniques. The introduction of drought- and disease-resistant cultivars into farming systems will present new opportunities for management for high yield.

Several important problems should receive added research emphasis. The role of water stress in the production and composition of root exudates should be examined in greater detail because of the possible importance of this source of organic and mineral nutrients to germination of propagules and growth of pathogens. Also, stress-induced changes in root physiology should be studied closely because of the apparent role of even mild stress in the infection process. Root senescence is an almost unexplored area of research that has importance to both drought and disease resistance. The total amount of dry matter lost as exudates and during root degeneration is unknown.

Information about the importance of individual mineral nutrient elements to the pathogen and to disease development is badly needed, especially for sorghum. Little is known about interactions between water and nutrient availability near the soil surface. The importance of root growth in near-surface regions of the profile should be explored because of the large requirements for mineral elements and their concentration in that region. Finally, the importance of mycorrhizal fungi in enhancing sorghum resistance to drought and nutrient stresses remains to be defined.

In summary the following is a list of specific topics for future research:

I. Effects of abiotic stresses on the host

A. Optimum root system and rooting pattern
   1. Define "optimum" in terms of the soil-plant-atmosphere system.
   2. What is the role of root resistance?
   3. Determine efficient use of water resources.

B. Root senescence and regeneration
   1. Determine the effects of low soil water potential on growth and survival, especially in the near-surface regions of the soil profile.
   2. What quantity of root dry matter is lost or remobilized?
   3. What is the role of root deterioration in microflora ecology?
   4. How much regeneration of roots is necessary for efficient water and nutrient uptake?

C. Root exudates; quantity and composition

D. Leaf firing
   1. What is the mechanism—heat or desiccation? To what degree does the retranslocation of carbon and minerals from the leaf affect its longevity?
   2. Determine genotypic differences.
   3. Determine progressive vs general firing.

E. Floret abortion
   1. What controls head blasting—heat stress, growth inhibition due to water stress, involvement of hormones, etc.?
   2. What are the limits to recovery via seed size adjustment?

F. Capacity and consequences of redistribution of stem and root reserves
   1. Is there some redistribution under all conditions?
   2. What is the relationship of reserves to yield under stress?

G. Interactions of biotic stresses

H. Role of individual nutrient elements and water x nutrient interactions on host growth, vigor, and senesence.

II. Effects of abiotic stresses on pathogens

A. Confirmation of synthetic media results in soil system
   1. Most media studies use osmotic stress. What is the impact of soil water deficits through matric effects?

B. Basis for apparent drought resistance
   1. Investigate stress levels at which propagules are resistant to heat and desiccation.
   2. What is the role of osmoregulation in maintenance of growth?

C. Specific nutrient effects

D. Temperature tolerance at low soil-water potential
   1. What is the effect of high temperatures near soil surface?
   2. Determine growth optimum shifts to higher temperature as water potential decreases in media studies.

E. I nvestigate the relation between soil physical properties and fungal survival

III. Host/pathogen interactions

A. Role of root exudates on germination, growth, and pathogenicity of fungi

B. Effect of pathogen on exudates

C. Mechanisms of infection

D. Mechanisms for control pf pathogen activity and spread in host tissues

E. Mechanisms for yield reduction

IV. Edaphic stress/host stress/pathogen interactions under field conditions

A. Inorganic nutrient availability to host and pathogen, including the role of mycorrhizal fungi

B. Mechanisms for stress-induced predisposition to infection

C. Direct vs indirect control of pathogen spread in tissues

D. Separation of effects of abiotic stresses from biotic stresses on grain yield

References

ALLEN, M.F. 1982. Influence of vasicular-arbuscular mycorrhizae on water movement through *Bouteloua gracilis* (H.B.K.) Lay ex Steud. New Phytologist 91:191-196.

AYANRU, D.K.G., and GREEN, R.J., Jr. 1974. Alteration of germination patterns of sclerotia of *Macrophomina phaseolina* on soil surfaces. Phytopathology 64:595-601.

BELL, A.A. 1982. Plant pest interaction with environmental stress and breeding for pest resistance: Plant diseases. Pages 335-363 *in* Breeding plants for less favorable environments (eds. M.N. Christiansen and C.F. Lewis). New York, New York, USA: John Wiley & Sons.

BLUM, A. 1970. Effect of plant density and growth duration on grain sorghum yield under limited water supply. Agronomy Journal 62:333-336.

BOYER, J.S. 1970. Leaf enlargement and metabolic rates in corn, soybean, and sunflower at various leaf water potentials. Plant Physiology 46:233-235.

COOK, R.J., and DUNIWAY, J.M. 1981. Water relations in the life-cycle of soilborne plant pathogens. Pages 119-139 *in* Water potential relations in soil microbiology (eds. J.F. Parr, W.R. Gardner, and L.F. Elliott), Special publication no. 9. Madison, Wisconsin, USA: Soil Science Society of America.

COOK, R.J., and FLENTJE, N.T. 1967. Chlamydospore germination and germling survival of *Fusarium solani* f. *pisi* in soil as affected by soil water and pea seed exudation. Phytopathology 57:178-182.

DHINGRA, O.D., and SINCLAIR, J.B. 1978. Biology and pathology of *Macrophomina phaseolina.* Vicosa, Brazil: Imprensia Universitaria, Universidade Federal De Vicosa. 166 pp.

DODD, J.L. 1980a. The photosynthetic stress-translocation balance concept of sorghum stalk rots. Pages 300-305 *in* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, A.P. 502324, India: ICRISAT.

DODD, J.L. 1980b. Grain sink size and predisposition of *Zea mays* to stalk rot. Phytopathology 70:534-535.

EDMUNDS, L.K. 1964. Combined relation of plant maturity, temperature, and soil moisture to charcoal stalk rot development in grain sorghum Phytopathology 54:514-517.

EDMUNDS, L.K., and VOIGT, R.L. 1966. Role of seed production in predisposition of sorghum to charcoal rot. Phytopathology 56:876 (abstract).

EDMUNDS, L.K., VOIGT, R.L., and CARASSO, F.M. 1964. Use of Arizona climate to induce charcoal rot In grain sorghum. Plant Disease Reporter 48:300-302.

EDMUNDS, L.K., VOIGT, R.L., and CARASSO, F.M. 1965. Charcoal rot induction and development in the field in Arizona. Proceedings of the Fourth Biennial Grain Sorghum Research and Utilization Conference, sponsored by the Grain Sorghum Producers' Association (GSPA) and the Sorghum Improvement Conference of North America, Lubbock, Texas. Available from GSPA, Abernathy, Texas, USA.

ESCALADA, R.G., and PLUCKNETT, D.L. 1975. Ratoon cropping of sorghum: 2. Effect of daylength and temperature on tillering and plant development. Agronomy Journal 67:479-484.

FISCHER, R.A. 1979. Growth and water limitations to dryland wheat yield in Australia: A physiological framework. Journal of the Australian Institute of Agricultural Science 54:83-94.

GARRITY, D.P, WATTS, D.G., SULLIVAN, C.Y., and GILLEY, J.R. 1982. Moisture deficits and grain sorghum performance: Effect of genotype and limited irrigation strategy. Agronomy Journal 74:808-815.

GERDEMANN, J.W. 1970. The significance of vesicular-arbuscular mycorrhizae in plant nutrition. Pages 125-129 *in* Root diseases and soil-borne plant pathogens (eds. T.A. Toussoum, R.V. Bega, and P.E. Nelson). Berkeley, California, USA: University of California Press.

GRAHAM, R.D. 1983. Effects of nutrients stress on susceptibility to disease with particular reference to the trace elements. Advances in Botanical Research 11:221 -274.

GRIFFIN, D.M. 1981. Water potential as a selective factor in the microbial ecology of soils. Pages 141 -151 *in* Water potential relations in soil microbiology (eds. J.F. Parr, W.R. Gardner, and L.F. Elliott), special publication no. 9. Madison, Wisconsin, USA: Soil Science Society of America.

HARRIS, R.F. 1981. Effect of water potential on microbial growth and activity. Pages 23-95 *in* Water potential relations in soil microbiology (eds. J.F, Parr, W.R. Gardner, and L.F. Elliott), Special publication no. 9. Madison, Wisconsin, USA: Soil Science Society of America.

HSIAO, T.C., ACEVEDO, E., FERERES, E., and HENDERSON, D. 1976a. Water stress, growth, and osmotic adjustment. Philosophical Transactions of the Royal Society of London, B Series, 273:479-500.

HSIAO, T.C., FERERES, E., ACEVEDO, E., and HENDERSON, D.W. 1976b. Water stress and dynamics of growth and yield of crop plants. Pages 281-305 *in* Water and plant life, problems and modern approaches (eds. 0 1 . Lange, L. Kappen, and E-D Schuize). Berlin, Federal Republic of Germany: Springer-Verlag.

HUBER, D.M. 1978. Disturbed mineral nutrition. Pages 163-181 *in* Plant disease, an advanced treatise, Vol. 3: How plants suffer from disease (eds. J.G. Horsfall and E B . Cowling). New York, New York, USA: Academic Press.

HUBER, D.M. 1980. The role of mineral nutrition in

defense. Pages 381-406 *in* Plant disease, an advanced treatise, Vol. 5: How plants defend themselves (eds. J.G. Horsfall and E.B. Cowling). New York, New York, USA: Academic Press.

HUISMAN, O.C. 1982. Interrelations of root growth dynamics to epidemiology of root-invading fungi. Annual Review of Phytopathology 20:303-327.

JORDAN, W.R. 1983. Whole plant responses to water deficits: An overview. Pages 289-317 (Ch. 7A) *in* Limitation to efficient water use in crop production (eds. H.M. Taylor, W.R. Jordan, and T.R. Sinclair). Madison, Wisconsin, USA: American Society of Agronomy.

JORDAN, W.R., DUGAS, W.A., and SHOUSE, P.J. 1983. Strategies for crop improvement for drought prone regions. Agricultural Water Management 7:281 -299.

JORDAN, W.R., and MILLER, F.R. 1980. Genetic variability in sorghum root systems: Implications for drought tolerance. Pages 383-399 *in* Adaptation of plants to water and high temperature stress (eds. N.C. Turner and P.J. Kramer). New York, New York, USA: John Wiley & Sons.

JORDAN, W.R., and MONK, R.L. 1980. Enhancement of drought resistance of sorghum: Progress and limitations. Pages 185-204 *in* Proceedings of the 35th Annual Corn and Sorghum Research Conference. Washington, D.C., USA: American Seed Trade Association.

JORDAN, W.R., and SULLIVAN, C.Y. 1982. Reaction and resistance of grain sorghum to heat and drought. Pages 131 -142 *in* Sorghum in the Eighties: Proceedings of the International Symposium on Sorghum, sponsored by INTSORMIL, ICAR, and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

KANNANGARA, T., SEETHARAMA, N., DURLEY, R.C., and SIMPSON, G.M. 1983. Drought resistance of *Sorghum bicolor:* 6. Changes in endogenous growth regulators of plants grown across an irrigation gradient. Canadian Journal of Plant Science 63:147-155.

KERR, A. 1964. The influence of soil moisture on infection of peas by *Pythium uitimum.* Australian Journal of Biological Sciences 17:676-685.

KRIEG, D.R. 1983. Sorghum. Pages 351-388 *in* Crop water relations (eds. I.D. Teare and M.M. Peet). New York, New York, USA: John Wiley & Sons.

LANE, H.C., and WALKER, H.J. 1961. Mineral accumulation and distribution in grain sorghum. Texas Agricultural Experiment Station Miscellaneous Publication 533. College Station, Texas, USA: Texas Agricultural Experiment Station. 9 pp.

LIVINGSTON, J.E. 1945. Charcoal rot of corn and sorghum. Nebraska Agricultural Experiment Station Bulletin No. 136. Lincoln, NE 68503, USA: University of Nebraska. 32 pp.

MARONEK, D.M., HENDRIX, J.W., and KIENAN, J. 1981. Mycorrhizal fungi and their importance in horticultural crop production. Pages 172-213 (Ch. 5) *in* Horticultural reviews. Westport, Connecticut, USA: AVI Publishing Co.

MENGE, J.A., ANAUSKAS, C.K., JOHNSON, E.L.V., and PLATT, R.G. 1978. Partial substitution of mycorrhizal fungi for phosphorus fertilization in the culture of citrus. Soil Science Society of America Journal 42:926-930.

MEYER, W.S., and RITCHIE, J.T. 1980. Resistance to water flow in the sorghum plant. Plant Physiology 65:33-39.

MONTEITH, J.L. 1977. Climate and the efficiency of crop production in Britain. Philosophical Transactions of the Royal Society of London, Series B, 281:277-294.

NORCIO, N.V. 1976. Effect of high temperature and water stress on photosynthesis and respiration rates of grain sorghum. Ph.D. thesis, University of Nebraska, Lincoln, Nebraska, USA.

ODVODY, G.N., and DUNKLE, L.D. 1979. Charcoal stalk rot of sorghum: effect of environment on host-parasite relations. Phytopathology 69:250-254.

PEACOCK, J.M. 1979. The effect of water on the growth, development and yield of sorghum *(Sorghum bicolor)* cv. RS610. Pages 29-52 *in* Dryland Farming Research Scheme (DLFRS) Botswana, Final Scientific Report, Phase II, Vol. II: Sorghum Physiology and Crop Climate Studies. Gaborone, Botswana: Ministry of Agriculture, Division of Agricultural Research.

PEACOCK, J.M. 1982. Response and tolerance of sorghum to temperature stress. Pages 143-159 *in* Sorghum in the Eighties: Proceedings of the International Symposium on Sorghum, sponsored by INTSORMIL, ICAR, and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

QUINBY, J.R., HESKETH, J.D., and VOIGT, R.L. 1973. Influence of temperature and photoperiod on floral initiation and leaf number in sorghum. Crop Science 13:243-246.

REICOSKY, D.C., and RITCHIE, J.T. 1976. Relative importance of soil resistance and plant resistance in root water absorption. Soil Science Society of America Proceedings 40:293-297.

ROSENOW, D.T., QUISENBERRY, J.E., WENDT, C.W., and CLARK, L.E. 1983. Drought tolerant sorghum and cotton germplasm. Agricultural Water Management 7:207-222.

SAFIR, G.R., BOYER, J.S., and GERDEMANN, J.W. 1972. Nutrient status and mycrorrhizal enhancement of water transport in soybean. Plant Physiology 49:700-703.

SCHNEIDER, R.W., and PENDERY, W.E. 1983. Stalk rot of corn: Mechanisms of predisposition by an early season water stress. Phytopathology 73:863-871.

SCHOENEWEISS, D.F. 1978. Water stress as a predisposing factor in plant disease. Pages 61 -99 *in* Vol.5, Water deficit and plant growth (ed. T.T. Kozlowski). New York, New York, USA: Academic Press. 323 pp.

SEETHARAMA, N., SUBBA REDDY, B.V., PEACOCK, J.M., and BIDINGER, F.R. 1982. Sorghum improvement for drought resistance. Pages 317-338 *in* Drought resistance in crops with emphasis on rice. Los Banos, Laguna, Philippines: International Rice Research Institute.

SHOKES, F.M., LYDA, S.D., and JORDAN, W.R. 1977. Effect of water potential on the growth and survival of *Macrophomina phaseolina.* Phytopathology 67:239-241.

SIEVERDING, E. 1981. Influence of soil water regimes on VA mycorrhizae. Zeitschrift fur Acker-und Pflanzenbau 150:400-411.

SIMPSON, G.M. 1981. Water stress on plants. New York, New York, USA: Prager Press. 324 pp.

SMITH, W.H. 1969. Germination of *Macrophomina phaseoli* sclerotia as affected by *Pinus lambertiana* root exudate. Canadian Journal of Microbiology 15:1387-1391.

STOUT, D.G., KANNANGARA, T., and SIMPSON, G.M. 1978. Drought resistance of *sorghum bicolor:* 2. Water stress effects on growth. Canadian Journal of Plant Science 58:525-533.

SULLIVAN, C.Y. 1972. Mechanisms of heat and drought resistance in grain sorghum and methods of measurement. Pages 247-263 *in* Sorghum in Seventies (eds. N.G.P. Rao and LR. House). New Delhi, India: Oxford and IBH Publishing Co.

SULLIVAN, C.Y., and ROSS, W.M. 1979. Selecting for drought and heat resistance in grain sorghum. Pages 263-281 *in* Stress physiology in crop plants (eds. H. Mussell and R.C. Staples). New York, New York, USA: Wiley-Interscience.

TAYLOR, H.M., and KLEPPER, B. 1974. Water relations of cotton: I. Root growth and water use as related to top growth and soil water content. Agronomy Journal 66:584-588.

TINKER, P.B. 1980. Role of rhizosphere microorganisms in phosphorus uptake by plants. Pages 617-654 *in* The role of phosphorus in agriculture (eds. F.E. Khasawnen, E.C. Sample, and E.J. Kamprath). Madison, Wisconsin, USA: American Society of Agronomy.

TINKER, P.B., and GILDON, A. 1983. Mycorrhizal fungi and ion uptake. Pages 21 -32 *in* Metals and micronutrients: uptake and utilization by plants (Phytochemical Society of Europe Symposium No. 21), (eds. D.A. Robb and W.S. Pierpoint). New York, New York, USA: Academic Press.

TURNER, N.C., and BURCH, G.T. 1983. The role of water in plants. Pages 73-126 *in* Crop water relations (eds. I.D. Teare and M.M. Peet). New York, New York, USA: John Wiley & Sons.

VANDERLIP, R.L. 1972. How a sorghum plant develops. Kansas Agricultural Experiment Station Cooperative Extension Circular 447. Manhattan, Kansas, USA: Kansas State University. 19 pp.

WILSON, J.H.H., and ALLISON, J.C. 1978. Effects of water stress on the growth of maize (*Zea mays* L). Rhodesian Journal of Agricultural Research 16:175-192.

WILSON, J.M., and GRIFFIN, D.M. 1975. Water potential and the respiration of microorganisms in the soil. Soil Biology and Biochemistry 7:199-204.

YARWOOD, C.E. 1976. Modification of the host responses—predisposition. Pages 701-718 *in* Encyclopedia of plant physiology, New series, Vol. 4 (eds. A. Pison and M.H. Zimmerman). Berlin, Federal Republic of Germany: Springer-Verlag.

Questions

Partridge:
In your conclusion you state that "stress triggers changes in host root resistance, allowing infection at an earlier stage of growth than expected...." Do you have or can you elucidate data to support this conclusion?

Jordan:
Reports by Schneider and Pendery [1983] with maize and Odvody and Dunkle [1979] present evidence that mild water stress during some period before anthesis promotes colonization of roots by organisms reported to cause root and stalk rots.

Partridge:
Do you have or can you elucidate data to support your conclusion that "disease spread is associated with an increasing demand for CHO...."

Jordan:
Disease spread (symptom expression in stalk) is associated with CHO deposition in grain. The assumption is that the presence of developing grain creates an increased demand for CHO, but in reality this may just be a competing sink. Disease spread into the stalk during grain fill of fertile, but not sterile isolines appears to be the best example of this presumed increased CHO demand. References are cited in the text.

Partridge:
Dr. Clark, have phytoalexins been demonstrated in sorghum, and if so, have they been shown to affect stalk rot?

Clark:
I saw no literature on phytoalexins in sorghum. However, my literature search was not directed to phytoalexins, but to nutrient elements.

The Association of Plant Senescence with Root and Stalk Diseases in Sorghum

R.R. Duncan*

Summary

Grain sorghum (Sorghum bicolor [L.] *Moench*) *generally undergoes sequential senescence in which the older leaves near the base of the stalk senesce and die first, followed by a wavelike progression of leaf senescence passing up the stem. Senescence is characterized by losses in chlorophyll, carbohydrates, protein, and dry weight, and can result in predisposition to attack by various pathogens. The interaction of environment, growth stage, and genetic potential determines the levels of endogenous growth regulators which govern senescence. Nonsenescent* (*indeterminate, "perennialistic," "stay-green"*) *sorghum genotypes maintain more green leaf area and higher stem carbohydrate contents than senescing gehotypes during late reproductive growth stages. Since the nonsenescent genotypes supposedly remain physiologically active during the late stages of growth, this characteristic probably contributes to the overall stress tolerance and disease resistance mechanisms in these plants. Environmental stress* (*drought, high temperature and humidity, and nutritional imbalances*) *can predispose sorghum to infection by stalk rot pathogens. Many of these organisms are weakly pathogenic or secondary invaders, while others* (*e.g.,* Colletotrichum *spp*) *damage healthy vigorous cell tissue. Stalk rotting frequently involves many organisms, and selection within genotypes for specific resistance mechanisms is often difficult. Concentration of research efforts to develop nonsenescing plants that are well buffered against environmental stresses will help to minimize the predisposing factors leading to stalk rot in sorghum.*

Sorghum is a versatile plant adaptable to a wide range of environmental conditions. Breeding efforts have centered on development of resistance to specific disease organisms in a particular environment. This paper will present the relationship between senescence/nonsenescence and potential root/stalk diseases in sorghum. The following topics will be discussed in detail: plant senescence; nonsenescence; host-parasite interactions, including predisposition and infection vs senescence; nonsenescence and potential stalk rot diseases in the southeastern USA; and future research priorities.

Plant Senescence

In the development of plants, senescence is a relatively gross change or series of changes, leading finally to the death of the plant. Comfort (1956) has described senescence as a decrease in viability with an increase in vulnerability. In plants these changes can be recognized as decreases in growth rates and vigor and increases in susceptibility to challenge by the environment (water or nutrient shortages) or by pathogens or physical disturbance (Leopold 1961). In senescing cells a gradual reduction in the capacity for protein syn-

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thesis occurs, coupled with degeneration of the endoplasmic reticulum and mitochondria as well as other organelles and membranes.

The rate at which these natural changes occur depends greatly on the species, the part of the plant involved, and the environmental conditions (photoperiod, temperature, soil fertility). Senescence may take place at about the same time in all parts of the plant (whole-plant senescence, which typically occurs following flowering and fruiting), or individual organs may senesce while the remainder of the plant retains vitality (leaf or fruit senescence).

Leaf senescence may be characterized by involvement of all the leaves at the same time (i.e., dropping of leaves in the fall), which is termed synchronous or simultaneous senescence. Or leaf senescence may pass up the stem in a "wave" (in which the older leaves at the more basal end of the stem become senescent and die first); this is known as sequential senescence. Senescence of fruits is a late stage in the ripening process, and does not generally begin until the developing seeds are fully formed (Leopold 1961; Phillips 1971, pp 116-124 and 158-159). Sorghum generally undergoes sequential leaf senescence, with the coleoptile and first few leaves senescing and dying during the vegetative stage and additional sequentially formed leaves continuing this senescence and death pattern throughout the reproductive growth stage. During the late reproductive stages, the tip of the panicle begins senescing downward. This senescence pattern continues into the peduncle and stem, moving toward the base of the stem. If environmental stress has not terminated the cells in the crown root region, basal buds will initiate and tillers will grow until stress (frost or water deficits, for example) or disease development kills them.

Senescence is clearly a distinct physiological and biochemical phase in plant development. A thorough review of plant senescence in general is presented by Thimann (1980). Its timing is controlled by both internal and external factors. Models for transcriptional and translational control of senescence have been summarized by Hoffman (1972). A good general review of the biochemistry of senescence has been presented by Varner (1961). In general senescence is characterized by losses of RNA, DNA, total nitrogen, chlorophyll, protein, and dry weight (Hoffman 1972, Potter 1971, Spencer and Titus 1972). These changes can be retarded by the addition of certain plant growth regulators.

Extensive research has been conducted concerning hormonal regulation of plant senescence (Carr and Pate 1967, Fletcher and Adedipe 1972, Fletcher and Osborne 1965, Garg and Kapoor 1972, Leopold 1961, Nichols 1973, Wareing and Seth 1967, and Woolhouse 1967). Fletcher and Adedipe (1972) have proposed a model for hormonal regulation of leaf senescence. They postulated that the interplay of environment, time, and genetic potential determines the levels of endogenous growth regulators. These regulators were classified into two broad groups: (a) senescence retardants, which include cytokinins, gibberellins, auxins, and ascorbic acid (Garg and Kapoor 1972); and (b) senescence accelerators, which include abscisic acid, ethylene, and other, unidentified "senescence factors." The plant species and age govern the effectiveness of any one of the senescence retardants in delaying senescence. A decline in the levels of senescence retardants and/or a subsequent rise in those of senescence accelerators leads to senescence. The role of senescence retardants is to maintain the synthesis of macromolecules such as chlorophyll, protein, and nucleic acids. This maintenance confers structural integrity and proper partitioning of cell metabolites. This is associated with a high photosynthetic rate and an increased turnover of metabolites, accompanied by an orderly metabolic coordination between energy production and utilization for biosynthetic and growth processes. Conversely, a rise in the levels of senescence accelerators and/or a decline in senescence retardants results in a decrease in the synthesis or an increase in the degradation of macromolecules. These changes, accompanied by a loss of membrane integrity, could lead to senescence and ultimately death. The numerous metabolic changes associated with senescence may be delayed by the exogenous application of hormones.

Secor et al. (1983) concluded that changes in ribulose-1, 5-biphosphate carboxylase activity and chlorophyll content were related to the onset of senescence in soybean (*Glycine max* [L] Merr.) leaves and that a similar mechanism seems to be operating in leaves that emerge after flowering. Trzebinski et al. (1972) demonstrated a positive correlation between the depression of sugar yield per plant attributable to virus yellows and mosaic infection and the decrease in chlorophyll (particularly chlorophyll b) content of sugar beet (*Beta vulgaris* L.) leaves.

Harada and Nakayama (1971) have suggested that dwarfness in rice (*Oryza sativa* L.) may be

related to leaf senescence. Short rice varieties had more chlorophyll per unit leaf area and slow chlorophyll degradation, while tall ones showed less chlorophyll content and rapid degradation. They concluded that close relationships exist among the chlorophyll content per unit leaf area, the leaf s degradation, and the plant type.

The removal of flowers or of developing fruits has been observed to delay senescence in a number of annual plants (Leopold 1961). Moss (1962) found that the leaves of maize (*Zea mays* L) plants on which pollination had been prevented were still green towards the end of the season and had a higher rate of assimilation than the leaves of normal plants, which were by then senescent. Prevention of pollination increased the sugar concentration in the stalks. Allison and Weinmann (1970) studied the relationship between carbohydrate content and senescence of maize leaves. They concluded that abnormal concentrations of nonstructural carbohydrates (resulting from pollination prevention or ear removal) might interfere with some of the leaf functions and possibly lead to premature senescence in maize. Mortimore and Ward (1964) showed that high levels of soluble sugars in maize stems at physiological maturity were associated with lodging resistance. Papers by McBee and Maranville and Clegg (these proceedings) provide more detailed explanations of carbohydrate metabolism and stalk strength, respectively.

Lewis (1953) proposed that host-parasite relations are governed by a combination of the biochemistry of the host and the nutritional requirements of the parasite. Since nutrients in the host at specific metabolic concentrations sometimes inhibit the parasite, a certain nutrient profile may be a necessary prerequisite for infection. Schoeneweiss (1975) determined that generally excess nitrogen favored infection, excess potassium reduced infection, and excess phosphorus gave variable results in host-parasite relationships.

Jyung (1975) found a close relationship between zinc nutrition and leaf senescence as suggested by a rapid decline in total chlorophyll and chlorophyll a:b in zinc-deficient leaf discs during aging. As a leaf grows older, its photosynthetic apparatus appears to become markedly less effective, as indicated by lower photosynthetic rates and depressed net. assimilation rates (Leopold 1961). Critical changes occur in the metabolism of leaf carbohydrates and proteins, as well as in respiratory activities. As a result, the chlorophyll pigments deteriorate in favor of the carotenoids and anthocyanins. As these metabolic shifts take place, there occurs a gross export of many of the organic and inorganic nutrients from the leaf, until abscission interrupts such traffic.

Colhoun (1973) stated that interactions among environmental factors and nutrition create problems in the interpretation of the predisposing effects of nutrient stresses on plant defense mechanisms. Huber (1978) has written an excellent paper on mineral nutrition, nutrient imbalances, and disease incidence in plants. Refer to "Disease Development and Mineral Nutrition" in the paper by W. Jordan et al. (these proceedings) for a more comprehensive evaluation of the relationships involving mineral element stress and stalk/root rots in plants.

Nonsenescence

Standard sorghum genotypes undergo a sequential leaf senescence in which the older leaves at the base of the stalk senesce and die first. Under environmental stress conditions, a pattern of leaf senescence and death progresses in wavelike fashion up the stem, culminating in overall plant death after reaching physiological maturity (maximum grain dry weight accumulation). In nonstress situations, the plant will senesce most of its leaves, but the basal and axillary buds are initiated and tillers will grow until frost or severe stress conditions terminate them. The phenomenon of green leaf retention after the grain has reached physiological maturity has been termed nonsenescence (Duncan 1977).

Senescent (BTx378, RTx7000, RTx2536, S.C0214[IS-1 598C]) and nonsenescent (SC0056[IS-12568], SC0599 [Rio derivative], SC0170[IS-12661]) genotypes were evaluated during the reproductive stage of development (Duncan et al. 1981). The nonsenescent (indeterminate, "perennialistic," "stay-green") genotype required 2 days longer to reach 50% anthesis, averaged 3 to 4 cm less height, produced 2 to 3 more basal tillers per plant, averaged 10% larger stem diameters, maintained 53% higher basal stem sugar concentrations, and produced 11% higher leaf blade chlorophyll contents than did the senescent genotype. Data involving leaves (green leaf number and weight, senesced leaf number and weight, leaf area index, leaf area duration [average leaf area index between two sampling periods as a

function of time], and leaf area ratio [green leaf blade area in relation to the weight of the total above-ground dry matter]) favored the nonsenescent genotype. Since the nonsenescing genotypes contained a higher chlorophyll content for a longer period of time, a 20% larger leaf area index, and a 23% longer leaf area duration than the senescent genotypes, the authors speculated that the nonsenescing lines possessed a greater photosynthetic capacity and possibly greater crop productivity. Leaf area duration was found to be a good indicator of leaf senescence on the sorghum plant. This parameter reflected the ability of the plant to maintain green leaf area on a given unit of land throughout the life of the crop. Consequently, it revealed not only the growth in leaf area during the vegetative stages, but also the maintenance of green (and supposedly physiologically active) leaf area over time during the reproductive stages of development. Since the nonsenescent genotypes remain physiologically active during the late stages of growth, this characteristic may also contribute to the overall disease resistance and stress tolerance mechanisms in these plants. The nonsenescent genotype SC0599 (Rio derivative) has shown very good dual resistance to *Fusarium* spp and *Colletotrichum* spp (Duncan and Sowell 1983).

A comparison of rooting patterns of senescent and nonsenescent sorghum genotypes revealed that the nonsenescent hybrid established its adventitious root system earlier than did the senescent hybrid (Zartman and Woyewodzic 1979). Root density of both hybrids increased until grain filling, with the senescent cultivars generally exhibiting greater root density. The root density of both hybrids decreased after grain filling, with a greater decrease noted for the senescent hybrid. The senescent hybrid also produced a lower grain yield.

McBee et al. (1983) studied the effect of senescence and nonsenescence on carbohydrates in sorghum during late stages of reproductive development. They found that stalk carbohydrates were significantly higher in nonsenescent cultivars during all stages of seed development (grain filling) than in senescent cultivars. Cultivars with the higher stalk carbohydrates produced panicles with significantly more grain weight. Duncan et al. (1981) found 10% higher test weights on grain harvested from nonsenescing genotypes than from senescing types. Refer to the paper by G.G. McBee (these proceedings) for a more comprehensive explanation of carbohydrates and carbohydrate metabolism in sorghum.

Host-Parasite Interactions

Predisposition

Schoeneweiss (1975) has defined predisposition as the tendency of nongenetic factors, acting prior to infection, to affect the susceptibility of plants to disease. He proposed that root- and stem-rotting organisms enter resistant and susceptible hosts with equal frequency in most cases. Whether a disease condition actually develops depends upon the influence of environmental factors on the genetically controlled response of the host plant to the presence of the pathogen or its metabolites, i.e., a host-environment interaction.

Dodd (1977) has proposed the photosynthetic stress-translocation balance concept as being important in maize stalk rots. Several stress-related factors can predispose plants to invasion by certain stalk rot organisms, which could lead to pathogenicity (Dodd 1980, Schoeneweiss 1975): hormonal imbalances, nutritional imbalances, environmental extremes, photosynthetic stress, injury, carbohydrate nutrition, and cellular senescence. Refer to the paper by G.G. McBee (these proceedings) for a more detailed explanation of these factors as they relate to senescence.

Infection Versus Senescence

Mortimore and Ward (1964) stated:

> Although rotting organisms may invade the root at a relatively early stage of plant growth, root and stalk rot is essentially a disease related to the onset of senescence and produces no visible symptoms until after the plant has reached physiological maturity. It is premature degradation of the stalk produced by a range of common decay organisms and appears to be essentially a physiological phenomenon. Resistance appears to depend upon the maintenance of a certain degree of physiological vigor, particularly in the stalks during the long post-maturity period in the autumn when the corn plant is left standing in the field until the moisture in the grain is reduced to the desired level. Conversely, susceptibility is due to a lack of vigor as the plant matures.
>
> Physiological vigor depends upon a steady respiration rate supported by a continuous supply of carbohydrate reserves as

well as an adequate and balanced level of nutrients. When the vigor of the plant drops below a certain level because of stress conditions, it becomes susceptible to invasion by certain saprophytes and weak parasites.

Farkas (1978) has presented a general discussion of senescence and plant disease, including separation of disease-induced senescence and senescence-induced diseases. He concluded that not everything that is stress related will necessarily lead to pathological problems in plants.

Mortimore and Ward (1964) determined that high levels of soluble sugars in the pith of corn (maize) stalks at physiological maturity are associated with resistance to root and stalk rots. A resistant hybrid had a higher sugar content than a susceptible hybrid when grown using recommended cultural practices. Treatments that predisposed plants to stalk rots (high population densities and late defoliation) caused a reduction in sugars. Treatments that gave "normal" plant resistance reactions (prevention of kernel development and low population densities) resulted in maintenance or increase of sugars in the pith. Craig and Hooker (1961) theorized that a decrease in the sugar levels of maize stalks caused senescence of pith tissue and enhanced susceptibility to diplodia stalk rot.

Lewis and Deacon (1982) found that natural senescence of the coleoptiles and root cortices of wheat (*Triticum aestivum* L.) and barley (*Hordeum vulgare* L) might increase the establishment of infection by the eyespot fungus (*Pseudocercosporella herpotrichoides* (Fron) Dei) either directly or through the activities of competing microorganisms that act as biological control agents. They proposed that shading decreases the rate of root production, which, in turn, reduces the plant's capacity to offset pathological infection of the existing roots by producing new ones. Deacon and Lewis (1982) concluded that *Cochliobolus sativus* (Ito & Kuribay) Drechsler ex Dastur and other weak parasites benefit from the early natural senescence of wheat root cortex, and that the degree of susceptibility or resistance to common root rot is at least partially determined by differences in cortical senescence.

Hodges and Madsen (1978) established that *Drechslera sorokiniana* (Sacc.) Subram. and Jain (= *Helminthosporium sorokinianum* Sacc. in Sorok.) and *Curvularia geniculata* (Tr. & Earle) Boed. may interact competitively or synergistically to decrease or increase the severity of leaf spot expression on leaves of *Poa pratensis* L. They found that C. *geniculata* grew at temperatures ($\geq$ 30°C) that were high enough to physiologically stress *P. pratensis.* The stress created leaf tissue conditions that were especially susceptible to infection and proliferation by a weak, primary pathogen with aggressive saprophytic capabilities at high temperatures. Hodges and Madsen (1979) subsequently concluded that the synergistic increase in disease produced by a combination of *D. sorokiniana* and *C. geniculata* at high temperatures was the result of increased high-temperature growth of the latter organism, which was probably more saprophytic than parasitic on senescing, heat-stressed leaves and did not reflect a high-temperature increase in virulence of this organism.

Katsanos and Pappelis (1965) and Pappelis and Katsanos (1966) conducted research on senescence of sorghum stalk tissue. In an attempt to understand the role of living cells in the resistance to entrance of pathogens into stalks under natural conditions, they tested the hypothesis that living cells of sorghum are involved in resistance to spread of several sorghum stalk rot pathogens. The plants were subjected to root injury, leaf injury, and panicle removal. The effects of root and leaf injury increased the rate of cell death in internode and nodal tissue, whereas the panicle-removal treatment resulted in a decrease in the rate of cell death. They speculated that if living cells were necessary in the resistance mechanism, root- and leaf-injured plants would be more susceptible than "normal" plants, whereas panicle removal would result in plants more resistant than "normal" plants.

Gourley et al. (1977) studied the effect of *Fusarium moniliforme* Sheldon on seedling development of sorghum cultivars. The fungus generally invades the seedling through insect or mechanical injuries, or through roots or stems weakened by other factors, and results in blight or damping off of susceptible cultivars. The organism and its toxin reduced primary root length by 53%, number of lateral roots by 25%, and epicotyl length by 32% in comparisons of inoculated and uninoculated juvenile plants.

Trimboli and Burgess (1983) found that typical symptoms of basal stalk rot and root rot of sorghum were reproduced in the greenhouse on plants that were grown under optimal soil moisture until flowering, then subjected to severe moisture stress, followed by rewetting. Stalk rot did not develop in plants grown under optimal conditions from planting to maturity, although many of the plants were

infected and partially colonized by *F. moniliforme.* The researchers determined that early infection and establishment of the fungus in the plant may not be essential for stalk rot development and that microconidia that formed on necrotic coleoptile residue and on the soil surface may have been the airborne inoculum source.

Reed et al. (1983) investigated the fungal colonization of stalks and roots of sorghum during the growing season. Isolation frequency patterns for several organisms are presented in Tables 1 and 2. *F. moniliforme* was the dominant species isolated from stalks, while F. *eguiseti* (Cda.) Sacc. was most frequently isolated from the roots. The colonization of stalk tissue appeared to coincide with the onset of anthesis, but roots were apparently inhabited by fungi regardless of the growth stage. Fungi became more abundant in both stalks and roots as the crop matured. The researchers proposed that a balance exists between fungal activity within plant tissue and the ability of the host to withstand such activity. The balance is then shifted by factors that adversely affect the host or by conditions that favor increased fungal activity. The fungi become destructive to stalk tissue and lead to the development of stalk rot.

Odvody and Dunkle (1979) investigated the effect of environment on host-parasite relations involving sorghum and *Macrophomina phaseolina* (Tassi) Goid. Charcoal rot occurred when sorghum plants (senescent genotypes) were subjected to drought stress as grain development approached the soft dough stage. High soil temperatures and low soil moisture caused a significant reduction in total stalk sugars, which correlated with increased development of charcoal rot. Nonfertilized male-sterile sorghum plants subjected to drought stress were less susceptible to charcoal rot than fertilized lines. Root systems of the stressed male-sterile plants were characterized by a high percentage of roots with latent infections, but no symptoms of rot. Most root infections of both fertile and male-sterile sorghums occurred only after the onset of stress conditions. The researchers also demonstrated that nutrients increased sclerotial germination and mycelial growth at low osmotic water potentials, which suggests a role for nutrients in overcoming soil fungistatic factors and promoting infection by *M. phaseolina.*

Doupnik et al. (1975) and Doupnik and Boosalis (1980) investigated the influence of tillage systems on stalk rot incidence in sorghum. *F. moniliforme* was the major pathogen that caused stalk rot in the sorghum. No-till plots had 72% less stalk rot incidence than conventionally tilled plots. Sorghum grain yields were 41 % higher on the no-till than on the conventional-till plots. The researchers speculated that the lower incidence of stalk rot in the no-tillage system was due to increased water conservation, reduced soil temperature fluctuations,

Table 1. Pattern of sorghum stalk colonization by various organisms. (Source: Reed et al. 1983.)

Organism	Isolation frequency		
	Vegetative growth	Reproductive growth	Post-physiological maturity
Fusarium moniliforme Sheldon	Low	High-A	Low
F. graminearum Schwabe	Low	Low	High-KF
F. "roseum" group [a]	Low	Low	High-KF
F. equiseti (Gda.) Sacc.	Low	Low	High
F. tricinctum (Cda.) Sacc.	Low	Low	High-KF
F. oxysporum Schlecht. emend. Snyd. & Hans. 'Redolens'	Low	Low	High
F. solani (Mart) Appel & Wr. emend. Snyd. & Hans.	Low	Low	High
Altemaria alternata (Fr.) Keissler	Low	High-A	Variable
Nigrospora sphaerica (Sacc.) Mason	Low	High-SD to PM	High
Trichoderma viride Pers. ex Gray	Low	Low	Low
Epicoccum spp	Low	Low	Low

a. *F. scirpi* Lambotte & Fautr. var. *acuminatum* (Ell. & Ev.) Wr.
F. avenaceum (Fr.) Sacc.
F. culmorum (W. G. Sm.) Sacc.
F.sambucinum Fckl.

PM - physiological maturity
SD = soft dough
KF = high incidence of infection after killing frost
A = anthesis

Table 2. Pattern of sorghum root colonization by various organisms. (Source: Reed et at 1983.)

Organism	Isolation frequency		
	Vegetative growth	Reproductive growth	Post-physiological maturity
Fusarium equiseti (Cda.) Sacc.	High	High	High
F. oxysporum Schlecht. emend. Snyd. & Hans. 'Redolens'	High	Low	Very low
F. solani (Mart.) Appel & Wr. emend.Snyd. & Hans.	High	Low	Very low
F. moniliforme Sheldon	Very low	High-BS to A	Medium
F. graminearum Schwabe	Low	High-SD	High
F. "roseum" group [a]	Very low	High-SD	High
F. tricinctum (Cda.) Sacc.	Very low	Medium	High
Alternaria alternata (Fr.) Keissler	Medium	High-BS to A	Medium
Trichoderma viride Pers. ex Gray	High	High	Medium
Nigrospora sphaerica (Sacc.) Mason	Very low	Very low	Low
Epicoccum spp	Very low	Very low	High-PM

a. *F. scirpi* Lambotte & Fautr. var. *acuminatum* (Ell. & Ev.) Wr.
F. avenaceum (Fr.) Sacc.
F. culmorum (W. G. Sm.) Sacc.
F. sambucinum Fckl.

BS = boot stage
A = anthesis
SD = soft dough
PPM = physiological maturity

and lower mean soil temperatures. Consequently, predisposition to stalk rot was minimized or delayed in a senescent cultivar by a cultural method-reduced tillage.

Nonsenescence and Potential Stalk Rot Diseases in the Southeastern USA

Duncan and Sowell (1983) have studied the relationship between anthracnose (*Colletotrichum graminicola* (Ces.) Wils.) and the *Fusarium* complex in causing disease problems on sorghum in the humid southeastern USA. These two organisms worked in tandem to reduce yield and cause serious disease problems in a breeding program in Georgia (USA) involving susceptible genotypes (Table 3). Other stalk rot organisms are potential threats to sorghum production in the Southeast. Charcoal rot generally occurs under conventional tillage systems without irrigation. Sorghum grown in no-tillage systems tends to have few or no problems with *Macrophomina* spp. Pythium stalk rot has been found in the Southeast in isolated fields and has occurred following rotation after tobacco (*Nicotiana tobaccum* L.) in the Coastal Plain region. Additional research indicates a possible association between winter cover crops (particularly annual legumes) and the escalation of disease problems with *Pythium* spp and other stalk rot organisms in conservation tillage production systems. Current research is focusing on this association.*Alternaria* spp and *Epicoccum* spp are generally secondary invaders of stalks and are apparently not competitive with the more dominant *Fusarium, Colletotrichum,* and *Macrophomina* stalk rot organisms in this region of the USA.

Table 3. Potential damage from sorghum root/stalk diseases and field grain deterioration in Georgia, USA.

Organism	Level of damage [a]	
	Plants [b]	Grain
Fusarium moniliforme Sheldon	4-5	5
F. "roseum" group (Toussoun & Nelson)	3	3
F. semitectum Berk & Rav.	3	3
Cofletotrichum graminicola (Ces.) Wils.	4-5	1-2
Macrophomina phaseolina (Tassi) Goid.	3	0
Pythium spp	2	0
Altemaria spp	1	4
Phoma spp	0	2-3
Epicoccum spp	1	2
Curvularia spp	0	3-4
Helminthosporium spp	0	3-4
Periconia circinata (Mangin) Sacc.	1	0
Bacteria	1-2	0

a. 1 = very little damage; 5=significant damage; 0 = no data available.
b. Plants at reproductive stage.

Host-environment interactions are key factors in the degree of damage caused by *Fusarium* and

Table 4. Pathogens isolated from field-grown seed in Georgia, USA, during 1982.

Pedigree	Pathogens [a]			Germination (%)
	Fusarium	*Colletotrichum*	*Curvularia*	
Martin	8.75	35.50	2.50	45.0
RCY ORO TXTRA	10.00	21.75	700	71.0
(SC599 x SC110)	10.50	25.00	7.75	87.0
BSC599-6	2.25	2.00	13.00	94.0
Funks G-522DR	14.75	10.00	0.00	38.0
RTx430	35.00	0.75	0.25	16.5

a. Mean number of colonies (4 replications).

Colletotrichum on sorghum. Evaluation of seed collected from the field after physiological grain maturity revealed that *Fusarium* spp and *Colletotrichum* spp were dominant genera in reducing germination (Table 4) of breeder seed, BSC599-6(a nonsenescent genotype) was the only cultivar that had a good level of resistance to both pathogens (Tables 4 and 5). Inoculation of the *Fusarium* susceptible, senescent genotype RTx2536 with the three *Fusarium* species that have been isolated in Georgia revealed substantial seed germination reductions (Table 6). *F. moniliforme* reduced germination threefold over the check when the plants were inoculated during the soft dough stage of seed development. *F. roseum* and *F. semitectum* decreased germination approximately twofold when the plants were inoculated during the hard dough stage.

Plant genetics, environmental stress, and senescence interact in conjunction with the pathogens to affect the growth and development of the plant. Sorghum yield losses from *Colletotrichum* spp in Georgia have been reported as high as 50% (Harris et al. 1964), while fusarium stalk rot and head blight have caused 40% yield reductions in the Jalisco region of Mexico (Sanchez and Betancourt 1983). Edmunds and Zummo (1975) have indicated that *Fusarium* spp can reduce sorghum yields up to 60%.

Table 5. The influence of *Fusarium* spp. on germination of field-grown sorghum seed in Georgia, USA, during 1982.

Pedigree	Colonies [a] (mean no.)	Germinatior (mean %)
BSC599-6	2.25	94.0
TP9R02-16	6.75	89.5
(SC599 x SC110)	10.50	87.0
B2219	14.25	70.0

a. Fifty seed samples.

Table 6. The influence of selected *Fusarium* spp on seed germination of RTx2536 inoculated during reproductive growth.

Treatment	Germination (%)
Check	92
Check (atomized with water)	83
Fusarium moniliforme Sheldon [a]	30
F. "roseum" group (Toussoun & Nelson)[b]	52
F. semitectum Berk & Rav. [b]	61

a. Treated during soft dough stage.
b. Treated during hard dough stage.

Future Research Priorities

Environmental stress factors can predispose sorghum plants to infection by a number of pathogens commonly known as stalk rot pathogens. The universal occurrence of many synergistically reactive microorganisms generally leads to multiple-organism involvement in stalk rotting (Turner 1982), Since many of the pathogens have a relatively wide range of host plants and characteristically may be normally weakly parasitic or secondary invaders, selection within genotypes of a plant species may not be very productive in locating unique genes for specific resistance (Van der Plank 1968).

Research efforts may need to be concentrated on selection of plants capable of withstanding pre-

disposing environmental stresses in an effort to maintain plant health longer (Turner 1982). Susceptibility to stresses that may impose irreversible physical and/or chemical changes in the plant should be avoided. Resistance to specific stresses or tolerance to stresses (reversible physical or chemical changes when the stress is removed) should be emphasized. A plant that is well buffered against environmental stresses and that remains metabolically and physiologically active (nonsenescent, indeterminate) during late reproductive stages of development should be in a better position to withstand attack by roots and stalk-rotting organisms. Improvements in genetic resistance/tolerance mechanisms, especially on the multiple gene level, will help to stabilize plant performance. Several areas of research should be emphasized during the next decade:

1. Nonsenescence characteristics vs disease population dynamics.
   a. Investigation of metabolic activity, nutritional profile, and associated internal plant factors that suppress disease buildup.
   b. Study of root system dynamics vs disease organism entry and determination of the "triggers" for disease enhancement within the plant.
   c. Evaluation of carbohydrate balance in roots and stems vs stalk disease relationships in senescing and nonsenescing genotypes.
2. Multiple gene resistance/tolerance to disease pathogens.
   a. Identification of genes and their transfer, and inheritance studies involving senescing and nonsenescing genotypes.
   b. Continued monitoring of the root and stalk-rotting organisms via international disease screening nurseries. Inclusion of senescing and nonsenescing genotypes in the nursery.

References

ALLISON. J.C.S.. and WEINMANN, H. 1970. Effect of absence of developing grain on carbohydrate content and senescence of maize leaves. Plant Physiology 46:435-436.

CARR, D.J., and PATE, J.S. 1967. Ageing in the whole plant. Pages 559-599 *in* Symposia of the Society for Experimental Biology XXI: Aspects of the biology of ageing. London, U.K.: Cambridge University Press.

COLHOUN, J. 1973. Effects of environmental factors on plant disease. Annual Review of Phytopathology 11:343-364.

COMFORT, A. 1956. The biology of senescence. New York. New York, USA: Rinehart. 414 pp.

CRAIG, J., and HOOKER, A.L. 1961. Relation of sugar trends and pith density to *Diplodia* stalk rot in dent com. Phytopathology 51:376-382.

DEACON, J.W., and LEWIS, S.J. 1982. Natural senescence of the root cortex of spring wheat in relation to susceptibility to common root rot (*Cochliobolus sativus)* and growth of a free-living nitrogen-fixing bacterium. Plant and Soil 66:13-20.

DODD, J.L. 1977. A photosynthetic stress-transiocation balance concept of corn stalk rot. Pages 122-130 *in* Proceedings of the 32nd Annual Corn and Sorghum Research Conference (eds. H.D. Loden and D. Wilkinson). Washington, D.C., USA: American Seed Trade Association.

DODD, J.L. 1980. The role of plant stresses in development of corn stalk rots. Plant Disease 64:533-537.

DOUPNIK, B., Jr., and BOOSALIS, M.G. 1980. Ecofallow—a reduced tillage system—and plant diseases. Plant Disease 64:31 -35.

DOUPNIK, B., Jr., BOOSALIS, M.G., WICKS, G.A., and SMIKA, D. 1975. Ecofallow reduces stalk rot in grain sorghum, Phytopathology 65:1021 -1022.

DUNCAN, R.R. 1977. Characteristics and inheritance of nonsenescence in *Sorghum bicoior* (L.) Moench. Ph.D. thesis, Texas A&M University, College Station, Texas, USA. 70 pp.

DUNCAN, R.R., BOCKHOLT, A.J., and MILLER, F.R. 1981. Descriptive comparison of senescent and nonsenescent sorghum genotypes. Agronomy Journal 73:849-853.

DUNCAN, R.R.,and SOWELL,G. 1983. The tandem association between anthracnose and the fusarium complex in sorghum grown under Georgia environmental conditions. Page 74 *in* Proceedings of the 13th Biennial Grain Sorghum Research and Utilization Conference, sponsored by Grain Sorghum Producers' Association (GSPA) and Sorghum Improvement Conference of North America. Available from GSPA, Abernathy, Texas, USA.

EDMUNDS, L.K., and ZUMMO, N. 1975. Sorghum diseases in the United States and their control. U.S. Department of Agriculture Handbook No. 468. Washington, D.C., USA: U.S. Government Printing Office, 47 pp.

FARKAS, G.L. 1978. Senescence and plant disease. Pages 391-412 *in* Plant disease, an advanced treatise,

Vol.3: How plants suffer from disease (eds. J.G. Horsfall and E.B. Cowling). New York, New York, USA: Academic Press.

FLETCHER, R.A., and ADEDIPE, N.O. 1972. Hormonal regulation of leaf senescence in intact plants. Pages 571 - 580 *in* Plant growth substances (ed. D.J. Carr). New York, New York, USA: Springer-Verlag.

FLETCHER, R.A., and OSBORNE, D.J. 1965. Regulation of protein and nucleic acid synthesis by gibberellin during leaf senescence. Nature 207:1176-1177.

GARG, O.P., and KAPOOR, V. 1972. Retardation of leaf senescence by ascorbic acid. Journal of Experimental Botany 23:699-703.

GOURLEY, L.M., ANDREWS, C.H., SINGLETON, L.L., and ARAUJO, L. 1977. Effects of *Fusarium moniliforme* on seedling development of sorghum cultivars. Plant Disease Reporter 61:616-618.

HARADA, J., and NAKAYAMA, H. 1971. Chlorophyll degradation in leaf sections from tall and short varieties of rice. Journal of Agricultural Science 76:573-574

HARRIS, H.B., JOHNSON, B.J., DOBSON, J.W., Jr., and LUTTRELL, E.S. 1964. Evaluation of anthracnose on grain sorghum. Crop Science 4:460-462.

HODGES, C.F., and MADSEN, J.P. 1978. The competitive and synergistic interactions of *Drechslera sorokiniana* and *Curvularia geniculata* on leaf spot development on Poa *prafensis*. Canadian Journal of Botany 56:1240-1247.

HODGES, C.F., and MADSEN, J.P. 1979. Leaf senescence as a factor in the competitive and synergistic interactions of *Drechslera sorokiniana* and *Curvularia geniculata* on *Poa pratensis.* Canadian Journal of Botany 57:1706-1711.

HOFFMAN, W.E. 1972. The relationship of amino acid pools, protein synthesis, proteinase activity, and tRNA methylase activity in senescence of corn tissue. Dissertation Abstracts 32:5016.

HUBER, D.M. 1978. Disturbed mineral nutrition. Pages 163-181 *in* Plant disease, an advanced treatise, Vol. 3: How plants suffer from disease (eds. J.G. Horsfall and E.B. Cowling). New York, New York, USA: Academic Press.

JYUNG, W.H. 1975. A similarity between zinc deficiency and senescence in navy bean leaves. Agronomy Abstracts 67:72.

KATSANOS, R.A., and PAPPELIS, A.J. 1965. Seasonal trends in density and cell death in sorghum stalk tissue. Phytopathology 55: 97-99.

LEOPOLD, A.C. 1961. Senescence in plant development. Science 134:1727-1732.

LEWIS, R.W. 1953. An outline of the balance hypothesis of parasitism. American Naturalist 87:273-281.

LEWIS, S.J., and DEACON, J.W. 1982. Effects of shading and powdery mildew infection on senescence of the root cortex and coleoptile of wheat and barley seedlings, and implications for root- and foot-rot fungi. Plant and Soil 69:401-411.

McBEE, G.G., WASKOM, R.M., III, MILLER, F.R., and CREELMAN, R.A. 1983. Effect of senescence and nonsenescence on carbohydrates in sorghum during late kernel maturity states. Crop Science 23:372-376.

MORTIMORE, C.G., and WARD, G.M. 1964. Root and stalk rot of corn in Southwestern Ontario, III: Sugar levels as a measure of plant vigor and resistance. Canadian Journal of Plant Science 44:451-457.

MOSS, D.N. 1962. Photosynthesis and barrenness. Crop Science 2:366-367.

NICHOLS, R. 1973. Senescence of the cut carnation flower: respiration and sugar status. Journal of Horticultural Science 48:111-121.

ODVODY, G.N., and DUNKLE, L.D. 1979. Charcoal stalk rot of sorghum: effect of environment on host-parasite relations. Phytopathology 69:250-254.

PAPPELIS, A.J., and KATSANOS, R.A. 1966. Effect of plant injury on senescence of sorghum stalk tissue. Phytopathology 56:295-297.

PHILLIPS, I.D.J. 1971. Introduction to the biochemistry and physiology of plant growth hormones. New York, New York, USA: McGraw Hill Book Co.

POTTER, J.R. 1971. The effect of senescence on protein synthesis and ribosomes in tobacco leaves. Dissertation Abstracts 31:5210-5211.

REED, J.E., PARTRIDGE, J.E., and NORDQUIST, P.T. 1983. Fungal colonization of stalks and roots of grain sorghum during the growing season. Plant Disease 67:417-420.

SANCHEZ, J.N., and BETANCOURT, A. 1983. Reaction of selected sorghum lines to fusarium stalk rot and head blight at El Bajio, Mexico. Pages 78-79 *in* Proceedings of the 13th Biennial Grain Sorghum Research and Utilization Conference, sponsored by Grain Sorghum Producers' Association (GSPA) and Sorghum Improvement Conference of North America. Available from GSPA, Abernathy, Texas, USA.

SCHOENEWEISS, D.F. 1975. Predisposition, stress, and plant disease. Annual Review of Phytopathology 13:193-211.

SECOR, J., SHIBLES, R.,and STEWART, C.R. 1983. Metabolic changes in senescing soybean leaves of similar plant ontogeny. Crop Science 23:106-110.

SPENCER, P.W., and TITUS, J.S. 1972. Biochemical and enzymatic changes in apple leaf tissue during autumnal senescence. Plant Physiology 49:746-750.

THIMANN, K.V. (ed.). 1980. Senescence in plants. Boca Raton, Florida, USA: CRC Press. 276 pp.

TRIMBOLI, D.S., and BURGESS, L.W. 1983. Reproduction of *Fusarium moniliforme* basal stalk rot and root rot of grain sorghum in the greenhouse. Plant Disease 67:891 - 894.

TRZEBINSKI. J., PAWELSKA-KOZINSKA, K., and WASAG, G. 1972. Studies on the correlation between changes in chlorophyll content of the leaves of sugar beet and tolerance of virus yellows. Plant Breeding Abstracts 42:No. 8500.

TURNER, M.T. 1982. An update on corn diseases and their pathogens. Pages 190-205 *in* Proceedings of the 37th Annual Corn and Sorghum Research Conference (eds. H.D. Loden and D. Wilkinson). Washington, D.C., USA: American Seed Trade Association.

VAN DER PLANK, J.E. 1968. Disease resistance in plants. New York, New York, USA: Academic Press. 206 pp.

VARNER, J.E. 1961. Biochemistry of senescence. Annual Review of Plant Physiology 12:245-264.

WAREING, P.F., and SETH, A.K. 1967. Ageing and senescence in the whole plant. Pages 543-558 *in* Symposia of the Society for Experimental Biology, XXI. Aspects of the biology of ageing. London, U.K.: Cambridge University Press.

WOOLHOUSE, H.W. 1967. The nature of senescence in plants. Pages 179-213 *in* Symposia of the Society for Experimental Biology XXI: Aspects of the biology of ageing. London, U.K.: Cambridge University Press.

ZARTMAN, R.E., and WOYEWODZIC, R.T. 1979. Root distribution patterns of two hybrid grain sorghums under field conditions. Agronomy Journal 71:325-328.

Questions

Scheuring:
How do you relate nonsenescence with juicy and dry-stem sorghums? If you admit that there exist dry-stem nonsenescent sorghums, how do you describe such plants, since the cortical stem cells are mostly senesced and dead? According to yours and forgoing discussions, such cells would be vulnerable to colonization by pathogens.

Duncan:
I really have not been too concerned with whether I have juicy- or dry-stemmed nonsenescing sorghums, and we have found both types. Rosenow's work with charcoal rot has shown basically no significant differences in disease ratings between the two types. How much actual difference in live and dead cortical stem cells exists between the two stem types in nonsenescing genotypes has not been researched to my knowledge. But that is not to say that differences do not exist and pathogen interactions should not be an important consideration.

Pande:
What is the environmental situation under which you have identified lines with the nonsenescent trait?

Duncan:
Normal field conditions in any breeding program, but the timing for making selections is at postphysiological grain maturity. If some stress is involved with senescence, then the plants may deteriorate and die faster than normal.

Pande:
Do these lines show consistency for nonsenescence over a period of time, location, and environment?

Duncan:
I have generally seen consistent reactions over locations and environments under southeast USA conditions; timing is influenced by interactions with environmental stresses and plant genetics.

Pande:
What scale do you use for quick estimation of nonsenescent lines and the lines that do not show green leaves but show only pale green juicy stems.

Duncan:
After working with the overall nonsenescence character for several years, I now use visual selection of nonsenescing plants and include leaves, stems, and panicle in the consideration process. Stems and panicle are sometimes split for visual observation—particularly where disease organisms (C. *graminicola* and *Fusarium* in my case) are involved.

Henzell:
You say that nonsenescence causes stress tolerance and relatively high carbohydrate levels in the stem. What evidence do you have for this cause-and-effect relationship, and would it not be more

likely that the reverse (i.e., stress tolerance causes nonsenescence) is the case?

Duncan:
I stated that since nonsenescent genotypes supposedly remain physiologically active during late stages of growth, this characteristic probably contributes to stress tolerance. I did not intend to say anything about a cause and effect relationship. The point was that an actively growing, physiologically active plant with perennialistic tendencies may be better buffered to withstand stresses such as drought or acid soils. The nonsenescent characteristic would only be a portion of the tolerance mechanisms. Jordan has conducted heat and desiccation tests between senescing and nonsenescing genotypes and found that nonsenescent types were more tolerant to these stresses. I am finding improved acid soil tolerance in nonsenescing genotypes, and it is probably tied in with a dynamic root system and efficiency in uptake of moisture and nutrients.

Partridge:
What did you mean by "postmaturity" relative to Reed et al.? In our research we assume this to be equal to postfrost kill and felt it to be entirely different from host-parasite interactions that existed in the living host.

Duncan:
Postphysiological maturity was indeed the postfrost period. I agree that the host-parasite interactions during this period are probably different than what occurs in the living host, but I was trying to portray the general pattern of disease development, contrasted to that which occurs after frost. The validity of the comparison could be extensively debated, but I still think that the trends are important.

Morphological and Physiological Factors Associated with Stalk Strength

J.W. Maranville and M.D. Clegg*

Summary

Substantial yield losses occur in lodged crops due to mechanical and physiological alterations. Morphological factors associated with stalk strength reside primarily in the rind. Stalk elasticity, breaking strength, and plant height are somewhat associated with lodging tendency, but stalk diameter, basal stalk weight, and rind thickness are highly associated. Rind penetrometer values and crushing strength best integrate the contribution of morphological factors to lodging.

Physiological factors contributing to stalk strength reside primarily in the pith. Factors that enhance plant vigor and well-being contribute to greater stalk strength. Depletion of basal stalk carbohydrates weakens the plant's ability to resist invasion by stalk rot organisms. The involvement of nitrogen metabolism and enzymes in the breakdown of parenchyma tissue is not well understood and needs further investigation.

Sorghum (*Sorghum bicolor* (L.) Moench) is an excellent example of the principle, "for a set of prescribed biological functions, an organism has the optimum possible design with respect to economy of material used and energy expenditure needed for the performance of the prescribed functions" (Rashevsky 1960). The sorghum stalk is tapered throughout its length to maintain strength in proportion to the load stress that generally is applied by forces of wind, passage of machinery, and other disturbances. Certain characteristics of the stalk determine the degree to which the natural taper is effective in resisting the load that tends to lodge it. These are morphological and physiological factors, many of which are inherent to the particular genotype. However, among the most important characteristics affecting stalk strength is the plant's ability to withstand, directly or indirectly, the invasion of stalk rot diseases. The following describes some of the plant characteristics most often associated with stalk strength, and discusses their involvement in resistance to lodging.

Lodging Effects on Yield and Quality

There is general agreement that yield losses from lodging are due primarily to a disruption in photosynthesis and translocation coupled with mechanical difficulties in harvesting the crop. Yield reductions are more severe if lodging occurs early. As much as 35% of the yield of winter wheat (*Triticum aestivum* L.) can be lost if lodging occurs either 1 to 2 weeks before heading or 1 to 2 weeks after heading. This loss is reduced to about 15% if lodging occurs at or just prior to heading (Laude and Pauli 1956). Weibel and Pendleton (1964) found progressively less yield reduction in wheat from heading to the hard dough stages, although up to 12% yield was still lost when lodging occurred at the hard dough stage. These trends have been substantiated in other crops such as maize (*Zea mays* L.) (Fisher and Smith 1960), barley (*Hordeum vulgare*) (Day 1957), oats (*Avena sativa*) (Pen-

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International Crops Research Institute for the Semi-Arid Tropics. 1984. Sorghum Root and Stalk Rots, aCritical Review: Proceedings of the Consultative Group Discussion on Research Needs and Strategies for Control of Sorghum Root and Stalk Rot Diseases, 27 Nov - 2 Dec 1983, Bellagio, Italy. Patancheru, A.P. 502 324, India: ICRISAT.

dleton 1954), and sorghum (Larson and Maranville 1977).

Studies with sorghum in which plants were artificially lodged indicated that the severity of lodging, as well as the time it occurred, is important to the magnitude of yield reduction (Larson and Maranville 1977). Yield was reduced 18% when plants were lodged at heading by manually forcing over and holding them in place, while breaking the stalk at the same growth stage resulted in a 31 % loss. Progressively less difference in yield reduction occurred between the two methods the later the treatments were imposed. Nonetheless, up to 13% yield reductions can occur in sorghum if lodging occurs due to stalk breakage as late as the soft dough stage, and reductions could reach as high as 30% at this growth stage due to disruption in metabolism alone. At the hard dough stage, yield losses ranged from 0% to 26%.

Alterations in grain protein content also occur in lodged grain crops. Generally, the protein concentration is increased (Laude and Pauli 1956, Esechie 1975, Larson and Maranville 1977), probably because of interference with carbohydrate assimilation. Protein in cereal grains originates largely from nitrogen, which is accumulated in the foliage prior to heading (Mulder 1954). High grain protein concentrations therefore arise from decreased carbohydrate deposition, which normally tends to dilute protein levels during the grain-fill period. Total protein on a per-area basis, however, is generally less in lodged portions of a field (Laude and Pauli 1956, Larson and Maranville 1977). This is, therefore, another critical loss that is of economic importance.

Morphological Factors and Stalk Strength

Elasticity of the stalk and its resultant breaking strength are two parameters that have been used to determine lodging characteristics of some crops. An instrument for testing the breaking strength of straw was used in very early research studies (Helmick 1915), and its use was later proposed as a means to form a lodging index (Salmon 1931). Varying results have been obtained using breaking strength as a parameter to judge lodging tendency. Davis and Stanton (1932) obtained relatively good agreement between standing ability in oats and breaking strength. Clark and Wilson (1933), however, found no correlation in several small grains, and McAuley (1973) found no correlation between breaking strength and lodging in sorghum. Breaking strength is apparently dependent on maturity and environmental conditions (Bartel 1937).

Suggs et al. (1962) were among the first to apply cantilever beam techniques to stalks in an attempt to characterize their elasticity. The modulus of elasticity in several sorghum types was determined using a two-point beam-loading technique (Bashford et al. 1976). This technique involved measurement of the force needed to bend the stalk a certain predetermined deflection distance. The modulus of elasticity was calculated from the bending moment equation derived from beam theory. This study found that stalk elasticity was not well associated with apparent standability in the field. When the stalk was deflected by the instrument until it failed (breakage) there was some correlation. Those sorghums classified as having a stiff stalk (more lodging-resistant) required more force to effect stalk failure. Plants acquired their maximum lodging resistance somewhere between the dough and physiological-maturity growth stages if disease was not prevalent. The stalk was shown to be strongest at the base, due, for the most part, to the contribution of the sheath. Esechie et al. (1977) reported that the weakest point on the sorghum stalk was the top internode immediately below the panicle. Other work, however, indicated that there was little difference in bending moment at stalk failure between the peduncle and the top node (Bashford et al. 1976).

Many morphological factors are associated with lodging tendency or resistance, with no single one being consistent enough to use in classifying plants in every situation. Plant height is an example of one characteristic related to lodging tendency. Sorghums prone to lodging tend to be taller than those that are lodging-resistant (Esechie et al. 1977, Bashford et al. 1976). Plant height is so subject to environmental factors, however, that its use as an index for isolating genotypes should be on a relative basis only. Any consideration of plant height as a factor in lodging must not overlook the effect of culm length x maturity interaction (Pinthus 1973). An early, short-stalk genotype close to maturity may be more prone to lodging than a late, long-stalk genotype that at the same time has only attained the heading or early dough stage.

The relationship between lodging and culm anatomy, particularly that of the basal internodes, has been extensively investigated. Larger basal stalk

diameters have been consistently correlated with lodging resistance in sorghum (Esechie et al. 1977), maize (Twumasi-Afriyie and Hunter 1982), and other small grains (Brady 1934, Hamilton 1941). This was also a characteristic of nonsenescent sorghums, which tend to resist lodging (Duncan et al. 1981). Similarly, the weight of a cut section of stalk can be correlated with lodging. Generally, a basal section is used, although other portions are satisfactory. Zuber and Grogan (1961) found a correlation of r = -0.73 between lodging and weight of a second internode of maize. Investigations with sorghum showed an r = -0.59 correlation between lodging and the weight of a basal intemode section, and r = -0.56 between lodging and the weight of the peduncle node. Stalk section weight was better correlated with rind thickness in many studies, whether rind thickness was determined mechanically (Twumasi-Afriyie and Hunter 1982) or by using the weight/circumference calculation (Esechie 1975, McAuley 1973).

Chang et al. (1976) concluded that stalk strength in maize resides principally in the rind tissue, and that rind characteristics are better indicators of stalk strength than those associated with the pith. This view is not shared by all researchers in the field. Nonetheless, the apparent importance of rind characteristics to lodging has led to the use of rind puncture methods to evaluate the lodging potential of plants. A penetrometer is often used, and the measure is determined as force in kilograms for rind penetration to occur (Thompson 1969). A correlation coefficient of r =-0.79 for rind puncture and lodging found by Twumasi-Afriyie and Hunter (1982) in maize is typical of the association generally shown. The method is useful in that it is nondestructive and obtained quickly at any plant growth stage.

Rind thickness, which undoubtedly influences or possibly accounts for rind penetration values, correlates well with plant lodging, but not as well as some other factors. Research in maize showed a correlation of r= -0.53 between lodging and rind thickness (Twumasi-Afriyie and Hunter 1982). Correlation of lodging with a rind thickness approximation used by Esechie et al. (1977) in sorghum was only slightly better (r = -0.61). The practical importance of rind thickness in maize was quantified by Thompson (1963). For every 1.56% increase in lodging due to increases in plant population, rind thickness decreased 0.02 mm. Indirectly selecting for lodging resistance in another maize study resulted in an increase in rind thickness of 0.09 mm after the fourth selection cycle (Zuber 1973).

A method for determining stalk strength that integrates several stalk characteristics was developed by Zuber and Grogan (1961). Five-cm stalk sections were crushed with a hydraulic press, and the force required was determined. This "crushing strength" method has been used extensively in many lodging studies and is highly correlated with anatomical characteristics associated with lodging scores, and therefore with lodging per se. The highest correlation appeared to be with stalk section weight (Zuber and Grogan 1961), although its association with rind thickness, rind weight, and even rind lignification was strong (Chang et al. 1976). The selection of genotypes of maize based solely on increased crushing strength resulted in a linear increase in stalk weight, rind thickness, rind puncture, decreased stalk lodging, and very importantly, improved diplodia stalk rot ratings (Zuber 1973).

An anatomical feature that appears to have a distinct relationship with lodging is the number of vascular bundles in the culm. A high number of vascular bundles has been found in strong stalks of cereals such as oats (Hamilton 1951) and maize (Chang et al. 1976). There appears to be a similar relationship in sorghum (Esechie and Maranville, unpublished data, 1977), although this was not confirmed with actual counts. Hamilton (1951) suggested that the number of bundles was directly associated with culm diameter, and therefore only indirectly with lodging tendency. Correlations of r = 0.77 between the number of vascular bundles and the stem diameter in maize have been reported (Martin and Hershey 1934). Positioning of the vascular bundles may be more important. If vascular bundles are considered as giving support to the stem, then bundles that are positioned close together and concentrated near the outside of the stem would give added strength. According to Hamilton (1951), who compared these factors in wheat and oats, denseness and outside positioning of vascular bundles were major factors in giving wheat superior lodging resistance. Chang et al. (1976) were able to increase the number of vascular bundles in maize by selection for increased crushing strength of the stalk, However, this selection criterion also increased stalk size and weight.

It would appear that several morphological characteristics contribute to stalk strength, many of which reside in the rind. Crushing strength seems to integrate these factors best, and this selection

criterion has been received favorably by researchers in the field. Interestingly, in one study, crushing strength of stalks gave a better measure of stalk rot response in infected plants than did rind thickness (Loesch et al. 1962). A selection index based on a measure of crushing strength in artificially infected plants might be useful in obtaining genotypes resistant to the weakening effects of stalk rot organisms, such as *Diplodia maydis* in maize.

Physiological Factors and Stalk Strength

Physiological factors that contribute to stalk strength are as numerous, or more numerous, than the morphological factors discussed in the previous section. These factors appear to reside more in the pith tissue than in the rind, but not exclusively so. Moreover, the physiological factors associated with stalk strength are those that have been associated with stalk rot resistance. Some factors, such as certain cellular constituents, appear to be associated with anatomical structure. However, there is mounting evidence that the physiological factors which make the greatest contribution to stalk strength do so by influencing, being directly responsible for, or being associated with the overall well-being of the plant. The following will deal briefly with anatomical influences, and then with the more controversial aspects of plant vigor.

One of the very early views was that the amount of silica in cells of the culm could determine the lodging tendency of cereals. Davidson and Phillips (1930), however, found more silica and ash in lodging-susceptible wheat than in wheat that was lodging-resistant. Hamilton (1941) concluded that silica offered no possibility as an indexto lodging in cereals.

A deficiency in lignin was considered to be the cause of lodging by some researchers (Welton and Morris 1931). They contended that lignin lent mechanical support to the stalks. Others had contrasting views (Davidson and Phillips 1930, Hamilton 1941); their work showed that high rather than low lignin contents were associated with lodging. The evidence was so strong in one instance that selecting for culms with low lignin was proposed as being a useful index in breeding for lodging resistance (Hamilton 1941). According to this view, high lignin contents made the culm brittle, and it was important to maintain some degree of elasticity in stalks during the selection process, High lignin contents, however, were also associated with high crushing strength values (Chang et al. 1976), which have been strongly associated with lodging resistance.

Stalk mineral content was investigated as a determinant in lodging tendency for some crops. The element potassium (K) perhaps received the most attention in speculations that it exerted a direct influence on stalk structural components. A positive correlation between K content and lodging was reported by Boswell and Parks (1957) and Leibhardt and Murdock (1965). In contrast, the work of Zuber and Loesch (1966) in maize and Esechie et al. (1977) in sorghum showed that high K values were associated with lodging-prone genotypes. These contrasting views regarding the relationship of K to stalk strength are not surprising, since this element is neither a permanent constituent of the plant nor is it laid down as a part of any specific compound. A deficiency of K can apparently lead to a breakdown in stalk tissue (Liebhardt and Munson 1976) by a direct effect on the parenchyma (Liebhardt et al. 1968). The mechanism by which this occurs is thought to be a polar translocation of carbohydrates from the basal stalk portions to sinks further up the stalk, which triggers parenchyma breakdown in these basal portions. Since photosynthate movement and velocity are limited in the leaves of K-deficient plants (Hart 1969), and their lower leaves are an insignificant source of photosynthate (Moss and Peaslee 1965), a greater portion of carbohydrate stored in the lower stalks of K-deficient plants must be mobilized to fill the demands of the developing inflorescence or ear. This exhaustive translocation from the lower stalks and roots results in parenchyma breakdown, producing a weaker, lodging-susceptible plant (Liebhardt et al. 1968). Leaf removal has been shown to induce parenchyma breakdown and typical K-deficient symptoms relating to kernel formation (Liebhardt et al. 1968). These implications may be valuable in stalk rot studies. For example, high K levels have been shown to have significant effects in reducing the incidence of stalk and root rot in maize (Parker and Burrows 1959).

A thorough understanding of the physiological phenomenon of pith deterioration should help to explain aspects of maintaining plant vigor and, very importantly, plant reaction to stalk and root rots. Pith condition indices have been developed and used to determine stalk strength (Pappelis and Katsanos 1965). The pith rating method has been pop-

ular in stalk rot studies, but its use is not confined to the incidence of disease infestations. Sorghum, for example, showed a marked difference in pith condition between lodging-prone and lodging-resistant genotypes at late stages of growth when disease was not present (Figure 1). Pith condition ratings were associated with the percentage of live tissue whose cells appeared turgid and hydrated. Pappelis and Smith (1963) demonstrated that pith moisture content on a volume basis was related to stalk rot incidence, and was most likely a direct result of a high percentage of live, turgid tissue in the stalk. Their histological observations indicated that the spread of *Diplodia zeae* was limited to dead cells, and that inherent susceptibility was related to the extent of the dead cells in the stalk. Spread of this disease was inhibited by live tissue.

Observations such as these have striking implications. Any phenomena, whether natural or artificial, that maintain living tissue in stalks should result in superior stalk quality and reduce the deleterious effects of rot infestations. Esechie et al. (1977) concluded that lodging-resistant sorghum genotypes appeared to be more perennial in nature, and thus were more resistant to postfreeze senescence than susceptible types. Nonsenescent genotypes studied by Duncan et al. (1981) were more disease-, drought-, and lodging-resistant than their senescent counterparts. Any factor that enhances the rate of cell death in the stalk increases the plant's susceptibility to disease (Pappelis 1963). Pith condition ratings based on this premise were developed for several crops in rot studies (Katsanos and Pappelis 1965, Pappelis and Katsanos 1965).

Depletion of carbohydrates at maturity appears to be associated with susceptibility to lodging in crops (Mortimore and Ward 1964, Esechie et al. 1977). Maranville (1974) suggested that carbohydrate content may be only an indirect indication of the healthiness and vigor of a plant rather than having a direct relationship to lodging. Certainly, any stress (moisture or mineral, for example) directly affects the plant's well-being. Under ideal conditions, plants manufacture sufficient carbohydrate to meet the requirements of both the inflorescence and the plant, but under stress conditions that restrict photosynthesis or alter any of the subsequent processes of carbohydrate metabolism, the amount of carbohydrate produced becomes insufficient to satisfy all demands. Under these circumstances, the requirements of a developing inflorescence are met first, resulting in reduction of stalk sugar levels. This, apparently, is why weak stalks and stalk rot susceptibilities are more prevalent under stress. Mortimore and Ward (1964) found that by artificially inducing stress, they decreased total sugars in maize stalks at physiological matur-

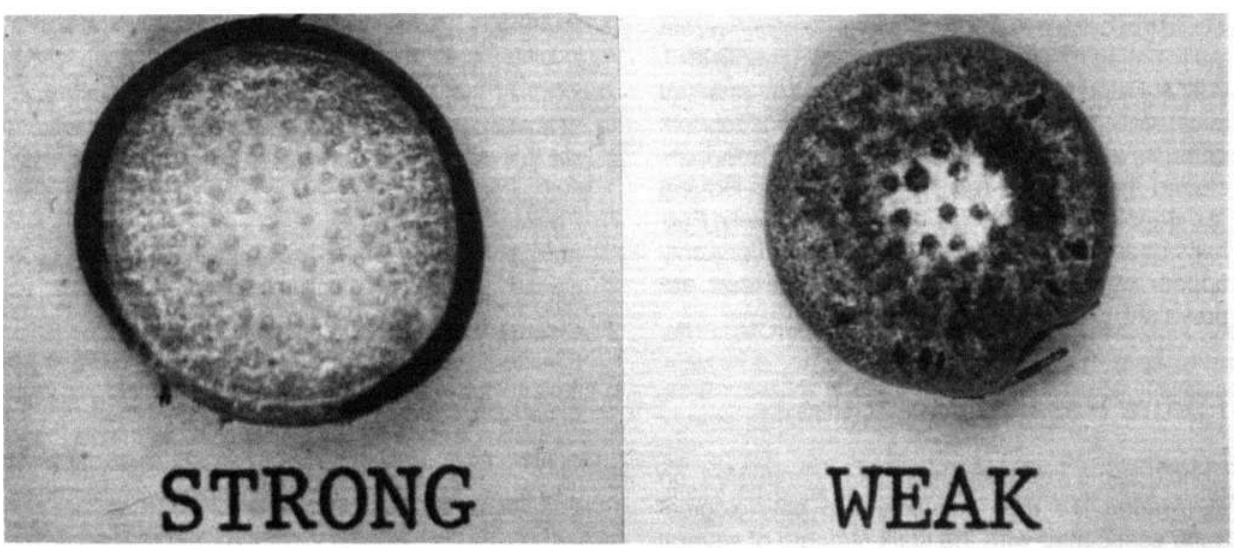

Figure 1. Left, strong: transverse section of the basal stalk of lodging-resistant sorghum lines KS19 x KS21. Right, weak: transverse section of the basal stalk of lodging-susceptible sorghum lines KS21xTX414.

ity, with a subsequent increase in the incidence of stalk rot. Stalk rot never occurred, or was minimal, in stalks of a susceptible plant whose sugar levels were artificially boosted to the same levels as those of rot-resistant plants. The relationship of stress-carbohydrate interactions to disease susceptibility has been named the "photosynthetic stress-translocation balance (PSTB)" concept. This concept has been proposed for both maize (Dodd 1977, 1980a) and sorghum (Dodd 1980b), and developed around the hypothesis that predisposition of stalk rot is associated with a carbohydrate shortage in root and lower stalk tissue, which is caused by the combination of reduced photosynthesis and a translocation of carbohydrate to the developing kernel. This weakens the tissue and allows invasion of rot organisms.

Nitrogen/carbohydrate ratios may be important in stalk quality, as suggested in early work by DeTurk et al. (1937). A decrease in soluble protein occurs during parenchyma breakdown (Liebhardt 1968), suggesting an internal degradation of enzymes that is directly associated with senescence in some manner. Aside from inadequate carbohydrate, a lack of sufficient enzymatic material needed for normal metabolism and synthesis reactions would also result in loss of plant vigor. Zuber et al. (1981) showed that the rind tissue was weakened after invasion by stalk rot. They were uncertain, however, whether this was a result of direct weakening due to enzyme degradation or indirect weakening due to loss of vigor and photosynthate-producing capacity. The former would not necessarily indicate loss of enzymes, but the initiation of a degradation system. Esechie et al. (1977) found that lodging-resistant sorghums had lower stalk protein concentrations than susceptible ones in a disease-free environment. They concluded that a direct relationship between lodging resistance and stalk protein seemed unlikely. Perhaps changes in protein and enzyme functions only appear when stalk rot or other stresses are prevalent.

Future Research Priorities

Considerable research has been conducted on elucidating the morphological and physiological traits associated with the stalk strength of several crops. Many of these investigations have dealt directly with the relationship of this factor to stalk rots. Since this relationship has not been fully clarified, future research should be conducted in the following areas:

1. Determining the exact influence of yield and maturity on inheritance and genetic stability of stalk strength.
2. Comparing a broader range of morphologically and physiologically different genotypes for stalk strength characters. A genotype with a high degree of stalk elasticity might be compared to one with a very stiff stalk, and these then evaluated for standability in the presence of stalk rot.
3. Elucidating the chemical and structural changes that occur in stalks when environmental stress is present.
4. Integrating studies on the senescence of leaves, stalks, and roots to better clarify its relationship to the incidence of stalk rot.
5. Determining the relationship of nitrogen metabolism to physiological factors influencing stalk quality and stalk rot reaction.

References

BARTEL, A.T. 1937. Changes in breaking strength of straw from heading to maturity. Journal of the American Society of Agronomy 29:153-156.

BASHFORD, L.L., MARANVILLE, J.W., WEEKS, S.A., and CAMPBELL, R. 1976. Mechanical properties affecting lodging of sorghum. Transactions of the American Society of Agricultural Engineers 19:962-966.

BOSWELL, F.C., and PARKS, W.L. 1957. The effects of soil K levels on yield, lodging and mineral composition of corn. Soil Science Society of America Proceedings 21:301-305.

BRADY, J. 1934. Some factors affecting lodging in cereals. Journal of Agricultural Science 24:209-232.

CHANG, H.S., LOESCH, P.J., and ZUBER, M.S. 1976. Effects of recurrent selection for crushing strength on morphological and anatomical stalk traits in corn. Crop Science 16:621-625.

CLARK, E.R., and WILSON, H.K. 1933. Lodging in small grains. Journal of the American Society of Agronomy 25:561-572.

DAVIDSON, J., and PHILLIPS, M. 1930. Lignin as a possible factor in lodging in cereals. Science 72:401 -402.

DAVIS, L.L., and STANTON, T.R. 1932. Studies on the

breaking strength of oat varieties at Aberdeen, Idaho. Journal of the American Society of Agronomy 24:290-300.

DAY, A.D. 1957. Effect of lodging on yield, test weight, and other seed characteristics of spring barley grown under flood irrigation as a winter annual. Agronomy Journal 49:536-539.

DeTURK, E.E., EARLY, E.B., and HOLBERT, J.L. 1937. Resistance of corn hybrids related to carbohydrates. Pages 43-45 *in* Illinois Agricultural Experiment Station Annual Report No. 49. Urbana, Illinois, USA: University of Illinois.

DODD, J.L. 1977. A photosynthetic stress-translocation balance concept of corn stalk rot. Pages 122-130 *in* Proceedings of the 32nd Annual Corn and Sorghum Research Conference (eds. H.D. Loden and D. Wilkinson). Washington, D.C., USA: American Seed Trade Association.

DODD, J.L. 1980a. Grain sink size and predisposition of *Zea mays* to stalk rot. Phytopathology 70:534-535.

DODD, J.L. 1980b. The photosynthetic stress-translocation balance concept of sorghum stalk rots. Pages 300-305 *in* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

DUNCAN, R.R., BOCHOLT, A.J., and MILLER, F.R. 1981. Descriptive comparison of senescent and nonsenescent sorghum genotypes. Agronomy Journal 73:849-853.

ESECHIE, H.A. 1975. Studies of physiological, morphological, and anatomical aspects of lodging in grain sorghum [*Sorghum bicolor* (L.) Moench]. Ph.D. thesis, University of Nebraska, Lincoln, Nebraska, USA. 145 pp.

ESECHIE, H.A., MARANVILLE, J.W., and ROSS, W.M. 1977. Relationship of stalk morphology and chemical composition to lodging resistance in sorghum. Crop Science 17:609-612.

FISHER, F.L, and SMITH, O.E. 1960. The influence of nutrient balance on yield and lodging of Texas Corn No. 8. Agronomy Journal 52:201 -204.

HAMILTON, D.G. 1941. Certain oat culm characteristics and their relationship to lodging. Science of Agriculture 21:646-676.

HAMILTON, D.G. 1951. Culm, crown and root development in oats as related to lodging. Science of Agriculture 31:286-315.

HART, C.E. 1969. Effect of potassium deficiency upon translocation of C^{14} in attached blades and entire plants of sugarcane. Plant Physiology 44:1461-1469.

HELMICK, B.C. 1915. A method for testing the breaking strength of straw. Journal of the American Society of Agronomy 7:118-120.

KATSANOS, R.A., and PAPPELIS, A.J. 1965. Seasonal trends in density and cell death in sorghum stalk tissue, Phytopathology 55:97-99.

LARSON, J.C., and MARANVILLE, J.W. 1977. Alteration of yield, test weight, and protein in lodged grain sorghum. Agronomy Journal 69:629-630.

LAUDE, H.H., and PAULI, A.W. 1956. Influence of lodging on yield and other characteristics in wheat. Agronomy Journal 48:452-455.

LIEBHARDT, W.C. 1968. Effect of potassium on carbohydrate metabolism and translocation. Pages 147-166 *in* The role of potassium in agriculture (eds. V.J. Kilmer, S.E. Younts, and N.C. Brady). American Society of Agronomy (ASA) Publication. Madison, Wisconsin, USA: ASA.

LIEBHARDT, W.C., and MUNSON, R.D. 1976. Effect of chloride and potassium on corn lodging. Agronomy Journal 68:425-426.

LIEBHARDT, W.C., and MURDOCK, J.T. 1965. Effect of potassium on morphology and lodging of corn. Agronomy Journal 57:325-328.

LIEBHARDT, W.C., STANGEL, P.J., and MURDOCK, J.T. 1968. A mechanism for premature parenchyma breakdown in corn (*Zea mays* L). Agronomy Journal 60:496-499.

LOESCH, P.J., Jr., CALVERT, O.H., and ZUBER, M.S. 1962. Interactions of Diplodia stalk rot and two morphological traits associated with lodging of corn. Crop Science 2:469-472.

MARANVILLE, J.W. 1974. What's new in sorghum physiology. Pages 22-28 *in* Proceedings of the 29th Annual Corn and Sorghum Research Conference (ed. D.Wilkinson). Washington, D.C., USA: American Seed Trade Association.

MARTIN, J.N., and HERSHEY, A.L. 1934. The ontogeny of the maize plant: The early differentiation of stem and root studies and their morphological relationships. Iowa State College Journal of Science 9:489-503.

McAULEY, R.L. 1973. The effects of potassium on stress-induced stalk lodging in grain sorghum. M.Sc. thesis, Kansas State University, Manhattan, Kansas, USA.

MORTIMORE, C.G., and WARD, G.M. 1964. Root and stalk rot of corn in southwestern Ontario, III: Sugar levels as a measure of plant vigor and resistance. Canadian Journal of Plant Science 44:451-457.

MOSS, D.N., and PEASLEE, D.E. 1965. Photosynthesis of maize leaves as affected by age and nutrient status. Crop Science 5:280-281.

MULDER, E.G. 1954. Effect of mineral nutrition on lodging of cereals. Plant and Soil 5:246-306.

PAPPELIS, A.J. 1963. Increased stalk rot susceptibility in

com following root and leaf injury. Phytopathology 53:24 (abstract).

PAPPELIS, A.J., and KATSANOS, R.A. 1965. An approach to the study of the physiology of senescence and parasitism in sugar cane. Phytopathology 55:620-622.

PAPPELIS, A.J., and SMITH, F.G. 1963. Relationship of water content and living cells to spread of *Diplodia zeae* in corn stalks. Phytopathology 53:1100-1105.

PARKER, D.T., and BURROWS, W.C. 1959. Root and stalk rot in corn as affected by fertilizer and tillage treatment. Agronomy Journal 51:414-417.

PENDLETON, J.W. 1954. The effect of lodging on spring oats yields and test weight. Agronomy Journal 46:265-267.

PINTHUS, M.J. 1973. Lodging in wheat, barley, and oats: The phenomenon, its causes, and preventative measures. Advances in Agronomy 25:209-263.

RASHEVSKY, N. 1960. Mathematical biophysics, Vol. I. New York, New York, USA: Dover Publications. 293 pp.

SALMON, S.C. 1931. An instrument for determining the breaking strength of straw and a preliminary report on the relation between breaking strength and lodging. Journal of Agricultural Research 43:75-82.

SUGGS, C.W., BEEMAN, J.F., and SPLINTER, W.E. 1962. Physical properties of tobacco stalks: Part I. Cantilever bean properties. Tobacco Science 6:146-151.

THOMPSON, D.L. 1963. Comparative strength of corn stalk internodes. Crop Science 3:384-386.

THOMPSON, D.L. 1969. Selection for stalk quality in corn. Pages 7-14 *in* Proceedings of the 24th Annual Corn and Sorghum Research Conference (eds. J.I. Sutherland and R.J. Falasca). Washington, D.C., USA: American Seed Trade Association.

TWUMASI-AFRIYIE, S., and HUNTER, R.B. 1982. Evaluation of quantitative methods for determining stalk quality in short-season corn genotypes. Canadian Journal of Plant Science 62:55-60.

WEIBEL, R.O., and PENDLETON, J.W. 1964. Effect of artificial lodging on winter wheat grain yield and quality. Agronomy Journal 56:487-488.

WELTON, F.A., and MORRIS, V.H. 1931. Lodging in oats and wheat. Ohio Agricultural Experiment Station Bulletin No. 471. Columbus, Ohio, USA: Ohio State University.

ZUBER, M.S. 1973. Evaluation of progress in selection for stalk quality. Pages 110-122 *in* Proceedings of the 28th Annual Corn and Sorghum Research Conference (ed. D. Wilkinson). Washington, D.C., USA: American Seed Trade Association.

ZUBER, M.S., AINSWORTH, T.C., BLANCO, M.H., and DARRAH, L.L. 1981. Effect of anthracnose leaf blight on stalk rind strength and yield in F_1 single crosses in maize. Plant Disease 65:719-722.

ZUBER, M.S., and GROGAN, C.O. 1961. A new technique for measuring stalk strength of corn. Crop Science 1:378-380.

ZUBER, M.S., and LOESCH, P.J. 1966. Total ash and potassium content of stalks as related to stalk strength in corn (*Zea mays* L). Agronomy Journal 58:426-428.

Questions

Doupnik:
By stalk strength, or standability, are you suggesting that those genotypes with good (high) resistance to breakage are also more resistant to stalk rot diseases? Some genotypes with severe stalk rot disease development can stand very well due to rind strength (structural). Isn't this possible?

Maranville:
The evidence in the literature indicates a very strong association between high resistance to breakage and less incidence of disease infection. I don't know the reason for this strong association—perhaps it's indirect. Your premise that plants can stand in the field even when severely diseased due to favorable morphological characteristics certainly should be true. We see in literature, however, that favorable morphological characteristics are not conducive to severe disease incidence.

Relation of Senescence, Nonsenescence, and Kernel Maturity to Carbohydrates and Carbohydrate Metabolism in Sorghum

G.G. McBee*

Summary

The sorghum plant produces high levels of carbohydrates. Senescence, nonsenescence, and stage of kernel maturity have been shown to significantly influence concentrations of sucrose, glucose, fructose, and starch within the plant. This paper presents a brief review of factors affecting senescence, cell changes, and the associated role of growth regulators. Studies on carbohydrate production among cultivars varying in rate of senescence are also summarized. Cultivars that are slower in senescing ha ve produced significantly higher levels of all the above carbohydrates for relative maturity stages of the plants. This factor also appears to be associated with improved yields, but more research over a wider land area and longer period of time is suggested to confirm these findings. Patterns of sugar accumulation within the stems vary during grain filling; some cultivars exhibit a decrease from anthesis to black layer, and then restoration of the stem sugars occurs. Tentative data indicate that sorghum for grain may also contain a higher percentage of structural cat bohydrates than forage types. These differences, plus the variations in accumulation patterns for the carbohydrates, may be critical to studies on pathogenesis and represent an area where more research is needed.

The sorghum plant (*Sorghum bicolor*[L.] Moench) produces tissue composed of high percentages of carbohydrates. By careful attention to management, including specific cultivar selection and cultural modifications, it is possible to significantly influence the structural and nonstructural carbohydrate composition within the plant. Due to increased interest in use of the biomass for energy, more attention has been directed to the levels and types of carbohydrates produced by cultivars. Because of the association between higher nonstructural carbohydrate content in stems (primarily sucrose, glucose, and fructose) and potentially increased grain yields (McBee et al. 1983), breeders are devoting more attention to developing cultivars with sweeter stems. This may alter susceptibility or resistance of sorghum plants to the various pathogens attacking them. These changes in stem carbohydrate content warrant attention, since "all plant pathogens have the ability to produce polysaccharide-degrading enzymes" (Albersheim and Anderson-Prouty 1975).

Two factors that appear to influence levels of nonstructural sugars within the stems are the inherent rate of progressive senescence and stage of kernel maturity at the time of sampling. Since plants senesce in various ways, the process should be defined. Bidwell (1979) defines senescence as "the latter part of the developmental process, which leads from maturity to the ultimate complete

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International Crops Research Institute for the Semi-Arid Tropics. 1984. Sorghum Root and Stalk Rots, a Critical Review: Proceedings of the Consultative Group Discussion on Research Needs and Strategies for Control of Sorghum Root and Stalk Rot Diseases, 27 Nov - 2 Dec 1983, Bellagio, Italy. Patancheru, A.P. 502 324, India: ICRISAT.

loss of organization and function." Further, he describes types of senescence, particularly progressive senescence where older parts of the plants die while the younger parts in the juvenile stage remain active. This appears to be the type that typifies sorghum. "Reduced progressive senescence" would, then, more accurately describe those sorghums referred to as nonsenescent.

Environmental and Other Factors Influencing Senescence

There are several factors that may influence normal, regulated plant senescence. These should be distinguished from the normal changes that occur as the plant matures or abnormal changes due to genetic inheritance. Such factors include light, mineral nutrition, temperature, water, pathogens, insects, physical restrictions, growth regulators, and wind. These factors will be briefly discussed, primarily in relation to monocots. Investigators should be careful to consider them when rating sorghum for the degree or completeness of senescence.

Light

Quality, duration, and intensity of light affect senescence (Thomas and Stoddart 1980). Plants are sensitive to changes in the quantity of light, and continued darkness results in chlorophyll loss or else the failure to synthesize it. Light delays leaf senescence (Thimann et al. 1977) and probably exerts its effect by photoproduction of adenosine-triphosphate. Phytochrome may also have an effect on the tendency to senesce. Red light appears to delay senescence, whereas illumination with far-red light overcomes this effect, thus implicating phytochrome (Beevers 1976, Mishra and Pradhan 1973). Additionally, sensitivity to photoperiod is a contributing factor. Schwabe (1970) has shown that senescence rates in *Kleinia* spp are determined by the daylength prevailing during early growth; daylengths adverse to flowering tended to enhance senescence. The reverse has been observed to occur in photoperiod-sensitive sorghums, which tend to be short-day plants (Martin 1970, p 18). Photoperiods that are either too short or too long to induce flowering tend to stimulate vegetative growth. The delay in flowering could then be regarded as a delay in senescence, or a delay in the physiological processes of senescence, which are discussed later. This effect would be much less significant for adapted cultivars located at increased distances from the equator, since the plants have tended to become less photoperiod-sensitive.

Mineral Nutrition

The effect of mineral nutrition level is closely related to the growth, development, and maturity of the plant. Deficiencies often result in chlorophyll degradation, subsequent necrosis, and perhaps senescence. Initially, it was suggested by Molisch (in Bidwell 1979) that nutritional deficiencies may be the cause of senescence, but later studies have not supported this theory. Since certain elements, such as nitrogen, phosphorus, and potassium, are mobile (Williams 1955) and may be translocated from old to younger tissue within the plant, senescence within those particular tissues may occur earlier than normal as a result of translocation or of the deficiency. Additionally, senescence occurs in annual plants such as maize *(Zea mays* L) after grain maturity, even when large applications of fertilizer have been previously applied.

Temperature

Since most plants can be characterized as exhibiting optimum growth within a certain range of temperatures, high or low extremes result in adverse effects. Frierabend and Mikus (1977) have noted that supraoptimal temperatures tend to inhibit chloroplast ribosome synthesis. Thomas and Stoddart (1980) have proposed that although heat stress may not be the total reason for senescence, it can adversely affect various pathways, producing effects typical of aging. Sullivan and Ross (1979) have noted that direct high-temperature injury occurs even in relatively tolerant crops such as sorghum. Such injury could then be expected to contribute to premature, localized, or partial senescence.

Water

Drought resistance and water relations in sorghum have been discussed by Blum (1979). At critical

levels of moisture stress, stomata close (Turner and Begg 1973) and growth approaches zero. Upon watering, growth resumption depends on the recovery potential of that genotype. Premature senescence and leaf firing may occur, thus suggesting that progressive senescence is also related to the drought resistance of the sorghum genotype. Inherent drought resistance and the varying degrees of drought to which the plant has been subjected must both be considered when evaluating plants for progressive senescence.

Pathogens

Attack by bacteria or viruses may result in either reduced or accelerated senescence of plants. As is known, symptoms may range from small, green, photosynthetic areas ringed by chlorotic haloes (Shaw 1963, Wood 1967, pp 393-397) to large, necrotic areas. Wheeler (1975) states that plant pathogens may employ enzymes, growth regulators, or toxins in pathogenesis. Both Wheeler (1975) and Albersheim and Anderson-Prouty (1975) present evidence that plant pathogens can produce polysaccharide-degrading enzymes. In fact, the latter authors state that together these enzymes can degrade all known glycosidic linkages occurring in primary plant cell walls. Reduced senescence in localized areas is sometimes attributed to the ability of the pathogen to produce cytokininlike substances, possibly an auxin or a gibberellin. The total effect on the plant, however, is generally premature senescence as distinguished from normal senescence exhibited by a healthy plant.

Insects

Various categories of insects and mites can be related to accelerated senescence of the sorghum plant. Piercing, sucking types such as the greenbug (*Schizaphis graminum*), other aphids (*Sipha* spp) and spider mite (*Oligonychus* spp) are prominent among those that produce significant destruction of sorghum blades (Hoelscher and Teetes 1981). Due to injection of toxin by the greenbug and the persistence of these various pests throughout the growing season, care should be taken to distinguish between normal senescence and that induced by these pests.

Physical Restrictions

There are various other factors that contribute directly or indirectly to senescence. We should also consider plant populations and consequences of physical constraints (Thomas and Stoddart 1980). It may be noted that the lower leaves of many currently planted sorghum cultivars tend to senesce when cultured in normal row patterns. Part of the initiation of senescence in the lower leaves can be attributed to darkness or to competition for light. With the more nonsenescent plant types being released, the lower leaves tend to remain green for a much longer period of time. A definite explanation for the lower leaves remaining green is not yet available. The answer may lie within the phytochrome and growth regulator complex. Photoperiod-insensitive sorghum plants produce approximately 20 leaves on the main culm during the growth cycle but contain only 7-10 (excluding side branches and tillers) at maturity. Normal morphological development will explain the senescence of the lower leaves of many monocot plants, especially sorghum. As the culm enlarges, the sheaths of leaves developed early are ruptured, thus resulting in their death (Vanderlip and Reeves 1972).

Growth Regulators

The influence of growth regulators within the plant may also explain some of the variations in the degree of senescence by plants (Bidwell 1979, Sacher 1973, Thomas and Stoddart 1980). Cytokines have been demonstrated to play a role, and some scientists propose that an antisenescence hormone translocated from the roots is a cytokinin or a group of them. Other growth regulators that have been shown to be active in promoting or retarding senescence are the gibberellins, auxins, and ethylene. These will be discussed in more detail later.

Thigmomorphogenesis

Another environmental factor associated with plant responses and senescence and rarely discussed is thigmomorphogenesis. Jaffe and Biro (1979) have discussed such effects. They include mechanical perturbation that may result from such factors as wind, raindrops, and machinery. Such

studies have indicated relationships to plant responses such as stem elongation, electrical resistance, ethylene production, and some resistance to stress. Certainly, incorporation of adequate lodging resistance to excessive wind and some delay of senescence are critical factors in sorghum cultivar development.

Degenerative Cell Changes

Several excellent reviews, chapters, and articles (Thomas and Stoddart 1980, Sacher 1973, Beevers 1976, Forward 1983, Bidwell 1979, Butler and Simon 1971) have been published on the regulated processes exhibited within plants during senescence. Most investigators agree that senescence follows an orderly process and the cell does not simply "collapse." Basically, most of the references indicate a sequential effect or change in chlorophyll, chloroplasts, ribosome content, mitochondria, RNA, proteins, lipids, and finally the nucleus. There are, of course, variations in patterns, such as between monocots and dicots as well as annuals and perennials.

Knowledge of normal cell degradation processes that occur during senescence may be desirable, and a generalized sequence (Butler and Simon 1971, Thomas and Stoddart 1980) has been described. During the early stages, chlorophyll degradation is evident, and, concurrent with this, a decrease in ribosome populations and chloroplast breakdown occurs. The process of chlorophyll breakdown is still not clearly understood, and doubts exist that it involves an enzymatic process. Chloroplast destruction apparently involves two systems, one of which acts on the stroma complex and the other on the thylakoid components. The stroma disappear, thylakoids swell and burst, and then a massing of osmophilic globules is apparent. During this period or soon thereafter, the endoplasmic reticulum swells and disintegrates or disappears, as do the Golgi dictyosomes. Other factors that occur include breakdown of the tonoplast, followed by the plasmalemma. Evidence indicates that there is a distortion of the mitochondria during the changes outlined above, but they tend to persist until the latter stages. Apparently the nucleus is stable until the last stage, when the nuclear membrane breaks down, the chromatin disappears, and the cell dies.

Other effects observed during stages of cell senescence, as described above, include a decline in protein synthesis. Within a leaf, turnover of protein is expected, but in a senescing leaf, protein synthesis is retarded and amino acids tend to accumulate (Beevers 1976). Some evidence exists, however, that not all proteins are degraded simultaneously. Thomas and Stoddart (1980) reason that a sensing reaction may occur as a result of reduced protein synthesis. The resulting gradual decrease in protein synthesis along with decreased chloroplast components may then trigger the sequential senescence steps through decreased export of metabolites to the cytoplasm.

Concurrent with declines in chlorophyll, chloroplasts, and protein, there is a decline in RNA (Thomas and Stoddart 1980, Beevers 1976, Bidwell 1979). This is based on a decline in incorporation of precursor into total RNA. Additionally, this has been explained as a failure of DNA to transcribe a template for RNA synthesis (Osborne 1962). Such findings support the idea that senescence is controlled at the transcriptional level. Concurrent with RNA decline, a decrease in ribosome content occurs. Although this would result in decreased protein synthesis, study of some works (Balz 1966, Matile 1968) also suggests that the content of remaining ribosomes may function to produce hydrolytic enzymes. Simultaneously with the previously described decreases, a decline also occurs in lipids. Not all lipids degrade at the same rate (Draper 1969), but lipid hydrolysis does occur during this period of degradation.

Growth Regulators

The relationship of growth regulators to senescence has been extensively studied, and numerous references are included in reviews of this subject (Thomas and Stoddart 1980, Bidwell 1979, Sacher 1973). A summary of the results, perhaps oversimplified, indicates that the cytokinins, auxins (particularly indoleacetic acid [IAA]), and gibberellins (GA) are associated with the prevention, retardation, or reversal of senescence, whereas abscisic acid (ABA) and frequently ethylene are associated with enhancement or promotion of senescence.

The interactive processes of these regulators have been described in various studies, but still many questions remain to be answered. Sacher (1973) states that cytokinins and GA are prominent as retardants of senescence in citrus fruit. In a review by Thomas and Stoddart (1980), several

references are cited to indicate that cytokinins are generally the most effective class of senescence-retarding hormones. Bidwell (1979) states that a cytokinin or group of cytokinins produced and translocated from the roots prevents or reverses senescence. Degradation of RNA during cell degeneration has been previously described, and some scientists propose that cytokinins may protect against such degradation. Thomas and Stoddart (1980) also present evidence that cytokinins function in sustaining the metabolic state and normal photosynthate-exporting phase of mature leaves. This in turn would prevent a decline below the threshold for senescence initiation (for example, reduced protein synthesis and amino acid accumulation). Ambler et al. (1983) have shown that cytokinin levels in the less senescent sorghum 77CS2 were about three times those of a more senescent entry, RTx7000. In addition to the cytokinin effects presented, Sacher (1973) proposes that auxin declines at about the time that ABA begins to increase. He further cites evidence to indicate that ABA enhances the hydrolytic enzymes and acts antagonistically with auxin, GA, or cytokinin. Additionally, Cracker and Abeles (1969) have reported that ABA enhances ethylene production. Not all plants respond in the same way to hormones, but apparently cytokinins are involved in the regulation of senescence in sorghums. Many of the reviewers above have cited references to experiments that detail the processes of enhancement or reversal of senescence by hormones. A detailed discussion of these processes, however, is beyond the scope of this paper.

An interesting observation is that photosynthesis decreases (Woolhouse 1967) and respiration continues at a fairly normal rate until the final phases of senescence (James 1953). With the degradation of chloroplasts and decrease in chlorophyll content, a decrease might be expected in the rate of photosynthesis. Although respiration proceeds as described above, changes have been recorded in the respiratory quotients. Protein degradation products may supply the respiratory substrate during senescence, thus partially explaining why we do not observe declines in respiratory rates until near the end of the senescence process.

From the previous discussion, it may be noted that senescence is a strictly controlled degradation process, and ultrastructural changes in the cell proceed in a reasonably defined sequence (Butler and Simon 1971). This begins with a decrease in ribosomes and chloroplast degradation, followed by a somewhat orderly process: mitochondria change, tonoplast rupture, organelle degeneration, and finally plasmalemma and nucleus deterioration.

Senescence and Reduced Progressive Senescence in Sorghum

Since initiation of the sorghum conversion program (Stephens et al. 1967), more emphasis is being placed on various studies comparing senescent to nonsenescent sorghum genotypes. Distinguishing characteristics have been described for both categories. Duncan et al. (1981) noted that the nonsenescent types produce higher leaf-blade chlorophyll content and retain green leaves for a longer period of time after grain maturity.

One of the major factors we have observed in our studies on the comparison of senescent to nonsenescent sorghum cultivars is a larger relative quantity of total nonstructural carbohydrates within the stems of the latter types. Several factors should be considered, however, in evaluating a sorghum cultivar for patterns of sugar development and correlation with the biochemistry of pathogenesis. Significant among these factors is correlation between the maturity stages of the sorghum plant and concentration of nonstructural carbohydrates. Minimum differences in nonstructural carbohydrates have been obtained prior to heading in both the senescent and nonsenescent cultivars studied. Webster et al. (1948) determined sugar composition in several cultivars of sorghum with selections from sorgo, kafir, milo, feterita, and others. They reported little difference in the percentage of sugar in the juice from the different entries prior to heading. After heading, significant differences in stem sugar levels began to develop among the entries. McBee and Miller (1982) observed a similar response in studies comparing Combine Kafir-60 (CK-60) and Rio at the preboot and early anthesis stages of maturity. As shown in Figure 1, the percentage of total nonstructural carbohydrates was very similar for the two cultivars at the preboot stage. After the beginning of anthesis, percentages increased significantly in both entries, with the increase being much larger for Rio. Starch is frequently higher in the stems during the preboot stage than during anthesis or grain fill, but this would not suffice to account for the significantly

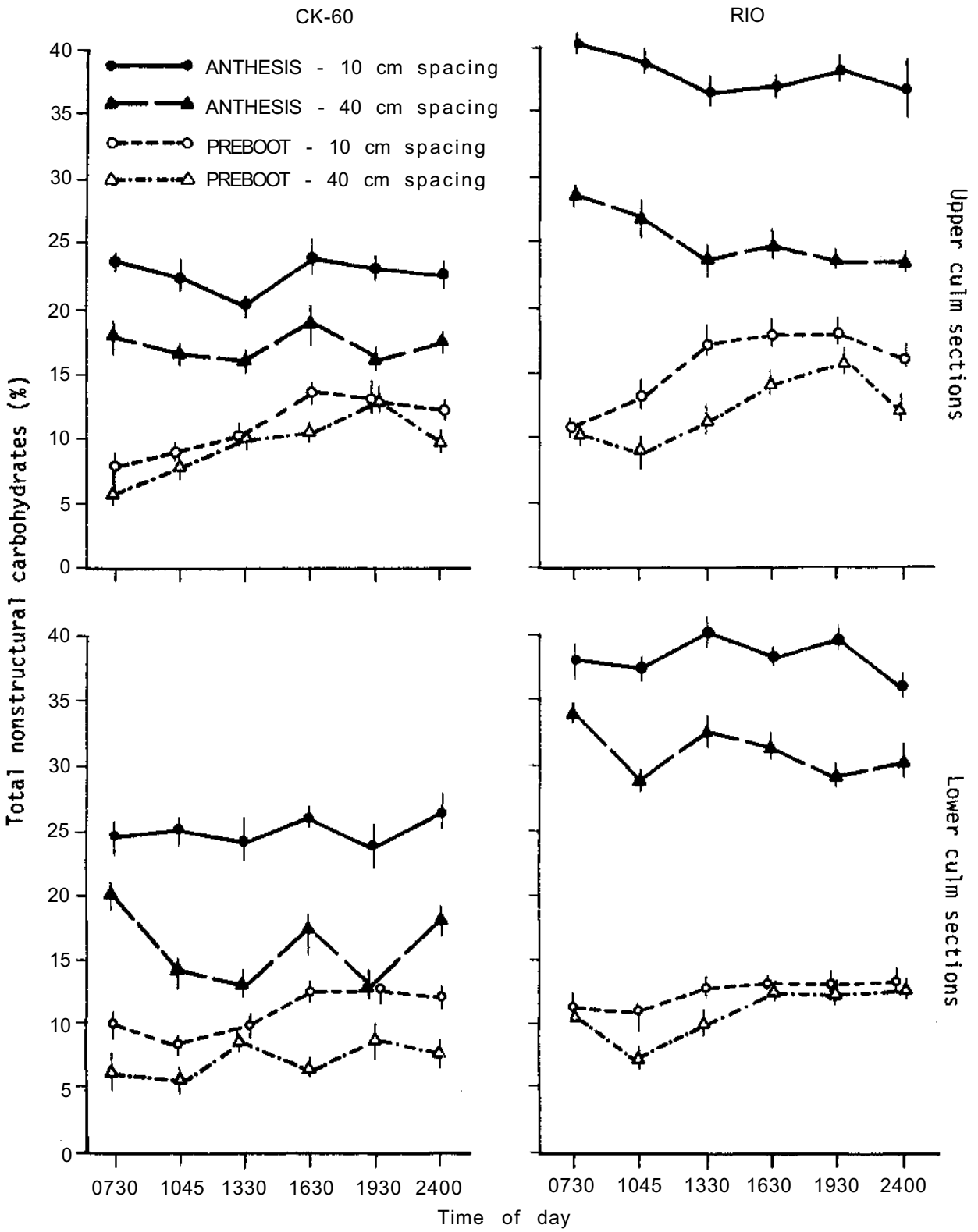


Figure 1. Percentage of total nonstructural carbohydrates over diurnal cycle in upper and lower culm sections of CK-60 and Rio during preboot and early anthesis stages at two plant spacings within rows. Values are mean ± SE.

higher levels of sugars produced after panicle emergence and anthesis.

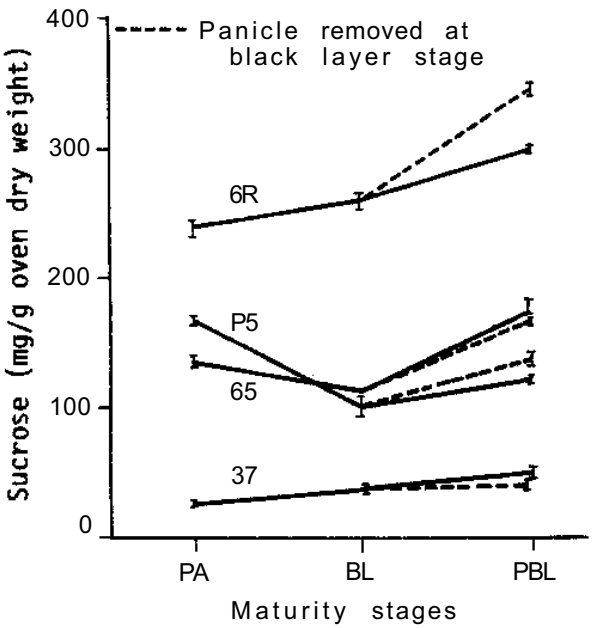
Plant population will apparently affect partitioning of nonstructural carbohydrates regardless of the senescent or nonsenescent influence. Among the first reports on this was a study conducted by Eilrich et al. (1964). They found the percentage of carbohydrates to be higher in sorghum planted in rows than in a drill system. It may also be noted in Figure 1 that plant spacing within rows significantly influenced the percentage of total nonstructural carbohydrates. More closely spaced plants contained a higher percentage of these sugars. Rows were spaced 100 cm apart in this study (McBee and Miller 1982).

Distribution of the nonstructural carbohydrates within the plant will vary somewhat. Janssen et al. (1930) noted that the central portion of the stem in sorgo contained the highest amount of soluble sugars, followed in sequence by the lower and uppermost sections. Ventre (1939) found more glucose in the lower portion of the stalk and more sucrose and starch in the upper portions. In general, we have noted lower glucose levels in the upper portion of the sorghum stem and rather uniform distribution for sucrose. Starch may vary some, but generally, more starch has been found in upper parts of the stems of closely spaced plants, whereas the quantities are sometimes higher in the basal part of widely spaced plants. This may be partially due to partitioning of carbohydrates into structural forms where more spacing exists between plants.

Leaf position definitely has an influence on the quantity of carbohydrates produced by the particular leaf. As previously stated, earlier senescence occurs in the first four or five leaves produced. This may result from a combination of factors such as competition for light, nutrient translocation, and rupture of the sheath due to stem enlargement. Various workers (Stickler and Pauli 1961, Goldsworthy 1970, and Fischer and Wilson 1971) have studied the effect of the remaining leaves on assimilate production. Goldsworthy (1970) reported that, for the cultivars used, the top leaves senesced more slowly than the middle leaves and much more slowly than those on the bottom of the plant. All of the investigators showed that the upper leaves contributed most to grain yield. Fischer and Wilson (1971) found that 93% of the grain yield was due to assimilation by the head and upper four leaves.

As the plant approaches maturity or the later stages of senescence, levels of glucose, fructose, sucrose, and starch within the plant will change. Webster et al. (1954) have noted that total sugars, especially sucrose in extracted sorghum juice, increased to a maximum after the soft dough stage. Ventre et al. (1948) reported that sucrose increased with maturity in sorghum, and Eilrich et al. (1964) stated that nonreducing sugars increased in forage sorghum after anthesis for about 7 weeks and then decreased. We (McBee et al. 1983) observed a significant increase in stem sugars after anthesis, with variable patterns obtained for the different cultivars (Figs. 2,3). The effect of the kernel maturity stage was very significant, as is shown in the figures.

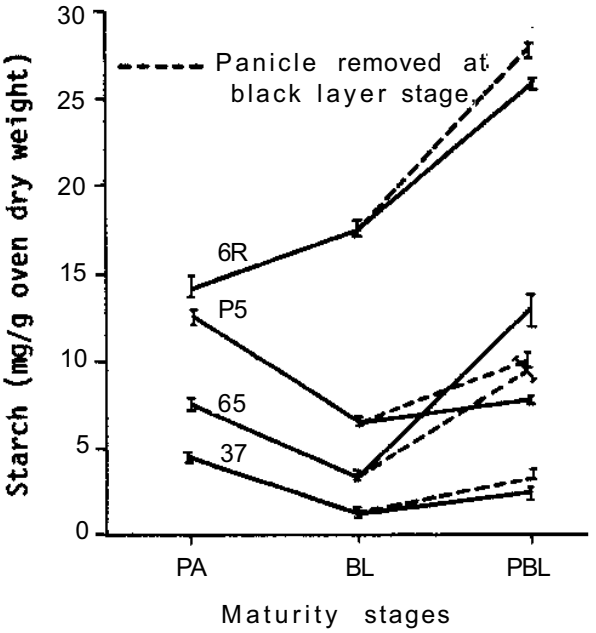
As may be noted, nonstructural carbohydrate production was greater in the nonsenescent entries. Variations in stem concentrations should



Maturity stages:
PA = 15 days postanthesis
BL = black layer
PBL = 15 days postblack layer

Cultivars used:
37 (senescent) = ATx378 x RTx7000
6R (nonsenescent) = ATx623 x Rio
P5 (nonsenescent) = APurl x R74CS5388
65 (nonsenescent) = ATx623 x R74CS5388

Figure 2. Effect of kernel maturity on sucrose levels in culms of four sorghum cultivars. Values are mean ± SE.



Maturity stages:
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Figure 3. Effect of kernel maturity on starch levels in culms of four sorghum cultivars. Values are mean ± SE.

be noted, however. Both P5 and 65 exhibited higher levels of sucrose and starch at PA, decreased at BL, and then increased. This appears to be a pattern that is followed by other nonsenescent cultivars (Clark 1981). Hybrid 6R exhibited an upward trend in stem levels at all kernel maturity stages. Apparently the supply of sugar was sufficiently greater than the sink demands so that no decrease occurred during the peak demand of the kernels.

Lengyel and Annus (1960) noted that sorghum for forage possesses less cellulose and hemicellulose than sorghum for grain. We have obtained similar results in our laboratory; however, these studies are in the preliminary stages. My preliminary data indicate that nonsenescent sweet sorghums contain about 20% hemicellulose and 21% cellulose, whereas a nonsenescent grain sorghum contains 24% hemicellulose and 23% cellulose. Information about variations in the structural fractions during development of the sorghum plant is very limited. Bettini and Proto (1960) reported that the percentage of crude cellulose content does not vary significantly as the plant progresses through its growth cycle.

Concentrations of cytokinins were approximately three times greater in a nonsenescent sorghum line than in a senescent one (Ambler et al. 1983). Major cytokinins in the stem exudate during the postblack-layer stage, were, in order of decreasing concentrations, *trans*-zeatin riboside, *trans*-zeatin, and isopentyl adenosine. This would substantiate the previous statement (see "Growth Regulators" above) that cytokinins are one of the most effective classes of hormones in retarding senescence. F.R. Miller (Texas A&M University, Soil and Crop Sciences Dept., USA; personal communication, 1983) reported that leaves on nonsenescent sorghum lines remain green longer and do not senesce on lower parts of the plant until significantly later than the more senescent entries. These observations together with the higher cytokinins concentrations indicate that this group of growth regulators is associated with delayed senescence in sorghum.

Conclusions

Based on the previous discussion, it can be proposed that reduced progressive senescence is genetically controlled (Butler and Simon 1971). Sorghum exhibits a somewhat uniform and typical pattern of senescence in the first leaves produced, due to sheath rupture. Sequentially, the lower leaves senesce, followed at a later period by the upper ones and subsequently by the stem. The pattern may be influenced by alterations in factors such as nutrition, light, etc., but reduced progressive senescence has also been shown to be associated with genetic inheritance. Under normal conditions, some controlled senescence occurs in the plant, as illustrated by the organized death of cells to form xylem tissue and naturally programmed leaf and chloroplast senescence (Woolhouse 1967, 1978; Butler and Simon 1971). A review by Heslop-Harrison (1967) also proposed that developmental changes in plants are based on changing patterns of gene repression and derepression. Evidence is also offered by Butler and Simon (1971) that DNA in the chloroplasts, mito-

chondria, and nucleus is involved. The evidence indicates that the rate of senescence in sorghum is genetically controlled and that the growth regulators, especially the cytokinins, are involved. Increased levels of sugar accumulation at comparative maturity stages in stems of various sorghum cultivars have been associated with delayed senescence. Accumulation of sugar in stems after black layer in the kernel is evidently due to the reduction in sink volume and continued photosynthesis resulting from incomplete senescence in some of the sorghum cultivars described. This factor appears to be important for improved yields and should be carefully observed for any correlations with pathogenesis.

Future Research Needs

1. Studies to determine conditions that influence partitioning of photosynthates between structural (SC) and nonstructural carbohydrates (NSC) at different plant growth stages.
2. Studies to obtain data for inherent levels of SC and NSC expected for various genotypes and cultivars.
3. Techniques to manipulate carbohydrate partitioning for improved quality of biomass.
4. Determination of the relationships of above items to resistance or susceptibility of plants to diseases.
5. Correlation of higher concentrations of NSC in cultivars to disease susceptibility or resistance.

References

ALBERSHEIM, P., and ANDERSON-PROUTY, A.J. 1975. Carbohydrates, proteins, cell surfaces, and the biochemistry of pathogenesis. Annual Review of Plant Physiology 26:31-52.

AMBLER, J.R., MORGAN, P.W., and JORDAN, W.R. 1983. Cytokinin concentrations in xylem sap of senescent and nonsenescent sorghums. Plant Physiology Supplement 72(1):168.

BALZ, H.P. 1966. Intracellular localization and function of hydrolytic enzymes in tobacco. Planta 70:207-236.

BEEVERS, L. 1976. Senescence. Pages 771-794 *in* Plant biochemistry (eds. J. Bonner and J.E. Varner). New York, New York, USA: Academic Press.

BETTINI, T.M., and PROTO, V. 1960. [Chemical composition of some hybrid sorghums.] Annali delta Sperimentazione Agraria 4:957-974 (in Italian).

BIDWELL, R.G.S. 1979. Plant physiology. New York, New York, USA: Macmillan Publishing Co. 726 pp.

BLUM, A. 1979. Genetic improvement of drought resistance in crop plants: a case for sorghum. Pages 428-445 *in* Stress physiology in crop plants (eds. H. Mussell and R.C. Staples). New York, New York, USA: John Wiley and Sons.

BUTLER, R.D., and SIMON, E.W. 1971. Ultrastructural aspects of senescence in plants. Advances in Gerontological Research 3:73-129.

CLARK, J.W. 1981. The inheritance of fermentable carbohydrates in stems of *Sorghum bicolor* (L.) Moench. Ph.D. thesis, Texas A&M University, USA. 88 pp.

CRACKER, L.E., and ABELES, F.B. 1969. Abscission: roles of abscisic acid. Plant Physiology 44:1144-1149.

DRAPER, S.R. 1969. Lipid changes in senescing cucumber cotyledons. Phytochemistry 8:1641-1647.

DUNCAN, R.R., BOCKHOLT, A.J., and MILLER, F.R. 1981. Descriptive comparison of senescent and nonsenescent sorghum genotypes. Agronomy Journal 73:849-853.

EILRICH, G.L, LONG, R.C., STICKLER, F.C., and PAULI, A.W. 1964. Stage of maturity, plant population, and row width as factors affecting yield and chemical composition of atlas forage sorghum. Kansas Agricultural Experiment Station, Technical Bulletin No. 138, Manhattan, Kansas, USA. 23 pp.

FISCHER, K.S., and WILSON, G.L. 1971. Studies of grain production in *Sorghum vulgare:* I. The contribution of pre-flowering photosynthesis to grain yield, II. Sites responsible for grain dry matter production during the post-anthesis period. Australian Journal of Agricultural Research 22:33-47.

FORWARD, D.S. 1983. Senescence. Pages 468-481 *in* Vol. 7, Plant physiology: a treatise (eds. F.C. Steward and R.G.S. Bidwell). New York, New York, USA: Academic Press.

FRIERABEND, J., and MIKUS, M. 1977. Occurrence of a high temperature sensitivity of chloroplast ribosome formation in several higher plants. Plant Physiology 59:863-867.

GOLDSWORTHY, P.R. 1970. The sources of assimilate for grain development in tall and short sorghum. Journal of Agricultural Science 74:523-531.

HESLOP-HARRISON, J. 1967. Differentiation. Annual Review of Plant Physiology 18:325-348.

HOELSCHER, C.E., and TEETES, G. 1981. Insect and mite pests of sorghum—management approaches. Texas A&M University, Bulletin No. 1220, College Station, Texas, USA. 23 pp.

JAFFE, M.J., and BIRO, R. 1979. Thigmomorphogenesis: The effect of mechanical perturbation on the growth of plants, with special reference to anatomical changes, the role of ethylene, and interaction with other environmental stresses. Pages 25-59 *in* Stress physiology in crop plants (eds. H. Mussell and R.C. Staples). New York, New York, USA: John Wiley and Sons.

JAMES, W.O.1953. Plant respiration. London (U.K.) and New York, New York (USA): Oxford University Press. 282 PP.

JANSSEN, a, MCCLELLAND, C.K., and METZGER, W.H. 1930. Sap extraction of sorghum and the localization of juice and sugars in internodes of the plant. Journal of the American Society of Agronomy 22:627-639.

LENGYEL, P., and ANNUS, S. 1960. [Domestic (Hungarian) plants suitable for pulp manufacture, IV]. Papiripar 4:100-105 (in Hungarian).

MARTIN, J.H. 1970. History and classification of sorghum *Sorghum bicolor* (Linn.) Moench. Pages 1 -27 *in* Sorghum production and utilization (eds. J.S. Wall and W.M. Ross). Westport, Connecticut, USA: AVI Publishing Co.

MATILE, P.H. 1968. Lysosomes of root tip cells in corn seedlings. Planta 79:181-196.

McBEE, G.G., and MILLER, F.R. 1982. Carbohydrates in sorghum culms as influenced by cultivars, spacing, and maturity over a diurnal period. Crop Science 22:381-385.

McBEE, G.G., WASKOM, R.M., III, MILLER, F.R., and CREELMAN, R.A. 1983. Effect of senescence and nonsenescence on carbohydrates in sorghum during late kernel maturity states. Crop Science 23:372-376.

MISHRA, D., and PRADHAN, P.K. 1973. Regulation of senescence in detached rice leaves by light, benzimidazole and kinetin. Experimental Gerontology 8:153-155.

OSBORNE, D.J. 1962. Effect of kinetin on protein and nucleic acid metabolism in xanthium leaves during senescence. Plant Physiology 37:595-602.

SACHER, J.A. 1973. Senescence and post harvest physiology. Annual Review of Plant Physiology 24:197-224.

SCHWABE, W.W. 1970. The control of leaf senescence in *Kleinia articulata* by photoperiod. Annals of Botany 34:43-55.

SHAW, M. 1963. The physiology and host-parasite relations of the rusts. Annual Review of Phytopathology 1:259-294.

STEPHENS, J.C., MILLER, F.R., and ROSENOW, D.T. 1967. Conversion of alien sorghums to early combine genotypes. Crop Science 7:396.

STICKLER, F.C., and PAULI, A.W. 1961. Leaf removal in grain sorghum: I. Effect of certain defoliation treatments on yield and components of yield. Agronomy Journal 53:99-102.

SULLIVAN, C.Y., and ROSS, W.M. 1979. Selecting for drought and heat resistance in grain sorghum. Pages 263-281 *in* Stress physiology in crop plants (eds. H. Mussell and R.C. Staples). New York, New York, USA: John Wiley and Sons.

THIMANN, K.V., TETLEY, R.M., and KRIVAK, B.M. 1977. Metabolism of oat leaves during senescence. Plant Physiology 59:448-454.

THOMAS, H., and STODDART, J.L. 1980. Leaf senescence. Annual Review of Plant Physiology 31:83-111.

TURNER, N.C., and BEGG, J.E. 1973. Stomatal behavior and water status of maize, sorghum, and tobacco under field conditions. Plant Physiology 51:31-36.

VANDERLIP, R.L. and REEVES, H.E. 1972. Growth stages of sorghum [*Sorghum bicolor* (L.) Moench]. Agronomy Journal 64:13-16.

VENTRE, E.K. 1939. Jellying and crystallization of sirups made from different parts of the sorgo stalk at different stages of maturity. Journal of Agricultural Research 59:139-150.

VENTRE, E.K., BYALL, S., and CATLETT, J.L. 1948. Sucrose, dextrose and levulose content of some domestic varieties of sorgo at different stages of maturity. Journal of Agricultural Research 76:145-151.

WEBSTER, J.E., BENEFIEL, D., and DAVIES, F. 1954. Yield and composition of sorghum juice in relation to time of harvest in Oklahoma. Agronomy Journal 46:157-160.

WEBSTER, J.E., SIEGLINGER, J., and DAVIES, F. 1948. Chemical composition of sorghum plants at various stages of growth, and relation of composition to chinch bug injury. Oklahoma Agricultural Experiment Station Bulletin T-30, Stillwater, Oklahoma, USA. 32 pp.

WHEELER, H. 1975. Plant pathogenesis. New York, New York, USA: Springer-Verlag. 106 pp.

WILLIAMS, R.F. 1955. Redistribution of mineral elements during development. Annual Review of Plant Physiology 6:25-42.

WOOD, R.K.S. 1967. Physiological plant pathology. Oxford/Edinburgh, U.K.: Blackwell. 570 pp.

WOOLHOUSE, H.W. 1967. The nature of senescence in plants. Symposia of the Society for Experimental Biology 21:179-214.

WOOLHOUSE, H.W. 1978. Cellular and metabolic aspects of senescence in higher plants. Pages 83-99 *in*

The biology of aging (eds. J.A. Behnke, C.E. Finch, and G.B. Moment). New York, New York, USA: Plenum Press.

Questions

Maranville:
Regarding the slide in your presentation that showed the source-sink relationship when the top didn't fill: if there was no environmental stress and the head fills from top to bottom, why didn't the top fill and the bottom become depleted if only the source was limiting?

McBee:
Lack of complete kernal fill occurred at the top of the panicle in those plants with a"high-sink/low-source" treatment. Preliminary data indicate the plant was producing insufficient carbohydrate during the period of fill. Apparently proximity was the reason that lower kernels filled first, because as the photosynthate was translocated acropetally, the lower kernels received the photosynthate at the expense of the upper ones.

Scheuring:
You commented on the hydrolysis of sucrose at the placental sac site. That's the deposition end of carbohydrate mobilization. Please comment on the starting point of carbohydrate mobilization, i.e., from cortex cells into vascular tissue: Does hydrolysis occur at that time? If so, might there be implications for pathogen nutrition and growth?

McBee:
I know of no work on hydrolysis of polysaccharides at the cellular level in sorghum for grain. There are reports for sugarcane. Those authors state and show data to support the fact that gradient levels of invertase are controlled by auxin. Consequently sucrose is hydrolyzed and moved symplastically from cell to cell as glucose and fructose and apoplastically in the vascular system. I would think the monosaccharides would be more conducive to growth of some pathogens. I might add, however, that as fructose and glucose are transported, the plant tends to combine them into sucrose again for ease of transport.

Sorghum Sensitivities to Environmental Stresses*

J.D. Eastin, C.Y. Sullivan, J.M. Bennett, A.M. Dhopte, T.J. Gerik, V.A. Gonzalez-Hernandez, K.-W. Lee, V. Ogunlela, and J.R. Rice**

Summary

Sorghum is relatively insensitive to heat and water stress during the vegetative stage. Stress has variable effects during panicle development, with the most sensitive times being about 3 to 6 days after floret differentiation (i.e., during microsporogenesis) and 7 to 11 days after floret differentiation (at megasporogenesis). Yield and seed number losses at 5°C above ambient night temperatures can easily be on the order of 30%. A marked loss of stomatal control occurs about 3 days after anthesis. Stress 7 to 10 days after anthesis can limit seed size, presumably because of cell division or cell wall elasticity problems in the endosperm. Sizeable shifts in the polarization of assimilate transport to grain at the expense of roots may predispose sorghum to charcoal rot and other root-stalk lodging problems during grain fill.

Other contributions at this symposium contain apt reviews of stresses imposed by pests,, including references to stresses from pathogens and insect feeders. Our purpose is to consider the plant's changing sensitivities to environmental stresses (heat and water) at various growth stages so that pathologists may better consider the sometimes compounding negative effects of pathogens on grain yield.

The sensitivity of a plant to an environmental stress may relate to its predisposition to diseases. Yarwood (1959) defined predisposition as the tendency of nongenetic factors (such as heat and water stress), acting prior to infection, to affect the plant's susceptibility to disease. This implies an effect on the host (plant) rather than on the pathogen, which is what we wish to consider. Schoeneweiss (1975) discussed this topic at length. Environmental stresses do force shifts in assimilates normally available for developing grain, which results in reduced grain production. Therefore, it is appropriate to consider the times and nature of sorghum sensitivity to environmental stresses as they relate to grain yield and its seed size and seed number components. This amounts to an exercise in both developmental physiology and, to some degree, process physiology. Developmental physiology is a vehicle for analyzing grain-yield reductions in terms of the seed weight and seed number components of yield. This establishes the time framework within which yield reductions occur, due to either stresses or diseases. Investigators may

*Contribution of the Nebraska Agricultural Experiment Station, Lincoln, Nebraska, USA.

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International Crops Research Institute for the Semi-Arid Tropics. 1984. Sorghum Root and Stalk Rots, a Critical Review: Proceedings of the Consultative Group Discussion on Research Needs and Strategies for Control of Sorghum Root and Stalk Rot Diseases, 27 Nov - 2 Dec 1983, Bellagio, Italy. Patancheru, A.P. 502 324, India: ICRISAT.

then consider physiological processes that may help them understand developmental limitations. We will not attempt an exhaustive coverage of the literature. The simple growth stage terminology of Eastin (1972) will be used for discussion. More complex growth stage terminology utilizing information gathered since 1972 would probably be useful in crop modelling.

Vegetative Development (GS1)

Whiteman and Wilson (1965) withheld water from sorghum to the point of suppressing all growth for 2, 3, and 4 weeks prior to panicle initiation and observed no adverse effects on panicle development when stress was relieved. General observations in the field seem to support their results, so long as growth is good enough to insure reasonably adequate leaf-area development prior to panicle initiation. Stressing plants during panicle development, however, can reduce yields drastically.

Floral Development (GS2)

Seed number per unit of land area usually correlates more positively with grain yield than does seed size (weight) (Stickler et al. 1961; Kambal and Webster 1966; Blum 1967, 1970; Quinby 1963 Doggett and Jowett 1967; Beil and Atkins 1967 Fischer and Wilson 1975; Eastin and Sullivan 1974; Ogunlela 1979). The seed size component of yield may become relatively more important as the levels of environmental stress increase (Eastin et al. 1983, Heinrich et al, 1983, Eastin 1984). Consideration of both yield components follows.

Vegetative-floral Competition

Eastin (1972) reported yield comparisons of 2-, 3-, and 4-dwarf RS 626 isogenic height hybrids (Table 1). Grain yields did not differ, but seed weights and total dry matter production did. Seeds were about 30% larger in the tall 2-dwarf hybrids. Since yields were the same, the seed number was proportionately lower. The reason for the higher seed number in the short hybrids (3x3 and 4x3 dwarfs) is probably explained by the comparative total dry weight figures. The tall hybrid produced 16% more total dry weight than the 3x3 dwarfs and 19% more than the 4x3 dwarfs. Since the heights of the hybrids were indistinguishable at panicle initiation, the extra vegetative dry weight was produced while the panicle was developing. Apparently competition between the simultaneously expanding panicle and vegetative parts for available assimilates was sufficient to reduce seed number. Panicles in the 2x3-dwarf hybrid were noticeably shorter, and the large bold seed could be discerned by eye. Panicle development is definitely influenced by other assimilate demands during GS2. Assimilate demands by insect vectors and diseases may be factors.

Panicle Development

Paulson (1962) and Lee et al. (1974) illustrated apex transformation when sorghum goes from vegetative to floral status (Fig. 1 a-c). Relatively photoperiod-insensitive temperate sorghums reach the panicle initiation (PI) stage about when the 10th to 13th leaf blade tip emerges from the whorl. The floral status is signalled by the appearance of primary panicle branch primordia, which

Table 1. Comparative production data for tall (2x3-dwarf), normal (3x3-dwarf), and short (4x3-dwarf) RS 626 sorghums grown under irrigation at Mead, Nebraska, USA (1970). Germination to maturity was 106 days. Dry matter is adjusted to 14% moisture. (Source: Eastin 1972.)

		Dry wt (kg/ha)			Dry matter/day (kg)				
Height	Grain-stover ratio[a]	Grain	Total	%of 2 dw	Grain	Total	%of 2 dw total	Seed wt (g/1000)	%of 2 dw over others
2dw	0.76	7741	17940	100	73.3	169	100	29.3	
3dw	1.14	7988	15080	84	75.4	142	84	22.7	29
4 dw	1.08	7528	14501	81	71.0	137	81	22.5	30

a Ratio is higher than normal due to leaf loss in a heavy unseasonal snow before final harvest.

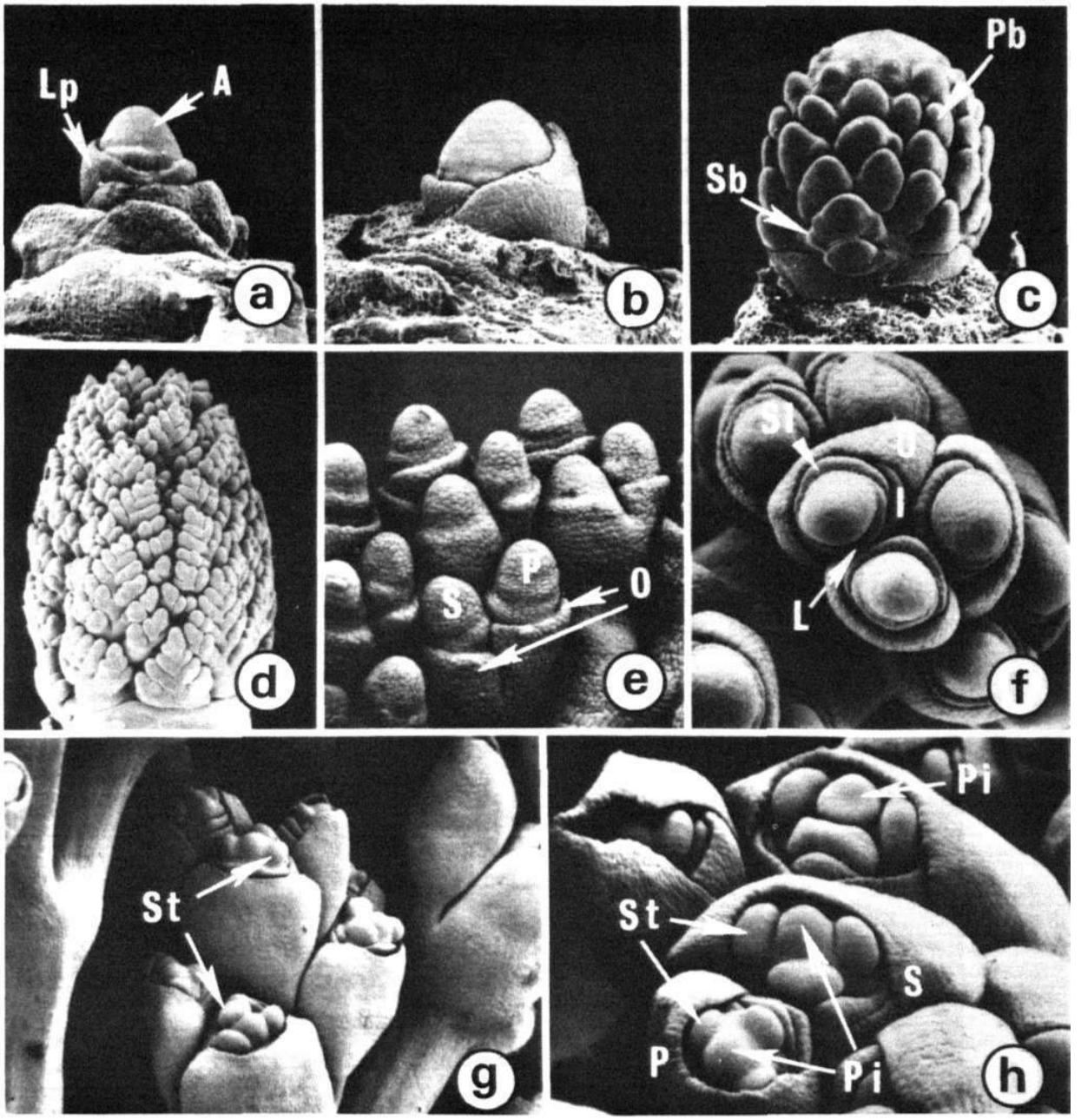

Figure 1. The vegetative and floral apices of **Sorghum bicolor (L.)** *Moench.*(*Eastin and Lee 1984.*)

a. Leaf primordium (*Lp*) *and shoot apex* (*A*) *of a 3-week-old seedling.*

b. Enlargement of the apex before floral development.

c. Initiation of primary branch primordia (*Pb*) *on the floral apex; secondary branch primordium* (*Sb*) *starts to form before primary branch primordia are completely formed at the apical dome.*

d. Enlargement of floral apex due to the formation of more branch primordia of higher order.

e. Initiation of the outer glume primordia (*O*) *on both sessile* (*S*) *and pedicellate* (*P*) *spikelets.*

f. Sterile lemma (*Sl*) *and lemma* (*L*) *are initiated on floret enclosed by outer* (*O*) *and inner* (*I*) *glumes.*

g. Initiation of stamen primordia (*St*) *in florets.*

h. Both stamen (*St*) *and pistil* (*Pi*) *primordia are initiated in the florets of the sessile* (*S*) *and pedicellate* (*P*) *spikelets.*

subsequently differentiate into higher order panicle branches, as shown in Figure 1 d. The appearance of glume ridges (Fig. 1 e) signals the formation of spikelet primordia. Figure 1 f illustrates the lemma and sterile lemma. Anthers are differentiated and surround the pistil primordium, which appears slightly later (Fig. 1g,h). Glumes then quickly enclose the stamens and pistil. A. Dhopte of our laboratory stripped away the glumes to get scanning electron microscope prints illustrating subsequent stamen expansion and development of the bilobed pistil (Fig. 2a-c) terminating in the stigmatic structures (Fig. 2c).

Regarding developmental timing for temperately adapted sorghum at Lincoln, Nebraska (USA), spikelet primordia (Fig. 1 e) differentiate about 10 days after PI, floret differentiation (FD) proceeds at about 2 weeks after PI, and bloom occurs from about 30

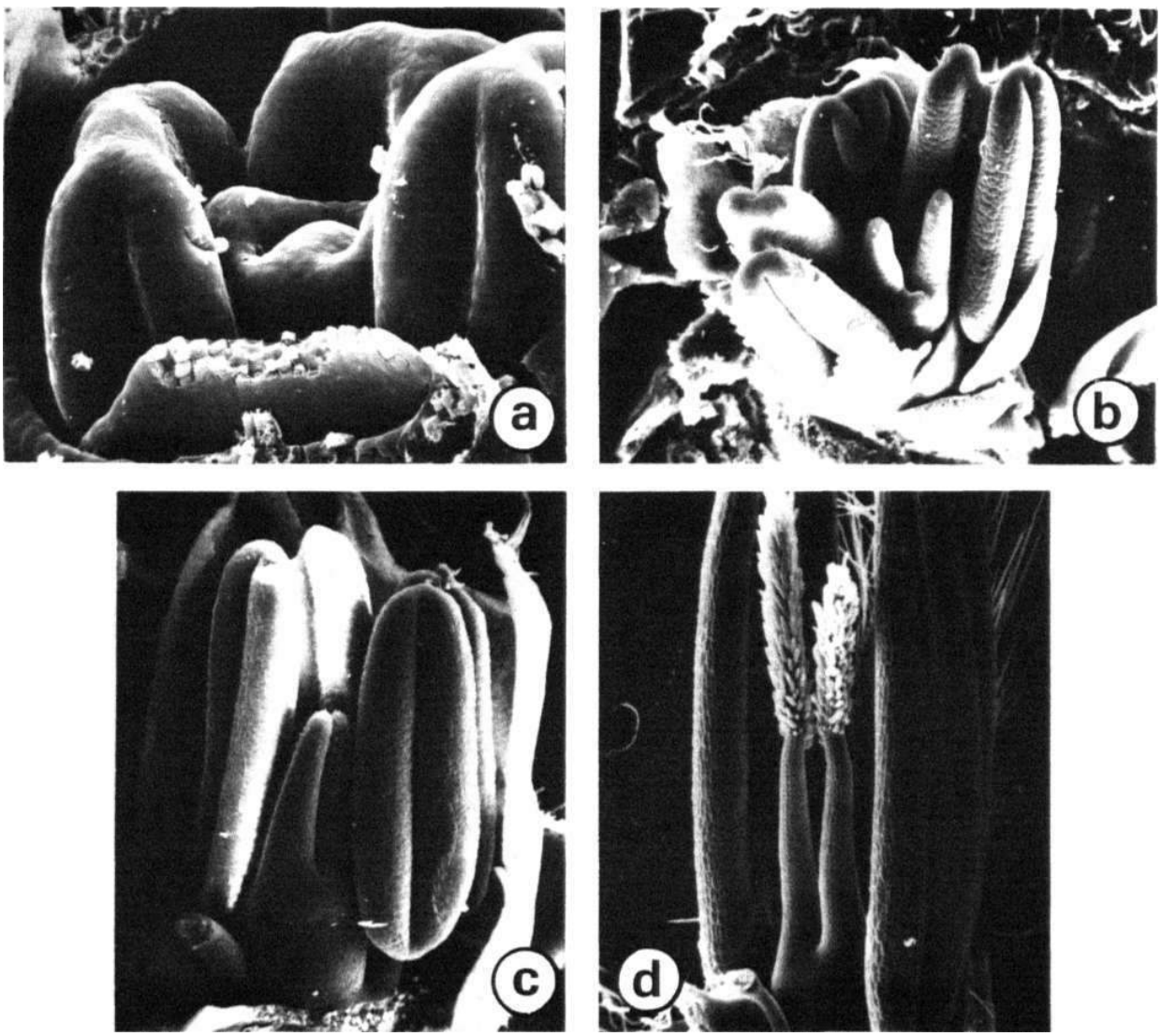

***Figure* 2. *Pistil and anther development in* Sorghum bicolor (L.) *Moench.* (*Source: A.M. Dhopte.*)**
a. *Glumes removed, exposing the three anthers surrounding the bilobed pistil primordium.*
b. Anthers surrounding the developing pistil.
c. Pistil with stigmatic structures.
d. Mature stigmas.

to 35 days after PI. Lengths of the various periods are very temperature-sensitive and are also influenced by daylength in photoperiod-sensitive plants (Lane 1963).

Sensitive Periods

Data from several sources relate to sensitivity to stress during GS2. Musick and Grimes (1961) first showed that the period approaching boot in their treatments was the most sensitive, while Shipley and Regier (1970) found the period from heading to bloom to be slightly more sensitive than mid-to-late boot. Lewis et al. (1974) used treatments that showed boot through bloom to be the most sensitive period.

Castleberry (1973) attempted to expand knowledge of sorghum's reaction to environmental changes by a series of thinnings that altered the amount of light energy available per plant. He thinned sorghum weekly and noted the effects on grain yield and its seed number and seed size components. Yields did not decrease until thinnings were done past the FD stage. While plant population was decreased by one-fourth, the seed number per head increased sufficiently to maintain yields until FD (about 2 weeks after PI, Fig. 1 g). At that point the plant could not compensate in terms of seed number per unit of land area, and the increase in seed size was insufficient to offset the seed number loss, so yields declined.

Building on Castleberry's results, Ogunlela (1979) elevated night temperatures in the field 5°C above ambient during weekly intervals, beginning at PI. The results in Table 2 show that the week after FD begins is the most sensitive. Note that yield per head was reduced proportionately to seed number per head. The obvious times to study mechanisms responsible for yield reductions are during anther and pistil development prior to anthesis. Ogunlela isolated these time periods more specifically than were indicated by the work of Downes (1972) and Eastin et al. (1976).

Dhopte of our laboratory is currently working on defining events causing seed number reductions. Weekly night-temperature treatments similar to those of Ogunlela have been used, plus growth-chamber treatments of 35/17°C, 35/23°C, and 35/29°C (day/night). He has sampled florets at FD, FD + 3, 7, 10, 14, 21, and 25 days (FD + 20 was anthesis). Field and growth-room results are similar. Therefore only growth-room results will be discussed, considering a 23°C night as normal, 29°C as high, and 17°C as cool for the hybrid RS 671.

Microsporogenesis was affected adversely at both 17° and 29°C. Cell vacuolation in the tapetum was somewhat premature. Meiosis was normal; however, meiotic division of the pollen mother cell was occasionally tangential to the tapetum, rather than perpendicular as in control plants. This abnormal relationship with the tapetum probably caused nutritional problems. Microspores past the tetrad stage were dissociated from the tapetum to a substantial degree. Those at 17°C were detached and shrivelled, while those at 29°C were detached but not shrivelled, Nonviability was high in both types. Structural changes occurring at this time appear to cause later pollen abortion.

Structural changes and functional behavior also changed in the tapetum. Vacuolation in the tapeturn apparently led to incomplete engorgement of the microspores, leaving unfilled or partially filled microspores by the time microsporogenesis was half completed. Mitotic division was generally normal at all temperatures, except for an increasing frequency of vegetative nuclei positioning themselves away from the annulus, which is not ideal for pollen germination. An increase of about 50% in pollen sterility resulted.

Table 2. Influence of night temperature on yield and other characteristics of RS 671 grain sorghum at Lincoln, Nebraska, USA, in 1979. Night temperatures (field) were regulated at 5°C above ambient. (Source: Ogunlela 1979.)

Treatment	Grain/ plant (g)	Seed no.	1000-seed wt(g)	Grain/GS3 [a] day/plant (g)
Control	66.9	2659	26.6	2.09
PI_1 to PI_7 [b]	59.3 (-11)[c]	2333 (-12)	27.2 (+2)	1.85 (-11)
PI_8 to FD_1 [d]	53.4 (-20)	2174 (-18)	27.8 (+5)	1.71 (-18)
DF_1 to FD_7	48.0 (-28)	1855 (-30)	29.7 (+12)	1.49 (-29)
FD_8 to BL_1 [e]	52.7 (-21)	2176 (-18)	27.8 (+5)	1.66 (-21)
BL_1 to BL_7 (-16)	55.9	2223 (-16)	25.5 (-4)	1.80 (-14)

a. GS3 = Grain filling stage.
b. PI = Panicle initiation (subscripts are days).
c. Values in parentheses are percent change from the control.
d. FD = Floret differentiation (stamen and pistil primordia).
e. BL = Bloom.

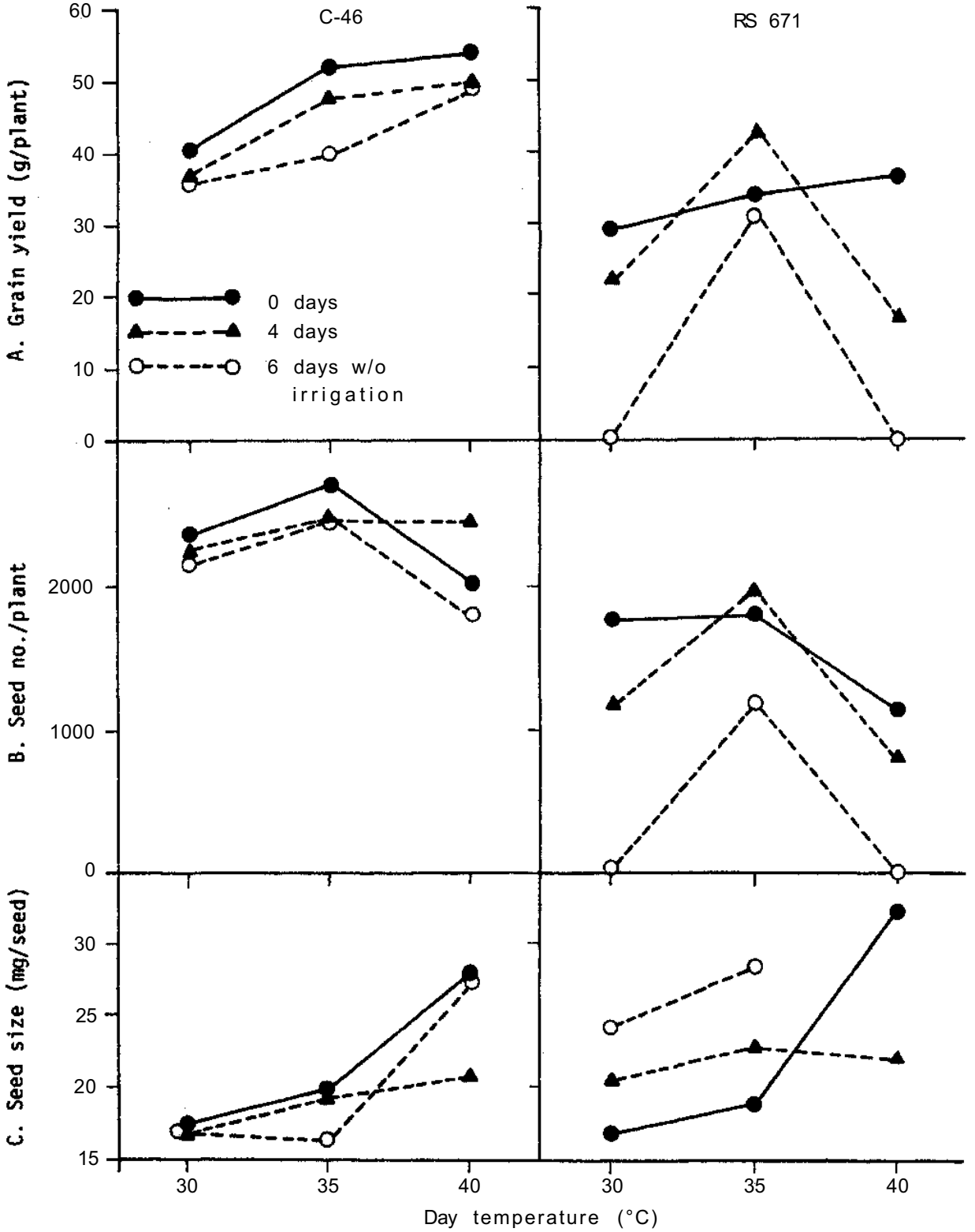

Figure 3. Grain yield **(A)**, ***seed number*** **(B)**, ***and seed size*** **(C)** ***of two sorghum hybrids grown in soil, as affected by temperature and drought stress imposed for 0, 4, and 6 days at the floret differentiation stage. LSDs(0.05) were 2.3 for*** **(A)**, ***576 for*** **(B)**, ***and 8.6 for*** **(C)**. **(*Source: Gonzalez-Hernandez 1982.*)**

Megasporogenesis was also affected adversely. Embryo sac development was normal, as was differentiation and development of the egg apparatus, synergids, polar nuclei, and antipodals. Abortion was preceded by separation of the integuments at the micropylar end and degeneration of nucellar tissue. Widening intercellular spaces led to collapse of the ovule, while the ovary wall remained intact. Of a sample of 30 florets, half had a poorly developed pistil with shrivelled stigmas, and 30% of the ovules were aborted. Results were similar in the 17°C treatments. Seed number per plant was appreciably reduced in both treatments.

The night-temperature elevation treatments of Oguniela (1979) and Dhopte were very modest but were, nonetheless, very effective in reducing seed number, as described by Dhopte above. There was no sign of stress in the plants. One then wonders whether the effects of early infection by stalk- and root-rot organisms may be equally subtle but still deleterious to yield.

Gonzalez-Hernandez (1982) compared a stress-resistant hybrid (C-46) with a normal hybrid (RS 671) in 11 -liter greenhouse pots. Plants were transferred to growth rooms set at 30/22°C, 35/22°C, and 40/22°C. Beginning at FD, water was withheld for 0, 4, and 6 days. Plants were then watered fully until maturity. These experiments are of particular interest because the genotype C-46 is a DeKalb stress-resistant and charcoal-rot-resistant hybrid, while RS 671 is a normal hybrid and is charcoal-rot susceptible. Figure 3 illustrates the extreme grain yield stability of C-46 compared to instability in RS 671. Note the distinct temperature x water stress interactions for RS 671 when water was withheld for either 4 or 6 days. The 40°C temperature in conjunction with water stress was particularly deleterious. Note also that damage was largely a function of seed number reduction. Seed size compensation was adequate to maintain grain yield at high temperatures and low water levels in C-46 but not in RS 671, where seed number losses were more severe.

Photosynthesis (PS), transpiration, and stomatal diffusive resistance were monitored in an effort to discover the mechanisms of stress resistance in C-46 (Fig. 4). Curves for the three plant properties were strikingly similar, except that PS appeared to decline at a slightly higher percentage of soil moisture in RS 671. Photosynthesis, transpiration, and diffusive resistance properties do not help much in explaining yield differences. Growth characteristics did show some differences. The number of new mature leaves that expanded did not appear useful in relating to yield differences (Table 3). In fact, leaf expansion during the 6-day stress period appears to be slightly better for RS 671. However, the relative changes (%) in functional leaf area per plant may be revealing. Note in Table 4 that the relative decline in functional leaf area at 40/22°C under both water stress levels was on the order of three to four times greater in RS 671. DeKalb C-46 apparently has osmotic adjustment characteristics under stress, which permit it to persist. This may relate also to the lower seed number reduction under stress (due to either floret abortion or lack of synthesis of florets). Dhopte's observations regarding tapetum vacuolation, poor development of floral tissues, abortion, etc., may result partially from osmoregulation inadequate to maintain cellular turgor. Blum and Sullivan (University of Nebraska, personal communication, 1983) showed that, of several physiological characteristics measured, only osmotic adjustment correlated with drought

Table 3. Number of new mature leaves expanded by sorghum hybrids C-46 and RS 671 grown in soil, as affected by temperature and drought stress imposed for 6 days during floret differentiation. (Source: Gonzalez-Hernandez 1982.)

Drought period (days)	Temperature (day/night, in °C)							
	C-46				RS 671			
	30/22	35/22	40/22	Mean	30/22	35/22	40/22	Mean
0	4.5	3.5	3.7	3.9	3.0	3.2	2.5	2.9
4	3.2	3.2	1.5	2.6	3.5	2.7	2.2	2.8
6	0.7	0.7	1.0	0.8	2.2	2.0	1.5	1.9
Mean	2.8	2.5	2.1		2.9	2.7	2.1	

LSD (0.05) = 1.08 leaves; CV = 30.0%

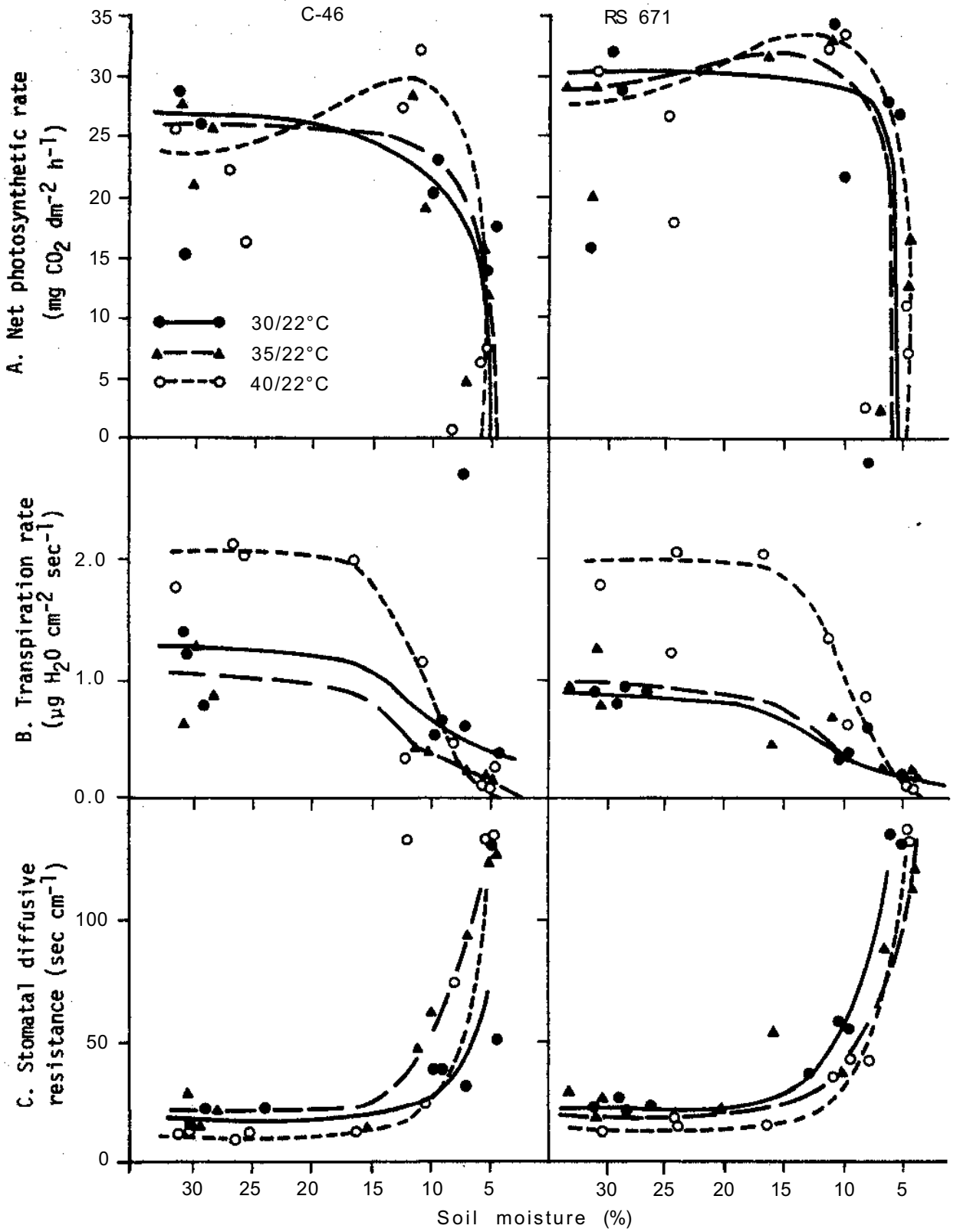

Figure 4. Net photosynthetic rates (A), transpiration rates (B), and stomatal diffuse resistances (C) of two sorghum hybrids as affected by temperature and soil moisture stress imposed at the floret differentiation stage. (Source: Gonzalez-Hernandez 1982.)

Table 4. Relative changes (%) in functional leaf area per plant of sorghum hybrids C-46 and RS 671 grown in soil, as affected by temperature and water deficits imposed for 6 days during floret differentiation. (Source: Gonzalez-Hernandez 1982.)

Drought period (days)	Temperature (day/night, in °C)							
	C-46				RS 671			
	30/22	35/22	40/22	Mean	30/22	35/22	40/22	Mean
0	79.9	63.7	56.9	66.8	70.0	57.7	36.5	54.7
4	19.8	45.4	17.7	27.7	35.9	32.8	-9.1	19.8
6	6.0	-5.4	-16.8	-5.4	-2.2	-4.0	-47.1	-17.8
Mean	35.3	34.6	19.3		34.5	28.8	-6.6	

LSD (0.05) = 29.0%; CV = 83.6%

resistance in sorghums evolved along a geographical rainfall gradient in India, Mali, and Sudan. Jordan et al. (1983) suggested that osmoregulation may permit lowering the threshold value of available soil water at which numerous turgor-dependent essential processes can occur. There is some evidence that the root systems of sorghums related to C-46 do have a high capacity to extract water.

In that respect Hultquist (1973) compared the performances of RS 626 and DeKalb C-42Y (female parent similar to C-46) under greenhouse stress conditions. RS 626, like RS 671, has poor stress tolerance and high charcoal rot susceptibility. When water-stressed during panicle development, the stress-resistant C-42Y transported a relatively greater proportion of its assimilates to the developing panicle than to the roots and lower nodes. C-42Y produced some grain under extreme water stress, whereas RS 626 translocated available assimilates to the roots while the aerial part of the plant died back. Regrowth occurred from the basal nodes when stress was relieved. By contrast, when C-42Y was stressed hard enough for the panicle to die back, the plant died. The growth pattern of RS 626 is geared to survival, while the growth pattern of C-42Y is geared to produce grain in the Great Plains environment of the USA, where rainfall is intermittent and unpredictable but the soils are often deep, with good water-holding capacity. Extensive water extraction capability is essential to production by C-42Y sorghums grown in season-limited and water-limited temperate environments, in contrast to some tropical environments. Concerning this, Hultquist (1973) noted that, while RS 626 exported a greater percentage of its photosynthetically fixed $^{14}CO_2$ to the roots, the root hairs (presumably the active sites) in C-42Y had a higher specific activity, suggesting that they were more active under stress. Perhaps a greater portion of the ^{14}C translocated to RS 626 roots was stored to be used for regrowth once water stress was relieved.

Rice (1979) compared production of C-46 and RS 671 in hydroponics and measured root growth and respiration from panicle initiation to the hard dough stage. Figure 5 shows root dry-matter accumulation to be slightly higher in G-46 during panicle development up to bloom, but differences were not great. By contrast, the root respiration rate in RS 671 was appreciably higher. The matter of root production efficiency then becomes a concern, since evidence given earlier suggests competition between simultaneously expanding above-ground vegetative and floral parts.

Average RS 671 root production day^{-1} divided by mg O_2 consumed per unit of dry weight day^{-1} = 1.30 mg root dry matter produced per mg O_2 consumed per gram of root dry weight. A comparable value for C-46 was 2.48. The division 2.48/1.30 suggests that C-46 was 1.9 times more efficient in root dry-matter production. This would appear to be a significant factor contributing to stability in seed number and yield under stress and in predisposition to diseases.

Another related C-46 characteristic appears interesting during grain fill. Note (Fig. 5) that from soft dough to maturity, when seed assimilate demand may be declining somewhat, the respiration rate in C-46 roots increased sharply while root respiration in RS 671 stayed flat or declined slightly. Just preceding and during this period, RS 671 expressed severe charcoal rot susceptibility, while

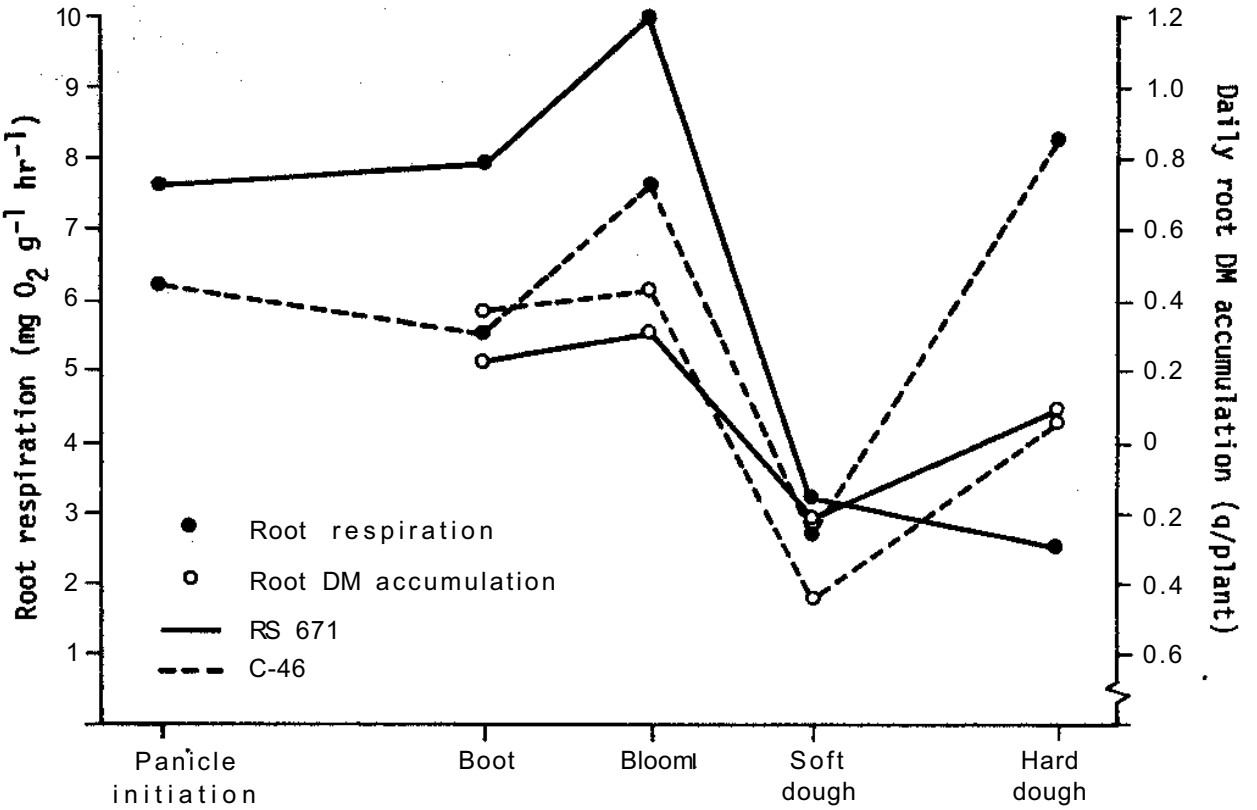

Figure 5. Root respiration and daily root dry-matter (DM) accumulation in RS 671 and DeKalb C-46 grain sorghum, under controlled conditions, from panicle initiation through the hard dough stage of development (Source: Rice 1979.)

C-46 was charcoal rot resistant. The ability of C-46 to maintain metabolic activity (exhibit perennial tendencies) must relate to its stalk-rot-resistance tendencies.

Bennett (1979) found sorghum sensitivities to water stress in hydroponics very similar to those shown by Hultquist (1973), Ogunlela (1979), and Gonzalez-Hernandez (1982). Bennett also noted a relatively high level of sensitivity right at bloom compared to slightly later.

Grain Development (GS3)

The sensitivity that Bennett (1979) recorded was both in loss of stomatal control about 3 days after bloom and in loss of seed number from stress right at bloom. Dickinson (1976) tested for sensitivity during grain fill by placing plastic bags over panicles every 3 days after anthesis and leaving them for different time periods. Temperature elevations for varying times created different stress levels. Seed abortion was generally modest. Effects on seed size limitations, however, were substantial. Stress applied 7 to 9 days after anthesis reduced seed size drastically. The influence could have been the inhibition of endosperm cell division, decreasing cell-wall elasticity, or other processes related to cell division and/or cell expansion.

Physiological relationships between stalk rots and grain-fill events are not well understood. It is clear that simultaneous heat and water stress, generally during the first 2 weeks of grain fill, are necessary for a serious attack of charcoal rot. Eastin (1972) showed that substantial ^{14}C-labelled photoassimilates were translocated to roots up to bloom. After that, increasingly larger percentages of labelled assimilates were translocated to the developing seeds. Whether or not lesser quantities of assimilate render roots more susceptible to invasion and damage by stalk rot organisms is not clear. As pointed out earlier, Rice's (1979) data do show that root respiration in C-46 (stalk rot resist-

ant) more than doubles between the soft dough and hard dough stages, while the respiration rate in RS 671 falls slightly, The increased root activity may relate to charcoal rot resistance in C-46. The higher respiratory efficiency of C-46 may also be a positive factor when photoassimilates are low due to water and heat stress.

The matter of temperature influence on respiration rate and its potential influence on the efficiency of respiratory energy utilization is intriguing. The suggestion that temperature may influence the efficiency of respiratory energy utilization comes from preliminary data in our laboratory. The test system involved seedling growth in the dark. Seeds of several genotypes were weighed and germinated (radicle appearance) at 22°C, and subsequently split into lots and grown in the dark for 5 to 7 days at 20°C, 25°C, 30°C, 35°C, and 40°C. The growth was then separated from the seed remnant, and both were dried and weighed. The ratio of grams of growth per gram of seed weight lost was used as an index of metabolic or growth efficiency. Most genotypes were similar at 30°C, but divergence at 5 to 10°C on either side of 30°C revealed significant differences. Some genotypes had high efficiencies at cool temperatures and some had high efficiencies at high temperatures, suggesting that one should be able to choose a genotype with an appropriate temperature response to fit a given temperature environment.

Given this generality, plus the notion that respiration is tightly coupled to many of the synthetic processes dictating plant growth, Gerik (1979) checked to see what kind of genotype variability in respiratory response to temperature might exist in the field. He checked respiration rates in the field in sorghum panicles in a random-mating population (fertile S_1 heads) during three different times of day to get three different temperatures. Table 5 shows the responses. First, temperature had a marked effect on respiration rate, as expected, in the 50 panicles sampled. Second, and more importantly, the ranges in respiration rates at each respective temperature were 1 to 2 times greater than the mean respiration rates. Results were confirmed in other populations. Obviously, great variability exists in respiration rates at any given temperature. Therefore, if temperature response is important in maximizing metabolic or growth efficiency, as appears to be the case (see Fig. 3), one should be able to select appropriate genotypes to fit various temperature environments and minimize stress effects.

Table 5. The mean, range, and coefficient of variation for panicle dark respiration at 17, 21, and 24°C for 50 randomly selected plants in the random-mating grain sorghum population. (Source: Gerik 1979.)

Temperature (°C)	Dark respiration (mg CO_2 evolved (g dry wt)$^{-1}$ hr^{-1}) 50-plant means	Range	Coefficient of variation (%)
17	0.51	0.21-0.90	30.7
21	0.72	0.36-1.18	39.5
24	1.20	0.50-2.75	33.9

Future Research Priorities

Some of the factors bearing on future research are that sorghum is relatively insensitive to heat and water stress during the vegetative stage. Stress has variable effects during panicle development, with the most sensitive times being about 3 to 6 days after FD (i.e., during microsporogenesis) and 7 to 11 days after FD (at megasporogenesis). Postanthesis sensitivities occur at 7 to 9 days, when difficulties can cause restrictions in seed size. Substantial heat and drought stress after anthesis predisposes sorghum to charcoal rot. This coincides with the time when increasing proportions of photoassimilates are transferred to the developing grain and decreasing amounts of assimilates go to the roots. One charcoal-rot-resistant hybrid retains a high level of root respiration during the dough stages, which may relate to stalk rot resistance. High metabolic efficiency in root growth may also be a factor contributing to stress resistance in general. Plant response to temperature may have a bearing on metabolic efficiency and predisposition to diseases.

Future research should be concerned with osmoregulation as it might relate to soil water extraction and turgor maintenance in florets during microsporogenesis and megasporogenesis, Partitioning of photoassimilates among competing plant parts and/or organisms may influence osmoregulation or be influenced by it. Differences in plant metabolic efficiency or dry-matter production efficiency at different temperatures need to be considered in relation to possible predisposition of plants to diseases. Consideration should be given to

selecting genotypes with appropriate temperature responses to fit a given environment.

Several types of investigation were cited to illustrate the sensitivities of grain sorghum to water and temperature stress. Similar types of experiments should be done superimposing stalk and root rot organisms on water and temperature treatments. Genotype x disease x environment interactions need to be defined, the mechanisms responsible for damage exposed, and the information used to devise cultural and/or genetic solutions for either avoiding or tolerating stalk and root rot diseases.

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References

BEIL, G.M., and ATKINS, R.E. 1967. Estimates of general and specific combining ability in F_1 hybrids for grain yield and its components in grain sorghum, *Sorghum vulgare* Pers. Crop Science 7:225-228.

BENNETT, J.M. 1979. Responses of grain sorghum (*Sorghum bicolor* (L) Moench) to osmotic stresses imposed at various growth stages. PhD. thesis, University of Nebraska, Lincoln, Nebraska, USA.

BLUM, A. 1967. Effect of soil fertility and plant competition on grain sorghum panicle morphology and panicle weight components. Agronomy Journal59:400-403,

BLUM, A. 1970. Heterosis in grain production by sorghum. Crop Science 10:28-31.

CASTLEBERRY, R.M. 1973. Effects of thinning at different growth stages on morphology and yield of grain sorghum *(Sorghum bicolor* (L.) Moench). Ph.D. thesis, University of Nebraska, Lincoln, Nebraska, USA.

DICKINSON, T.E. 1976. Caryopsis development and the effect of induced high temperatures in *Sorghum bicolor* (L) Moench. M.Sc. thesis, University of Nebraska, Lincoln, Nebraska, USA.

DOGGETT, H., and JOWETT, D. 1967. Yield increase from sorghum hybrids. Nature (London, U.K.) 216:798-799.

DOWNES, R.W. 1972. Effect of temperature on the phenology and grain yield of *Sorghum bicolor.* Australian Journal of Agricultural Research 23:585-594.

EASTIN, J.D. 1972. Photosynthesis and translocation in relation to plant development. Pages 214-216 *in* Sorghum in seventies (eds. N.G.P. Rao and L.R. House). New Delhi, India: Oxford & IBH Publishing Co.

EASTIN, J.D. [1984.] Sorghum development and yield. In proceedings of the Symposium on Potential Productivity of Field Crops under Different Environments (ed. S Yoshida), Los Banos, Laguna, Philippines. 22-26 Sept 1980. Los Banos, Laguna, Philippines: International Rice Research Institute, (in press.)

EASTIN, J.D., BROOKING, IAN, and TAYLOR, S.A. 1976. Influence of temperature on sorghum respiration and yield. Page 71 *in* Agronomy Abstracts. Madison, Wisconsin, USA: American Society of Agronomy.

EASTIN, J.D., CASTLEBERRY, R.M., GERIK, T.J., HULTQUIST, J.H., MAHALAKSHMI, V., OGUNLELA, V.B., and RICE, J.R. 1983. Physiological aspects of high temperature and water stress. Pages 91 -112 *in* Crop reactions to water and temperature stresses in humid, temperate climates (eds. C.D. Raper, Jr., and P.J. Kramer). Boulder, Colorado, USA: Westview Press.

EASTIN, J.D., and LEE, K.-W.[1984.] *Sorghum bicolor* (L) Moench. *in* Handbook of flowering (ed. A.H. Hallevy) Boca Ratan, Florida, USA: Chemical Rubber Co. Press (in press).

EASTIN, J.D., and SULLIVAN, C.Y. 1974. Yield considerations in selected cereals. Pages 871 -877 *in* Mechanisms of regulation of plant growth (eds. R.L. Bielski et al.), Bulletin No. 12. Wellington, New Zealand: Royal Society of New Zealand.

FISCHER, K.S., and WILSON, G.L 1975. Studies of grain production [*Sorghum bicolor* (L) Moench]: V. Effect of planting density on growth and yield. Australian Journal of Agricultural Research 26:31-41.

GERIK, T.J. 1979. The relationship of photosynthesis and dark respiration in grain sorghum [*Sorghum bicolor* (L.) Moench] to yield, yield components, and temperature. Ph.D. thesis, University of Nebraska, Lincoln, Nebraska, USA.

GONZALEZ-HERNANDEZ, V.A. 1982. Sorghum responses to high temperature and water stress imposed during panicle development. Ph.D. thesis, University of Nebraska, Lincoln, Nebraska, USA.

HEINRICH, G.M., FRANCIS, C.A., and EASTIN, J.D. 1983. Stability of grain sorghum yield components across diverse environments. Crop Science 23:209-212.

HULTQUIST, J.H. 1973. Physiologic and morphologic investigation of sorghum *[Sorghum bicolor* (L.) Moench]: I. Vascularization; II. Response to internal drought stress. Ph.D. thesis, University of Nebraska, Lincoln, Nebraska, USA.

JORDAN, W.R., DUGAS, W.A., and SHOUSE, P.J. 1983. Strategies for crop improvement for drought prone regions. Agricultural Water Management 7:281-299.

KAMBAL, A.E., and WEBSTER, O.J. 1966. Manifestation of hybrid vigor in grain sorghum and the relations among the components of yield, weight per bushel and height. Crop Science 6:513-515.

LANE, J.C. 1963. Effect of light quality on maturity in the milo group of sorghum. Crop Science 3:496-499.

LEE, K.-W., LOMMASSON, R.C., and EASTIN, J.D. 1974. Developmental studies on the panicle initiation in sorghum. Crop Science 14:80-84.

LEWIS, R.B., HILER, E.A., and JORDAN, W.R. 1974. Susceptibility of grain sorghumto water deficit at three growth stages. Agronomy Journal 66:589-591.

MUSICK, J.T., and GRIMES, D.W. 1961. Water management and consumptive use of irrigated sorghum in Western Kansas. Kansas Agricultural Experiment Station Technical Bulletin No. 113. Manhattan, Kansas, USA: Kansas State University. 20 pp.

OGUNLELA, V.B. 1979. Physiological and agronomic responses of grain sorghum *[Sorghum bicolor* (L.) Moench] hybrid to elevated night temperatures. Ph.D. thesis, University of Nebraska, Lincoln, Nebraska, USA (Diss. Abstr. 80:10871).

PAULSON, I.W. 1962. Embryology and seedling development to floral transition of *Sorghum vulgare* Pers. Ph.D. thesis, Iowa State University, Ames, Iowa, USA.

QUINBY, J.R. 1963. Manifestation of hybrid vigor in sorghum. Crop Science 3:288-291.

RICE, J.R. 1979. Physiological investigations of grain sorghum [*Sorghum bicolor* (L) Moench] subjected to water stress conditions. Ph.D. thesis, University of Nebraska, Lincoln, Nebraska (Diss. Abstr. 40(1 -2): 527B). University Microfilm no. 7918697, Ann Arbor, Michigan, USA.

SCHOENEWEISS, D.F. 1975. Predisposition, stress, and plant disease. Annual Review of Phytopathology 13:193-211.

SHIPLEY, J. and REGIER, C. 1970. Water response in the production of irrigated grain sorghum, High Plains of Texas, 1969. Texas Agricultural Experiment Station Progress Report No. 2829. College Station, Texas, USA: Texas A&M University. 24 pp.

STICKLER, F.C., PAULI, A.W., LAUDE, H.H., WILKINS, H.D., and MINGS, J.L. 1961. Row width and plant population studies with grain sorghum at Manhattan, Kansas. Crop Science 1:297-300.

WHITEMAN, P.C., and WILSON, G.L. 1965. Effects of water stress on the reproductive development of *Sorghum vulgare* Pers. University of Queensland Papers (Queensland, Australia) 4:288-289.

YARWOOD, C.E. 1959. Predisposition. Pages 521-562 *in* Vol. 1. Plant Pathology (eds. J.G. Horsfall and A.E Diamond). New York, New York, USA, and London, U.K.: Academic Press. 674 pp.

Questions

Partridge:
In your paper was any cognizance made of the presence, absence, or pathogenicity of any internal parasite and/or their potential role affecting your conclusions?

Eastin:
Tests for the presence of pathogens were not made. Plants were green and healthy in appearance. Pathogenicity was not considered in these presumed normal plants.

Physiological and Environmental Factors in Root and Stalk Rot Diseases

Summary and Synthesis

D.F. Schoeneweiss*

The excellent papers presented in this section provide a wealth of information on physiological and environmental factors associated with lodging, senescence, and root and stalk rot diseases in monocot grain crops. Much of the information has been derived from studies on maize and extrapolated to sorghum because of the similarity of the two species. In most cases, results obtained from research on maize appear to apply to sorghum as well, but such interpretations should be confirmed before they are accepted as valid. Although patterns of growth, senescence, nutrient transport, and source-sink relations are similar and basically the same pathogens are involved in root and stalk rots of maize and sorghum, the two species not only differ genetically but are often grown in different areas of the world under different cropping practices. It seems questionable Whether high management approaches to control disease in maize (i.e., breeding for narrow-based genetic characters, modifications in row spacing, and tillage practices or soil fertility management) can be incorporated into marginal sorghum cropping systems in developing countries in the semi-arid tropics. Sorghum lines that produce satisfactory yields under highly variable, marginal growing conditions will need to possess broad-based genetic tolerance to physical and environmental stresses that are involved in predisposition to root and stalk rot pathogens.

There appears to be a consensus among researchers that any factor that contributes to plant vigor, particularly to a retardation of senescence of pith parenchyma cells, enhances resistance to root and stalk rots. Unfortunately, the mechanisms of resistance or defense reactions of parenchyma cells to pathogen attack in grain sorghum are essentially unknown. The weight of evidence strongly indicates that the amount and distribution of nonstructural carbohydrates are closely correlated with stalk rot resistance, as summarized in the photosynthetic stress-translocation balance concept proposed by Dodd (1980). Physiological and environmental factors that predispose sorghum to stalk rots cause either a reduction in synthesis of carbohydrates or a depletion in nonstructural carbohydrates due to uneven transport to the grain sink. Since carbohydrate metabolism is involved in most physiological processes in higher plants, many hypotheses could be advanced to explain the role of carbohydrates in host resistance. Albersheim et al. (1969) stated that high levels of glucose repress the synthesis by fungal pathogens of polysaccharide-degrading enzymes that are universally involved in plant pathogenesis. The close association between sugar levels and predisposition to stalk rot fungi in grain sorghum supports this concept and merits further research.

Wavelike or sequential senescence from base to top is characteristic of grain sorghum. In genotypes possessing nonsenescence or delayed progressive senescence characters, carbohydrate levels remain high longer and parenchyma cells remain physiologically active into later stages of plant maturity. After black layer formation in the kernel,

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Pappelis:
I believe this lack of contact will occur only in a severe disease situation.

McBee:
The model would need to have a phenology component in that susceptibility to stress may vary with stage of development, e.g., pre- or postanthesis.

Drought Resistance

Mughogho:
In my experience at ICRISAT the model is supported. However, a question arises from this: Is a drought-resistant plant (i.e., one that resists damage in the absence of a pathogen even under large stress) also resistant to disease?

Duncan:
In sorghum, Rosenow considers that there are two types of drought-resistance: one expressed during preflowering and the other during postflowering. We need to consider the latter in relation to stalk rot pathogen resistance. There is genetic variation for both types Of drought stress, but no known genotypes are resistant to both.

Yield Reductions/Energy Loss

Jordan:
Dr. Mughogho, you say you accept the model. What explanation do you have for the increased grain yield reduction caused by the pathogen?

Mughogho:
Literature suggests that the reduction is through decreased grain size, but the same literature does not separate the effects of the pathogen from the effects of the predisposition factors, although Mayers in Australia has some data which show that there is an added reduction in yield in stalk-rotted plants and that it is due to reduction in both grain size and number.

Eastin:
What are the energy costs to the plant from fungal invasion?

Jordan:
There may not be much energy loss due to tissue destruction by the pathogen since very little additional material would be moved to the grain in the absence of the pathogen.

Schneider:
I would like to make a point here that with fusarium or charcoal rot where invasion is primarily into dead cells there is probably no energy loss, but in the situation with *Colletotrichum,* which invades living tissue, there will be a net energy loss.

Temperature Effects

Maunder:
I'd like to hear a discussion of temperature as well as moisture deficits.

Jordan:
We don't know the role of temperature in the model.

Partridge:
Pathologists have information on the effect of temperature on fungal growth. The problem is finding the effect of temperature on the interaction of the host and pathogen. One problem is finding a control, i.e., an uninfected plant.

Nutrient Effects

Maunder:
Are plants subjected to a sudden moisture stress more likely to develop stalk rot than those grown under continuous, low-level stress, and is it true that high nitrogen will induce greater stalk rot incidence?

Clark:
Yes, there is a relationship between N and stalk rot.

Schneider:
Leaf area/root ratio is high in N-fertilized wheat plants, and these therefore are more likely to run into stress.

Pappelis:
We've shown that cell senescence is increased by high N.

Partridge:
I don't know of any evidence suggesting that plants can outgrow fungi in response to nutrition.

Jordan:
We have data that show that both grain yield and stalk rot increase with N.

Nonsenescence

Seetharama:
Nonsenescence is associated with a cost, and that is the production of extra roots. This is of little use in the Indian situation where soils are shallow. Secondly, I do not consider that all drought resistant genotypes are charcoal rot resistant.

Rosenow:
Dr. Duncan, please comment on your stated association of nonsenescence with anthracnose.

Duncan:
Yes, there seems, in our environment, to be an association; for example, in sorghum lines SC-170, SC-56, and SC-599.

Scheuring:
Please comment on the genotype x environment interaction problem with expression of nonsenescence.

Duncan:
We don't have this problem in southeastern USA.

Rosenow:
In Texas we do get the genotype x environment problem in evaluating nonsenescence. It's probably related to the cause of the senescence; for example, the cause is moisture stress in West Texas, whereas in other areas it may be leaf disease, insecticide burn, etc.

Maunder:
What does the F_2 look like in a senescent x nonsenescent cross?

Rosenow:
In some crosses nonsenescence acts as a recessive character and in others the opposite. We need to know more about this.

Stalk Quality

Pappelis:
The sorghum people must not lose sight of the two aspects of the lodging problem: stalk rot resistance and stiff stalk characteristics.

Maunder:
Where does morphology fit into breakage of stalks?

Maranville:
I would like to ask another question. Are the anatomical characters associated with lodging resistance also found in the nonsenescent types? I would also like to comment that it appears that selecting for stalk quality characters, for example, crushability, also carries along resistance to stalk rots.

Duncan:
We found larger stem base diameter in nonsenescent types.

Rosenow:
We select simultaneously for both characters.

Zummo:
The variety Brandes is an exceptionally good stander—no one knows why. Certainly there is no apparent anatomical reason for it. Its disease resistance, I consider, is not the cause.

Pappelis:
There are problems in using crushability: for example, stem segments high in sugar when dried under heat will turn out like bricks; these should be considered artifacts.

McBee:
The variety Giza, which has a very stiff stalk, has a very high lignin content in the stem.

Maranville:
Crushing strength correlates with rind puncture—$r = 0.98$.

Pappelis:
In maize, rind puncture taken at preflowering is a very useful tool. I think it should be adopted by sorghum workers.

Maunder:
Most sorghum breeders would agree that selecting for stalk quality is an important component of selecting for lodging resistance.

Partridge:
I agree that significant progress has been made in

maize in selecting for lodging resistance via stalk morphological characters, but I make a plea that breeders do not neglect the pathological aspects of this problem.

Mughogho:
I would like to support Dr. Partridge in that, in sorghum, lodging resistance based on physiological/pathological characters will at times break down, and it's then that stalk quality characters will assume importance.

Carbohyclrate Relationships

Rosenow:
Dr. McBee, your data reported today were collected under well-watered conditions.—Do you have any from moisture-stress situations?

McBee:
Dr. Fred Miller [Professor, Texas A&M University, College Station, USA] had a student who found that ATx623 x RTx423 yielded very well under drought conditions—there is some correspondence.

Schneider:
You reported a plant spacing effect. What was that?

McBee:
Plants spaced closely in the row had a higher percentage of nonstructural carbohydrates in the stems in both senescent and nonsenescent types.

Schneider:
So spacing affects translocation patterns? In maize there is good evidence that spacing affects stalk rot due to *Fusarium* and *Verticillium* spp. Maybe your finding provides an explanation for this.

McBee:
This seems relevant, and as a result of this meeting I'm going to take much more notice of pathogens in the stalk. The distribution of carbohydrate may be of importance. For example, the sucrose level tends to be uniform all along the stalk; but the glucose level is high in the top early in plant development, and then later there is more glucose in the base.

Schoeneweiss:
Very rarely has it been demonstrated that pathogen growth is limited by host nutrients. More likely high CHO has a suppressant effect on pathogen growth.

Pappelis:
The sugar level in maize stalk tissue is irrelevant. The question is whether it's dead or not, You must look at the cell level.

Grain Yield Relationships

Rosenow:
Dr. Eastin, in your presentation you talked about differences in drought resistance with respect to grain yield. Is this average yield or yield under stress conditions?

Eastin:
Average yield over a wide range of environments.

Environment/Temperature

Seetharama:
I would like to show data from an experiment involving one genotype and four planting dates and the response in yield and stalk rot incidence as it is affected by the environment, specifically temperature. The first planting in September was inferior and the fourth planting in November was superior, both in highest yield and lowest stalk rot incidence. Early growth was affected by temperature extremes in all cases, but in the fourth planting growth was very fast after flowering. This was a time of increasing temperature, which was also critical for this important grain-filling period. This illustrates the importance of environment during a period of high grain filling.

Carbohydrate Relations to Stress

Partridge:
Dodd's photosynthetic stress-translocation concept has been often quoted in papers prepared for this meeting. It's been out now for 6 or 8 years. Does anyone have any experimental evidence to support or refute it?

Mughogho:
Chamberlin's Ph.D. degree thesis does not support it in that his research did not indicate that mobilization occurred in response to stress.

Partridge:
I take it then that Dodd's hypothesis is not supported.

Mughogho:
No, not entirely, because Chamberlin looked at only two genotypes.

Henzell:
I believe that the crux of Dodd's hypothesis is that the shortage of carbohydrate in the stem and roots results in cell death and consequent predisposition to pathogen attack. If Dodd is saying that reallocation of assimilate in response to stress contributes to this CHO shortage in the stem, then Chamberlin's work does not support this part of his hypothesis.

Scheuring:
This kind of discussion on CHO content of stems has problems unless we can talk about CHO content at the cellular level.

Henzell:
Dodd considers this purely hypothetical.

Pappelis:
This is not hypothesis: it cannot be tested and therefore it is pure speculation. Let's call it what it is.

Partridge:
I agree with Dr. Pappelis.

Experience with Root and Stem Rots of Crops Other than Sorghum

The Maize Root Rot, Stalk Rot, Lodging Syndrome

A.J. Pappelis and J.N. BeMiller*

Summary

Studies of diplodia and gibberella stalk rots of maize and related breeding programs were advanced by the recognition that stalk rot resistance is associated with living parenchyma cells and stalk rot susceptibility is associated with dead cells. Anthracnose stalk rot of sorghum has a similar etiology.

In maize, injury to roots, stalks, or leaves and water stress accelerate the expression of parenchyma cell death in roots and stalks. Root rot pathogens spread upward into the stalks as areas of dead cells in these are linked. Hence, one way to prevent (or delay) root and stalk rotting is to prevent (or delay) parenchyma cell death. We established that nuclear and nucleolar degeneration, loss of tRNA methylase activity, abnormal protein synthesis, and increased synthesis and activity of nucleases and proteases precede cell death. We propose that research (cytological, biochemical, physiological, pathological) needs to be continued to determine the effects of genes and environment on the expression of cell death patterns, to develop practical methods to delay cell death (related to disease responses), and to determine the nature of resistance and susceptibility to major fungal pathogens that incite root rot, stalk rot, and lodging in maize and sorghum.

The root rot, stalk rot, lodging (RSL) syndrome is economically more important than any other disease of maize. Annual world losses due to the RLS syndrome exceed 1 billion bushels. Thirty years ago, the nature of resistance to the RSL syndrome was considered too difficult to solve and unimportant in that period of surplus. However, in 1954, a team effort was begun by A.L. Hooker, A.J. Pappelis, and F.G. Smith to seek a physiological basis for resistance to diplodia stalk rot. This effort led to the discovery that susceptibility to the disease was due to parenchyma cell death (pithiness); resistance to spread was associated with living cells. These research efforts resulted in a change in attitude. Data now exist that permit a better understanding of the nature of resistance and susceptibility of maize to several stalk rot pathogens that play a central role in the RSL syndrome. We have extended these principles to similar diseases in sorghum.

Information on the sequential diseases of maize should not be separately analyzed, since a combination of diseases causes a reduction in field stands, reduced health and vigor of plants surviving seedling stages of growth, root and stalk rotting and lodging in developing and maturing plants, and a reduction in yield and grain quality at maturity. The interrelationships between parasitic organisms, the genetic constitution of the host, the expression of host and pathogen genes, cultural practices, and the age and physiological state of the host must be considered as a function of variations in environment throughout the season. A number of review articles present various aspects of the problem, state some of the major contributions to this field of study, and give a sense of direction to the present

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research (Bruehl 1983; Christensen and Wilcoxson 1966; Hooker 1976,1978; Koehler 1960; Pappelis et al. 1971; Schneider and Pendery 1983; Shurtleff 1980; Thompson 1970; Twumasi-Afriyie and Hunter 1982a, 1982b; Ullstrup 1961,1977; White et al. 1979).

Root rots that begin during seed germination, or as seedlings develop, can predispose stalks to stalk rot. Pathogens from rotted roots spread upward and eventually penetrate the crown (below-ground internodes and nodes). Crown rot may remain quiescent for weeks. If rotting extends upward through brace roots, basal stalk rot may become severe. Severe root rotting can cause root lodging, and thus cause direct yield loss.

In addition to spreading from roots to stalks, stalk rot pathogens can penetrate the mature stalk directly and through corn [maize] borer tunnels. Several stalk-rotting fungi penetrate nodes. Others attack the rind. Extensive spread in the stalk may result in stalk lodging or breakage that interferes with machine harvesting, thus causing indirect yield loss.

By producing maximum disease response (or predicting it) at any geographic location, researchers have improved our understanding of the underlying causes of disease resistance and susceptibility. Because of the importance of these methods to maize breeding, we will briefly describe their use. We will, in particular, discuss the pith condition rating system, based on the distribution of dead parenchyma cells. In doing so, we will review research on stalk rots incited by *Diplodia maydis* (Berk.) Sacc., *Gibberella zeae* (Schw.) Petch, and *Fusarium moniliforme* Sheld. These three pathogens can incite seed rot, seedling root rot and blight, root and stalk rot of maturing plants, root and stalk lodging, shank rot, and ear rot. The factors for resistance to diplodia and gibberella stalk rots are considered to be the same or closely related (Hooker 1956). Fusarium stalk rot is difficult to distinguish from gibberella stalk rot (Shurtleff 1980, Ullstrup 1977). We propose that parenchyma cell death in root and stalk tissue predisposes maize to root and stalk rots incited by these fungal pathogens.

RSL Syndrome

It is generally believed that stalk rot infections start from roots (Britton and Hooker 1963, Craig and Hooker 1961, McKeen 1953, McNew 1937, Pappelis 1970a, Pappelis and Boone 1966b, Schneider and Pendery 1983, Whitney and Mortimore 1957) and through nodes of the lower stalk (Durrell 1923, Pappelis and Boone 1966b). Susceptibility increases in all plants with time after flowering. Late-maturing cultivars are more resistant to naturally occurring basal stalk rot than those maturing earlier (Koehler 1960).

Incidence of diplodia stalk rot has been shown to be highly correlated with susceptibility to artificial inoculation, as is the incidence of natural infection with broken stalks (Smith et al. 1938, Cloninger et al. 1970, Horrocks et al. 1972). Methods of inoculation were reviewed by Koehler (1960). Although stalk-lodging resistance has been improved over the past four decades, stalk breakage and stalk rot continue to be a maize production problem (Zuber 1983).

Hooker (1957) found a progression in internode susceptibility to stalk rot following inoculation: the lowest elongated internode above the uppermost brace roots were the least susceptible, and the fifth internodes above the uppermost brace roots were the most susceptible. He recommended that similar internodes be inoculated to measure comparative resistance to stalk rot among maize plants or varieties. Inoculation of the first (basal) or second elongated internode above the ground between 1 and 3 weeks after silking was recommended as most satisfactory for this purpose, with stalk rot ratings preferably made 4 weeks after inoculation. This procedure classifies inbreds and hybrids into disease-response groups that are highly correlated with natural stalk rot observations. In susceptible cultivars, the rate of spread of the inoculum is rapid in the first 2 weeks after inoculation. In cultivars of intermediate resistance, the rate of spread is less rapid but constant during the 4-week interval following inoculation. In resistant cultivars, no spread occurs after the 1 st week following inoculation. However, later in the season the rate of spread changes and all cultivars become susceptible (Pappelis 1957).

Parenchyma Cells and Resistance to RSL Pathogens

Pappelis (1957, 1965) and Pappelis and Smith (1963) were the first to relate resistance to the spread of *D. maydis* and G. *zeae* with living parenchyma cells of nodal and internodal tissue, and areas of susceptibility to these pathogens with

areas of stalk tissue composed primarily of dead parenchyma. These observations also apply to anthracnose stalk rot of sorghum incited by *Colletotrichum graminicola* (Cesati) Wilson (Katsanos and Pappelis 1965, 1966a, 1966b, 1967, 1968, 1969a, 1969b; Pappelis and Katsanos 1966) and red rot of sugar cane incited by *Physalospora tucumanesis* Speg. (Bare et al. 1971; Pappelis and Katsanos 1965a, 1965b; Schmid et al. 1966). As plants of these three species undergo developmental changes associated with flowering, the number of dead stalk parenchyma cells increases greatly. Areas of dead cells in internodes are observed as white tissue between vascular bundles. In each species, the patterns of stalk cell death vary from basal to upper internodes. Discoloration associated with stalk rot response following inoculation occurs where living cells are present along the vascular tissue, rind, and in the nodes. Injuring plants during this developmental period changes the rate of cell death in stalks: cutting roots or removing leaves increases the rate and removal of the ear of maize and inflorescence of sorghum delays it. There is much additional support for the conceptual scheme of host-pathogen interaction in maize involving *D. maydis* and G. *zeae* (Gates 1970; Kang et al. 1974; Pappelis 1970a, 1970b; Pappelis and Boone 1966a; Pappelis et al. 1971, 1973a; Pappelis and Katsanos 1969).

Rating Systems for Pith Condition and Stalk Rot

Pappelis (1957) and Hooker (1957) used the same diplodia stalk rot rating system for inoculated maize plants. The pith-condition system developed by Pappelis (1957) was based on the same numerical rating units used for tissue discoloration following inoculation, but was limited to one internode. These rating systems and the high correlations between them were described by Pappelis and Smith (1963). The two systems were expanded to improve both ends of the rating scales (Pappelis 1963, 1965, 1970a, 1970b; Pappelis and Boone 1966b). Cell death in nodal tissue (after massive cell death in internodes) was highly correlated with naturally occurring stalk rots that spread from rotted roots and penetrated the stalk through nodes. If no cell death occurs in the pith tissue, the pathogen does not spread in the inoculated internode.

The improved diplodia stalk rot rating system was as follows: 0.0 = less than 1% of inoculated internodes discolored; 0.5 =1-12.5% discolored; 1 = 12.6-25%; 2 = 26-50%; 3 = 51 -75%; 4 = 76-100%; 4.5—like 4, with less than 50% of the adjacent internode discolored; 5—like 4, with more than 50% of the adjacent internode discolored; 5.3 = discoloration of three internodes (including the inoculated internode); 5.4 = discoloration of four internodes; 5.5 = discoloration of five or more internodes; and 6 = premature death of plant. The report of Hooker et al. (1962) contained a modification of the improved system.

The improved pith-condition rating system was as follows: 0.0 = no white, fluffy pith in rated internode; 0.1 = less than 1 % white; 0.5 = 2-12.5% white; 1 = 12.6-25% white; 2,3, and 4, as described above; 4.1 —like 4, with dead cells between intercalary meristem and node and/or in the nodal plate; 5—like 4, with dead parenchyma cells in node linking areas of dead cells in adjacent internodes; and 6—like 5, with premature death of plant, no green color in leaves or rind.

When rapid cell death occurs in previously resistant internodes, the spread of D. *maydis* or G. *zeae* may require 1 or 2 weeks to reach living cells along the rind and in nodes, where additional discoloration can occur. Thus, late-season stalk rot ratings may not be well correlated with pith-condition ratings (Abney 1964).

Pappelis and Boone (1966b), using the 4.1 and 5.0 pith-condition ratings, predicted the spread of stalk rot pathogens from rotted roots into the lower stalk and penetration of the pathogen into the upper nodes from infected leaf sheaths. They concluded that the physiological changes in the stalk reported by McNew (1937), McKeen (1953), and Whitney and Mortimore (1957,1961) to occur prior to penetration of stalk-rotting organisms appear to be related to cell death in internodal and nodal tissue of the stalk, especially the latter. We have made further improvements in the pith-condition rating system as follows: 4.1 —like 4, with dead cells between node and nodal plate (intercalacy meristem); 4.5—like 4.1, with dead cells in the nodal plate, as well as between the node and the nodal plate; and 5.3,5.4, and 5.5 indicate that dead cells are linked from the first through the third, fourth, and fifth internodes above the uppermost brace roots, respectively.

Sorghum stalk-rot and pith-condition rating systems were also developed (Katsanos and Pappelis 1965, 1966a). The pith-condition rating system suggested for sorghum stalk tissue is as follows: 0.0 = no white, fluffy tissue composed of dead

parenchyma in the internode; 0.1 = less than 1% white; 0.5 = 2-12% white; 1.0 =13-25% white; 2.0 = 26-50% white; 3.0 = 51 -75% white; 4.0 = 76-100% white; and 6.0 = plant dead. The letter "T" is added to the internode when cell death in the upper node links dead cells in adjacent (upper) internodes. The stalk-rot rating system for plants inoculated with C. *graminicola* uses the same 0.0 through 6.0 rating units and the letter T, but the area of discoloration is rated, rather than the area of white tissue. As with maize, the pith-condition ratings were highly correlated with stalk rot ratings.

Pappelis (1963) rated parenchyma cell death in cortical and stelar tissue of maize roots after determining that cells in the root that appeared white were dead and others were living (neutral red plasmolysis-deplasmolysis method). The extent of dead cells in roots is difficult to quantify, but cell death patterns in adventitious roots can be documented. As dead cells in the adventitious roots link with areas of dead cells in the stalk, root-rotting pathogens spread into the stalk because there is no barrier of living cells in the root-stalk junction. These predictions can be recorded with basal internode pith-condition ratings by adding the letters RS.

Root and Stalk Rot Symptoms and Inoculation

In severely root-rotted plants, stalk rot and lodging may occur concurrently with the premature death of plants. Severely affected plants show sudden changes in leaves similar to those caused by early frost. The green color of the lower stalk fades. Ears become chaffy. Stalk pith tissue is discolored and has a shredded appearance (Ullstrup 1961,1977).

After some success in inducing stalk rot symptoms by cutting roots (Pappelis 1970a, 1970b), Pappelis (1963) was able to reproduce all the symptoms ascribed to stalk rot by cutting the roots of maize inbreds B2, C103, 38-11, and Os420 before tasseling. The development of the following symptoms varied in each inbred: stunting, areas of gray on leaves lighter green in color than those of normal plants, wilting of lower leaves followed by death, drooping of developing tassel, death of two to three upper leaves around tassel, inhibition of leaf expansion (width) associated with potassium deficiency symptoms, reduction in ear size and number (plants normally having two ears developed one stubby ear), ears generally chaffy, and premature death of some plants. Growth of new secondary roots past the point of root cutting was observed often in plants of B2 and C103 (resistant to stalk rot and least affected by root cutting), occasionally in 38-11 (intermediate; severely affected by root cutting), and seldom in Os420 (susceptible; very severely affected by root cutting—many plants died prematurely). Root cutting induced all the symptoms of stalk rot without any basal stalk rot, crown rot, or root rot evident in either control plants or those whose roots were cut. However, as time passed, the latter developed severe root rot (stalk-rot-resistant plants showing less than stalk-rot-susceptible plants); and in susceptible inbreds, spread of the pathogen into the stalk through the root-stalk junction followed. The death of root cortical parenchyma and stelar parenchyma preceded the spread of root rot pathogens (this was determined using the neutral red plasmolysis-deplasmolysis method), and the death of internodal pith parenchyma at the root-stalk junction-preceded the spread of the pathogen into the stalk. The root and stalk rots were similar to those incited by G. *zeae,* and the diseased tissues subsequently became invaded by the charcoal-rot pathogen, *Macrophomina phaseolina* (Tassi) Goid. Cutting roots always increased susceptibility to diplodia and gibberella stalk rot following inoculation. These findings have disease implications with respect to cultivation practices and root worm damage.

Stalk rot results obtained using the single inoculation method with first and fourth internodes were highly correlated with results obtained using a double inoculation method (both first and fourth internodes inoculated within the same plant) (Pappelis 1965,1970a). The results were as follows: D. *maydis* -1957, r = 0.95 and 0.98; 1960, r = 0.98; and G. *zeae* - 1960, r = 0.98. The diplodia and gibberella stalk rot ratings obtained in these tests were also highly correlated with pith-condition ratings for the first and fourth internodes of control plants: *D, maydis*-1957, r = 0.95 and 0.99; 1960, r = 0.95; and G. zeae-1960, r = 0.95.

Inoculations with D. *maydis* and G. *zeae* to produce stalk rot in breeding plots at maturity are traditionally made in the first elongated internode above the brace roots. The reports that internodes above this location are susceptible have not caused changes in the plant breeder's routine. However, the study of two internodes within the same plant is attractive. Inbreds can be selected to provide resistant first and susceptible fourth internodes for study at the time of flowering and for

several weeks thereafter. This eliminates genetic differences encountered when a number of inbreds showing a wide range of stalk rot responses are studied for differences at maturity. Similarly, it is not necessary to study resistance at flowering and susceptibility at maturity within the same inbred (first internodes), thus eliminating seasonal variations. The variables of leaf size, photosynthesis rates, translocation patterns, ear development, mineral nutrition, and drought stress can all be included for study within the same plant. A model now exists to relate physiological changes to changes in parenchyma cell death within the same plant. Methods to select appropriate inbreds for study have been described (Pappelis and Williams 1966, Pappelis et al. 1975).

In the past, as maize breeders improved yield, they were also unknowingly selecting for cell death in roots and stalks (as evidenced by the continuing problems of the RSL syndrome). By including cell death ratings as an important trait in breeding programs, breeders can now improve disease and lodging resistance as well as yield and other agronomically desirable traits.

Cell Death Patterns

Gas spaces (aerenchyma) in the root cortex of maize seedlings grown in water culture were first reported by Norris in 1913 (McPherson 1939). Dunn (1921) confirmed and extended the findings to include wheat. In 1934, Bryant reported that the same condition existed in barley roots when seedlings were grown in nonaerated solutions, but not when they were grown in aerated solutions (McPherson 1939).

Dunn (1921) observed cortical aerenchyma in maize seedlings grown in sphagnum, sand, or soil culture under summer greenhouse conditions but not winter conditions. She suggested that the rate of root growth (temperature effect) and oxygen supply seemed to be the factors determining the time of the appearance of aerenchyma, not seedling age. Gas spaces were largest in the upper part of the root (4 cm below the seed) and smallest 3 cm from the root tip (roots 10 to 12 cm long).

McPherson (1939) studied the progressive changes in the cortex of maize roots that were related to aerenchyma formation (cell death and degeneration). Nuclear degeneration preceded the loss of cytoplasmic streaming and failure of the cell to plasmolyze. Cell walls collapsed near the root tip and degenerated in the area of cell elongation. The aerenchyma was surrounded by dead cortical cells. When roots 15 cm long with no dead Cortical cells were placed in unfavorable conditions, cell death was extensive and aerenchyma formation ensued both in the mature tissue formed before transference and in newly formed cortical tissue. A few rows of cells adjacent to the epidermis appeared to resist deterioration, but sometimes all cortical cells died. Roots grown in well-aerated soils were not as severely affected. Roots of plants grown for half a season in the field contained aerenchyma cells in the cortex. Relatively dry soils led to smaller and fewer air spaces than water-laden soils. In the latter, aerenchyma formed within 4 days (at 20°C). The rate of aerenchyma formation increased as the temperature increased. Oxygen prevented or greatly reduced cell death in all culture conditions. Poorly aerated roots and soils have recently been shown to cause increases in ethylene and ethylene trapped in roots induces the formation of lysigenous cortical cavities (aerenchyma) due to cell death (Konings 1982). The identical course of aerenchyma development was observed in roots of maize, wheat, barley, and oats (McPherson 1939).

Cytochemical tests revealed that the cell walls of young cells in maize root tips contained cellulose, pectic acid, and protein (McPherson 1939). As cells became older, the cell-wall proteins were lost, pectic acid decreased, and insoluble pectin increased. Endodermal cell walls became lignified.

Cell death patterns in the epidermis and cortex of wheat and barley have been reported (Holden 1975, 1976; Deacon and Henry 1978a, 1978b, 1980). Root hairs and cortical cells died at a faster rate in wheat than in barley over the first 4 weeks of growth. Cell death in cortical tissue first occurred near the epidermis and then progressively toward the endodermis. Pathogens grew into tissue composed of dead cells and induced discoloration of adjacent living cells. Several types of host reactions were observed in response to parasitism; cortical cell walls thickened; cell walls became brown (with or without thickening); and living cells often produced lignitubers (fingerlike wall ingrowths surrounding infection hyphae). Xylem plugging occurred in advance of hyphae growing up the stele. The methods used to study root rots of wheat and barley may have application in the study of r6ot rots of maize and sorghum. D. *maydis* has been reported to induce lignitubers in cells of the maize root (Craig and Hooker 1961), Schneider and

Pendery (1983) reported reduced water uptake following maize root rot.

We discovered that death of parenchyma cells in the lower nodes was delayed in no-till and conservative-tillage plots (unpublished data, 1970). This may explain the observation by Mock (1982) that less stalk rot occurred in no-till plots. No-till plots contain 20 to 30% more water, more organic matter (due to reduced aerobic microbial oxidation and increased anaerobic activities), are several degrees colder than plowed soil (Doran 1982), and are devoid of any root damage from cultivation.

Mechanisms of Resistance

Recurrent selection has enabled breeders to improve yield, root and stalk strength, and stalk rot resistance (Miles et al. 1980, Smith 1983, Thompson 1982, Zuber et al. 1980). Quantitative methods have been developed to evaluate stalk strength and to determine the contribution of rind and pith to strength (Twumasi-Afriyie and Hunter 1982a, 1982b; Zuber and Kang 1978; Zuber 1983). Root volume was found to be highly correlated with root-pulling resistance (Zuber et al. 1971). Methods that could be used to study this aspect of the syndrome were reviewed by Donovan et al. (1982), Arihara and Crosbie (1982), and Peters et al. (1982). Selection for size of the root system is not expected to reduce grain yield. Methods to study root morphology (Maizlish et al. 1980) may be helpful in testing for root rot resistance. Fungal population studies have been completed by many researchers (Kommedahl et al. 1979). Hornby and Ullstrup (1967a, 1967b), using methods to study fungal and nematode populations on maize roots, reported higher microflora populations on susceptible plants than on resistant plants. Similarly, ear-rot screening methods have been improved (Sutton 1982, Sutton and Proctor 1982). These new research methods and reports should guide pathologists, physiologists, and biochemists through resistance mechanism studies.

Physiology, Biochemistry, and Cytology of RSL

Senescence and Cell Death

When it became evident that the way to maintain field resistance to diplodia and gibberella stalk rots was to keep a barrier of living parenchyma cells in nodes, internodes (especially along the rind), and adventitious roots, we began to explore the events related to senescence and cell death. We defined the moment of *cell death* as that time when the cytoplasmic membrane becomes irreversibly permeable and *cellular senescence* as irreversible degeneration that leads to cell death. We defined *cellular autolysis* as the degenerative events that occur following cell death. (Some of our early studies along this line have been reviewed: Pappelis et al, 1971). Biochemical changes in senescing cob parenchyma tissue, stalk pith tissue, and first developed leaf of maize were examined (BeMiller et al. 1969a, 1970, 1972/73, 1973, 1976a, 1976b; BeMiller and Hoffmann 1972), and characteristics that make each of these tissues useful for studies of senescence were described and evaluated (BeMiller et al. 1972/73, 1976a).

Because pith tissue included both parenchyma cells and vascular tissue, the relationship between changes in concentrations of components in the sample and cellular senescence and death was not clear. Although parenchyma cells of the stalk died, many cells around vascular tissue remained alive, and the vascular tissue continued to function. For this reason, BeMiller et al. (1969a) used maize cob parenchyma tissue, free of vascular bundles, as a model for study of senescence. BeMiller et al. (1970) concluded that the only valid basis for comparing cell constituents in stalk and cob parenchyma tissue was the per-cell basis.

In a 3-week study beginning at silking, BeMiller et al. (1970) determined changes in concentrations of various nutrients that might give a clue to changes in membrane integrity and compared the data with those of previous studies. They found that K, Si, P, Fe, and Co concentrations per cell increased during the 1 st week (period of greatest cell elongation), then decreased slowly as the cells senesced and died. There were continuous accumulations of Sr, Cu, crude fiber, and ether-soluble substances per cell over the study period. Mo per cell increased during the 1st week, then remained constant. Zn, Ba, and B per cell increased until the 2nd week, then decreased. Mg and total N per cell remained constant during the study period. There was no index that appeared to forecast cell senescence and death.

BeMiller and Hoffman (1972) determined the 80% ethanol-soluble carbohydrate content of maize cob tissue (parenchyma cells) on a per-cell basis before and during the period of cellular

senescence and death. In four of the five populations studied, a period of cell elongation was followed by a decrease in the amounts of total and reducing sugars per cell. Before cells died, at least 90% of the total sugars were reducing sugars, Within a day or two after the drop in the per-cell sugar contents, many cells in the tissue died. After this period, the total sugar content increased to a maximum, then decreased (and in some populations increased again). Changes in per-cell content of D-glucose, D-fructose, sucrose, and total sugar were also calculated. Glucose and fructose content almost paralleled each other throughout the study in all varieties. When cells died, the reducing sugars had declined to 10% or less of the maximum level. The sucrose content, however, was very low until after the 1 st week, at which time it began to increase (at the same time massive cell death was beginning in all cultivars).

Betterton (1963), using pith tissue from fully elongated first and fourth internodes and expressing his data on a volume basis, did not observe the early abrupt decrease of total and reducing sugars to a very low level, followed by an increase as cells died. On the contrary, he found that sucrose content, and thus total sugars, increased as cells in the two locations were dying, and only reducing sugars decreased. His data showed that water content of the tissue decreased before the decrease in reducing sugars, suggesting that leakage occurs as a result of senescence and cell death rather than as a cause of these events. Using both water and sugar content data (per cc of tissue), he found that first-internode pith tissue generally contained greater amounts of reducing sugars and sucrose than did fourth internodes, but the molar concentrations of these in the two locations were similar. The data from cob tissue (BeMiller and Hoffman 1972) showed similar relationships; i.e., decreases in reducing sugar content per cell were correlated with senescence and cell death (density decreases), while sucrose and total sugar content per cell were not. The soluble carbohydrates that were lost from stalk parenchyma cells were probably transported to ears to increase yield.

The literature on carbohydrate synthesis, distribution, and utilization in maize is vast and needs critical evaluation since the bases of data comparisons differ widely and can result in misleading views about physiological trends. Attempts to relate some of the data to stalk rot resistance were made by Schneider and Pendery (1983) and Dodd (1980). Many additional considerations of the physiological baste of genetically controlled increases in yield, with emphasis on control and improvement in distribution and storage of photosynthetic assimilates, were discussed by Gifford and Evans (1981).

Hoffmann (1968) reported a drop in the per-cell phenylanine content to zero and an increase in total amino acid (especially aspartic acid) content preceding the onset of cell death in cob parenchyma tissue. The content of total fatty acids did not appear to be correlated with anything else, but the highest fatty-acid content occurred at the time of lowest sugar content, just prior to water loss (cell death). Two unknown acids peaked in content shortly after silking, then disappeared; subsequently, one was identified as aconitic acid (BeMiller and Hoffman, unpublished data, 1969).

In searching for changes in the per-cell content of potential regulatory molecules in stalk internodal pith tissue, we obtained the following results. The putrescine:spermidine ratio (per-cell basis) in stalk pith tissue decreased with distance from the intercalary meristem, increased sharply to the original level just below the region in which cells were beginning to die, and then decreased in the region in which cells were dying (Curran 1971). In cob parenchyma tissue the putrescine:spermidine ratio increased continuously during the period of cell elongation and senescence preceding cell death. Spermine was either absent from or at very low levels in the tissues sampled.

Recent literature on polyamines (biosynthesis, precursors, ubiquitous distribution, involvement in various growth processes, and senescence) has been reviewed by Altman (1982). Among their other physiological effects, polyamines are involved in the control of several stress-related phenomena. Exogenous application of polyamines and related precursors retard the progressive senescence of oat leaf protoplasts, stabilize them against lysis, and support a higher incorporation of uridine and leucine. These events may be due to the effect polyamines have on preventing chlorophyll loss and preventing the rise of RNase and protease, and the stabilizing effect they have on both nucleic acids and membrane function during senescence. Because polyamines were highly effective in retarding protease and RNase activity prior to chlorophyll loss, Altman concluded that polyamines affect early senescence-linked events that are not light-dependent. While their modes of action are not known, the cationic nature of these compounds may produce an effect similar to that of calcium (stabilization of chloroplast thylakoids, sta-

bilization of membranes against leakage, and inhibition of RNase and protease).

Curran (1971) in our laboratory, found that senescence of cells in the first leaf, cob parenchyma, and stalk pith tissue of maize is accompanied by considerable loss of total polyamines. Research in our laboratory (Liu 1972) also showed that the 3', 5'-cyclic adenosine monophosphate (cAMP) content (per-cell basis) decreased slightly, but not significantly, in the first three sections above the intercalary meristem (young elongating cells), then increased as the mature cells aged; this pattern exactly parallels those of protein synthesis, RNA synthesis, RNase activity, and DNase activity, and is the inverse of the total RNA pattern. In cob parenchyma tissue, the cAMP content decreased for the first 7 days after silking, then increased, though not significantly, as cell death began. Inthis case, the pattern was the inverse of that of protein synthesis and RNase activity, patterns and paralleled RNA synthesis. Therefore, correlations of contents of possible regulatory molecules and cell development and senescence were obtained. This is not surprising since senescence of higher plants is known to involve a series of highly synchronized events under hormonal control, but it is not yet possible to use these data to develop a unified concept of plant cell senescence and death.

Decreases in the amount of soluble protein per cell were observed during senescence in cob parenchyma, but not in senescing stalk parenchyma (BeMiller et al. 1972/73). Associated with the decrease in soluble protein in cob parenchyma cells was an increase in the free amino acid content of the cells. However, as the number of dead cells increased in the tissue, the free amino acid content began to decline rapidly. In both cob and stalk tissue, protein synthesis (incorporation of labeled leucine) decreased with tissue age until late in the senescence period, when there was a large increase in synthesis. The increased synthesis could be reduced with actinomycin D. The amount of free leucine per cell increased with senescence, indicating that the increase in protein synthesis was not simply an apparent increase owing to an increase in specific activity of the labeled leucine because of a smaller pool of leucine. The nucleic acid content of cob parenchyma tissue (volume basis) dropped to less than 20% of the original value as cells elongated, underwent senescence, and died (BeMiller et al. 1969a).

In a later study in our laboratories, Fong (1973) found that different cultivars appeared to have different patterns of age-related metabolic changes. In general, there was an increased synthesis of a protein fraction during tissue senescence. In both cob parenchyma and stalk pith tissue of Pioneer hybrid 314, it was high-molecular-weight protein molecule(s) or particle(s) whose synthesis increased, but synthesis of this fraction was unchanged in both cob parenchyma and stalk pith tissue of WF9 x 38-11 single cross. In stalk pith tissue of both Pioneer hybrid 314 and WF9 x 38-11, synthesis of a low-molecular-weight protein increased with age. In cob parenchyma tissue of both cultivars, synthesis of this fraction decreased with age. In Pioneer hybrid 314, there was an increased synthesis of intermediate-molecular-weight proteins with age. RNA synthesis in both tissues of both cultivars, if it changed at all, increased. Ribonuclease activity also increased, indicating an increasing rate of RNA turnover.

Cob and stalk parenchyma cell senescence and death also involved decreases in total RNAcontent (primarily a loss in RNA which preceded a loss in DNA) and in RNA synthesis, and a sharp increase in nuclease activity a few days prior to cell death (BeMiller et al. 1976b). Based on additional cytochemical data from our laboratories (with the emphasis on a per-cell basis extended by analytical and quantitative cytology), BeMiller et al. (1976b) proposed that early decreases in DNA and degeneration of nucleoli were indicators of cell senescence in maize stalk and cob parenchyma tissue.

tRNA methylase activity of cob parenchyma tissue, stalk pith tissue, and the first developed leaf of maize disappeared or declined to low levels as cells senesced preceding death (BeMiller et al. 1973). The fact that similar changes in tRNA methylase activity were found in all three tissues suggests that the decrease in activity is a general characteristic of senescence in maize tissue. Measurements of the cob parenchyma tissue began on the day of silking and continued until cell death about 10 days later. Maize stalk tissue was taken from the fourth internode before tassel elongation (plants about 1.22 m tall), when parenchyma cell death was beginning to occur in the upper part of the internode (sections from pith cores were analyzed; the pith cores were from the intercalary meristem through the area containing dead parenchyma cells). Leaf tissue (2nd- and 3rd-cm sections from the tip) was coltected on the 8th through the 20th days after planting. The undermethylation or nonmethylation of tRNA in senesc-

ing tissue could account for changes in the cellular content of specific enzymes, perhaps by misreading of codons. Decreases in essential enzymes could then cause senescence. The breakdown of fidelity of protein synthesis could result both in enzymically inactive proteins and in an increased rate of synthesis of particular proteins affected by misincorporation of amino acids into polypeptide chains via feedback mechanisms.

These studies support the idea that gene-level activity such as RNA synthesis (BeMiller et al. 1976a) and protein synthesis (BeMiller et al. 1976b) continues, but in such a way that errors in polypeptides accumulate, causing senescence and death of cells. The increase in nuclease and protease activities may simply indicate an increase in turnover as cells try to make correct polypeptide sequences.

Several people in our laboratories have used quantitative Feulgen cytochemistry to determine changes in DNA during cell growth, development, maturation, and senescence, and quantitative interference microscopy to determine changes in nuclear and nucleolar dry mass and size. Commean (1974) found that nuclear and nucleolar dry mass and size increased during parenchyma cell development and elongation in both maize stalk and cob tissue (attributed to increases in nuclear proteins and RNA), then declined during senescence (often rapidly). The DNA content in cob parenchyma cells remained constant through the early period of cell elongation and declined as cells senesced and died.

Bhattacharya and Pappelis (1983) reported that eight nuclear traits (total nucleic acid, DNA, RNA, total nuclear protein, histone protein, nonhistone protein, protein-bound arginine, and protein-bound lysine) decreased as cell senescence occurred in two models in onion bulb leaf base tissue. In the sequential leaf-senescence model, tissue was selected from similar sites in young and older (physiological and chronological) leaves. Cells in young leaves had the least amounts of the macromolecules measured, and those in older, normal leaves had four to five times these amounts due to polyploidy. Cells in the oldest, dying leaves had little or no measurable amounts of macromolecules. We believe that a similar "all-or-nothing" effect was encountered in maize, using the random selection method in cross sections of tissue.

In the apical-cell senescence model in individual onion leaf bases, normal cells were encountered within 3 mm of dead cells (Bhattacharya and Pappelis 1983), and drastic decreases in the eight nuclear traits were obtained when successive, contiguous cells were studied. We believe that this sampling method should be used in future studies.

We concluded that the models we selected for study could be improved by measuring multiple nuclear and nucleolar traits, selecting successive (contiguous) cells from dead to normal types for measurements, and selecting cells of the same size (normal and polyploid cells within the sample should not be mixed). The same methods should be used to study the host-pathogen interactions.

Using quantitative interference microscopy, Pappelis et al. (1973b) found that *D. maydis* induced increases in nuclear dry mass, nuclear size, and nucleolar size in parenchyma cells of the first internodes of two single-cross maize hybrids. Similar results were obtained using inoculated cobs from one of these hybrids. This may be an inhibition response of all living parenchyma cells. Since the multigenic mechanisms that control these responses are not known, they represent an important starting point that should be applied to the study of inbreds. It may be that other fungal pathogens have the opposite effect on host cells (killing in advance of spread into tissue). We have obtained data on induced host-cell senescence and death in several studies of onion pathogens (Kulfinski and Pappelis 1976, Bhattacharya and Pappelis 1982).

Karagiannis et al. (1984) characterized the nucleolar enlargement when quiescent onion cells are activated without pathogens. Small, round nucleoli enlarge to form elongated and dumbbell-shaped nucleoli within a few hours. As nucleoli enlarge, nucleolar vacuolarization occurs. This is indicative of transcriptional activity in the nucleolar organizer regions and represents a significant physiological change. Karagiannis and Pappelis (unpublished data, 1983) have found that many growth-regulating substances cause nucleolar activation, as well as inhibition of this process.

The use of quantitative interference microscopy to measure loss of dry mass during fungal spore germination was first accomplished with two-celled spores of D. *maydis* (Pappelis et al. 1979). We extended that work with studies of asexual spores of G. *zeae* (Mumford and Pappelis 1978). We expected and found dry-mass losses in both cases. In addition, we found that both accumulated dry mass after germination, suggesting that this process in water is not merely a utilization of endogenous spore reserve. The dry-mass increases

could be accounted for by the uptake of secreted or leached substances from the spores that did not germinate. Murphy et al. (1976) found that spores of *D. maydis* contained relatively large amounts of Si, P, Cl, and K; smaller amounts of S and Ca; and trace amounts of Mg and Al. K and Cl were concentrated in the cells without a germ tube, and Mg and P were concentrated in the germinating cells. X-ray image maps revealed that K and Cl were located together at one end of the spore. No such studies have been conducted with spores in inoculated host tissue. These methods may be very helpful in studying highly resistant varieties of maize that may kill germinating spores following inoculation, and also in bioassay studies of fungistatic and fungitoxic substances from maize.

Murphy (1977) examined the infection process of D. *maydis* in maize stalk tissue using both transmission and scanning electron microscopy. Using a technique to detect cellulase activity, she studied cellulose degradation in culture and in penetration of the host-cell wall (Murphy et al. 1973, 1974, 1976, 1977, 1980). Before penetration of dead parenchyma cell walls in inoculated stalk tissue, hyphal "flattening" occurred and adhesion material was secreted. As hyphal constriction occurred, the release of cellulase was detected and penetration followed. The frayed appearance of the host-cell walls occurred only where there was direct contact with hyphal surfaces and where cellulase activity was detected. The spread of the pathogen in dead host cells appeared to be random. Cellulase was discovered to be constitutive (i.e., the enzyme was associated with vesicles in the cytoplasm and on the wall of the pathogen and did not require induction). These studies supported earlier findings by BeMiller et al. (1969b) that the release of cellulase from hyphae did not occur when the pathogen was grown in the presence of glucose.

Studies of the relationship of the nutrient element content in maize parenchyma cells to senescence in field-grown plants (BeMiller et al. 1970, Imbamba et al. 1966, Pappelis and Boone 1966a) were expanded, with studies of plants grown in a gravel-nutrient culture using high-low NPK nutrient solutions (Meyer 1966, Pappelis and Liu 1966, Pappelis et al. 1967). Cell death in internodes was hastened and stalks lodged when plants were grown in low-potassium solutions. When gravel beds were infested with G. *zeae,* severe root and stalk rots were produced in a susceptible inbred; moderate root and stalk rots in an intermediate inbred; and trace amounts of root and stalk rots in a resistant inbred. Because the results were comparable in every way with results obtained with these inbreds in field studies, we concluded that the gravel culture method would be the best possible way to study agronomic stresses and their interaction with the RSL syndrome.

It was obvious from our field studies (Pappelis and Myers 1970, Miller and Myers 1974) that genetic control of cell death (associated with susceptibility) and genetic control of physiological activities in living cells (associated with resistance) were separate phenomena. Evidence that cell death is controlled by one or few genes was obtained. The number of genes that control the production of fungistatic and fungitoxic compounds found in living cells is not known (BeMiller and Pappelis 1965a, 1965b; BeMiller et al. 1967; Dabler et al. 1969).

Water Stress and Cell Death

Probably the most exciting development that will lead to the long-sought cause of cell death in tissues of the stalk and other organs of maize and sorghum has come from research on water stress. Petiole pithiness in celery was shown to develop rapidly after the plants were subjected to a short period of water stress (Aloni and Pressman 1979) and could also be induced by treatment with abscisic acid (ABA) solutions. Additional research (Aloni and Pressman 1981) demonstrated water-stress-induced wilting of younger leaves of tomato and the nonreversible onset of pithiness in stems. Increasing the duration of water stress increased the extent of pithiness. ABA applied through the root system induced the same effect with or without water-stress treatments, using polyethylene glycol to obtain an osmotic potential of -2.0 bars. Kinetin enhanced pithiness only after water stress had occurred. Pithy cells in water-stressed plants (white tissue) lost their stainability with 2, 3, 5-triphenyltetrazolium chloride (no staining = senescing or dead cell).

Ackerson (1983) found that leaves of water-stressed plants contained higher-than-normal amounts of ABA, and he discussed his findings in relation to earlier reports of ABA accumulation and other physiological indices in leaves of water-stressed cotton, maize, wheat, and pearl millet. Durley et al. (1983) and Kannangara et al. (1983) were able to evaluate genotype drought resistance to a given stress treatment in sorghum by examin-

ing ABA and phaseic acid (PA) concentrations in leaves. PA is a principal metabolite of ABA. ABA levels were increased and PA levels reduced by stress, Indole-3-acetic acid levels could not be related to stress. Kannangara et al. reviewed earlier work with hormones and water stress in relation to work in their laboratories and concluded that leaf ABA levels are a sensitive indicator of the degree and type of drought stress for sorghum plants. Increases in ABA levels were also correlated with marked leaf senescence, decline in plant height, and reduced yield. Leaf area development was more sensitive to stress than stem elongation. Water stress was associated with increased leaf temperature.

Eze et al. (1983) demonstrated that leaf temperature extremes did not induce changes in ABA levels if plants were not water-stressed. However, under conditions of water stress, leaf temperature did affect ABA levels (the highest increase was tenfold at 25°C). ABA has also been shown to inhibit sucrose uptake by leaf tissue; i.e., it inhibits phloem loading (Vreugdenhil 1983). Although one plausible explanation of the inhibition of phloem loading by ABA might be that ABA diminishes the transmembrane proton gradient that is coupled to and drives this process (Giaquinta 1983), Vreugdenhil (1983) found no such effect. He suggested an alternative explanation: ABA could induce or activate a passive sucrose leak and result in stimulated fruit and seed import.

Water stress is a major predisposing factor to stalk rot of maize (Koehler 1960, Schneider and Pendery 1983, Ullstrup 1955). Ullstrup (1955) observed that the severity of diplodia stalk rot was increased by wet weather near the end of the growing season, especially when preceded by unusually dry weather. Schneider and Pendery (1983) verified this experimentally. Late-season stalk rot (incited by *F. moniliforme*) in early water-stressed plants was more than twice that of control plants. Pith density in water-stressed plants was significantly lower than that in control plants. Also, pith tissue in water-stressed plants was more spongelike and white. Root senescence in the upper soil strata was inferred from increased root infections and systemic colonization. Root infections resulted in inefficient water uptake. Schneider and Pendery inferred that chronic water stress may follow such conditions and result in (a) the remobilization of stored assimilates from roots and stalks that enhance yields, (b) senescence and cell death in roots and stalks, and (c) root and stalk rot susceptibility.

Schneider and Pendery (1983) also observed that although postpollination water stress caused little or no change from the control treatment (disease response), water stress at grain filling reduced the incidence of naturally occurring stalk rot. They did not attempt to explain this observation. Whether parenchyma cells in the upper roots and lower nodes remain alive longer by enduring such a brief period of stress remains to be determined. Possibly ear-filling ceased under these conditions. The relationship between root and stalk rot was clear. Pith condition ratings would have greatly aided interpretation of the data.

Conclusions

We conclude that many of the symptoms of early stalk rot (retarded leaf development, stunting, wilting, reduction in ear size and number, chaffy ears, etc.) may be caused by a hormone imbalance induced by severe early-season water stress. Cell death in roots predisposes them to root rot. Root rots predispose the stalks to an increased rate of parenchyma cell death and stalk rot, and root rot pathogens spread into the lower internodes when areas of dead cells in roots are linked to areas of dead cells in stalks. The roles of ABA, ethylene, and cytokinin in cell death and patterns of cell death throughout the plant need to be studied. If ABA effects membrane leakage, glycosides (BeMiller and Pappelis 1965a, 1965b) may leak from vacuoles, their cytotoxic (fungistatic) aglycones may be released enzymically into the cytoplasm, and cell death may follow.

We agree with Zuber (1983) and Mahon (1983) that the next big breakthrough in disease resistance, water-stress tolerance, standability, and yield is likely to be related to physiological/biochemical research.

Future Research Needs

1. Patterns of parenchyma cell death in roots and stalks of sorghum should be determined in the screening programs designed to select sources of resistance to root and stalk rots.
2. The inheritance of parenchyma ceil death patterns should be determined since genetically-controlled variability in this trait is

expected to be related to disease-response variability.

3. Inoculation procedures should be developed that predict disease responses in disease nurseries. The results of inoculation trials should be studied in relation to parenchyma cell death patterns in sorghum.

4. Cell death patterns in midribs of sorghum leaves have been reported to predict pithiness in stalks. This may be an important trait to use in disease nursery studies of stalk rot resistance and susceptibility, and thus the relationships between these traits should be studied.

5. Gravel culture methods should be developed for sorghum to enable researchers around the world to study a wide range of variables under similar conditions.

6. Cytological, physiological, and biochemical studies on the nature of resistance and susceptibility to root and stalk rots of sorghum must be given high priority. Included should be studies on the mechanism(s) of cellular senescence, death, and autolysis; studies on fungitoxic substances in living cells that inhibit the spread of fungal pathogens; and studies on the mechanism(s) of induced host cell death that are associated with the spread of some fungal pathogens in nonsenescing sorghum tissue; and the effect of water stress on all these phenomena.

7. The effects of soils and soil environments on the longevity of parenchyma cells in roots and stalks of sorghum should be determined and related to tillage methods and cultural practices used in sorghum production.

8. Pathogen variability needs to be studied to prevent unexpected worldwide production problems.

References

ABNEY, T.S. 1964. Seasonal trends in total nitrogen content of corn stalk tissue in relation to stalk rot resistance. M.Sc. thesis, Southern Illinois University at Carbondale, Carbondale, Illinois, USA. 48 pp.

ACKERSON, R.C. 1983. Comparative physiology and water relations of two corn hybrids during water stress. Crop Science 23:278-283.

ALONI, B., and PRESSMAN, E. 1979. Petiole pithiness in celery leaves: Induction by environmental stresses and the involvement of abscisic acid. Physiologia Plantarum 47:61-65.

ALONI, B., and PRESSMAN, E. 1981. Stem pithiness in tomato plants: The effect of water stress and the role of abscisic acid. Physiologia Plantarum 51:39-44.

ALTMAN, A. 1982. Retardation of radish leaf senescence by polyamines. Physiologia Plantarum 54:189-193.

ARIHARA, J., and CROSBIE, T.M. 1982. Relationships among seedling and mature root system traits for maize. Crop Science 22:1197-1202.

BARE, C.E., PAPPELIS, A.J., and SCHMID, W.E. 1971. Cell death patterns in sugar cane stalk tissue following injury and flowering. Sugar Journal 33:30-32.

BeMILLER, J.N., and HOFFMANN, W.E. 1972. Relationship of soluble carbohydrates to age of corn cob parenchyma tissue. Phytochemistry 11:1321-1325.

BeMILLER, J.N., HOFFMANN, W.E., and PAPPELIS, A.J. 1972/1973. Relationship of protein metabolism to senescence of corn (*Zea mays* L.) stalk parenchyma, cob parenchyma and first developed leaf tissue. Mechanisms of Ageing and Development 1:387-401.

BeMILLER, J.N., HOFFMANN, W.E., and PAPPELIS, A.J. 1973. Changes in tRNA methylase activity in senescing cob and stalk parenchyma tissue and the first developed leaf of corn *(Zea mays* L). Mechanisms of Ageing and Development 2:363-369.

BeMILLER, J.N., JOHNSON, D.C., and PAPPELIS, A.J. 1969a. Relationship of nucleic acids to cell death in corn cob parenchyma tissue. Phytopathology 59:989-991.

BeMILLER, J.N., JOHNSON, D.C., and PAPPELIS, A.J. 1970. Relationship of nitrogen, crude fiber, ether-soluble substances and mineral nutrients to cell death in corn cob parenchyma tissue. Phytopathology 60:513-517.

BeMILLER, J.N., LIU TU, Y.L., LIU, C.R., and PAPPELIS, A.J. 1976b. Relationship of nuclease activity and synthesis to senescence of corn (*Zea mays* L.) stalk pith, cob parenchyma and first developed leaf tissues. Mechanisms of Ageing and Development 527-436.

BeMILLER, J.N., and PAPPELIS, A.J. 1965a. 2, 4-Dihydroxy-7-methoxy-1, 4-benzoxazin-3-one glucoside in corn: I. Relation of water-soluble, 1-butanol-soluble glycoside fraction content of pith cores and stalk rot resistance. Phytopathology 55:1237-1240.

BeMILLER, J.N., and PAPPELIS, A.J. 1965b. 2, 4-Dihydroxy-7-methoxy-1, 4-benzoxazin-3-one glucoside in corn: II. Isolation of 6-methoxy-2(3)-benzoxazolinone fraction as a measure of glucoside content and tissue differences of glucoside content. Phytopathology 55:1241-1243.

BeMILLER, J.N., RIMSAY, R.L., and PAPPELIS, A.J. 1967. Extraction and purification of phenolic acids in corn. Transactions of the Illinois State Academy of Science 60:68-71.

BeMILLER, J.N., SPARKS, M.C.B., FONG, T.W., and PAPPELIS, A.J. 1976a. Relationship of senescence of corn (*Zea mays* L.) stalk pith, cob parenchyma and first developed leaf tissues to RNA content and synthesis. Mechanisms of Ageing and Development 5:419-426.

BeMILLER, J.N., TEGTMEIER, D.O., and PAPPELIS, A.J. 1969b. Constitutive cellulolytic enzymes of *Diplodia zea.* Pages 188-196 *in* Cellulases and their applications. Vol. 95, Advances in Chemistry Series. Washington, D.C., USA: American Chemical Society.

BETTERTON, H.O. 1963. Seasonal trends in sugar content and cell death in corn stalk tissue. M.Sc. thesis, Southern Illinois University at Carbondale, Carbondale, Illinois, USA. 99 pp.

BHATTACHARYA, P.K., and PAPPELIS, A.J. 1982. Cytofluorometric study of onion epidermal nuclei in response to wounding and *Botrytis allii* infection. Physiological Plant Pathology 21:217-226.

BHATTACHARYA, P.K., and PAPPELIS, A.J. 1983. Nucleic acid, protein, and protein-bound lysine and arginine patterns in epidermal nuclei of the mature and senescing onion bulb. Mechanisms of Ageing and Development 21:27-36.

BRITTON, M.P., and HOOKER, A.L. 1963. Failure to control corn stalk rots with above-ground applications of protectant fungicides. Plant Disease Reporter 47:470-471.

BRUEHL, G.W. 1983. Nonspecific genetic resistance to soilborne fungi. Phytopathology 73:948-951.

CHRISTENSEN, J.J., and WILCOXSON, R.D. 1966. Stalk rot of corn. Monograph No. 3. St. Paul, Minnesota, USA: American Phytopathological Society. 59 pp.

CLONINGER, F.O., ZUBER, M.S., CALVERT, O.H., and LOESCH, P.J., Jr. 1970. Methods of evaluating stalk quality in corn. Phytopathology 60:295-300.

COMMEAN, V.L. 1974. Changes in DNA, nuclear and nucleolar area and dry mass in aging corncob and stalk parenchyma tissue. M.Sc. thesis, Southern Illinois University at Carbondale, Carbondale, Illinois, USA. 40 pp.

CRAIG, J., and HOOKER, A.L. 1961. Diplodia root and stalk rot of dent corn. Phytopathology 51:382-385.

CURRAN, G. 1971. Relationship of polyamine content to tissue senescence in corn. M.Sc. thesis, Southern Illinois University at Carbondale, Carbondale, Illinois, USA. 86 pp.

DABLER, J.M., PAPPELIS, A.J., and BeMILLER, J.N. 1969. Effect of phenolic acids and corn extracts upon spore germination of *Diplodia zeae.* Phytopathology 59:1098-1101.

DEACON, J.W., and HENRY, C.M. 1978a. Death of cereal root cortex: Its relevance to biological control of take-all. Annals of Applied Biology 89:100.

DEACON, J.W., and HENRY, C.M. 1978b. Studies on virulence of the take-all fungus, *Gaeumannomyces graminis,* with reference to methodology. Annals of Applied Biology 89:401-409.

DEACON, J.W., and HENRY, C.M. 1980. Age of wheat and barley roots and infection by *Gaeumannomyces graminis* var. *tritici.* Soil Biology and Biochemistry 12:113-118.

DODD, J.L. 1980. Grain sink size and predisposition of *Zea mays* to stalk rot. Phytopathology 70:534-535.

DONOVAN, L.S., JUI, P., KLOEK, M., and NICHOLLS, C.F. 1982. An improved method of measuring root strength in corn (*Zea mays* L). Canadian Journal of Plant Science 62:223-227.

DORAN, J.W. 1982. Tilling changes soil. Crops and Soils Magazine 34:10-12.

DUNN, G.A. 1921. Note on the histology of grain roots. American Journal of Botany 8:207-211.

DURLEY, R.C, KANNANGARA, T., SEETHARAMA, N., and SIMPSON, G.M. 1983. Drought resistance of *Sorghum bicolor.* 5. Genotypic differences in the concentrations of free and conjugated abscisic, phaseic and indole-3-acetic acids in leaves of field-grown drought-stressed plants. Canadian Journal of Plant Science 63:131-145.

DURRELL, L.W. 1923. Dry rot of corn. Iowa Agricultural Experiment Station Research Bulletin No. 77.

EZE, J.M.O., DUMBROFF, E.B., and THOMPSON, J.E. 1983. Effects of temperature and moisture stress on the accumulation of abscisic acid in bean. Physiologia Plantarum 58:179-183.

FONG, T.W. 1973. An investigation of protein synthesis, RNA synthesis, and ribonuclease activity in senescing cob and stalk parenchyma tissues and the first developed leaf of corn (*Zea mays* L). M.Sc. thesis, Southern Illinois University at Carbondale, Carbondale, Illinois, USA.

GATES, L.F. 1970. Relationships between pith cell conditions as assessed by tetrozolium chloride and incidence of Gibberella stalk rot of corn. Canadian Journal of Plant Science 50:679-684.

GIAQUINTA, R.T. 1983. Phloem loading of sucrose. Annual Review of Plant Physiology 34:347-387.

GIFFORD, R.M., and EVANS, C.T. 1981. Photosynthesis, carbon partitioning and yield. Annual Review of Plant Physiology 32:485-509.

HOFFMANN, W.E. 1968. Relationship of lipids, soluble carbohydrates, and free amino acids to cell death in corn

cob parenchyma tissue. M.Sc. thesis, Southern Illinois University at Carbondale, Carbondale, Illinois, USA. 96 pp.

HOLDEN, J. 1975. Use of nuclear staining to assess rates of cell death in cortices of cereal roots. Soil Biology and Biochemistry 7:333-334.

HOLDEN, J. 1976. Infection of wheat seminal roots by varieties of *Phialophora radicicola* and *Gaeumannomyces graminis* . Soil Biology and Biochemistry 8:109-119.

HOOKER, A.L. 1956. Association of resistance to several seedling, root, stalk, and ear diseases in corn. Phytopathology 46:379-384.

HOOKER, A.L. 1957. Factors affecting the spread of *Dipiodia zeae* in inoculated corn stalks. Phytopathology 47:196-199.

HOOKER, A.L. 1976. Corn anthracnose leaf blight and stalk rot. Pages 167-182 *in* Proceedings of the 31st Annual Corn and Sorghum Research Conference. Washington, D.C., USA: American Seed Trade Association.

HOOKER, A.L. 1978. Genetics of disease resistance in maize. Pages 319-332 *in* Maize breeding and genetics (ed. D.B. Walden). New York, New York, USA: John Wiley and Sons.

HOOKER, A.L., HENDERSON, C.B., and YATES, D.E. 1962. Agronomic characteristics of corn inbreds and their reaction to leaf blights and stalk rot. Report on Plant Diseases No. 204. 28 pp.

HORNBY, D., and ULLSTRUP, A.J. 1967a. Fungal populations associated with maize roots: Quantitative rhizosphere data for genotypes differing in root rot resistance. Phytopathology 57:76-82.

HORNBY, D., and ULLSTRUP, A.J. 1967b. Fungal populations associated with maize roots: Composition and comparison of mycofloras from genotypes differing in root rot resistance. Phytopathology 57:869-875.

HORROCKS, R.D., CLONINGER, F.D., and ZUBER, M.S. 1972. The relationship between stalk lodging and corn leaf blight. Plant Disease Reporter 56:11 -14.

IMBAMBA, S.K., SCHMID, W.E., and PAPPELIS, A.J. 1966. Nutrient element content of corn parenchyma as related to cell senescence. Proceedings of the Association of Southern Agricultural Workers 63:289-290 (abstract).

KANG, M.S., PAPPELIS, A.J., MUMFORD, P., MURPHY, J.A., and BeMILLER, J.N. 1974. Effect of cob and shank inoculations (*Diplodia maydis*) on cell death in stalk internodes of corn. Plant Disease Reporter 58:1113-1117.

KANNANGARA, T., SEETHARAMA, N., DURLEY, R.C., and SIMPSON, G.M. 1983. Drought resistance of *Sorghum bicolor.* 6. Changes in endogenous growth regulators of plants grown across an irrigation gradient. Canadian Journal of Plant Science 63:147-155.

KARAGIANNIS, C.S., PAPPELIS, A.J., and RUSSO, V.M. [1984.] The effects of physiological activation on nucleolar morphology in *Allium* cepa. Cytologia (in press).

KATSANOS, R.A., and PAPPELIS, A.J. 1965. Seasonal trends in density and cell death in sorghum stalk tissue. Phytopathology 55.97-99.

KATSANOS, R.A., and PAPPELIS, A.J. 1966a. Relationship of cell death patterns and spread of *Colletotrichum graminicola* in sorghum stalk tissue. Phytopathlogy 56:468-469.

KATSANOS, R.A., and PAPPELIS, A.J. 1966b. Effect of root injury on cell death in sorghum stalk tissue and susceptibility to *Colletotrichum graminicola.* Plant Disease Reporter 50:287-288.

KATSANOS, R.A., and PAPPELIS, A.J. 1967. Relationship of root injury to cell death and spread of *Colletotrichum graminicola* in sorghum. Plant Disease Reporter 51:957-959.

KATSANOS, R.A, and PAPPELIS, A.J. 1968. Patterns of cell death in sorghum stalk tissue as a measure of the susceptibility to spread of *Colletotrichum graminicola* in fifty-five sorghum varieties. Plant Disease Reporter 52:68-70.

KATSANOS, R.A., and PAPPELIS, A.J. 1969a. Relationship of living and dead cells to spread of *Colletotrichum graminicola* in sorghum stalk tissue. Phytopathology 59:132-134.

KATSANOS, R.A., and PAPPELIS, A.J. 1969b. Patterns of cell death in stalks of normal and injured sorghum. Transactions of the Illinois State Academy of Science 62:3-7.

KOEHLER, B. 1960. Corn stalk rots of Illinois. Agricultural Experiment Station Bulletin No. 658. Urbana, Illinois, USA: Illinois Agricultural Experiment Station.

KOMMEDAHL, T., WINDELS, C.E., and STUCKER, R.E. 1979. Occurrence of *Fusarium* species in roots and stalks of symptomless corn plants during the growing season. Phytopathology 69:961 -966.

KONINGS, H. 1982. Ethylene-promoted formation of aerenchyma in seeding roots of *Zea mays* L under aerated and non-aerated conditions. Physiologia Plantarum 54:119-124.

KULFINSKI, F.B., and PAPPELIS, A.J. 1976. The use of quantitative interferometry in the study of epidermal tissue. Pages 637-657 *in* Advances in the microbiology of aerial surfaces of plants (eds. T.F. Preece and O H. Dickinson). London, U.K.: Academic Press.

LIU, R. 1972. The relationship of ribonuclease and deoxyribonuclease activities to tissue senescence in corn. M.Sc. thesis, Southern Illinois University at Carbondale, Carbondale, Illinois, USA. 48 pp.

MAHON, J.D. 1983. Limitations to the use of physiological

variability in plant breeding. Canadian Journal of Plant Science 63:11-21.

MAIZUSH, N.A., FRITTON, D.D., and KENDALL, W.A. 1980. Root morphology and early development of maize at varying levels of nitrogen. Agronomy Journal 72:25-31.

McKEEN, W.E. 1953. Preliminary studies of root and basal stalk rot of maturing corn in Ontario. Canadian Journal of Botany 31:132-141.

McNEW, G.L. 1937. Crown infection of corn caused by *Diplodia zeae,* Iowa Agricultural Experiment Station Research Bulletin No. 216.

McPHERSON, D.C. 1939. Cortical air spaces in the roots of *Zea mays* L New Phytologist 38:190-202.

MEYER, A. 1966. Growing corn without soil. IAA Record (Illinois Agricultural Association, Normal, Illinois, USA) 44(4):26-27.

MILES, J.W., DUDLEY, J.W., WHITE, D.G.,and LAMBERT, R.J. 1980. Improving corn population for grain yield and resistance to leaf blight and stalk rot. Crop Science 20:247-251.

MILLER, T.L., and MYERS, O.1974. Correlation of pith cell death with various stalk quality characteristics in two synthetic populations of maize. Crop Science 14:217.

MOCK, J.J. 1982. Breeding corn for no-till farming. Pages 103-117 *in* Proceedings of the 37th Annual Corn and Sorghum Research Conference. Washington, D.C, USA: American Seed Trade Association.

MUMFORD, P.M., and PAPPELIS, A.J. 1978. Dry mass of *Fusarium roseum* spores before and after germination. Mycopathologia 64:63-64.

MURPHY, J.A. 1977. Ultrastructure of *Diplodia rnaydis* and of its infection process in corn (*Zea mays*), Vols. I, II, and III. Ph.D. thesis, Southern Illinois University at Carbondale, Carbondale, Illinois, USA. 757 pp.

MURPHY, J.A., CAMPBELL, L.L., and PAPPELIS, A.J. 1973. Morphological observations of *Diplodia rnaydis* on synthetic and natural substrates as revealed by scanning electron microscopy. Applied Microbiology 27:232-250.

MURPHY, J.A., PAPPELIS, A.J., THOMPSON, M A. and CAMPBELL, L.L. 1974. Morphological aspects of *Diplodia rnaydis* and its role in the stalk rot of corn. Electron Microscopy 1974 (Part II):405-412.

MURPHY, J.A., THOMPSON, M.R., and PAPPELIS, A.J. 1976. Ultra-structure and elemental composition of dormant and germinating *Diplodia rnaydis* spores. Journal of Bacteriology 127:1465-1471.

MURPHY, J.A., THOMPSON, M.R., and PAPPELIS, A.J. 1977. Ultrastructural localization of cellulose in *Diplodia maydis*-infected corn stalk tissue. Proceedings, Electron Microscopy Society of America 35:454-455.

MURPHY, J.A., THOMPSON, M., and PAPPELIS, A.J. 1980. Ultra-structure of *Diplodia rnaydis* grown on selected synthetic media. Mycopathologia 71:171-191.

PAPPELIS, A.J. 1957. Nature of resistance to diplodia stalk rot of corn. Ph.D. thesis, Iowa State University, Ames, Iowa, USA.

PAPPELIS, A.J. 1963. Corn stalk rot symptoms induced by root injury. Phytopathology 53:624 (abstact).

PAPPELIS, A.J. 1965. Relationship of seasonal changes in pith condition and density to gibberella stalk rot of corn. Phytopathology 55:623-626.

PAPPELIS, A.J. 1970a. Double inoculation for corn stalk rot studies. Forschungen Gebiet Pflanzenkrankheiten (Shokubutsu Byogai Kenkyu) 7:85-91. (Kyoto University, Kyoto, Japan.)

PAPPELIS, A.J. 1970b. Effect of root and leaf injury on cell death and stalk rot susceptibility in corn. Phytopathology 60:355-357.

PAPPELIS, A.J., BeMILLER, J.N., SCHMID, W.E., MYERS, O., and MURPHY, J.A. 1971. Stalk rot of corn. Pages 148-164 *in* Proceedings of the 26th Annual Corn and Sorghum Research Conference. Washington, D.C, USA: American Seed Trade Association.

PAPPELIS, A.J., and BOONE, L.V. 1966a. Effects of soil fertility on cell death in corn stalk tissue. Phytopathology 56:850-852.

PAPPELIS, A.J., and BOONE, L.V. 1966b. Effect of planting date on stalk rot susceptibility and ceil death in corn. Phytopathology 56:829-831.

PAPPELIS, A.J., and KATSANOS, R.A. 1965a. An approach to the study of the physiology of senescence and parasitism in sugar cane. Phytopathology 55:620-622.

PAPPELIS, A.J., and KATSANOS, R.A. 1965b. Spread of *Physalospora tuccumanensis* in stalk tissue of sugar cane. Phytopathology 55:807-808.

PAPPELIS, A.J.. and KATSANOS, R.A. 1966. Effect of plant injury on senescence of sorghum stalk tissue. Phytopathology 56:295-297.

PAPPELIS, A.J., and KATSANOS, R.A. 1969. Ear removal and cell death in corn stalk tissue. Phytopathology 59:129-131.

PAPPELIS, A.J., and LIU, K.C. 1966. Effects of NPK on cell senescence in corn stalk tissue. Proceedings of the Association of Southern Agricultural Workers 63:296 (abstract).

PAPPELIS, A.J., MAYAMA, S., MAYAMA, M., BeMILLER J.N., MURPHY, J.A., MUMFORD, P., and PAPPELIS, G.A. 1973a. Parenchyma cell death and *Diplodia rnaydis* susceptibility in stalks and ears of corn. Plant Disease Reporter 57:308-310.

PAPPELIS, A.J., MAYAMA, S., PAPPELIS, G.A., MAYAMA, M., and BeMILLER, J.N. 1973b. Increases in host nuclear

dry mass, nuclear area and nucleolar area in diplodia-. infected corn. Plant Disease Reporter 57:1043-1045.

PAPPELIS, A.J., MUMFORD, P.M., ABNEY, T.S., and PAPPELIS, G.A. 1975. Classification of corn inbreds using pith cell death patterns and the prediction of stalk rot response. Cereal Research Communications 3:227-232.

PAPPELIS, A.J., MUMFORD, P.M., and WU, L. 1979. Dry mass changes in germinating spores of *Diplodia maydis.* Mycopathologia 67:111-114.

PAPPELIS, A.J., and MYERS, O.1970. Parenchyma cell death patterns in the stalk, shank, cob and leaf midrib of maize. Agronomy Abstracts No. 27.

PAPPELIS, A.J., SCHMID, W.E., KOIKE, H., and MUSGRAVES, R.A. 1967. Development of a hydroponic unit for growth of corn to maturity. Transactions of the Illinois State Academy of Science 60:90-94.

PAPPELIS, A.J., and SMITH, F.G. 1963. Relationship of water content and living cells to spread of *Diplodia zeae* in corn stalks. Phytopathology 53:1100-1105.

PAPPELIS, A.J., and WILLIAMS, J.R. 1966. Patterns of cell death in elongating com stalks. Transactions of the Illinois State Academy of Science 59:195-198.

PETERS, D.W., SHANK, D.B., and NYQUIST, W.E. 1982. Root-pulling resistance and its relationship to grain yield in F_1 hybrids of maize. Crop Science 22:1112-1114.

SCHMID, W.E., PAPPELIS, A.J., and IMBAMBA, S.K. 1966. Nutrient content as related to senescence in sugar cane. Transactions of the Illinois State Academy of Science 59:201-204.

SCHNEIDER, R.W., and PENDERY, W.E. 1983. Stalk rot of corn: Mechanisms of predisposition by an early-season water stress. Phytopathology 73:863-871.

SHURTLEFF, M.C. 1980. Compendium of corn diseases, 2nd edition. St. Paul, Minnesota, USA: American Phytopathological Society.

SMITH, A.L., HOPPE, P.E., and HOLBERT, J.R. 1938. Development of a differential inoculation technique for diplodia stalk rot of corn. Phytopathology 28:497-504.

SMITH, O.S. 1983. Evaluation of recurrent selection in BSS, BSCB1, and BS13 maize populations. Crop Science 23:35-40.

SUTTON, J.C. 1982. Epidemiology of wheat head blight and maize ear rot caused by *Fusarium graminearum.* Canadian Journal of Plant Pathology 4:195-209.

SUTTON, J.C., and PROCTOR, R. 1982. Dispersion analysis of gibberella ear rot in maize. Canadian Journal of Plant Pathology 4:254-258.

THOMPSON, D.L. 1969. Selection for stalk quality in corn. Pages 7-14 *in* Proceedings of the 24th Annual Corn and Sorghum Research Conference. Washington, D.C., USA: American Seed Trade Association.

THOMPSON, D.L. 1982. Grain yield of two synthetics of corn after seven cycles of selection for lodging resistance. Crop Science 22:1207-1210.

TWUMASI-AFRIYIE, S., and HUNTER, R.B. 1982a. Evaluation of quantitative methods for determining stalk quality in short-season corn genotypes. Canadian Journal of Plant Science 62:55-60.

TWUMASI-AFRIYIE, S., and HUNTER, R.B. 1982b. Lodging-enhancing techniques for use on corn performance trials in short-season areas. Canadian Journal of Plant Science 62:299-304.

ULLSTRUP, A.J. 1955. Diseases of corn. Pages 465-536 *in* Corn and corn improvement (ed. G.F. Sprague). New York, New York, USA: Academic Press.

ULLSTRUP, A.J. 1961. Corn diseases in the United States and their control. U.S. Department of Agriculture Handbook No. 199. Washington, D.C., USA: USDA. 38 pp.

ULLSTRUP, A.J. 1977. Diseases of corn. Pages 391 -500 *in* Corn and corn improvement (ed. G.F. Sprague). Madison, Wisconsin, USA: American Society of Agronomy.

VREUGDENHIL, D. 1983. Abscisic acid inhibits phloem loading of sucrose. Physiologia Plantarum 57:463-467.

WHITE, D.G., YANNEY, J., and NATTI, T.A. 1979. Anthracnose stalk rot. Pages 1 -15 *in* Proceedings of the 34th Annual Corn and Sorghum Research Conference. Washington, D.C., USA: American Seed Trade Association.

WHITNEY, N.J., and MORTIMORE, C.G. 1957. Root and stalk rot of field corn in southwestern Ontario: I. Sequence of infection and incidence of the disease in relation to maturation of inbred lines. Canadian Journal of Plant Science 37:342-346.

WHITNEY, N.J., and MORTIMORE, C.G. 1961. Root and stalk rot of field corn in southwestern Ontario: II. Development of the diseases and isolation of organisms. Canadian Journal of Plant Science 41:854-861.

ZUBER, M.S. 1983. Challenges for maize breeders-today's challenges for increased maize production tomorrow. Pages 88-102 *in* Proceedings of the 37th Annual Com and Sorghum Research Conference. Washington, D.C., USA: American Seed Trade Association.

ZUBER, M.S., COLBERT, T.R., and DARROH, L.L. 1980. Effect of recurrent selection for crushing strength on several stalk components in maize. Crop Science 20:711 - 717.

ZUBER, M.S., and KANG, M.S. 1978. Corn lodging slowed by sturdier stalks. Crops and Soils Magazine 30:13-15.

ZUBER, M.S.,MUSICK,G.J.,and FAIRCHILD, M.L. 1971. A method of evaluating corn strains for tolerance to western corn root-worm. Journal of Economic Entomology 64:1514-1518.

Questions

Scheuring:
Did you develop a scoring system for root cell pith death in conjunction with your stem pith scoring system? Wouldn't you think such a root and stem scoring system would be essential for adequately identifying resistance to stalk rots?

Pappelis:
I did use a root parenchyma cell death system: 0, no dead cells; 1, less than half of the cortical cells dead; and 2, more than half of the cortical cells dead. For stalk rot, I recommend the pith condition and discoloration rating systems I described in my manuscript prepared for this meeting.

Root and Stalk Rots Caused by *Macrophomina phaseolina* in Legumes and Other Crops

J.B. Sinclair*

Summary

Symptoms of charcoal rot and the isolation and identification of the causal fungus, Macrophomina phaseolina, *are described. The disease cycle and factors that affect the epidemiology of the disease are reviewed. Studies on various control practices are presented, A number of studies suggest resistance may be available in several crops. Since* M. phaseolina *causes disease in stressed plants, maintaining vigorous plants through recommended cultural practices, particularly by providing adequate organic matter and moisture, should be followed. Systemic fungicides may be used when economically feasible.*

Macrophomina phaseolina (Tassi) Goid. (*Rhizoctonia bataticola*(*Taub*.) Butler) causes charcoal rot of root and stems, foliage blight, and fruit and tuber decay. The fungus infects more than 300 plant species, including a wide range of cultivated crops, and although present in most cultivated soils of the world, charcoal rot is prevalent in the warm temperate and tropical cropping areas when dry conditions prevail or when plants are under water stress. The disease often appears on irrigated soybeans when water is withheld to promote maturity. Magalhaes et al. (1982) showed that the incidence of common bean plant death caused by the fungus increased from 8.6% under ideal soil moisture conditions to 63.9% with 18 days of water deficit. Losses due to this disease are difficult to determine since diagnostic symptoms usually appear when infected plants are in progressive senescence or under low-moisture or other stress condition. However, infection may take place throughout the growing season, often causing continuous debilitation of the host. When severe, the pathogen can reduce stands, plant vigor, yields, and seed quality. Losses up to 77% have been estimated on soybeans due to the disease. However, it is often difficult to determine yield losses due to the pathogen and those due to the stress factors that encourage the disease.

A report on the state of the knowledge of *M. phaseolina* was published as an annotated bibliography (Dhingra and Sinclair) in 1977 and a review of the literature (Dhingra and Sinclair) in 1978. This present review uses in part the material from these two references and that presented in the Compendium of Soybean Diseases (Sinclair 1982).

Symptoms

Symptoms of the disease are usually confined to the roots, crowns, and lower stalks, but infection of the above-ground parts of many crop plants has been reported (Dhingra and Sinclair 1977,1978).

Infected seedlings may show a reddish-brown discoloration at the emerging portion of the hypocotyl, which may be confused with symptoms produced by infection by *Rhizoctonia solani* Kuehn. Infected melon seeds have given rise to infected

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International Crops Research Institute for the Semi-Arid Tropics. 1984. Sorghum Root and Stalk Rots, a Critical Review: Proceedings of the Consultative Group Discussion on Research Needs and Strategies for Control of Sorghum Root and Stalk Rot Diseases, 27 Nov - 2 Dec 1983, Bellagio, Italy. Patancheru, A.P. 502 324, India: ICRISAT.

seedlings and increased the inoculum potential in the soil (Reuveni et al. 1983). If infection occurs through the roots, the discoloration appears at the soil line and above. The discolored area turns dark brown to black, and infected seedlings may die under hot, dry conditions. Symptom development may be retarded under wet, cool conditions but continues once again with the return of hot, dry weather.

In older plants, even though colonized earlier in the season, symptoms appear when infected plants are in progressive senescence or under stress by low soil moisture and high temperature or other factor(s). After flowering, a light gray or silvery discoloration develops in the epidermal and subepidermal tissues of the taproot and lower stem. When the epidermis is removed, small, black microsclerotia may be so numerous as to give a grayish-black color to the tissue, resembling powdered charcoal (Fig. 1). Splitting of stems and taproots reveals a reddish-brown discoloration of the vascular and pith tissues, with black streaks in the woody portions. Sclerotia may be found in the pith and vascular elements (Fig. 2). Infected plants produce smaller leaves than normal, a subtle loss of vigor, and in a more advanced stage, leaves turn yellow and wilt, but remain attached (Sinclair 1982, pages 30-33).

Fruit and vegetable decays have been described for many crops, including various cucurbits, papaya, and root crops (Dhingra and Sinclair 1978). These are usually dry rots, unless accompanied by other soft-rotting organisms, and show the presence of the microsclerotia of the fungus.

Foliage infection has been described for many crops, including guava, jute, various *Phaseolus* spp, and tobacco (Dhingra and Sinclair 1978).

Pod and seed infection is reported on a variety of

***Figure 1. Symptoms of charcoal rot of soybeans caused by* Macrophomina phaseolina; *when the epidermis of an infected plant is removed, small black sclerotia are apparent* (*Courtesy: U.S. Department of Agriculture*.)**

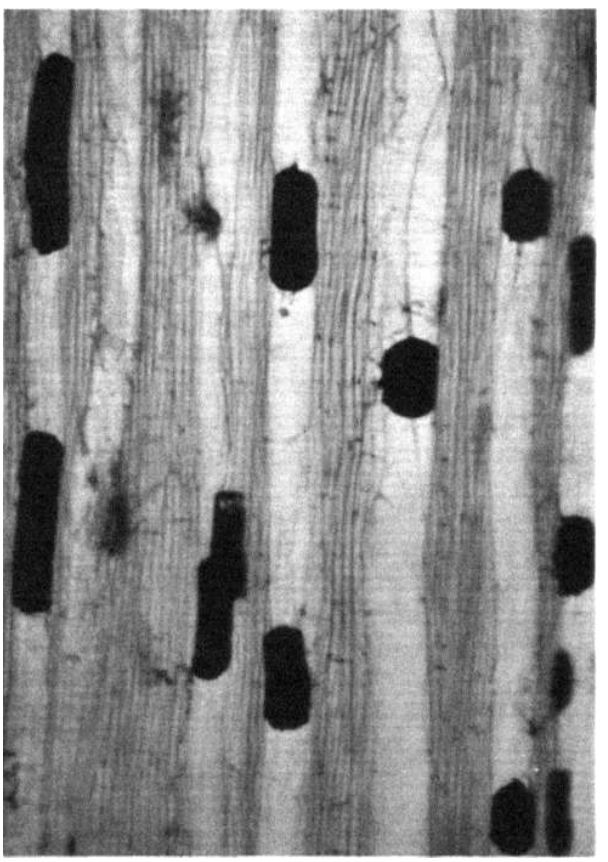

***Figure 2. Charcoal rot of soybeans caused by* Macrophomina phaseolina: *microsclerotia in the xylem vessels of a young plant.* (*Source: Ilyas and Sinclair 1974*.)**

legumes, as well as other crops. The fungus is known to be seedborne in common bean, subterranium clover, cowpea, groundnut (peanut), jute, maize (corn), melon, foxtail millet, okra, sesame, and soybean (Dhingra and Sinclair 1978). In sesame, infection of the capsule was found in the inner wall, septum, placenta, and seeds, spreading from base to apex (Singh and Singh 1982).

Causal Organism

M. phaseolina is highly variable, differing among isolates in cultural characteristics, sclerotial production and size, presence or absence of pycnidia, and conidia size and shape. Isolates are ecologically and morphologically specific; one isolate recovered from one part of the plant may not cause disease on another part (Dhingra and Sinclair 1973). This characteristic of the fungus can cause problems in selecting breeding lines for resistance.

The fungus produces colonies in culture that range from white to brown to gray and become darker with age (Fig. 3a). Aerial mycelia, with completely or partially appressed growth, may or may not be produced. Some isolates form concentric growth rings. Hyphal branches generally arise at right angles to parent hyphae, but branching at an acute angle is common (Fig. 3b). Most branches show a characteristic constriction at the point of union, and a septum separates the lateral and mother hyphae, as in other *Rhizoctonia* spp. The optimum temperature for growth in culture ranges from 28° to 35°C.

The jet black sclerotia of the fungus are smooth and round to oblong or irregular (Fig. 3c). Their size and shape vary within an isolate and on different substrates. Sclerotia are uniformly reticulate and show no special structural modification in internal form.

Pycnidia, initially immersed in host tissues, are erumpent at maturity. They are more or less globose, membranous or subcarbonaceous, dark to grayish—becoming black with age, and generally 100-200 µ m in diameter. The small truncate ostiole may be inconspicuous or have a definite opening.

The conidia (pycnidiospores), which develop at the tips of conidiogenous cells lining the inner wall of the pycnidium, are cut off by maturity and fill the pycnidial cavity. The conidia are single-celled; ovate, elongate or elliptical; sometimes curved or irregularly contoured; and hyaline and variable in size, with a 3:1 ratio of length to width. Pycnidia and conidia are produced under continuous light and under intermittant light in some isolates, but not in complete dark in culture (Machado 1980, Machado and Kimati 1975). Michail et al. (1977) induced pycnidia on soybean seeds in a water-agar-leaf medium at 20°C under 12-hour alternations of dark and ultraviolet light for 7-10 days. The role of conidia in spread of the disease is not understood.

The fungus grows well on potato dextrose agar and produces sclerotia often 75-150 µ m in diameter, depending upon the nutritional level of the substrate (Fig. 3c).

A number of selective media have been developed for the isolation of M. *phaseolina:* two containing chloroneb, mercuric chloride, streptomycin sulfate, potassium penicillin G, and rose bengal (Meyer et al. 1973); two containing chlortetracydine hydrochloride, and streptomycin sulfate plus either fenaminosulf, oxgall, and quintozene or fenaminosulf, oxgall, and rose bengal (Papavizas and Klag 1975); and one using chloroneb and streptomycin sulfate (Mihail and Alcorn 1982). A modified agar plate technique was described for detecting the fungus in pea seeds (All et al. 1982).

M. phaseolina and *Botryodiplodia theobromae* can be confused in culture (Sinclair 1982, pages 30-33).

Disease Cycle and Epidemiology

Activity Before Penetration

The activity of *M. phaseolina* in the soil before penetration and colonization of the host tissue was summarized by Dhingra and Sinclair (1978). Most colonies of *M. phaseolina* from naturally-infested soils originate from free sclerotia in the soil (Papavizas and Klag 1975). The optimum conditions for the germination of sclerotia in water agar was 24 hours at 32°C, followed by 72 hours of drying between germination flushes (Locke and Green 1977). A number of compounds stimulate sclerotia germination. Crude root exudates and sugar fractions from okra roots stimulate sclerotial germination and mycelial growth of *M. phaseolina,* and amino acids are inhibitory (Goel and Mehrotra 1975). Germinating sesame seeds and seedlings have stimulated sclerotia germination and attracted developing mycelia to the host roots (Abdou et al. 1979). In the spermosphere of soybean, sclerotia germinate within 2-3 mm of the

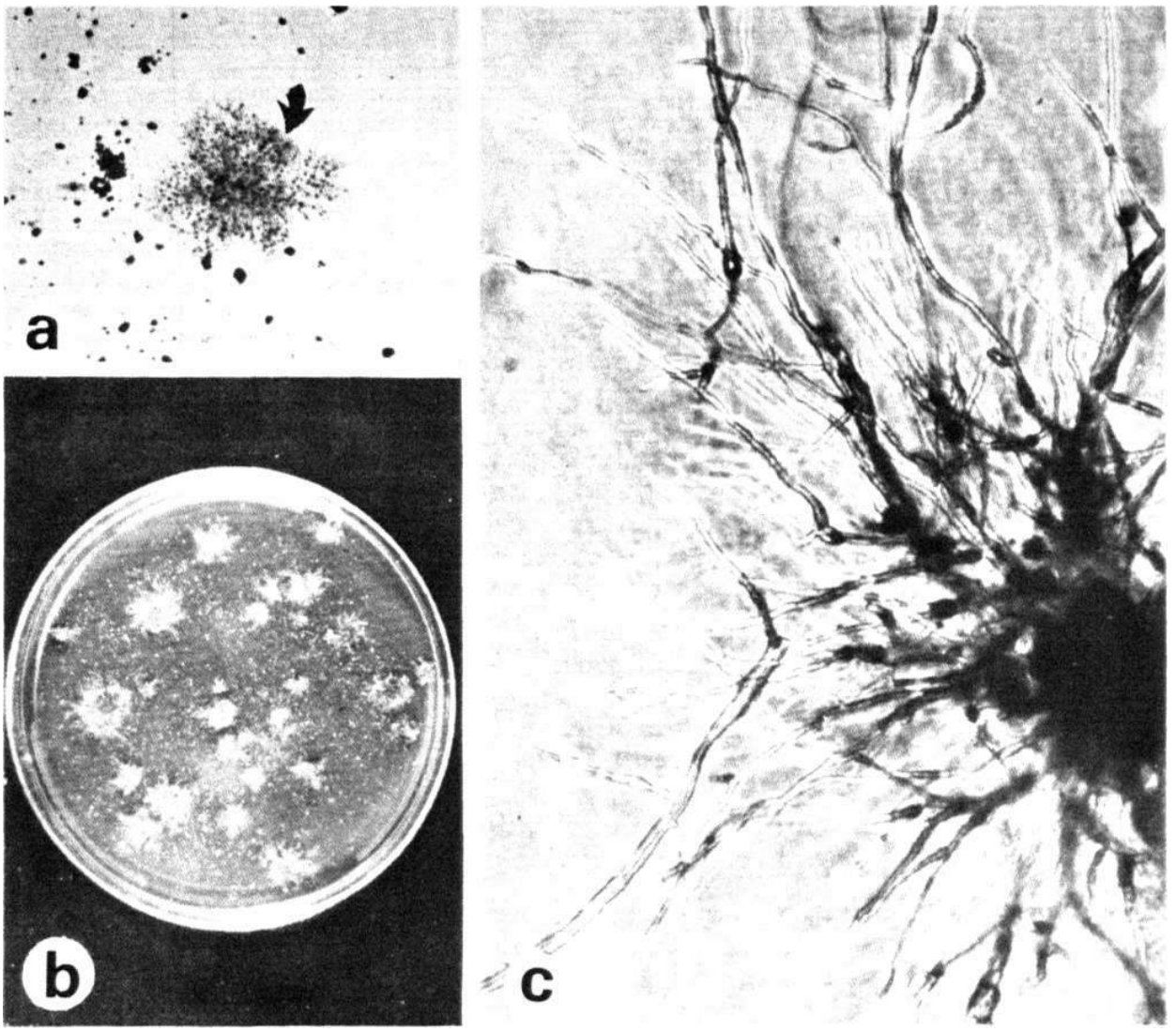

***Figure* 3. *Development of* Macrophomina phaseolina *on selective media:* (a) *a 7-day-old colony* (*arrow*) *showing sclerotia production from a soil sample naturally infested with the fungus and plated on chloroneb/Ceresan Wet medium;* (*b, c*) *plates of chloroneb/mercuric chloride/rose bengal medium, with* (b) *showing individual colonies 6 days after plating a soil sample artificially infested with the test fungus; and* (c) *mycelial development from a single sclerotium 4 days after plating. (Source: Meyer, Sinclair, and Khare 1973.*)**

seed surface (Short and Wyllie 1978). Available nutrients of the substrate affect sclerotia size: the richer the medium, the larger the sclerotia (Short and Wyllie 1978).

Gangopadhyay and Wyllie (1979) showed that an excessive nutrient pool, i.e., high sugar and protein, resulted in rapid germination, abundant growth, and saprophytism of *M. phaseolina* in culture, while a low nutrient pool resulted in increased parasitism. However, Dhingra and Chagas (1981) found that the addition of nitrogen to soil completely inhibited saprophytic colonization. Cerkauskas and Sinclair (1982) reported that paraquat inhibited growth of *M. phaseolina* incorporated into potato dextrose agar and in Fries medium, and inhibited colonization of soybean stem pieces in culture.

Sclerotia may survive free in the soil or embedded in host residue in dry soils for long peri-

ods. Short et al. (1980) showed that the severity of charcoal rot of soybean was directly related to the population of germinable sclerotia in soils and that yields were inversely related to the severity of the disease. Moustafa and Wyllie (1974) found that soybean stubble was a major source of inoculum for seedlings.

Sclerotia of *M. phaseolina* are sensitive to soil f ungistasis (Short and Wyllie 1978) and cannot survive in wet soils for more than 7-8 weeks, and mycelia cannot survive in wet soils for more than 7 days. Thus *M. phaseolina* is a poor competitor in soil. The nonpersistence of *M. phaseolina* in stems in soil suggests that the saprophytic activity of the fungus does not effectively increase its inoculum density in soil (Cerkauskas et al. 1982).

Growth of the fungus in a soil phase is limited by the availability of nutrients. Populations of the fungus in soil increase when hosts are grown continuously in the same field, and the disease thus becomes more severe in successive crops. The role of conidia in the disease cycle is not understood.

Colonization

The penetration and colonization of host tissue by *M. phaseolina* was reviewed by Dhingra and Sinclair (1978). Sclerotia germinate on the surface of roots and produce numerous germ tubes. Penetration of the roots generally occurs from appressoria formed over anticlinal walls of epidermal cells or through natural openings. The fungal hyphae first grow intercellulary, then intracellularly through the xylem, and form sclerotia that plug the vessels. Sclerotia can be formed in green or juvenile tissues but are usually formed as a result of moribundity and release of nutrients.

Dhingra and Chagas (1981) studied the colonization of bean and wheat stems by M. *phaseolina* in two soils. Colonization was maximum at 15-20°C and decreased with increasing soil temperature. At 15°C more wheat than bean stems were colonized; at higher temperatures the reverse was true. Maximum colonization occurred at 15-25% moisture-holding capacity, and there was a decrease with increasing soil moisture. In controlled experiments it was found that infection of soybean seedlings took place in a 15-37°C range of soil temperatures, with infection of seedling stems occurring only at the higher temperatures; infection increased with increased exposure time and temperature (Locke and Green 1976).

M. phaseolina probably causes disease via the mechanical plugging of xylem by sclerotia (Fig. 2), and via toxin production, enzymatic action (pectolytic and cellulytic enzymes), and mechanical pressure exerted by penetration of the middle lamellae (Dhingra and Sinclair 1978, Sinclair 1982).

Control Strategies

A disease management program designed to minimize yield losses should include: (a) adapted resistant or tolerant cultivars, or cultivars with a tendency to escape infection; (b) balanced fertility; (c) good water management; (d) crop rotation; (e) care in weed and insect control; (f) use of high-quality planting seeds; (g) use of fungicides, either as seed, soil, or foliage treatments, if appropriate; and (h) use of antagonists and organic matter.

Disease Resistance

The use of disease-resistant or tolerant cultivars is the most economical and efficient way to control plant diseases. However, resistance to *M. phaseolina* is not widely reported. Resistance in safflower was reported by Qadri and Deshpande (1982); susceptibility appeared to be associated with low sugar content or a rapid drop in sugar following infection. In resistant and susceptible Indian mustard cultivars, there was a general increase in phosphatidase activity in inoculated susceptible cultivars, and activity was considerably lower in resistant ones (Srivastava and Dhawan 1982). Soybean plants were reported to decrease in susceptibility with increased age (Chowdhuri and Karmakar 1978). Short et al. (1978) suggested that the differences in the numbers of propagules in diseased tissues were a measure of the degree of compatibility between soybean cultivars and *M. phaseolina.*

Balanced Fertility

Adequate, balanced fertility is important in reducing disease losses since it appears that high nitrogen tends to reduce the saprophytic ability of the fungus. Also, plants under stress from deficient or toxic levels of nutrients are more susceptible to M.

phaseolina than those grown in soil with well-balanced fertility.

Water Management

Water management practices influence charcoal rot development (Palti 1983). Flooding a field for 3-4 weeks before planting will reduce the viability of soilborne sclerotia and mycelia. The onset of charcoal rot can be delayed by postponing the last application of irrigation water, since the disease develops rapidly under dry, hot conditions and when plants are under water and maturation stress.

Crop Rotation

Although *M. phaseolina* has a wide host range, isolates tend to be somewhat host selective and are ecologically specific. Thus, crop rotation will tend to reduce disease. Bristow and Wyllie (1975) found that the inoculum density of *M. phaseolina* in the soil at planting time from continuous soybean plots was twice that of maize-soybean rotation plots, and that the extent of root colonization by the charcoal rot fungus averaged 33% more ih continuous soybean plots. They concluded that early planting and rotation with maize reduced charcoal rot development. Tillage practices, the crops to use in a rotation, and the length of rotation have not been studied intensively for the control of charcoal rot. However, Bisht (1983) found that row spacings (25 and 76 cm) and six tillage practices in either continuous soybeans or in a maize-soybean rotation did not affect the occurrence of charcoal rot.

Weed and Insect Control

Plants under stress from weed competition, insect injury, or herbicide or insecticide damage will be more susceptible to *M. phaseolina.* Therefore, carefully applied agricultural chemicals to control weeds and insects will help prevent losses from charcoal rot, as well as from other plant diseases.

Seed Quality

Planting undamaged seeds as free as possible from pathogens produces vigorous seedlings and plants that will tend to escape infection by *M. phaseolina* and sustain fewer losses from other pathogens.

Use of Fungicides

A number of fungicides have been tested on a variety of crops for the control of *M. phaseolina.* A selection of reports on some of these tests is summarized in Table 1. Systemic as well as topical fungicides have been used as seed and soil treatments and as foliage sprays.

Systemic fungicides offer the most promise for chemical control of charcoal rot. Carbendazim as a jute seed treatment controlled the disease (Barman and Prasad 1981), but not when used as a soil treatment on bean (Satischandra et al. 1979). Benomyl, carbendazim, and mancozeb increased fiber yield of jute when used as a seed treatment (Barman and Prasad 1981). Thiophanate controlled charcoal root rot on sunflower (El-Dahab et al. 1980) and clover (El-Tobshy et al. 1981b), and thiophanate-methyl controlled the same disease on cowpea, sesame, and sunflower and controlled leaf blight on mungbean (Taneja and Grover 1982). Carboxin, thiabendazole, thiophanate, and ethridiazole controlled macrophomina root rot of Egyptian clover (El-Tobshy et al. 1981b),

Topical fungicides, such as quintozene, controlled bean root rot (Satischandra et al. 1979); and thiram and mancozeb controlled postemergence root and collar rot of groundnut (Natarajan et al. 1983), but mancozeb did not control root rot of bean (Satischandra et al. 1979). Captan alone (Satischandra et al. 1979) or in combination with carboxin (El-Tobshy et al. 1981 b) controlled root rot of bean and clover, respectively.

Four isolates of *M. phaseolina* became temporarily adapted to four fungicides in culture, and as reversion to the parental type occurred, morphological characters also changed (Pan and Sen 1982).

Use of Antagonists and Organic Matter

Two antagonists, *Trichoderma viride* and T. *harzianum,* were active in reducing *M. phaseolina* sclerotial viability in sterilized and nonsterilized soils (Sharma and Bhowmik 1983). Wheat straw and rice hulls used as soil additives controlled root rot of bean caused by *M. phaseolina,* but not farmyard manure or green grass (Satischandra et al. 1979).

Table 1. Fungicides reported to be effective against *Macrophomina phaseolina* either in vivo or in vitro or both.

Fungicide	Crop	Reference
Benomyl	Jute	Barman and Prasad 1981
	Soybean	Ilyas et al. 1976
	In culture	Menten et al. 1976
	In culture	Singh and Chohan 1981
	Sesame, mungbean, sunflower	Taneja and Grover 1982
Captan	Soybean	Ilyas et al.1976
	Bean	Satischandra et al. 1979
Carbendazim	Jute	Barman and Prasad 1981
	Sunflower	El-Dahab et al. 1980
	Sesame, mungbean, sunflower	Taneja and Grover 1982
Carboxin	In culture	Menten et al. 1976
Carboxin + captan	Clover	El~Tobshy et al.1981a,1981b
Copper 8-quinolinolate	Clover	El-Tobshy et al.1981a,1981b
Etridiazole	Clover	El-Tobshy et al. 1981b
Mancozeb	Jute	Barman and Prasad 1981
	Groundnut	Natarajan et al. 1983
Metham	Soybean	Gray 1979
Methyl 4-[2(2-dimethylamino acetamide) phenyl]-3-thioallophanate	Sesame, mungbean, sunflower	Taneja and Grover 1982
Methoxy ethylmercury chloride+thiram	Cotton	Raju et al. 1982
Quintozene	In culture	Menten et al. 1976
	Sunflower	Narasimhan and Prakasam 1983
	Cotton	Raju et al. 1982
	Bean	Satischandra et al. 1979
Thiabendazole	Clover	El-Tobshy et al. 1981a, 1981b
	Soybean	Ilyas et al.1976
Thiophanate-methyl	Sunflower	El-Dahab et al. 1980
	Clover	El-Tobshy et al. 1981a
	Soybean	Ilyas et al. 1976
	Sesame, mungbean, sunflower	Taneja and Grover 1982
Thiram	Soybean	Ilyas et al.1976
	Groundnut	Natarajan et al. 1983
Triforine	Soybean	Ilyas et al. 1976
Zineb	In culture	Kaur and Deshpande 1981

Future Research Priorities

1. **Yield losses.** Field studies need to be conducted to accurately determine yield losses due to M. *phaseolina* and those due to the stress conditions that favor development of charcoal rot. Ideally, genetically related "resistant" and "susceptible" cultivars should be

compared under stress conditions favorable to disease development. However, it must be known that the cultivars do not react differently to the stress situation.

2. **Nonchemical control.** Field studies are needed to compare certain cultural practices, including the addition of potential antagonists, for control of charcoal rot. The control of soil moisture levels and the use of organic amendments should be studied in relation to disease development. Results from published studies are contradictory as to the importance of rotation, tillages, spacing, and other cultural practices. Field studies on the effect of these factors on charcoal rot development in sorghum should be studied.

3. **Chemical control.** Published data suggest that further studies need to be conducted on whether soil or plant application of systemic fungicides provides the most efficient and economical means of controlling charcoal rot.

4. **Integrated control.** To provide the most efficient means of controlling charcoal rot, the best combination of nonchemical and chemical control methods should be determined.

References

ABDOU, Y.A., AL-HASSAN, S.A., and ABBAS, H.K. 1979. Effects of exudation from sesame seeds and seedlings on sclerotial germination and mycelium behaviour of *Macrophomina phaseoiina,* the cause of sclerotial wilt in the soil. Agricultural Research Review (Cairo) 57:167-174.

ALI, S.M., PATERSON, J., and CROSBY, J. 1982. A standard technique for the detecting seed-borne pathogens in peas, chemical control, and testing commercial seed in South Australia. Australian Journal of Experimental Agriculture and Animal Husbandry 22:348-352.

BARMAN, B., and PRASAD, Y. 1981. Seed treatment of jute against stem and root-rot incited by *Macrophomina phaseoiina,* Journal of Research, Assam Agricultural University (Assam, India) 22:48-249.

BISHT, V.S. 1983. Effect of *Cercospora sojina* and *Phomopsis* spp. on soybean seed quality and yield, and of production systems on diseases and yield of soybean. PhD. thesis, University of Illinois at Urbana-Champaign, USA. 303 pp.

BRISTOW, P.R., and WYLLIE, T.D. 1975. The effect of crop rotation and date of planting on charcoal rot of soybean. Proceedings of the American Phytopathological Society 2:83 (abstract).

CERKAUSKAS, R.F., DHINGRA,O.D.,and SINCLAIR, J.B. 1982. Effect of herbicides on competitive saprophytic colonization by *Macrophomina phaseolina* of soybean stems. Transactions of the British Mycological Society 79:201-205.

CERKAUSKAS, R.F., and SINCLAIR, J.B. 1982. Effect of paraquat on soybean pathogens and tissues. Transactions of the British Mycological Society 78:495-502.

CHOWDHURI, A.K., and KARMAKAR, S.K. 1978. The influence of plant age on the susceptibility of soybean (*Glycine max* L. Merrill) to *Macrophomina phaseoiina* (Tassi) Goid. Indian Agriculturalist 22:181-183.

DHINGRA, O.D., and CHAGAS, D. 1981. Effect of soil temperature, moisture, and nitrogen on competitive saprophytic ability of *Macrophomina phaseoiina.* Transactions of the British Mycological Society 77:15-20.

DHINGRA, O.D., and SINCLAIR, J.B. 1973. Location of *Macrophomina phaseoli* on soybean plants related to culture characteristics and virulence. Phytopathology 63:934-936.

DHINGRA, O.D., and SINCLAIR, J.B. 1977. An annotated bibliography of *Macrophomina phaseoiina* 1905-1975. Vicosa, Brazil: Imprensia Universitaria, Universidade Federal de Vicosa. 244 pp.

DHINGRA, O.D., and SINCLAIR, J.B. 1978. Biology and pathology of *Macrophomina phaseoiina.* Vicosa, Brazil: Imprensia Universitaria, Universidade Federal de Vicosa. 166 pp.

EL-DAHAB, M.K.A, TARABETH, A.M., and MOHAMED, S.E. 1980. Studies on sunflower diseases in Egypt: 1. Studies on charcoal rot and its control. Egyptian Journal of Phytopathology 12:113-122.

EL-TOBSHY, Z.M., EL-SAYED, E.I., RAMMAH, A., and ABD EL-SATTAR, M.A. 1981a. Pathogenicity and control of three fungi associated with damping-off and root-rot of the Egyptian clover *Trifolium alexandrinum* L Faculty of Agriculture, Ain Shams University, Research Bulletin No. 1674.14 pp.

EL-TOBSHY, Z.M., EL-SAYED, E.I., ABD EL-SATTAR, M.A., and RAMMAH, A. 1981b. Studies on the control of damping-off and root-rot diseases of the Egyptian clover, Faculty of Agriculture, Ain Shams University, Research Bulletin No. 1718. 11 pp.

GANGOPADHYAY, S., and WYLLIE, T.D. 1979. The effect of carbon:nitrogen ratios on sclerotial germination and pathogenicity of *Macrophomina phaseoiina.* Phytopathology 67:1028 (abstract).

GOEL, S.K., and MEHROTRA, R.S. 1975. Biochemical nature of root exudates of *Abelmoschus esculentus* in relation to pathogenesis of root and collar rot caused by *Rhizoctonia bataticola.* Acta Phytopathologica Academiae Scientiarum Hungaricae 10:41-49.

GRAY, L.E. 1979. Effect of soil fumigation on survival of *Macrophomina phaseolina* in soybean stem residue. Phytopathology 69:540 (abstract).

ILYAS, M.B., ELLIS, M A , and SINCLAIR, J.B. 1976. Effect of soil fungicides on *Macrophomina phaseolina* sclerotium viability in soil and in soybean stem pieces. Phytopathology 66:355-359.

ILYAS, M.B., and SINCLAIR, J.B. 1974. Effects of plant age upon development of necrosis and occurrence of intraxylem sclerotia in soybean infected with *Macrophomina phaseolina.* Phytopathology 64:156-157.

KAUR, M., and DESHPANDE, K.B. 1981. *In vitro* evaluation of fungicides and antibiotics against *Macrophomina phaseolina* (Tassi) Goid., the incitant of leaf spot of soybean (*Glycine max*). Hindustan Antibiotics Bulletin 23:41 - 44.

LOCKE, J.C., and GREEN, R.J., Jr. 1976. The effect of soil temperature on infection of soybean seedlings by *Macrophomina phaseolina.* Proceedings of the American Phytopathological Society 3:276 (abstract).

LOCKE, J.C., and GREEN, R.J., Jr. 1977. The effect of various factors on germination of sclerotia of the charcoal rot fungus, *Macrophomina phaseolina.* Proceedings of the American Phytopathological Society 4:97 (abstract).

MACHADO, C.C. 1980. [Sporulation of *Macrophomina phaseolina* (Tassi) Goid. and feasibility of a spore inoculation method in screening germplasm for resistance.] M.Sc. dissertation, University of Sao Paulo, Piracicaba, Brazil. 66 pp. (In Portuguese, English summary).

MACHADO, C.C., and KIMATI, H. 1975. [Effect of light on formation of pycnidia of *Macrophomina phaseolina* in culture.] Summa Phytopathologica 1:65-66 (in Portuguese).

MAGALHAES, A.A. de, CHOUDHURY, M.M., MILLAR, A.A., and ALBUQUERQUE, M.M. de. 1982. [Effect of soil water deficit on *Macrophomina phaseolina* infection on bean.] Pesquisa Agropecuaria Brasileira 17:407-411 (in Portuguese).

MENTEN, J.O.M., MACHADO, C.C., MINUSSI, E., CASTRO, C., and KIMATI, H. 1976. [Effect of some fungicides on the growth of *Macrophomina phaseolina* (Tassi) Goid. in vitro.] Fitopatologia Brasileira 1:57-66 (in Portuguese, English summary).

MEYER, W.A., SINCLAIR, J.B., and KHARE, M.N. 1973. Biology of *Macrophomina phaseoli* in soil studied with selective media. Phytopathology 63:613-620.

MICHAIL, S.H., ABD EL-REHIM, M.A., and ABU ELGASIM, E.A. 1977. Pycnidial induction in *Macrophomina phaseolina* in seed samples of four soybean cultivars. Acta Phytopathologica 12:311-313.

MIHAIL, J.D., and ALCORN, S.M. 1982. Quantitative recovery *of Macrophomina phaseolina* sclerotia from soil. Plant Disease 66:662-663.

MOUSTAFA, A.M., and WYLLIE, T.D. 1974. Preliminary studies on overwintering and survival of *Macrophomina phaseolina.* Proceedings of the American Phytopathological Society 1:127 (abstract).

NARASIMHAN, V., and PRAKASAM, N. 1983. Efficacy of certain fungicides in the control of root rot of sunflower. Pages 87-88 *in* Proceedings of the National Seminar on Management of Diseases of Oilseed Crops. Madurai, India: Tamil Nadu Agricultural University.

NATARAJAN, S., NARAYANASAMY, P., and KANDASWAMY, T.K. 1983. Control of post-emergence root rot and collar rot diseases of groundnut. Pages 29-30 *in* Proceedings of the National Seminar on Management of Diseases of Oilseed Crops. Madurai, India: Tamil Nadu Agricultural University.

PALTI, J. 1983. Coordination of disease control options in irrigated crops. Page 69 *in* Abstracts of papers presented at the Fourth International Congress of Plant Pathology, University of Melbourne, Queensland, Australia.

PAN, S., and SEN, C. 1982. Persistence of tolerance and cross resistance among fungicide-adapted isolates of *Macrophomina phaseolina.* Zeitschrift fur Pflanzenkrankheiten und Pflanzenschutz 89:399-405.

PAPAVIZAS, G.C., and KLAG, N.G. 1975. Isolation and quantitative determination of *Macrophomina phaseolina* from soil. Phytopathology 65:182-187.

QADRI, S.M.H., and DESHPANDE, K.S. 1982. Studies on root/collar rot of safflower caused by *Rhizoctonia bataticola.* Indian Botanical Reporter 1:141-142.

RAJU, K.S., PANDU, S.R., and ADISESHAIAH, J. 1982. Varietal reaction and evaluation of fungicides against root rot of cotton. Indian Journal of Mycology and Plant Pathology 11:294-295.

REUVENI, R., NACHMIAS, A., and KRIKUN, J. 1983. The role of seedborne inoculum on the development of *Macrophomina phaseolina* on melon. Plant Disease 67:280-281.

SATISCHANDRA, K.M., HIREMATH, R.V., and HEGDE, R.K. 1979. Effect of organic amendments and fungicides on the saprophytic activity of *Rhizoctonia bataticola* causing root rot of beans. Indian Phytopathology 32:543-546.

SHARMA, R.C., and BHOWMIK, T.P. 1983. Sclerotial population of *Macrophomina phaseolina* in field soil under tropical climate and the effects of inorganic amendments and soil antagonists on their viability. Page 254 *in* Abstracts of papers presented at the Fourth International Congress of Plant Pathology, University of Melbourne, Queensland, Australia.

SHORT, G.E., and WYLLIE, T.D. 1978. Inoculum potential

of *Macrophomina phaseolina.* Phytopathology 68:742-746.

SHORT, G.E., WYLLIE, T.D., and AMMON, V.D. 1978. Quantitative enumeration of *Macrophomina phaseolina* in soybean tissues. Phytopathology 68:736-741,

SHORT, G.E., WYLLIE, T.D., and BRISTOW, P.R. 1980. Survival of *Macrophomina phaseolina* in soil and in residue of soybean. Phytopathology 70:13-17.

SINCLAIR, J.B. 1982. Compendium of soybean diseases. St. Paul, Minnesota, USA: American Phytopathological Society. 104 pp.

SINGH, R.A., and CHOHAN, J.S. 1981. Effect of Benlate on growth and spore germination of *Macrophomina phaseolina.* Pesticides 15(7):8.

SINGH, T., and SINGH, D. 1982. Transmission of seedborne inoculum of *Macrophomina phaseolina* from seed to plant. Proceedings of the Indian Academy of Sciences 91:357-370.

SRIVASTAVA, S.K., and DHAWAN, S. 1982. Phosphatidase activity in *Brassica juncea* plants infected with isolates of *Macrophomina phaseolina* and its role in pathogenesis. Bulletin of the Torrey Botanical Club 109:508-512.

TANEJA, M., and GROVER, R.K. 1982. Efficacy of benzimidazole and related fungicides against *Rhizoctonia solani* and *R. bataticola.* Annals of Applied Biology 100:425-432.

Questions

Odvody:
Were all of the variant isolates of *M. phaseolina* from the same plant still virulent pathogens on soybean?

Sinclair:
Yes.

Odvody:
Could you speculate on the importance of pycnidia in the disease cycle of *M. phaseolina* where it does occur on the host plant?

Sinclair:
Nothing has been published on the importance of pycnidia in the life cycle. Pycnidia production can occur on any portion of the soybean plant.

Williams:
You said that the pathogen has over 400 hosts. Then you said it specialized between maize and sorghum. What is your message?

Sinclair:
The pathogen isolated from various crops like maize, soybean, squash, etc., can be cross-inoculated. However, the isolates from the same crop in continuous maize or soybean are more virulent than if coming from a different crop (rotation). This has been shown from 5 years' data on continuous maize or continuous soybeans.

Eastin:
If charcoal rot is taking its toll from nearly the beginning in soybeans as you suspect, how do you measure the toll?

Sinclair:
It would require studies under controlled conditions.

Pappelis:
When you pass the organism through a crop several times, does the organism become more virulent?

Sinclair:
I don't believe in the bridge-post theory. It's only a theory. The isolate adapts to the crop by passing through the same crop many years.

Omer:
Did you state that the fungus can attack active cells?

Sinclair:
Yes, it's in 2-week-old seedlings.

Omer:
Is it not the case that the fungus attacks only dead or senescing cells?

Sinclair:
This is the challenge I am posing. Sorghum scientists should look for the fungal attack early in the crop growth stage, since in other crops this pathogen is becoming virulent much earlier.

Williams:
How does the seed infection occur?

Sinclair:
It is through the pod and not systemic.

Experience with Root and Stem Rots of Crops Other than Sorghum

Summary and Synthesis

J.E. Partridge*

It is difficult, if not impossible, to find another crop plant to compare with sorghum, considering that sorghum is primarily grown as a nonsenescing plant that produces a "dry" seed as the agricultural product of interest. After considering some of the possibilities, we find that the woody perennials are the closest "crop," but the biochemical, physiological, and phenotypical differences are sufficient to dissuade us in that comparison.

Other crops that may be useful for comparison are the oil seed crops, including sunflower, safflower, and castor bean. In these crops, a *Fusarium* complex such as we find in sorghum (as reviewed by Dr. Zummo in these proceedings) or maize (as reviewed by Dr. Pappelis in these Proceedings) has not been characterized. The *Fusarium* species that are pathogenic to these crops typically cause a root rot and/or wilt, and not the stalk deterioration typical in sorghum. Conversely *Macrophomina phaseolina* does occur in sunflower.

Macrophomina Stalk Rots

M. phaseolina has a wide host range, causing disease in over 293 plant species. Known in South America as *Pesta Negra,* it is the most destructive stalk rot of sunflower under high temperature and drought conditions. Its occurrence is unpredictable and, while frequent in southern areas where high temperature and drought are common, it is rare in northern areas.

According to Cobia and Zimmer (1979, pp 27-28):

> Usually, symptoms are not apparent until after flowering, when poorly filled heads are evident and premature ripening and drying of the stalks occur. The diseased stalks normally are discolored at the base, the pith is disintegrated, and the vascular fibers have a shredded appearance. After a period of hot and dry weather, the fibers become covered with small black sclerotia.

This description of the disease and its etiology leads one to wonder if the internal shredding is a component of the disease contributed by the pathogen irrespective of the host, while the lodging component is contributed by the particular host. This is not to say that lodging does not occur in nongramineous hosts.

Fusarium Stalk Rot of Maize and Natural Senescence

The following is a discussion of research conducted by Partridge et al. (1984) and presently awaiting publication, It is presented here because It was not available for review by Dr. Pappelis and in hopes that its presentation might aid in our understanding of the disease:

Although different methods have been used to evaluate hybrids for stalk rot reaction, there is at

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least one common factor present in most evaluations. Because of the size of most experiments, investigators have found it necessary to collect data as a single event. We reasoned that since stalk rot is of pathogenic origin, the disease should develop progressively over time. Since not all maize hybrids reach maturity on the same day, we felt that useful information on disease development could be obtained by planting hybrids of similar maturity and monitoring stalk manual-crushability by taking data at weekly intervals.

At the beginning of data acquisition, isolations were made and it was determined that all plants of all hybrids were infected by *Fusarium moniliforme* and/or *Fusarium graminearum* and/or *Fusarium equiseti.* Neither *Diplodia maydisnor Macrophomina phaseolina* was present. Beginning when stalks of all hybrids were still green and continuing until 5 days after killing frost, we took weekly data on manual crushability of the second internode above the brace roots.

Briefly, the data (Fig. 1) indicate that stalk rot symptom expression progresses as a simple interest disease. In terms of Van der Plank (1963, pp 40-51), the r value (infection rate) is constant for all hybrids because the infection was 100% at the time data collection was begun. The rate of symptom expression appears to be characteristic of the individual hybrid.

Any discussion of a disease that occurs in senescing, senescent, or moribund tissue is inherently fraught with the difficulty of separating the natural loss of integrity due to the senescence process from the pathological decomposition that is occurring at the same time. We prefer to use the term "crushability" where loss of integrity is due primarily to the natural senescence process and the role of internal parasites has not actually been determined. The term "stalk rot" we reserve for pathological decomposition that occurs from pathogenic origin. In our experiments, stalk rot is held to be a condition resulting from the activity of pathogens or parasites and clearly apparent only after physiological maturity or killing frost.

Our data indicate that even when the role of microorganisms in the destruction of stalk tissue has been amply demonstrated, it is erroneous not to consider the role of natural stalk senescence in the disease. Accordingly, we interpret the linear increase of stalk crushability prior to physiological maturity as primarily a measure of the rate of senescence peculiar to each hybrid. In the absence of other data, one cannot ignore the possible role of organisms in modifying the rate of senescence; however, it is equally true that one cannot disregard the fact that senescence in maize occurs with or without the involvement of microorganisms.

A = N7a x Mo17
B = B59 x N152
C = H99 x A632
D = W64a x B73
E = N159 x N160
F = Mo17 x B73
G = H99 x B73
H = Mo17 x H99
I = W64a x W117
J = N139 x B73

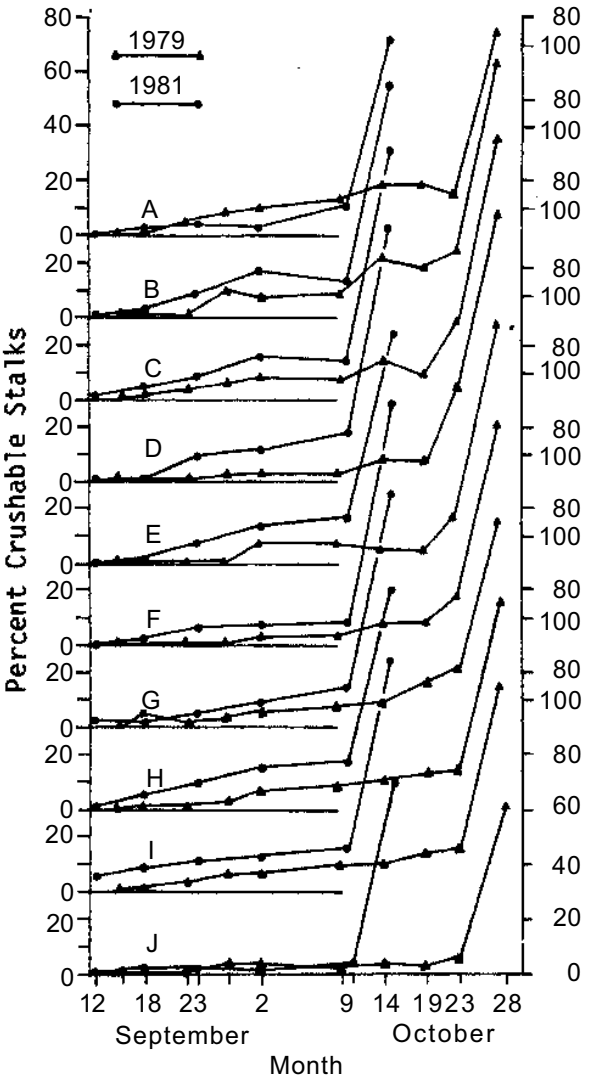

Figure 1. Percent crushable stalks versus age.

Once physiological maturity of the stalk has been reached or senescence is complete, the role of microorganisms becomes apparent and their contribution to stalk rot is pronounced.

In the absence of true physiological resistance, the parasites infect early in the life of the plant, but become pathogens only as the plant senesces. The key to disease management of this type of stalk rot of maize (and possibly sorghum) lies in the potential to develop hybrids that have a rate of ear (head) senescence (dry down) sufficiently more rapid than the rate of senescence of the lower stalk to provide a time interval for harvest.

In summary, it is apparent that the stalk rot disease phenomenon that involves the pathogenic decomposition of the stem or stalk is not restricted to sorghum, grasses, or even annual plants. Additionally, those organisms responsible for stalk rot (i.e., *Fusarium* spp and *Macrophominaphaseolina*) have similar environmental requisites for disease development in a large and varied number of hosts. And finally, the host is not an inert member in the interaction leading to the disease. Its physiological condition as affected by age, stage, maturity, and environment plays a very key role in determining the time of onset of pathogenesis and the severity of the disease.

References

COBIA, D.W., and ZIMMER, D.E. 1979. Sunflower production and marketing. North Dakota Agricultural Experiment Station Bulletin 25.

PARTRIDGE, J.E., WYSONG, D.S., and DOUPNIK, B.L. [1984.] Natural senescence and stalk rot expression in corn. Plant Disease (in press).

VAN DER PLANK, J.E. 1963. Plant diseases: epidemics and control. New York, New York, USA: Academic Press.

Discussion

Relation of Nutrient Deficiencies to Root and Stalk Diseases of Maize

Schneider:
Potassium affects cell death in maize.

Pappelis:
We have gotten K deficiency even at normal levels of K, especially when N is low. When we added increments of K, it could be overcome; but we could also reduce K deficiency by adding N.

Schneider:
Is there a mechanism for this?

Pappelis:
No. We used complete nutrients in our studies (Hoagland solution).

Schneider:
Is there a role of K in carbon translocation? Induced senescence can occur with low K.

Clark:
As for cell death, I have no comment. Potassium is involved in cycling or translocation of carbon. Potassium also interacts with N; NH_4 inhibits K uptake and NO_3 enhances K uptake.

Doupnik:
There are no low K soils in Nebraska, and we found no influence of K on stalk rot.

Maranville:
Nebraska soils are high in K, and we have never found a K deficiency by lowering N.

Pappelis:
By altering N and K, we got what we think was a K deficiency in our greenhouse studies.

Clark:
From mineral deficiency studies, it is sometimes difficult to separate K deficiency from some other deficiencies.

Schneider:
From my studies on mineral nutrition and *Fusarium* on celery, K affected the disease, California soils are high in K also, but when I added K with Cl the

disease was controlled, in contrast to K_2SO_4 and KNO_3. The source of N also affected the disease. Earlier studies indicated similar types of effects of stalk rot on maize [Younts, S.E., and Musgrave, R.B. 1958. Chemical composition, nutrient absorption, and stalk rot incidence of corn as affected by chloride in potassium fertilizer. Agronomy Journal 50:426-429].

Claflin:
Does temperature (especially cooler temperature) affect K uptake?

Clark:
Theoretically yes, but this may not be of much practical importance since cooler temperatures also decrease plant growth.

Maranville:
In soils, NH^+_4 is converted to NO_3^-, and NO_3^- is the predominant form of N taken up by plants.

Clark:
Is the K deficiency really an Mg deficiency? They look very much alike in many cases.

Charcoal Rot (*Macrophomina phaseolina*)

Vidyabhushanam:
Dr. Sinclair, in your presentation, you mentioned that resistance to charcoal rot is complicated. Has any work been done in this regard on soybean? If so, what are the findings?

Sinclair:
No, the problem of charcoal rot on soybean is not considered significant enough in the U.S. to breed for resistance. The disease occurs in some years, but the losses are never of such a level that breeding for resistance is economical.

Partridge:
Soybean plants tend to compensate for losses of plants in the row. Could this be the reason one would not detect certain amounts of seedling losses due to *Macrophomina?*

Sinclair:
Yes, in part. It's always difficult to measure yield losses in soybean due to seedling disease, because soybean plants tend to branch and thus compensate for reduced stands.

Williams:
You recorded seed transmission of *Macrophomina* in soybean. How does it get into the seed?

Sinclair:
We have not done any histopathology on this pathogen. But we have evidence of the presence of the pathogen in the tissues from the base to the top of the plant. I'm sure the pathogen penetrates the seed—if not systemically, then through the pods.

Claflin:
Are you saying that this is a passive transmission in the seed for this organism?

Sinclair:
The seeds were surface sterilized with 70% ethanol, Chlorox, then washed with distilled water, before being placed on filter paper. The transmission was internal.

Pappelis:
Our experience was that we could never get charcoal rot on immature seedlings from soybean seeds collected from the lower part of the plant. We did not know where the organism on mature plants came from, but it did not come from the seed. Where the organism came from is an open question. We have never seen immature plants with charcoal rot. We have seen many other organisms such as *Fusarium* and *Alternaria* on immature plants.

Sinclair:
Did you find *Macrophomina* on immature seed? Mature seed?

Pappelis:
Not on immature seed. We sterilized pods, but we never got the organism. For 5 years, we got extremely low levels or no charcoal rot in our experiments.

Odvody:
Are pycnidia involved?

Sinclair:
The role of pycnidia in the life cycle of this organism has not been studied. I now have students working on this.

Odvody:
On sesame, I've found a lot of pycnidia. On

sorghum and maize, I didn't find any natural occurrence. Isolates from these plants produce pycnidia in specific culture media under long-wave ultraviolet light, but not as readily as sesame isolates.

Sinclair.
I would like to see sorghum scientists try a technique we use. We treat soybean stems, pods, petioles, and leaves with paraquat or glyphosate and can detect latent infection of *Macrophomina, Colletotrichum, Phomopsis, Circospora kikuchii,* and C. *sojina.* Green stem tissues without disease symptoms are dipped in the herbicide (glyphosate is safer to use) and plated on filter paper. In 5 to 7 days, fruiting structures of the fungi appear. We can detect the fungi 2 to 3 weeks in advance of that noted in the field. It's a good technique to detect disease. Other scientists are also using it. I don't know whether it will work for a monocot like sorghum.

Odvody:
We were readily able to isolate *Macrophomina* from symptomless infected roots by incubating them in laboratory humidity chambers after roots were surface sterilized.

Pande:
We have had two kinds of experiences: In one we could not get any *Macrophomina* from symptomless roots. But in the sorghum plant there are primary roots that die early and hang from the crown of the plant. *Macrophomina* can be obtained from these roots, but not from the healthy roots of the same plant. We have successfully isolated the pathogen from the seedling stage to maturity. The second situation occurred when we artificially inoculated the young seedlings that were grown in sterilized Hoagland culture. On the 12th day, irrespective of the variety, we got a kind of discoloration when the *Macrophomina* was put aseptically into the medium. When infected seedlings were planted in sterilized soil, they stayed alive for a while before dying. However, this requires more detailed investigations, which we are presently engaged in.

Omer:
Does infection start at the cotyledon or crown?

Sinclair:
Macrophomina is in the soil all the time and penetrates soybean directly. It does not require wounds or natural openings. The sclerotia will germinate near host roots and penetrate them directly. The organism doesn't need to penetrate the cotyledons or leaves or other plant parts.

Rosenow:
Because sorghum intemodes aren't of equal length like those in maize, and the lower part of the sorghum stem has many nodes, this might cause difficulties in looking at specific internodes, as has been suggested in maize. Dr. Pappelis, do you have any suggestions based on your experience in maize?

Pappelis:
The way we do it is published, and we also have a lot of unpublished data. This can be determined easily. We have looked at numerous maize lines from many stalks.

Jordan:
As temperature increases, the optimum water or osmotic potential for growth is low. Often growth is reduced to a point where optimum growth shifts from -5 to -20 or -30 bars. Has anyone an explanation?

Schneider:
We note this for *Verticillium* and various *Fusarium* species. J. Levitt gave an explanation for this, although I don't remember what it was, in his book on stress physiology [1972. Responses of plants to environmental stresses. New York, N.Y, USA: Academic Press].

Pappelis:
It may be a pH phenomenon. For pathogens taken from maize, if the pH changed from 3 to 8, no growth occurred at 3, but growth did occur at 5. If the tissue was ground and added to media of a resistant variety, the growth of the fungus was inhibited. The pH effects are different for *Gibberella zeae.* An inhibitor may not be present; it may be a matter of a pH change. The pH of an onion cell may be 5.5, but right next to it the pH of a fungus cell may be 3 to 3.5. Other organisms show different results, and I don't know what these mean.

Rosenow:
We have a hard time sticking a toothpick into the same internode all the time, especially when 90% of the stem is peduncle. With nodes so close together, how do we determine which internode to

use? Should we count down from the top of the plant?

Pappelis:
If I can't feel the node, I drill (I don't use a toothpick) to the center. If I don't hit the center, I don't rate that plant. I usually inoculate at least 15 plants, knowing I won't hit the center on all, and take ratings only of plants that have been inoculated in the center. Sometimes I slice the stem to make sure that the inoculum is in the appropriate place. Plants in the row are evenly spaced to reduce other variables. The job isn't easy, and a lot of variability can arise.

Is anyone aware of recent studies on ethylene biosynthesis and how those data might be related to methylase activity in senescing cells? [Editor: See Adams, D.D., and Yang, S.F. 1979. Ethylene biosynthesis: Identification of 1-amino-cyclopropane-1 -carboxylic acid as an intermediate in the conversion of methionine to ethylene. Proceedings, National Academy of Science (USA) 76:170-174.]

Control of Sorghum Root and Stalk Rots

The Role of Fungicides in the Control of Sorghum Root and Stalk Diseases

R.J. Williams and O. Nickel*

Summary

The present knowledge and research activity on the use of fungicides to control sorghum root and stalk diseases are reviewed. Seedling diseases caused by seed- and soilbome fungi are readily controlled by treating seed with small quantities of appropriate fungicides, with combinations of systemic and nonsystemic compounds finding increasing use. Virtually nothing is known about the potential role of fungicides for the control of root and stalk rots of adult sorghum plants, though fungicides are available with activity against the causal organisms. More information is needed on the biology and epidemiology of these diseases in order to better assess the practical possibilities for their control by fungicides. The below-ground infection and early colonization sites are difficult targets for fungicide application, but systemic products, particularly the new generation with high activity at low rates, could offer useful possibilities for integration with host-plant resistance and crop-management control practices.

The importance of sorghum as a food crop in the tropics, the need to rapidly increase sorghum production in many less-developed countries where it is a staple food, and the importance of pest and disease control for the achievement of increased production have been strongly emphasized in recent conferences and publications (ICRISAT 1980, ICRISAT 1982, Williams et al. 1983).

During the past 15 years a major international plant breeding effort has been underway to develop high-yielding cultivars of sorghum. It has not been difficult to develop new sorghum genotypes with high yield potential, but it has been difficult to achieve these potentials on farms in the tropics due to the adverse effects of a wide range of environmental and biotic stresses (ICRISAT 1982), some of which are considerably more severe on the new high-yield-potential cultivars than on traditional landrace cultivars, e.g., grain molds (Williams and Rao 1981) and stalk rots (Dodd 1980).

It is now well accepted that for sorghum cultivars with high-yield potential to be useful in the tropics and subtropics they need protection against a wide range of pests and pathogens. There is a strong research effort in several countries to find and use host-plant resistance to many of the biotic enemies of sorghum, and given time and adequate resources, this approach can be successful for many of them. However, the development of cultivars with resistance to a wide range of pests and pathogens will take time, the resistant cultivars could be vulnerable to "breakdown" through adaptive changes by their genetically variable enemies, and adequate resistance to the full range of pests and pathogens attacking the crop may not be available or may not be feasible to incorporate. There is, therefore, a need to consider the role that other disease control measures can play in the urgent and important endeavor of rapidly increasing on-farm production of sorghum in the less-developed world.

The future of successful, stable disease control must involve the careful integration of all appropriate disease control methods, to maintain disease levels below those that cause significant losses in production and to minimize the opportunities for

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pathogens to adapt to and overcome the disease control measure(s). There is a need for pragmatism, innovation, and above all open-mindedness in considering the possible measures that could be used to control the sorghum pest and disease complex. An impressive array of fungicides is available with strong protective and curative activity against a wide range of plant pathogenic fungi, and more are likely to become available in the near future. While it is recognized that a large proportion of sorghum farmers in the tropics and subtropics are resource poor and cultivate small farms and that therefore they cannot be expected to use fungicides on a large scale as the sole means of control of the sorghum disease complex, we believe it would be a mistake to dismiss the possibilities for the judicious and integrated use of appropriate fungicides in sorghum disease management programs.

In this paper, at the invitation of the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), we have reviewed the role of fungicides in the control of sorghum root and stalk diseases. The basic objectives, as specified in the ICRISAT invitation, are to:

a. assess present knowledge and research activity,
b. determine gaps of knowledge and research,
c. determine future research and strategies for control and management.

Key Questions for Consideration

The key biological questions that need to be considered are:

a. which are the important organisms that incite root and stalk diseases?
b. do fungicides exist that are biologically effective against these organisms?
c. can the fungicides be brought to the critical infection/colonization sites at the critical stages of crop growth to effectively control the organisms before they incite damaging levels of disease?

The key technical, economic, and social considerations concern:

a. the technical skills and complexity of application equipment required to deliver the fungicides to the target sites at the appropriate dosages and crop growth stages;
b. the costs of the fungicides, application equipment, and the actual application relative to the increased value of the treated crop;
c. the safe use of the fungicide on the farm, with regard to crop tolerance, human safety, and environmental compatability.

We believe it to be important to first thoroughly explore the biological questions, to establish what is biologically possible, and not to preclude the use of fungicides at the outset because of apparent technical and/or economic constraints. These latter parameters can change rapidly and dramatically with changes in such factors as government pricing policies and crop production levels.

The Target Organisms and Diseases

The important stalk and root disease problems defined by ICRISAT in the invitation to prepare this review are:

a. fusarium root and stalk disease complex
b. charcoal rot (*Macrophomina phaseolina/Rhizoctonia bataticola*)
c. pythium root and seedling rots
d.. anthracnose stalk rot (*Colletotrichum graminicola*)
e. periconia root and stalk rots
f. acremonium wilt
g. cephalosporium wilt
h. other root and stalk rots

It is interesting, and correct, that seedling diseases have been included among the root and stalk diseases, for, as will be discussed, the attack on the primary roots and shoots of the germinating seeds and the pre- and postemergent seedlings by seed- and soilborne fungi represents an important area for disease control.

Seed and Seedling Diseases

The Problem

The germinating seeds and young seedlings of sorghum are vulnerable to invasion and infection by a large number of seed- and soilborne fungi that

cause seed rot, seedling death, and stunted seedling growth, resulting in reduced plant populations and nonuniform crop growth (Tarr 1962). Adverse environmental factors that reduce the rate of germination and seedling development, such as low soil temperature, waterlogging, and drought, increase the problems of seed rot and seedling disease, while the seedling pathogens, through their damaging effects on young roots and stems, reduce the ability of seedlings to withstand and recover from pre- and postemergence stress problems such as shootfly attack and drought.

Many diverse seed- and soilborne fungi are reported to be associated with seed rot and seedling disease in sorghum (Table 1), including Oomycetes (e.g., *Pythium* spp), Basidiomycetes (e.g., *Rhizoctonia solani*), and Ascomycetes (e.g., *Gibberella* spp, *Glomerella* spp, *Dreschlera* spp). Fungal pathogens that incite diseases of aerial organs of adult plants, such as *Helminthosporium turcicum, Gloeocercospora sorghi,* and *Phoma insidiosa,* and more saprophytic fungi such as *Aspergillus* spp and *Penicillium* spp, can, under suitable conditions, also cause sorghum seed rot and seedling disease (Tarr 1962, Dhanraj 1979).

In addition to the fungi that cause seed rot and seedling disease, sorghum seedlings are vulnerable to infection by highly specialized fungi, such as *Peronosclerospora sorghi* and *Sphacelotheca* spp, that develop systemically within the growing plant to cause disease in the adult plant (*Sphacelotheca* spp) and seedling and adult plant *(P. sorghi*). There are also indications that the fungi involved in stalk rots of the maturing plants may first infect at a much earlier stage of plant development, but more evidence is needed on how early the infection occurs.

Table 1. Sorghum seed and seedling pathogens. (Source: Tarr 1962.)

Disease	Causal fungus
Seed rot	*Alternaria* spp [a]
	Aspergillus spp [a]
	Helminthosporium turcicum
	Penicillium spp
	Phoma insidiosa
	Pyrenochaeta terrestris
	Pythium spp [b]
	Rhizopus spp [a]
Damping-off	*Corticium rolfsii*
	Fusarium culmorum [b]
	Fusarium moniliforme [b]
	Penicillium oxalicum
	Pythium graminicola [b]
	Pythium spp [b]
	Rhizoctonia solani [b]
Seedling blight	*Coltetotrichum graminicola*
	Fusarium graminearum
	Helminthosporium turcicum
	Macrophomina phaseolina
	Phenicillium oxalicum
Root rot	*Pyrenochaeta terrestris*
Downy mildew	*Peronosclerospora sorghi*
Covered smut [c]	*Sphacelotheca sorghi*
Loose smut [c]	*Sphacelotheca cruenta*
Head smut [c]	*Sphacelotheca reiliana*

a. Primarily saprophytic.
b. Main fungi causing poor emergence of sorghum.
c. Infection at seedling stage, but symptoms not expressed until flowering.

Control Measures

Although there are differences among sorghum cultivars in vulnerability to seed rots based on degree of seed hardness (Tarr 1962), several factors act against the successful development of sorghum cultivars resistant to seedling diseases, including:

a. the wide range of organisms involved,
b. the unspecialized facultative pathogenicity of some of the most important causal fungi, and
c. the high degree of vulnerability of seedling tissues to colonization by soil- and seedborne fungi.

Cultural measures that can be used to reduce these disease problems include the selection of clean undamaged seed for planting, and, in some regions, avoiding planting in cold wet soils.

The most widely applicable and consistently effective method to control seed rot, seedling disease, and seedling infection by systemic pathogens of adult plants is the treatment of seed or planting furrows with fungicidal chemicals. The systemic fungicides, which protect against a wide spectrum of plant pathogenic fungi, are particularly valuable, as they enter the seedling tissues and protect the entire seedling for relatively long periods.

In the first half of this century inorganic compounds such as copper carbonate, copper sul-

phate, and sulphur, and mercury-based products were used to treat sorghum seed in various soaking, steeping, sprinkling, and dusting treatments (Tarr 1962). In cold soils, sulphur caused emergence reduction that was not apparent in tropical countries, and the mercurials also had problems of phytotoxicity above certain critical application rates. The spectra of activity of the organomercurial fungicides were broader than those of the inorganic copper and sulphur fungicides, and they were somewhat less phytotoxic at the relative dosages required for effective control of seed- and soilborne fungi. Until the end of the 1970s the organomercurial fungicides were widely used throughout the world in seed treatments against a diverse range of plant pathogenic fungi. However, because of their high mammalian toxicity and persistence, and the growing realization of the hazards of such products, the use of the organomercurial fungicides has by today been severely restricted or even completely banned (as in Australia, Algeria, Canada, Morocco, New Zealand, South Africa, and the USA). Their withdrawal from several other countries can be expected in the near future (Bowling 1978).

From the late 1940s well-tolerated nonmercurial organic compounds, which were relatively low in toxicity compared with the organomercurials, such as thiram (tetramethylthiuram disulphide) and captan [N-(trichloromethylthio)-cyclohex-4-ene-1, 2-dicarboximide] began to be widely used in dust and slurry treatments, often combined with an insecticide such as gamma-BHC, for the protection of sorghum seed and seedlings (Tarr 1962), These fungicides have a strong protective/eradicant activity against a wide range of plant pathogenic fungi, and sorghum seed treatment with them, often in combination with insecticides/is still practiced today, with farmers in some countries able to buy inexpensive sachets of premixed products at the local village store.

The development of systemic fungicides in the 1960s and 1970s has provided a powerful new group of weapons to use in the war on seed and seedling diseases, e.g., (a) the benzimidazole fungicides, which show considerable activity against many diverse pathogens, some of which (e.g., *Colletotrichum* spp, *Fusarium* spp, *Rhizoctonia* spp, *Penicillium* spp) are involved in the seed-rot/seedling-disease complex; (b) the oxathiin derivatives, carboxin and oxycarboxin, which are highly effective against Basidiomycete fungi, thus providing opportunities for the control of *R. solani* and the smut fungi; and (c) the acylalanine fungicides, first

Table 2. Recent literature on the control of sorghum seed mycoflora and on the effectiveness of fungicides in increasing sorghum plant establishment in India through seed treatment.

Source	Type of test	Fungal genera involved	Most effective fungicides (in rank order)	Dosage (% W/W)
Munghate and Raut 1982	Blotter test of seed mycoflora control	*Alternana, Cladosporium, Curvularia, Drechslera, Fusarium, Phoma*	thiram, captan	0.25
Raut and Wangikar 1982	Blotter test of seed mycoflora control	*Alternaria, Cladosporium, Curvularia, Drechslera, Fusarium*	thiram	0.23
Bhale and Khare 1980	Pot test of plant establishment	*Curvularia*	thiram (and) 2-methoxyethylmercury chloride	0.25
Bidari et al. 1978	Pot test of plant establishment	*Fusarium, Curvularia, Helminthosporium*	ferbam, benomyl, thiram	0.2
Sharma et al. 1976	Field test of plant establishment	*Curvularia, Fusarium, Alternaria, Verticillium*	thiram, captan	0.4
Patil-Kulkarni et al. 1972	Field test of plant establishment	*Rhizoctonia* (inoculum introduced)	carboxin, benomyl	0.6

represented by the highly active metalaxyl (Urech et al. 1977), with specific activity against Oomycetes, of which *Pythium* spp play a major role in seedling damping-off, particularly when seed is planted in cold wet soil. Metalaxyl has also been shown to provide excellent protection in sorghum, and other cereals, against the systemic downy mildews (Anahosur 1980, Frederiksen and Odvody 1979,Schwinn 1980).

In Tables 2 and 3 we have summarized results from publications from India and the USA in which reports were made on control of seed- and soilborne fungal pathogens of sorghum seedlings. The current trend in the USA appears to be the combination of the broad-spectrum nonsystemic fungicides such as thiram or captan with one of the modern fungicides, whereas in India the single compounds are still mainly used.

It is quite apparent from the above that there exists today a wide range of fungicides effective against virtually the whole range of fungi that rot seeds and cause death and disease in seedlings. If local researchers can determine what are the important local seed and seedling pathogens of sorghum, suitable pesticide mixes can probably be developed for use in seed treatments for effective control.

Seed treatment with fungicides at the farm level is a simple operation that requires little technical skill and no expensive or complicated equipment. The quantities of fungicide products needed for effective seedling disease control are small (generally in the 0.3-1 g a.i./kg range), making seed treatment more economically feasible for a larger number of farmers than any other means of fungicide treatment of the sorghum crop.

Table 3. Recent literature on the effectiveness of fungicides used as seed treatments in increasing sorghum plant establishment in field tests in the USA.

Source	Fungal genera involved	Most effective fungicides	Dosage	
			U.S. Units	Metric equivalents [a]
Hansing 1974	*Fusarium, Pythium, Rhizoctonia*	thiram + carboxin	1.9 oz/bu + 1.7 oz/bu	2.1 g/kg + 1.9 g/kg
Philley and Frederiksen 1975	Not specified	thiram + carboxin PPG-152 50 W	4.0 oz a.i./cwt + 4.0 oz a.i./cwt 5.0 oz/bu	2.5 g a.i./kg + 2.5 g a.i./kg 5.5 g/kg
Hansing 1975	*Fusarium, Pythium, Rhizoctonia*	Olin OAC 5-4787 50 W	4.0 oz/bu	4.4 g/kg
Hansing 1976	*Fusarium, Pythium, Rhizoctonia*	PCNB 23.2% + etridiazole 5.8% + OAC 5-1563 48 F	2.0 oz/bu 2.1 oz/bu	2.2 g/kg 2.3 g/kg
Hansing 1978	*Fusarium, Pythium, Rhizoctonia* et al.	captan-60% + dieldrin-15%	1.67 oz/bu	1.8 g/kg
Anzalone 1980	*Pythium, Fusarium*	captan 4 carbendazim 7% + maneb 70%	3.4 fl. oz/bu 4.0 oz/bu	3.9 ml/kg 4.4 g/kg
Anzalone 1982	*Fusarium, Pythium, Rhizoctonia*	thiram 17% + carboxin 17%	4.0 oz/bu	4.4 g/kg

a. Approximate values, converted from U.S. Units.

Stalk and Root Rots of Adult Plants

There are very few reports in the literature of research on the fungicidal control of stalk and root rots of sorghum after the crop has passed the seedling stage (Tables 4 and 5). For maize, such reports are somewhat more numerous, but they are few compared with the numbers on the fungicidal control of seedling diseases and smuts of sorghum and of chemical control of nematodes in maize (Table 5). This dearth of 'available literature could indicate that:

a. these diseases are not regarded as sufficiently important to warrant much research effort,
b. they are difficult to work with,
c. they are difficult to control with fungicides, and thus positive results have been few,
d. these are more easily used, more effective, or more economically viable means of control.

Table 4. The relative frequency of entries on various categories of sorghum pathology in Review of Plant Pathology 1973-1982.

Category	% of entries
All diseases	100 [a]
Seed fungi/germination/establishment	10
Fungicide seed treatments	3.5
Root and stalk rots	7
Chemical control of root and stalk rots	0.7

a. Total number was 434 entries.

Table 5. The relative frequency of reports of research on the use of fungicides/nematicides for the control of various diseases and nematodes in sorghum and maize in Fungicide and Nematicide Tests from 1974 to 1983.[a]

Disease group	The numbers (%) of reports for	
	Sorghum	Maize
All diseases & nematodes	26(100)	67(100)
Seedling diseases	11 (42.3)	3(4.5)
Root rots	0(0)	1 (1.5)
Stalk rots	0(0)	5(7.5)
Smuts	10(38.5)	0(0)
Nematodes	3(11.5)	33(49.3)

a. i.e., results of 1973-1982.

Experience gained in attempts to increase sorghum production over the past 20 years shows clearly that stalk rots are a major constraint to the achievements of high grain yield, and that as yet no easily used economically viable, effective means of control is available (ICRISAT 1980, ICRISAT 1982). The root and stalk rots are difficult to work with, because of (a) their physical location, below ground level and within the stalk tissue; (b) the several pathogens that can be involved; and (c) their interactions with environmental stress and crop productivity levels.

The Causal Organisms

The three fungi of greatest importance in stalk rot etiology in sorghum appear to be *M. phaseolina* (R. *bataticola*), *Fusarium moniliforme,* and C. *graminicola.* A fourth, *Cephalosporium acremonium* (=*Acremonium strictum* ?) can also cause serious stalk rot, but appears to be more local in occurrence. The relative importance of these in the tropics has not been systematically determined, but it is believed that charcoal rot, caused by *R. bataticola* (the sclerotial stage of *M. phaseolina*), and fusarium stalk rot are the most widespread on sorghum.

Apart from the milo disease caused by *Periconia circinata,* which has been well studied and controlled with host plant resistance on a sustained basis for many years, little is known of root rot of sorghum, and the only major studies of sorghum root problems appear to be those on the root parasites belonging to the *Striga* spp (Ramaiah and Parker 1982).

Control with Fungicides

Current Knowledge.

We have not been able to find any reports of conclusive research results on the fungicidal control of sorghum stalk and root rots. Clinton (1960) observed a slight but nonsignificant reduction in lodging in a crop in which thiram was used as a drench (173 g/ha) applied shortly after flowering. Reports of control of R. *bataticola* incited root and stalk rots in other crops are summarized in Table 6. In the most recent report found, that of Taneja and Grover 1982, complete control of sunflower and sesame root rot was achieved by seed treatment with benomyl or thiophanate-methyt at 2 g product per kg seed.

Table 6. Summary of reports on control of *Rhizoctonia bataticola* induced diseases in several crops.

Source	Type of test	Crop	Disease	Effective control compounds	Application rates
Taneja and Grover 1982	Field tests with seed application	Sunflower	Root rot	benomyl, thiophanate methyl	0.2% W/W
		Sesame	Root rot	carbendazim-60, benomyl, thiophanate methyl	02% W/W
Goel and Mehrotra 1973	Pot test with seed application	Okra	Seedling damping-off	Ceresan, thiram, PCNB	0.3% W/W
Clinton 1960	Field test with the fungicide watered into the soil shortly after flowering	Sorghum	Lodging	(thiram)[a]	173g/ha
Seymour and Cordell 1979	Field test with soil fumigation	Pine	Seedling mortality + charcoal root rot	methyl bromide (67%) + chloropicrin (33%)	390 kg/ha
Vir et al. 1972	Field test with foliar sprays/ plant drench (~1100 litres/ha)	Soybean	Charcoal rot	thiophanate, furcarbanil	1000 ppm (1.1 kg/ha)

a. Lodging reduced only slightly.

In contrast to field studies, there are numerous reports of the in vitro effectiveness of fungicides to inhibit the growth of the major stalk rot pathogens. However, activity in vitro is not necessarily related to utility for disease control in the growing crop, and thus we do not believe it appropriate to present a detailed review of these in vitro studies.

It appears that the only field crops that are regularly treated with fungicides for control of a basal stalk disease of the adult plants are barley and wheat in Europe, where 70-80% of the cropped area is treated with foliar sprays of, until very recently, mainly benzirnidazole fungicides, for the control of eyespot (*Pseudocercosporella herpotrochoides).* The degree of control achieved depends upon the disease pressure, which is primarily related to the weather, but reductions in the disease index from 80% to 20% is an acceptable and achieveable target. In the past 2 years, problems of pathogen resistance to benzirnidazole fungicides have arisen, and different fungicide groups are being examined to cope with this problem (Trow-Smith 1983).

The major difficulty, in the absence of an effective basipetally translocated fungicide is to get sufficient product to the critical infection and early colonization sites. However, the trend is toward systemic fungicides with high activity at low rates, such as the sterol-inhibiting triazole compounds, and these new products need to be evaluated for their activity.

Gaps in Knowledge and Research

The major areas in which gaps in our knowledge will need to be filled if we are to objectively assess the potential role of fungicides in contributing to the practical control of root and stalk rots in the adult sorghum crop are:

a. the biology and epidemiology of the diseases,
b. relationships with other diseases and stress factors.
c. the relationship between disease levels and crop loss,
d. the availability of and ease of handling host-plant resistance.

The more complete the state of knowledge on the biology and epidemiology of the diseases, the greater will be the opportunity to identify critical points in the disease life cycles when the pathogens would be most vulnerable to fungicidal action. The key parameters are:

a. the "overwintering'' mechanisms
b. the sources of primary inoculum
c. the stages of growth when primary infection occurs
d. the physical location of the primary infection sites
e. the colonization and disease development dynamics from the primary infection sites
f. the role of secondary infection

Fungicides can be used as a valuable research tool to help obtain the information to fill these gaps (e.g., the role of seedbome inoculum, or whether infection of seedlings is important, can be evaluated through the use of appropriate fungicide seed dressings), and thus it would be wrong to wait until we fully understand the biology and epidemiology of the diseases before we bring fungicides into the research action. However, once the targets are more clearly known, more accurate experimentation on the biological potentials for fungicide use will be possible. Once biological efficacy is demonstrated, the questions of economic and technical feasibility will need to be examined.

There are many gaps, and the way to fill them is through research. The objectives of this meeting are to identify the gaps and define the research, with priorities, to fill them.

Suggestions for Future Research

We cannot give precise suggestions for experimentation because of our lack of knowledge of some of the key elements of the target diseases. However, we can describe some possible scenarios and the approaches that would appear to be appropriate, e.g.:

a. **pathogen entirely seedborne -** choose appropriate fungicides based on the fungal species (Table 7) and examine the effects of

Table 7. Information on fungicide groups[a] that are appropriate for use in research trials for the control of sorghum root and stalk diseases (including seedling disease).

Fungicide group	Activity	Transport character	Target pathogens
Acylalanines	Systemic	Primarily acropetal, though some basipetal movement reported	Oomycetes: *Pythium, Phytophthora, Peronosclerospora, Sclerospora, Sclerophthora*
Benzimidazoles	Systemic	Acropetal	Many, excluding Oomycetes and dark-spored Ascomycetes; particularly effective on *Fusarium* spp and *Colletotrichum* spp
Oxathiin derivatives	Systemic	Acropetal	Basidiomycetes: *Rhizoctonia* spp; smut fungi; *Helminthosporium: Curvularia*
Phthalimides	Nonsystemic protectant	———	Many diverse pathogens
Thiocarbamates	Protective/eradicant nonsystemic	———	Many diverse fungi
Triazoles	Systemic	Acropetal	*Typhula,* smut fungi, and many cereal foliar pathogens

a. Omission of any fungicide group does not necessarily indicate that certain fungicides belonging to those groups could not be potentially useful for the control of sorghum root and stalk diseases.

seed treatments using from 0.2 to 1 g a.i./kg seed;

b. **pathogen soilbome but initiates infection at the seedling stage -** choose appropriate fungicide mixtures (Table 7) (a protectant/ eradicant plus a systemic) and examine the effects of seed treatments using from 0.2 to 1 g a.i./kg seed;

c. **pathogen soilborne and infection occurs on roots near the stem base some unknown time after the seedling stage is completed -** choose appropriate fungicides based on pathogen identity (Table 7) and make applications around the stem bases at fixed growth stages, accompanied by some destructive sampling and attempted isolation of the pathogen from stem base tissues.

Concluding Remarks

The use of fungicides in seed treatments is effective in sorghum for the control of seed - and soilborne pathogenic fungi that cause seed rots and seedling disease. It remains to be determined what effect seed treatment can have on the root and stalk rots of the adult plants. The time of occurrence and location of infection and colonization sites are key factors that will determine the type of experimentation required to test the potential effectiveness of fungicides to control the later occurring root and stalk rots. Fungicides can be a useful research tool in investigations of the biology and epidemiology of these diseases. Given the spectrum of activity of the systemic fungicides, and the potential availability of products with high activity at low application rates, it may be feasible to devise biologically effective control treatments. Only when this has been done will it be possible to determine (a) whether practical economically viable on-farm use of such control is possible, and (b) the possibilities for the development of integrated control systems, with fungicides combined with host-plant resistance and other crop management control procedures.

References

ANAHOSUR K.H. 1980. Chemical control of sorghum downy mildew in India.Plant Disease 64:1004-1006.

ANZALONE, L. 1980. Fungicide and Nematicide Tests (American Phytopathological Society, St. Paul, Minnesota) 35:190.

ANZALONE, L. 1982. Fungicide and Nematicide Tests (American Phytopathological Society, St. Paul, Minnesota) 37:170.

BHALE, M.S., and KHARE, M.N. 1980. Significance of *Curvularia lunata* associated with *Sorghum vulgare* seeds. Phytopathologische Zeitschrift 99:357-361.

BIDARI, V.B., SATYANARAYAN, H.V., HEGDE, R.K., and PONNAPPA, K.M. 1978. Effect of fungicides against the seed rot and seedling blight of hybrid sorghum CSH-5. Mysore Journal of Agricultural Sciences 12:587-593,

BOWLING, C.C. 1978. Rice seed treatments. Pages 74-78 *in* Seed treatments, Collaborative International Pesticides Analytical Council (CiPAC) Monograph 2 (ed. K.A. Jeffs).

CLINTON, P.K.S. 1960. Some pests and diseases of sorghum and their control in the central rainlands of the Sudan. Empire Journal of Experimental Agriculture 28:294-304.

DHANRAJ, K.S. 1979. Seedling blight of sorghum, Current Science 48:588-589.

DODD, J.L. 1980. The photosynthetic stress-translocation balance concept of sorghum stalk rots. Pages 300-305 *in* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

FREDERIKSEN. R.A.,and ODVODY, G. 1979. Chemical control of sorghum downy mildew. Sorghum Newsletter 22:129.

GOEL, S.K., and MEHROTRA, R.S. 1973. Rhizoctonia root rot and damping-off of okra and its control. Acta Botanica Indica 1:45-48.

HANSING, E.D., 1974. Fungicide and Nematicide Tests (American Phytopathological Society, St. Paul, Minnesota, USA) 29:144-146.

HANSING, E.D. 1975. Fungicide and Nematicide Tests (American Phytopathological Society, St. Paul, Minnesota, USA) 30:147-148.

HANSING, E.D. 1976. Fungicide and Nematicide Tests (American Phytopathological Society, St, Paul, Minnesota, USA) 31:187-188.

HANSING, E.D. 1978. Fungicide and Nematicide Tests (American Phytopathological Society, St. Paul Minnesota, USA) 33:172-173.

ICRISAT. 1980. Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored jointly by Texas A&M University (USA)

and ICRISAT. Patancheru. A.P. 502 324, India: ICRISAT. 469 pp.

ICRISAT. 1982. Sorghum in the Eighties: Proceedings of the International Symposium on Sorghum, 2 Vols. Sponsored by INTSORMIL, ICAR, and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

MUNGHATE, A.G., and RAUT, J.G. 1982. Efficacy of nine different fungicides against fungi frequently associated with sorghum seed. Pesticides 16(8):10-18.

PATIL-KULKARNI, B.G., PATIL, N.K., and MALEBENNUR, N.S. 1972. Studies with oxathiin derivatives and other chemicals for the control of seed rot, damping-off and downy mildew of sorghum. Mysore Journal of Agricultural Sciences 6:1-4.

PHILLEY, G.L.,and FREDERIKSEN, R.A.1975. Fungicide and Nematicide Tests (American Phytopathological Society, St. Paul, Minnesota, USA) 30:148.

RAMAIAH, K.V., and PARKER, C. 1982. Striga and other weeds on sorghum. Pages 291-302 *in* Sorghum in the Eighties: Proceedings of the International Symposium on Sorghum, sponsored by INTSORMIL, ICAR, and ICRISAT. Patancheru, A.P, 502 324, India: ICRISAT.

RAUT, J.G., and WANGIKAR, P.D. 1982. Efficacy of elemental sulphur fungicides in the control of seed-borne fungi of sorghum other than smuts. Pesticides 16:23-24.

SCHWINN, F.J. 1980. Prospects for chemical control of the cereal downy mildews. Pages 220-222 *in* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, AP. 502 324, India: ICRISAT.

SEYMOUR, C.P., and CORDELL, C.E. 1979. Control of charcoal root rot with methyl bromide in forest nurseries. Southern Journal of Applied Forestry 3(3):104-108.

SHARMA, H.C., JAIN, N.K., and AGARWAL, R.K. 1976. Effect of seed treatment with fungicides alone and in combination with carbofuran on stand and yield of *Sorghum bicolor* (L.) Moench. JNKVV Research Journal 10(1)(Suppl): 83-84,

TANEJA, M., and GROVER, R.K. 1982. Efficacy of benzimidazole and related fungicides against *Rhizoctonia solani* and R. *bataticola.* Annals of Applied Biology 100:425-432.

TARR, S.A.J. 1962. Diseases of sorghum, sudan grass and broom corn. Kew, Surrey, U.K.: Commonwealth Mycological Institute. 380 pp.

TROW-SMITH, R. 1983. Resistant eyespot is hitting winter cereal crops. Farmers Weekly (U.K.) 99(14):52.

URECH, P.A., SCHWINN, F., and STAUB, T. 1977. CGA 48988, a novel fungicide for the control of late blight, downy mildews, and related soil-borne diseases. Pages 623-631 *in* Proceedings of the 1977 British Crop Protection Conference. Croydon, Surrey, U.K.: British Crop Protection Council.

VIR, D., GANGOPADHYAY, S., and GAUR, A. 1972. Evaluation of some systemic fungicides and antibiotics against *Macrophomina phaseoli.* Pesticides 6:25.

WILLIAMS, R.J., DAVIES, J.G. and MUGHOGHO, LK. 1983. Prospects for successful integrated control of biotic yield-reducing agents of sorghum and pearl millet in the tropics. Pages 904-912 *in* Vol. 3, Proceedings of the 10th International Congress of Plant Protection. Croydon, Surrey, U.K.: British Crop Protection Council.

WILLIAMS, R.J., and RAO, K.N. 1981. A review of sorghum grain molds. Tropical Pest Management 27:200-211.

Questions

Vidyabhushanam:
Considering the soilborne nature of charcoal rot and the expression of the disease at the later stage of plant growth, do you think that seed dressing with systemic fungicides would be effective?

Williams:
We don't know and we need to find out. If, as several persons here have indicated, infection occurs early in the life of the plant, there will certainly be possibilities for the control of this infection with fungicide seed treatment.

Odvody:
Do you see any continuing problem with the acropetal translocation of most systemic fungicides when trying to control root and stalk problems, and do you feel that soil treatments for long-term and seed treatments for short-term controls will overcome these problems?

Williams:
It is certainly a problem. The use of soil-applied granules, particularly with a slow release component, could provide the answer. We need to test these in the field.

Cultural and Biological Control of Root and Stalk Rot Diseases of Sorghum

B. Doupnik, Jr.*

Summary

Recommendations for the control of root and stalk rot diseases of sorghum have basically remained unchanged over the past 50 years. These recommendations integrate several cultural practice decisions and host resistance into a crop production management system that reduces stress to the crop during the critical periods of anthesis and grain filling. Some of the more important cultural practice decisions are discussed. These include: (1) *variety selection,* (2) *seed quality and seed treatment,* (3) *plant population,* (4) *nutrition,* (5) *crop rotation,* (6) *conservation tillage,* (7) *control of other diseases and insects,* (8) *planting date,* (9) *irrigation, and* (10) *early harvest*

The state of the art in utilization of biological control as an aid to reduce root and stalk rot diseases of sorghum is briefly discussed. There is no precedent for the successful biocontrol of soilborne pathogens at this time except under highly artificial conditions. The potential integration of new developments in genetic engineering and rhizosphere technology, however, offer promise for the future development of biological control.

Areas of research needed to improve cultural and biological control of root and stalk rot diseases of sorghum are listed.

Recommendations for the control of stalk rot diseases of sorghum (*Sorghum bicolor* (L) Moench) and maize (*Zea mays* L) have basically remained unchanged over the past 50 years. The following references from private seed companies, land grant colleges, and the USDA are just a few of the many examples that could be cited to illustrate this point: Anonymous (1974), Berry (1979), Christensen and Wilcoxson (1966), Doupnik et al. (1983), Edmunds and Zummo (1975), Home and Berry (1980), Jacobsen et al. (1979), Koehler (1960), Koehler and Holbert (1938), Livingston (1945), Shurtleff (1980), Ullstrup (1978), and Wrather and Palm (1983). These recommendations integrate host resistance (standability?) with a number of cultural or crop management practices. Most of this integration is aimed towards reducing stress and/or delaying senescence of the host plant.

Compared to that in maize, relatively little research has been carried out with sorghum on the integration of cultural practices with host resistance to control stalk rot. Many researchers have extrapolated information derived from studies on maize to sorghum because of the similarity of the two species. In many cases these extrapolations do appear to be valid. Since there are some obvious differences between the two species, however, these interpretations should be confirmed. In addition to the genetic differences, many sorghum varieties differ from maize in their ability to: (1) tiller, (2) remain nonsenescent, and (3) become semidormant during periods of extreme stress. Sorghum is also quite often grown under drastically

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different environments and cultural practices than maize. The current status of cultural control of root and stalk rot diseases of sorghum is discussed below.

Another potential method to reduce plant diseases is the utilization of biological control. Interest in the use of biological agents to control plant diseases has increased dramatically during the past decade (Linderman et al. 1983). Extensive reviews of the literature on biological control by Baker (1968), Baker and Cook (1974), Cook and Baker (1983), and Papavizas and Lumsden (1980) indicate, however, that at the present time there are no successful reports of controlling soilborne diseases except under highly artificial conditions. The future feasibility of integrating biological control with cultural practices and host resistance to control root and stalk rot diseases of sorghum is discussed below.

Cultural Control

Cultural control of plant diseases is obviously not a new concept. Crop rotation, for example, has long been recognized as an effective means of reducing losses from certain diseases, even when a susceptible host is planted. In the following discussion, I will attempt to briefly summarize our present knowledge on the cultural control of root and stalk rot diseases of sorghum. As mentioned above, much of our information relative to cultural practices that can help reduce sorghum root and stalk rot diseases has been extrapolated to sorghum from research on maize.

Cultural control of stalk rot diseases primarily involves the integration of several crop production management practices and host resistance. One of the main objectives of cultural control is to reduce stress to the crop at critical times (anthesis and grain filling) during the growing season. Since stress is such an important factor in predisposing the sorghum plant to the development of stalk rot diseases, the following publications are cited as resource references: Colhoun (1973), Cook (1973), Cook and Papendick (1972), Dodd (1980a, b, c), Doupnik and Frederiksen (1983), Edmunds (1964), Odvody and Dunkle (1979), Schneider and Pendery (1983), Schoeneweiss (1975), and Sumner (1968).

A general review on cultural control of infectious crop diseases has been published by Palti (1981). Excellent reviews on the cultural control of stalk rot diseases of maize have also been published by Christensen and Wilcoxson (1966), Shurtleff (1980), and Pappelis and BeMiller (these proceedings). In addition, nearly every author in these proceedings has made reference to the role that certain cultural practices play in the integration of their assigned subject matter areas to the general problem of root and stalk rot diseases of sorghum. It is for this reason, then, that an in-depth discussion of each cultural practice covered in this paper will not be made if it has been covered in other papers in these proceedings.

Variety Selection

One of the first and most critical crop production management decisions a grower will make is which sorghum variety to plant. Since there are differing levels of host resistance available to most growers, this decision is very important since the more stalk-rot-susceptible varieties may even develop stalk rot in the absence of any obvious predisposing stress conditions. Past performance under similar environmental and geographic conditions offers a very good basis for selection. Satisfactory performance should include such factors as lodging resistance (standability), yield, maturity, resistance to stress (especially water deficits), resistance to other diseases, and resistance to insects.

A generalization with regard to maturity is that shorter season varieties are more susceptible to stalk-rotting diseases than are longer season varieties. This puts many growers in a "Catch 22" situation, especially in the underdeveloped, semi-arid, tropical production areas since most of the improved high-yielding sorghum varieties are actually shorter-season than the lower yielding varieties that they replaced. Since this relates to a more senescent type of sorghum that is more sensitive to stress at anthesis and grain filling than a more nonsenescent type, you end up with a potentially high-yielding sorghum that is supersusceptible to stalk rot diseases.

Whether the resulting lodging problem has been stress-and/or pathogen(s)-induced is probably unimportant since the end result is the same. The goal of the sorghum breeder and the hope of the grower is to eventually have available high-yielding, nonsenescent sorghum varieties that are well adapted for their particular environmental and geographic conditions. Additional information on the importance of host resistance and variety

selection can be obtained from the articles by Buddenhagen (1983), Christensen and Wilcoxson (1966), Shurtleff (1980), and Henzell et al., Maunder, and Rosenow (these proceedings).

Seed Quality and Seed Treatment

Most researchers feel that the primary infection site for the sorghum stalk rot pathogens is the root system (Frederiksen, Mughogho and Panda, Odvody and Forbes, Partridge et al., Pappelis and BeMiller, and Zummo, these proceedings). The planting of sound, disease-free seed that has been treated with a broad-spectrum fungicide should then give some protection against these pathogens by either reducing or delaying infection. Thus, the planting of sound, fungicide-treated seed is a cultural practice that should be integrated into the crop production management system (Christensen and Wilcoxson 1966, Shurtleff 1980).

Williams and Nickel (these proceedings) have thoroughly reviewed the effectiveness of fungicides to control stalk rot diseases. At the present time, however, fungicides do not offer much for control other than as a seed treatment to delay early infection. The complex etiology and epidemiology of the many seed- and soilborne root pathogens that may be encountered make it an extremely difficult problem to control (Bowen and Rovira 1976, Curl 1982, Park 1963). The development of new-generation, systemic fungicides that will be effective in controlling stalk rot diseases offers future possibilities (Williams and Nickel, these proceedings).

Plant Population

The association of high plant populations with increased incidences of stalk rot has been known for a long time. In fact, many sorghum breeders take advantage of this very predictable response to improve their screening programs for stalk rot resistance (Henzell et al., Maunder, and Rosenow, these proceedings). This association is thought to be primarily related to water-deficit-induced stress due to increased competition for the available soil moisture (Duncan, Eastin et al., Maranville and Clegg, and McBee, these proceedings). Unfortunately, many growers do not give enough attention to their plant population decisions; yet it is a cultural practice that they have a lot of control over.

Nutrition

The influence of nutrition on the incidence and severity of stalk rot of sorghum and maize has been reviewed by Huber and Watson (1974), Murphy (1975), and Jordan et al. (these proceedings). Unfortunately, much of the information on the effects of nutrition on stalk rot has been extrapolated to sorghum from research on maize and other crops (Abney 1971, Otto and Everett 1956, Taylor et al. 1983, Warren et al. 1975, and Younts and Musgarave 1958). To summarize the influence of nutrition upon stalk rot: high rates of nitrogen/potassium deficiency, and/or unbalanced ratios of nitrogen and potassium will increase stalk rot disease. Fertilizer application should be based on soil tests and realistic yield goals. As with plant populations, decisions on fertility are often not given enough attention; yet these are cultural practices over which many growers have considerable control

Planting Date

Depending on the length of the growing season and the geographic location, this is a cultural practice decision that can be used to reduce stalk rot. The goal here is to avoid environmental stress during the critical periods of anthesis and grain filling (Dodd 1980a; Duncan, McBee, these proceedings). This would have more impact in the tropic and semitropic zones than in the temperate zones.

Crop Rotation

For many diseases crop rotation, as opposed to monoculture, is an effective cultural practice to help control plant diseases (Curl 1963, Shipton 1977). Such is not the case, however, for the stalk rot diseases of sorghum This is due to the diversity and wide host range of the stalk rot pathogens (Dhingra and Sinclair 1978; Reed et al. 1983; Frederiksen, Mughogho and Pande, Odvody and Forbes, and Zummo, these proceedings) and their ability to survive saprophytically on crop residues and/or as specialized structures in the soil for long periods of time (Cook et al. 1973; Katsanos and Pappelis 1969; Nyvall and Kommedahl 1968,1970; and Vizvary and Warren 1982). However, as discussed below, crop rotation in conjunction with conservation tillage may offer some control of stalk rot.

Conservation Tillage

Conservation tillage offers a useful tool to conserve soil moisture as well as to reduce wind and water erosion In addition, the residue maintained on the surface will reduce soil temperature and temperature fluctuation. The plant disease consequences of conservation tillage have been thoroughly reviewed (Boosalis et al. 1969, Boosalis and Doupnik 1976, Boosalis et al. 1981, Cook et al. 1978, and Sumner et al. 1981).

In many cases conservation tillage has resulted in an increase in disease problems. This is especially true when crops are monocultured. However, a unique 3-year conservation tillage rotation system (wheat-sorghum-fallow) known as ecofallow that has been developed for the semi-arid Central Great Plains area of the United States has actually reduced the incidence and severity of sorghum stalk rot while increasing yields (Doupnik et al. 1975, Doupnik and Boosalis 1980). In this case it is believed that the increased soil moisture storage and the lower, more constant soil temperatures are the major factors accounting for the reduced stalk rot problems. The sorghum rotation with wheat allows one to plant directly into the residue of another crop, which has very few common pathogens, rather than into the residue of the same crop. The growing of two different crops appears to be a useful mechanism, then, to avoid the disease consequences encountered when monoculturing is practiced under conservation tillage.

Control of Other Diseases and Insects

As with many of the other cultural practices discussed above, the control of other diseases and of insects is part of an overall crop production system objective to reduce stress to the sorghum plants (Christensen and Wilcoxson 1966, Gates and Mortimore 1972, Pappelis and Katsanos 1966, and Shurtleff 1980). The "other disease" category should also include nematodes (Claflin, these proceedings).

Host resistance should be utilized when available; and, where feasible, chemicals should be employed to help control these other diseases and insects.

Irrigation

When available and where applicable, timely irrigation can be used to reduce early-season stress (Schneider and Pendery 1983), as well as stress at anthesis and grain filling (Dodd 1980a, b, c; Eastin et al., and McBee, these proceedings).

Timely Harvest

If field symptoms and visible signs suggest that stalk rot disease is developing in a given field after physiological maturity of the grain is reached, plans should be made to harvest early. This will help prevent excessive field losses due to lodging and unharvestable heads (Doupnik et al. 1983).

Biological Control

The concept of biological control as a mechanism to reduce plant disease is not new. The literature on biocontrol is very extensive and has been recently reviewed by Cook and Baker (1983), Linderman et al. (1983), and Papavizas and Lumsden (1980). The current "state-of-the-art" of biocontrol suggests, however, that there is no precedent for the successful control of any soilborne disease except under highly artificial conditions. Most of the successful examples of biocontrol involve "single pathogen:single host" systems under container-grown greenhouse conditions. Attempts to demonstrate these same biocontrol systems under field conditions, however, have generally failed. Most of the reported field successes of biocontrol involve diseases of woody plants, propagated plant pieces, and seedlings. This should not be interpreted as an indictment of the feasibility of developing biological control measures for root and stalk rot diseases of sorghum; however, it should be pointed out that in order for successful biological control systems to be developed, the etiology and epidemiology of the disease(s) must be thoroughly known. The basic gaps in our knowledge concerning the etiology and epidemiology of sorghum root and stalk diseases have been repeatedly emphasized throughout these proceedings.

Even with the thorough understanding of the etiology and epidemiology of the root and stalk rot disease complex of sorghum, biological control will undoubtedly be very difficult to obtain. This is due to the fact that there are several different pathogens involved (i.e., *Fusarium moniliforme* Sheldon, *Macrophomina phaseolina* (Tassi) Gold, and *Colletotrichum graminicola* (Cesati) Wilson), In addition, these pathogens have a wide host range and

the ability to survive saprophytically as well as to act as weak parasites in sorghum if the opportunity arises (i.e., host cell death).

The general concept of biological control encompasses a wide array of potential mechanisms. These include, among others, antagonism (i.e., hyperparasitism—Boosalis 1964 and De La Cruz and Hubbell 1975) and suppressive soils (Hornby 1983, Scher and Baker 1980, Schneider 1982, Weller 1983). Another area of much interest and activity at this time is the role of mycorrhizae in the development of root diseases (Gerdemann 1968, Marx 1972, Marx and Schenck 1983, Schenck 1981, Zak 1964). Sorghum roots appear to be good hosts of mycorrhizal fungi. The potential for exploiting these sorghum mycorrhizae as biological buffers against a variety of biotic and abiotic stresses is of great interest. This is especially true in view of the extensive research developments occurring in biotechnology. Genetic engineering may offer very powerful tools in the near future for the designing of specific biological control agents.

Areas of Research Needed

The complex etiology and epidemiology of the root and stalk rot diseases of sorghum have been major stumbling blocks for improved disease control. Changing attitudes in disease management and future developments in the integration of cultural and biological controls with improved host resistance, however, offer promise for the future (Andrews 1983, Baker 1983, and Delp 1983).

Based on previous discussions (Anonymous 1980) and information presented during these proceedings on cultural and biological control, the following research needs are identified:

1. Conservation tillage—Determine long-term effects of conservation tillage on stalk rot diseases, including multilocational evaluations for disease suppression similar to the suppression achieved in the Central Great Plains areas of the USA.

2. Plant population and nutrition—Determine if nonsenescent/nontillering sorghums react the same as senescent/tillering sorghums to changes in population and nutrition with regard to disease incidence.

3. Mycorrhizae—Identify mycorrhizae associated with sorghum roots and explore the role they might play in the development of root and stalk rot diseases.

4 Fungicides and plant growth regulators- Explore the possibilities of using new-generation systemic fungicides and/or plant growth regulators (such as antitranspirants) as additional tools to reduce stalk rot.

5. Integration of cultural practices and host resistance—Make multilocational evaluations of crop production management systems (integration of cultural practices and host resistance) that have been shown to suppress root and stalk rot diseases.

References

ABNEY, T.S., and FOLEY, D.C. 1971. Influence of nutrition on stalk rot development in *Zea mays.* Phytopathology 61:1125-1129.

ANDREWS, J.H. 1983. Future strategies for integrated control. Pages 431 -440 *in* Challenging problems in plant health. St. Paul, Minnesota, USA: American Phytopathological Society.

ANONYMOUS. 1974. How to prevent and identify troublesome grain sorghum diseases. A management manual. Des Moines, Iowa, USA: Asgrow Seed Co. 15 pp.

ANONYMOUS. 1980. Recommendations of the workshop on sorghum diseases: Head blight and stalk rots. Pages 463-464 *in* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, Sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

BAKER, K.F. 1983. The future of biological and cultural control of plant diseases. Pages 422-430 *in* Challenging problems in plant health. St. Paul, Minnesota, USA: American Phytopathological Society.

BAKER, K.F., and COOK, R.J. 1974. Biological control of plant pathogens. San Francisco, California, USA: W.H. Freeman and Co. 433 pp.

BAKER, R. 1968. Mechanisms of biological control of soilborne pathogens. Annual Review of Phytopathology 6:263-294.

BERRY, C. 1979. Sorghum lodging is a manageable problem. Crops Quarterly (PAG inhouse publication) 1(1):14-15.

BOOSALIS, M.G. 1964. Hyperparasitism. Annual Review of Phytopathology 2:363-376.

BOOSALIS, M.G., COLVILLE, W.L., and SUMNER, D.R. 1969. Effect of intensive cultural practices on soil-borne and related corn diseases. Pages 12-20 *in* Disease consequences of intensive and extensive culture of field crops (ed., JA Browning) Jowa State University Depart-

ment of Botany and Plant Pathology Special Report No. 64, Ames, Iowa.

BOOSALIS. M.G., and DOUPNIK, B. 1976. Management of crop diseases in reduced tillage systems. Bulletin of the Entomological Society of America 22:300-302.

BOOSALIS, M.G., DOUPNIK, B., and ODVODY, G. 1981. Conservation tillage in relation to plant diseases. Pages 445-474 *in* CRC Handbook of Pest Management, Vol.1. Boca Raton, Florida, USA: CRC Press. Inc.

BOWEN, G.D., and ROVIRA, A.D. 1976. Microbial colonization of plant roots. Annual Review of Phytopathology 14:121-144.

BUDDENHAGEN, I.W. 1983. Breeding strategies for stress and disease resistance in developing countries. Annual Review of Phytopathology 21:385-409.

CHRISTENSEN, J.J., and WILCOXSON, R.D. 1966. Stalk rot of corn. American Phytopathological Society Monograph No. 3, St. Paul, Minnesota. 59 pp.

COLHOUN, J. 1973. Effects of environmental factors on plant disease. Annual Review of Phytopathology 11:343-364.

COOK, G.E, BOOSALIS, M.G, DUNKLE, L.D., and ODVODY, G.N. 1973. Survival of *Macrophomina phaseoli* in corn and sorghum stalk residue. Plant Disease Reporter 57:873-875.

COOK, R.J. 1973. Influence of low plant and soil water potentials on diseases caused by soilborne fungi. Phytopathology 63:451-458.

COOK, R.J., and BAKER, K.F. 1983. The nature and practice of biological control of plant pathogens. St. Paul, Minnesota, USA: American Phytopathological Society. 539 pp.

COOK, R.J., BOOSALIS, M.G., and DOUPNIK, B. 1978. Influence of crop residues on plant diseases. Pages 147-163 *in* Crop residue management systems. American Society of Agronomy Special Publication Number 31, Madison, Wisconsin,

COOK, R.J., and PAPENDICK, R.I. 1972. Influence of water potential of soils and plants on root disease. Annual Review of Phytopathology 10:349-374.

CURL, E.A. 1963. Control of plant diseases by crop rotation. Botanical Review 29:413-479.

CURL, E.A. 1982. The rhizosphere: Relation to pathogen behavior and root disease. Plant Disease 66:624-630.

DE LA CRUZ, R.E., and HUBBELL, D.H, 1975. Biological control of the charcoal root rot fungus *Macrophomina phaseolina* on slash pine seedlings by a hyperparasite. Soil Biology and Biochemistry 7:25-30.

DELP, C.J. 1983. Changing emphasis in disease management Pages 416-421 *in* Challenging problems in plant health, American Phytopathological Society, St. Paul, Minnesota.

DHINGRA, O.D., and SINCLAIR, J.B. 1978. Biology and Pathology of *Macrophomina phaseolina.* Vicosa, Brazil: Imprensia Universitaria, Universidade Federal de Vicosa. 166 pp.

DODD, J.L. 1980a. The photosynthetic stress-translocation balance concept of sorghum stalk rots. Pages 300-305 *in* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, Sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

DODD, J.L. 1980b. The role of plant stresses in development of corn stalk rots. Plant Disease 64:533-537.

DODD, J.L. 1980c. Grain sink size and predisposition of *Zea mays* to stalk rot. Phytopathology 70:534-535.

DOUPNIK, B., and BOOSALIS, M.G. 1980. Ecofallow-A reduced tillage system—and plant diseases. Plant Disease 64:31 -35.

DOUPNIK, B., BOOSALIS, M.G., WICKS, G., and SMIKA, D. 1975. Ecofallow reduces stalk rot in grain sorghum. Phytopathology 65:1021-1022.

DOUPNIK, B., DUNKLE, L.D., and WYSONG, D.S. 1983. Stalk rots of corn and sorghum. University of Nebraska Cooperative Extension Service NebGuide Series No. G83-643, Lincoln, Nebraska. 2 pp.

DOUPNIK, B., and FREDERIKSEN, R.A. 1983. Diseases of major crops. Pages 525-534 *In* Dryland agriculture. American Agronomy Society, Agronomy Monograph Number 23, Madison, Wisconsin.

EDMUNDS, L.K. 1964. Combined relation of plant maturity, temperature, and soil moisture to charcoal stalk rot development in grain sorghum. Phytopathology 54:514-517.

EDMUNDS, L.K., and ZUMMO, N. 1975. Sorghum diseases in the United States and their control. U.S. Department of Agriculture Handbook No. 468, Washington, D.C. 47 pp.

FUEHRING, H.D., and FINKNER, M.D. 1983. Effect of folicote antitranspirant application on field grain yield of moisture-stressed corn. Agronomy Journal 75:579-582.

GATES, L.F., and MORTIMORE, C.G. 1972. Effects of removal of groups of leaves on stalk rot and yield in corn. Canadian Journal of Plant Science 52:929-935.

GERDEMANN, J.W. 1968. Vesicular-arbuscular mycorrihiza and plant growth. Annual Review of Phytopathology 6:397-418.

HORNBY, D. 1983. Suppressive soils. Annual Review of Phytopathology 21:65-85.

HORNE, C.W., and BERRY, R.W. 1980. Sorghum disease

atlas. Texas A&M University Cooperative Extension Service Publication B-1323, College Station, Texas. 16 pp.

HUBER D.M., and WATSON, R.D. 1974. Nitrogen form and plant disease. Annual Review of Phytopathology 12:139-165.

JACOBSEN, B.J., WHITE, D.G., HOOKER, A.L, SHURTLEFF, M.C., and NOWLIN, B.E. 1979. Corn stalk rots. University of Illinois Department of Plant Pathology Report on Plant Diseases No. 200, Urbana, Illinois. 6 pp.

KATSANOS, R.A., and PAPPELIS, A.J. 1969. Relationship of living and dead cells to spread of *Colletotrichum graminicola* in sorghum stalk tissue. Phytopathology 59:132-134.

KOEHLER, B. 1960. Corn stalk rots in Illinois. University of Illinois Agricultural Experiment Station Bulletin 658, Urbana, Illinois. 90 pp.

KOEHLER, B., and HOLBERT, J.R. 1938. Combating corn. diseases. University of Illinois Agricultural Experiment Station and Extension Service Circular 484, Urbana, Illinois. 35 pp.

LINDERMAN, R.G., MOORE, L.W., BAKER, K.F., and COOKSEY, D.A. 1983. Strategies for detecting and characterizing systems for biological control of soilborne plant pathogens. Plant Disease 67:1058-1064.

LIVINGSTON, J.E. 1945. Charcoal rot of corn and sorghum. University of Nebraska Agricultural Experiment Station Research Bulletin 136, Lincoln, Nebraska. 32 pp.

MARX, D.H. 1972. Ectomycorrhizae as biological deterrents to pathogenic root infections. Annual Review of Phytopathology 10:429-454.

MARX, D.H,, and SCHENCK, N.C. 1983. Potential of mycorrhizal symbiosis in agricultural and forest productivity. Pages 334-347 *in* Challenging problems in plant health, St. Paul, Minnesota, USA: American Phytopathological Society.

MURPHY, L.S. 1975. Fertilizer efficiency for corn and grain sorghum. Pages 49-72 *in* Proceedings of the 30th Annual Corn and Sorghum Research Conference. Washington, D.C., USA: American Seed Trade Association.

NYVALL, R.F., and KOMMEDAHL, T. 1968. Individual thickened hyphae as survival structures of *Fusarium moniliforme* in corn. Phytopathology 58:1704-1707.

NYVALL, R.F., and KOMMEDAHL, T. 1970. Saprophytism and survival of *Fusarium moniliforme* in corn stalks. Phytopathology 60:1233-1235.

ODVODY, G.N., and DUNKLE, L.D. 1979. Charcoal stalk rot of sorghum: Effect of environment on host-parasite relations. Phytopathology 69:250-254.

OTTO, H J., and EVERETT, H.L. 1956. Influence of nitrogen and potassium fertilization on the incidence of stalk rot in corn, Agronomy Journal 48:301 -305.

PALTI, J. 1981. Cultural practices and infectious crop diseases. Berlin, Heidelburg, New York: Springer-Verlag. 243 pp.

PAPAVIZAS, G.C., and LUMSDEN, R.D. 1980. Biological control of soilborne fungal propagules. Annual Review of Phytopathology 18:389-413.

PAPPELIS, A.J., and KATSANOS. R.A. 1966. Effect of plant injury on senescence of sorghum stalk tissue. Phytopathology 56:295-297.

PARK, D. 1963. The ecology of soil-borne fungal diseases. Annual Review of Phytopathology 1:241-258.

PUTNAM, A.R., and DUKE, W.B. 1978. Allelopathy in agroecosystems. Annual Review of Phytopathology 16:431-451.

REED, J.E., PARTRIDGE, J.E., and NORDQUIST, P.T. 1983. Fungal colonization of stalks and roots of grain sorghum during the growing season. Plant Disease 67:417-420.

SCHENCK, N.C. 1981. Can mycorrhizae control root disease? Plant Disease 65:230-234.

SCHER, F.W., and BAKER, R. 1980. Mechanism of biological control in a *Fusarium*-suppressive soil. Phytopathology 70:412-417.

SCHNEIDER, R.W. (ed). 1982. Suppressive soils and plant disease. St. Paul, Minnesota, USA: American Phytopathological Society. 88 pp.

SCHNEIDER, R.W., and PENDERY, W.E. 1983. Stalk rot of corn: Mechanism of predisposition by an early-season water stress. Phytopathology 73:863-871.

SCHOENEWEISS, D.F. 1975. Predisposition, stress, and plant disease. Annual Review of Phytopathology 13:193-211.

SHIPTON, P.J. 1977. Monoculture and soilborne plant pathogens. Annual Review of Phytopathology 15:387-407.

SHURTLEFF, M.C. 1980. Compendium of corn diseases (2nd ed.). St. Paul, Minnesota, USA: American Phytopathological Society. 105 pp.

SUMNER, D.R. 1968. The effect of soil moisture on com stalk rot. Phytopathology 58:761-765.

SUMNER, D.R., DOUPNIK. B., and BOOSALIS, M.G. 1981. Effects of reduced tillage and multiple cropping on plant diseases. Annual Review of Phytopathology 19:167-187.

TAYLOR, R.G., JACKSON, T.L., POWELSON, R.L., and CHRISTENSEN, N.W. 1983. Chloride, nitrogen form, lime, and planting date effects on take-all rot of winter wheat. Plant Disease 67:1116-1120.

ULLSTRUP, A.J. 1978. Corn diseases in the United States

and their control (Revised). U.S. Department of Agriculture Handbook Number 199. Washington, D.C..55 pp.

VIZVARY, M.A., and WARREN, H.L. 1982. Survival of *Colletotrichum graminicola* in soil. Phytopathology 72:522-525.

WARREN, H.L., HUBER, D M , NELSON, D.W., and MANN, O.W. 1975. Stalk rot incidence and yield of corn as affected by inhibiting nitrification of fall-applied ammonium. Agronomy Journal 67:655-660.

WELLER, D.M. 1983. Colonization of wheat roots by a fluorescent Pseudomonad suppressive to take-all. Phytopathology 73:1548-1553.

WRATHER, J.A., and PALM, E.W. 1983. Controlling diseases of grain sorghum. University of Missouri Extension Division Agricultural Guide No. 4354, Columbia, Missouri. 6 pp.

YOUNTS, S.E., and MUSGRAVE, R.B. 1958. Chemical composition, nutrient absorption, and stalk rot incidence of corn as affected by chloride in potassium fertilizer. Agronomy Journal 50:426-429.

ZAK, B. 1964. Role of mycorrhizae in root disease. Annual Review of Phytopathology 2:377-392.

Questions

Maranville:
Measurements of soil temperature at 5-centimeter depth may reflect meaningful differences in germination and early growth. Do you feel, however, those graphs you showed were valid for meaningful differences later in the season when any root activity is much deeper?

Doupnik:
We suspect (but don't have the data) that soil temperatures were affected at deeper depths. Certainly moisture loss through evaporation is greatly reduced under the reduced tillage system, and heat reflection would be greatly reduced. What effect this has is not known, but probably a cooler plant environment in general would be less stressful. Temperature is just one component of stress, but an important component along with moisture in predisposition to stalk rot development.

Pande:
Would you like to comment on the behavior of plant populations under the following two cultural practices, with respect to available moisture: sorghum hybrid CSH 6 grown under rainfed condition in India at the three plant population densities of 66675, 133350,266 700 plants/ha did not show any significant differences in lodging and charcoal rot, and as such all three populations behaved equally susceptible. However, under irrigated conditions with irrigation stopped at 50% flowering or earlier, lower plant populations showed less lodging and charcoal rot than higher plant populations.

Doupnik:
In the rainfed situation, the threshold level of stress probably wasn't reached. In other words, you may still have opportunity to increase plant populations and yield potential without increasing problems with stalk rot. In regard to irrigation, early-season environments favorable for higher yields followed by drought stress at grain filling stage would result in less stress effect under the lower populations, hence, less stalk rot.

Schneider:
At what depth were soil temperatures measured?

Doupnik:
At 2.5 to 5 centimeters.

Odvody:
Allen and Boosalis showed that endomycorrhizal incidence in wheat was greater following crested wheat grass than in wheat monoculture. Have you considered a mutual or single endomycorrhizal benefit from sorghum because of the crop rotations involved in the ecofallow system?

Doupnik:
Boosalis et al. have initiated a study to look at this aspect. The question is that, since sorghum and maize are much better hosts for mycorrhizal fungi, will a rotation of these crops with wheat increase the mycorrhizal population of wheat roots versus monoculturing of wheat?

Breeding for Resistance to Root and Stalk Rots in Texas

D.T. Rosenow*

Summary

The moisture stress/charcoal rot/lodging complex is the most important type of stalk rot in Texas, and much breeding work has been directed toward this problem. Selection is done under field conditions in large nurseries where irrigation is withheld to allow moisture stress to develop during the grain-filling stage. In the past, charcoal-rot-infested toothpicks were inserted into the stalks, and the spread of infection, stalk rot, and lodging were used as indicators in selection. At present, charcoal rot resistance is considered primarily a postflowering drought response trait—generally referred to as "stay-green" or nonsenescence. The response we select is the ability of plants to remain alive and fill the grain normally, with stalks that remain alive and resist lodging and charcoal rot when under severe moisture stress during the late stages of grain development. The presence of the stay-green trait correlates well with resistance to charcoal rot and lodging. Significant progress has been made in the incorporation of the stay-green trait into high-yielding, agronomically desirable lines. Sources of resistance and the breeding and selection techniques are discussed.

Stalk and root rots are serious problems in sorghum. Plants weakened by stalk and root rots lodge easily, with loss in harvestable grain. Also, stalk rots 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 stresses commonly occur in the sorghum-producing areas of Texas.

Lodging in sorghum is often associated with stalk rots. Considerable research is reported on the causal factors and the relationship between moisture stress, stalk rots, and lodging—especially with respect to charcoal rot (Edmunds and Zummo 1975, Hoffmaster and Tullis 1944, Hsi 1961, Malm and Hsi 1965, Voigt 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, the red rot phase of anthracnose (*Colletotrichum graminicola* (Cesati) Wilson) is important, although fusarium stalk rot can also be severe. 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 1964a, Odvody and Dunkle 1979). Techniques to screen for charcoal rot were developed by Hsi (1961), Malm and Hsi (1965), Edmunds (1964a, 1964b), and Edmunds et al. (1965). The techniques involve either artificial or natural (dry-climate) moisture stress during the grain-development stage, combined with inoculation of the stalk by an infested toothpick. Conditions favor-

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International Crops Research Institute for the Semi-Arid Tropics. 1984. Sorghum Root and Stalk Rots, a Critical Review: Proceedings of the Consultative Group Discussion on Research Needs and Strategies for Control of Sorghum Root and Stalk Rot Diseases, 27 Nov - 2 Dec 1963, Bellagio, Italy. Patancheru, A.P. 502 324, India: ICRISAT.

ing 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 important and are sometimes involved in the stalk rot problem (Edmunds et al. 1973, Johnson et al. 1966). *Phythium* spp appeared to cause extensive lodging and serious grain loss in northwestern Texas in 1971 (Edmunds et al. 1973), and in specific genotypes in more recent years (Odvody, personal communication). 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, 1982) 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 of greenbug-infested plots. In 1978, J.W. Johnson (Texas Agricultural Experiment Station, Lubbock, Texas; personal communication) found that under natural conditions more charcoal rot developed in greenbug-susceptible hybrids than in resistant hybrids when moisture stress and large numbers of greenbugs were present. The Banks grass mite and the sugarcane root stalk weevil are also believed to accelerate stalk rot.

Since lodged plants are often the end result of rotted stalks, research on selection for lodging resistance will be discussed. In maize, Zuber (1973) improved stalk strength through the use of various selection techniques. Al-Tayar (1974) and Schertz et al. (1978) tried various stalk-strength measurements on sorghum, and found that bending dry plants and green stalk penetration were the most promising. Mechanical properties associated with lodging-resistant genotypes were reported by Bashford et al. (1976) and Esechie et al. (1977). These included shorter, stockier plants with extensive leaf sheath coverage, shorter peduncles, and a thicker rind. Resistant lines matured later, were more perennial in habit, and contained higher total nonstructural carbohydrates.

Anatomical variation in sorghum stalk intemodes was studied by Schertz and Rosenow (1977). Large differences were found in the number of cells with lignifted walls and in the degree of lignification in the epidermis, subepidermis, and vascular bundles. Lodging-resistant lines generally had the most lignification.

Several earlier reports describe differences among sorghums in resistance to stalk rots (Edmunds et al. 1965, Frederiksen and Rosenow 1971, Malm and Hsi 1965, Tarr 1962, Voigt and Edmunds 1970). However, none of the lines possessed a sufficiently high level of resistance to contribute substantially to improved stalk-rot-resistant types. New Mexico-31 was the first sorghum line developed and released primarily for its charcoal rot resistance.

In 1972, Rosenow reported on a promising procedure for selecting for lodging and charcoal rot resistance. Five years later, Rosenow (1977) and Rosenow et al. (1977) indicated excellent progress in developing sorghums with resistance to charcoal rot and lodging in Texas. In recent years, sorghums with a high degree of resistance to charcoal rot and lodging have been reported in Texas by Frederiksen and Rosenow (1980), Rosenow (1980), and Rosenow and Frederiksen (1982); at ICRISAT by Rao et al. (1980); and in West Africa by Frowd(1980).

The use of leaf and plant death ratings (degree of premature plant senescence) to predict subsequent stalk rot (primarily charcoal rot) was first discussed by Rosenow et al. (1977) and Rosenow (1977). Ratings were made when plants were under moisture stress during the late grain development stage. They found significant correlations between nonsenescence, charcoal rot resistance, and lodging resistance. Duncan (1977) and McBee et al. (1983) described some characteristics of nonsenescing sorghums, including carbohydrate levels in their stalks. Katsanos and Pappelis (1965) reported a direct relationship between senescing tissue and susceptibility to stalk rots. Dodd (1977, 1980) described a "photosynthetic stress-translocation balance" concept of predisposition to stalk rot in maize and sorghum. The concept proposed agrees well with observations I have made on sorghum. Dodd's theory is that root and stalk rot predisposition begins with the senescence of root tissue because of an insufficient supply of carbohydrate. The senescing cells are invaded by microorganisms that may be only weakly pathogenic or nonpathogenic to vigorous cells, reducing the ability of the plant to obtain water. Eventually transpiration rates exceed water uptake and permanent wilting occurs, followed by the death of leaves and stalk. At this stage several different organisms may invade the stalk, resulting in visible

stalk rot and lodging. The predisposition is therefore affected by the rate of photosynthesis and the rate of translocation of carbohydrates to the roots. Stresses that reduce the rate of photosynthesis include water deficit, leaf destruction, light reduction, and mineral deficiency. The size of the carbohydrate sink in developing grain is very important in determining the level of stress necessary to induce stalk rot predisposition.

The relationship of drought stress to charcoal rot in sorghum was discussed by Rosenow et al. (1983). They identified two distinct stress responses. The "preflowering" response occurs when plants are under significant moisture stress prior to flowering. The "postflowering" response occurs when plants are under severe water stress during the grain-filling stage. Symptoms of postflowering drought-stress susceptibility include premature plant (leaf and stem) death or premature plant senescence, stalk rot (charcoal rot), stalk disintegration and lodging, and reduced seed size. Rosenow et al. proposed that selecting for tolerr ance to postflowering drought stress by selecting against premature plant death and lodging is an efficient and effective method of developing charcoal-rot-resistant sorghums.

Screening and Evaluation Techniques

The relationship between moisture stress during the late grain development stage (postflowering) and charcoal rot is the basis for our breeding program. We believe that most charcoal rot resistance can be explained by tolerance to postflowering drought stress, as explained by Rosenow et al. (1983). We commonly use the term "stay-green" to describe plants or lines that possess postflowering drought tolerance. Other terms sometimes used synonymously with stay-green are nonsenescence and late-season drought tolerance.

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 lodging resistance or nonsenescence by any worker in any nursery, (b) initial screening in single-row observation or in individual pedigree breeding plots in a large field nursery allowed to stand for a long period after maturity, and (c) screening for the stay-green trait, as well as for other types of lodging resistance, in replicated trials at several locations.

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 lodging pressure, moisture stress, stalk or root rots, and any other natural diseases or insect pests. In West Texas they are planted in postflowering drought-tolerance screening nurseries. In these nurseries, ideal growing conditions are maintained during early plant growth, especially regarding moisture availability. As 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.

In the past we inoculated plants by inserting toothpicks infested with the causal organism, *M. phaseolina,* into an internode of the stalk, usually 2.5 to 5 cm above the soil surface. Inoculation was generally done 2 weeks after flowering, but timing did not appear to be critical as long as it was after flowering, but before physiological maturity. Normally five plants were inoculated in each of two or three replications. After 3 to 4 weeks or later, inoculated stalks were split and the stalk disintegration and charcoal rot invasion were rated on a 1 to 5 scale, where < 1 = less than one intemode affected; 1 = one internode invaded but rot did not pass through any nodal area; 2 = two internodes invaded; 3 = more than two invaded; 4 = more than three invaded, sometimes with sclerotia; and 5 = extensive invasion, shredding, death, and sclerotia present. We sometimes also rated for stalk disintegration, other than charcoal rot, as an indication of possible resistance to other stalk rots.

The breeding and screening techniques we presently use to develop resistance to moisture-stress-related stalk rot and how it relates to our drought resistance breeding program were presented by Rosenow and Clark (1982, 1983), Rosenow et al. (1981,1982,1983), and Woodfin et al. (1979). In nurseries where screening is for postflowering drought tolerance and lodging, stay-green is evaluated at the late grain-development stage or shortly after maturity. Each entry or plot is subjectively rated for the amount of premature leaf and stem death on a scale of 1 to 5, where 1 = completely green and 5 = dead, Leaf and stem ratings can be made together or separately. The nursery is often altowed to stand for an extended

period following maturity to allow stalk lodging to occur This facilitates the identification of entries with weakened but not severely rotted stalks. Entries can also be rated for yield and other traits and selections made. Maturity is critical because plants in the vegetative stage are very resistant to senescence. Also there is a period just prior to physiological maturity when sorghum is very susceptible to plant senescence and stalk rot. Plants a few days earlier or later in maturity may show little senescence. Therefore, flowering notes are taken on all plots and comparisons of charcoal rot, senescence, and lodging are made only among plants at similar stages of maturity.

Additional data taken on the replicated tests include plant height/head exsertion, desirability (an estimate of yield), and in some cases, grain yield. Lodging notes, recorded as the percentage of lodged plants, are taken periodically throughout the season whenever significant lodging occurs.

Results and Discussion

Excellent progress has been made in breeding sorghum lines for improved charcoal rot and lodging resistance (Table 1). Note the vast improvement in lodging and charcoal rot resistance of the research breeding lines compared to the standard check lines. The average flowering dates are not sufficiently different to account for the differences in lodging and charcoal rot.

Charcoal rot and lodging ratings for 12 of the best source lines, along with five check varieties, are presented in Table 2. All 12 lines had lower charcoal rot ratings and lodging percentages than New Mexico-31. All but one of the 12 entries were derived from lines developed in the sorghum conversion program (Stephens et al. 1967). The stay-green trait of these lines has proven very stable across environments, both in Texas and internationally (e.g., Sudan).

Although lines such as those described above have a high degree of stay-green or postflowering drought tolerance, most perform poorly when severe drought stress occurs prior to flowering. Conversely, most sorghum lines with excellent preflowering moisture stress tolerance are susceptible to postflowering stress. However, some genotypes with moderate levels of stay-green also perform well under preflowering stress. Crosses have been made between and among pre- and postflowering stress tolerant source lines and elite, high-yielding lines in an attempt to develop high-yielding sorghums with good levels of stay-green, combined with a high level of preflowering drought tolerance. The breeding materials are planted for drought evaluation at five locations in West Texas:

Halfway - limited irrigation (postflowering stress)
Lubbock - limited irrigation (postflowering stress)
Lubbock - dryland (season-long stress)
Big Spring - dryland (preflowering stress)
Chillicothe - dryland (preflowering stress)

In these nurseries, selection is based on the stay-green trait and lodging resistance, with emphasis on entries that also perform well under preflowering stress. In 1983, F_6s from the initial crosses were evaluated. Good progress appears to have been made in combining good levels of stay-green with preflowering drought tolerance into lines with improved yield potential. Comparison of some promising new B-line breeding materials with check varieties are presented in Table 3.

Data presented in Table 4 show that hybrids involving charcoal- and lodging-resistant parental lines have superior charcoal rot ratings, leaf-plant-

Table 1. Summary of agronomic, lodging, and charcoal rot data from the Texas Agricultural Experiment Station Statewide Sorghum Lodging Test.

	1975				1976	
					Lodging (%)	
Entries	Date of 50% flower	Lodging[a] (%)	Charcoal rating[a]	LPD rating[a]	2/10	3/8
Research lines (20)	Aug 14	9.3	1.3	2.8	0.5	13.8
Standard (5)	Aug 13	64.6	3.3	3.4	68.1	90.7

a Flowering data, charcoal rot, and leaf-plant death (LPD) ratings from Halfway, Texas. Lodging rating taken from Lubbock, Texas (data taken in late winter or on date indicated).

Table 2. Charcoal rot and lodging of selected sorghum lines, Lubbock and Halfway, Texas, USA; 4-year averages.

Designation	Type or pedigree	Charcoal rot rating [a]	Lodging [b] (%)
SC35-6	IS12555 der. (Durra)	1.5	2.8
SC56-6	IS12568 der. (Caud/Niger)	1.4	5.6
SC56-14	IS12568C (Caud/Niger)	0.6	2.8
R9188	IS17459 der. (SC599-6 sel.)	1.2	34.6
R9247	IS17459 der. (SC599-6 sel.)	0.8	11.0
NSA440	Kafir der.	1.0	3.0
1790E	(SC56 x SC33) der.	1.6	19.3
1790L	(SC56 x SC33) der.	1.2	3.6
1778	(SC56 x SC33) der.	0.6	10.2
R1584	(SC56 x SC170)der.	0.8	2.0
B4R	(BTx406 x Rio) der.	1.0	3.8
SC170-6-17	IS12661 der. (Zerazera)	1.1	39.4
New Mexico-31 (check)		1.7	54.0
BTx378(check)	Redlan	2.2	89.8
Tx7000 (check)	Caprock	2.7	90.3
BTx399 (check)	Wheatland	1.7	65.2
TAM428(check)	IS12610 der. (Zerazera)	2.6	86.4

a. Rated on 1-5 scale: ¡<1 = < one internode, 1 = one internode, 2 = two internodes, 4 = > three internodes, 5 = death.
b. Taken late in winter following exposure to strong winds.

Table 3. Comparison of breeding and parental sorghum lines for charcoal rot and other characteristics, Lubbock, Texas, USA, 1983.

Designation	Lodging [a] (%)	Charcoal rating [b]	LPD rating [c]	Stem base rating [d]	Peduncle rating [d]	Grain yield (kg/ha)
(BTx625xB35-6)-HL19	0	0.70	2.9	1.2	1.8	3030
(BTx625 x B35-6)-LDE73	0	0.45	3.5	1.2	2.1	3215
(BTx625 x B35-6)-LEC	0	0.83	2.8	1.1	1.5	3080
B35-6	0	0.48	2.7	1.1	1.7	2010
BTx625	38	3.40	4.6	4.3	3.5	3500
BTx623	40	2.00	4.7	2.8	3.5	3140
Tx7000	13	3.40	4.6	4.1	.3.9	3740

a. Moisture-stress-type lodging (November 7).
b. Charcoal rot rating of toothpick-Inoculated plants: 0 = no infection, 1 = one internode infected, 5 = death, sclerotia, shredded.
c. Leaf-plant-death rating (emphasis on premature leaf death): 1 = no leaf death, 5 = all dead (November 7).
d. Base of stalk and peduncle "stay-green" rating: 1 = completely green and alive, 5 = all dead (November 21).

death ratings, and lodging resistance, especially if both parents are resistant. Also, some hybrids made with one highly stay-green parent and one highly senescing parent show excellent pre- and postflowering drought tolerance (stay-green). We have not conducted inheritance studies on charcoal rot or lodging resistance, but F_1 and F_2 data indicate that in many lines resistance is recessive, while in a few lines it appears to be quite dominant. Hybrids involving such lines as R9188 and A599

(Table 4) perform like the susceptible parent. In some lines, especially those involving SC35-6 (A35), resistance appears to be dominant (Rosenow 1980). Lodging and charcoal rot resistance is quite heritable, but not by a single gene as reported by Coleman and Stokes (1958) in sorgo. The stay-green trait we have selected appears to be very stable over a wide environmental range. It has been screened in Arizona, throughout Texas, Mexico, and in Sudan.

Grain yield should be carefully considered when breeding and selecting for the stay-green trait and charcoal rot resistance. In general, as grain yield is increased and lower grain-to-stover ratios achieved, stalk rot susceptibility is increased. Also, hybrids are more susceptible than varieties. Until recently, few U.S. commercial hybrids have exhibited a high degree of stay-green or charcoal rot resistance; however, some new commercial hybrids are now appearing that possess good stay-green.

In a study of lodging resistance, the green-stalk puncture-pressure screening technique was used (Rosenow 1977) on previously selected lodging and charcoal-rot-resistant lines that had high puncture pressures. However, a selection study within a random-mated population, TP9, showed that selection based on individual plant puncture pressure was ineffective in increasing lodging resistance from that of the base population. Selection based on lack of lodging of individual plants within the population or on lodging percentages of S_1 rows resulted in a significant increase in lodging resistance (unpublished data).

In another study of a possible selection technique, aerial infrared photography as described by Blum et al. (1978) was used on nurseries under moisture stress. However, differences in canopy color showed no association with plant senescence, lodging, or drought ratings (Rosenow 1977). It appeared that color (and therefore canopy temperature) differed considerably among genotypes, but appeared to be a trait of the genotype and not associated with response to moisture stress.

Another study evaluated charcoal rot resistance in isogenic genetically juicy-stem and dry-stemmed sorghum lines. There was no difference in their charcoal rot reaction (unpublished data).

Selection for the stay-green trait in sorghum has indirectly resulted in high levels of resistance to fusarium head and peduncle blight and possibly to f usarium stalk rot. The Rio (SC599) derivative lines, which are among the most stay-green and resistant to charcoal rot, are very resistant to fusarium head blight (Frederiksen et al. 1973) and Banks grass mite (Foster et al. 1977). Although no specific breeding work has been done on pythium root rot, and it only occurs occasionally, highly susceptible lines can be identified by premature plant death

Table 4. Senescence, lodging, charcoal rot, and grain yield of selected sorghum hybrids at Lubbock and Halfway, Texas, USA.

Designation		LPD rating[a]	Charcoal rating[b]	Lodging[c] (%)	Yield (kg/ha)
A35 x SC56-14	R x R[d]	1.9	1.1	2	5910
A35xR9188	RxR	2.1	1.0	13	6050
A599 x SG56-14	RxR	1.8	1.0	73	6940
A1778 x R9247	RxR	1.9	1.0	15	5940
A599 x NSA440	RxR	2.2	1.1	22	5910
ATx399 x SC56-14	SxR	1.9	1.6	65	5010
ATAM618 x R9188	SxR	3.6	1.9	88	5500
ATAM618 x NSA440	SxR	2.2	1.5	72	5500
ATx399 x 1790E	SxR	2.0	1.1	42	6600
A599 x TAM428	RxS	3.0	2.8	100	6190
ATx399 x Tx2536	SxS	2.7	1.7	99	5220
ATx378 x Tx2536	SxS	2.7	2.7	100	5360

a. Leaf-plant death rated on 1-5 scale: 1 = none, 5 = dead.

b. Rated on 1-5 scale: < 1 = < one Internode, 1 = one internode, 4 = > three internodes, 5 = death.

c. Lodging data taken in February.

d. Parental line rating on charcoal and lodging: R = resistant, S = susceptible.

and lodging, so resistance has likely been selected in some of our materials.

The identification and extensive use of anthracnose-resistant sorghums from converted materials in the past 10 years has essentially eliminated anthracnose stalk and peduncle rot from South Texas. Screening for anthracnose is done primarily in Georgia, with some in Puerto Rico. Periconia root rot resistance is an outstanding example of breeding success. Resistant lines were selected in the late 1930s, and resistance (single gene inheritance) has remained stable for over 40 years.

Conclusions

Although stalk rot resistance is a complex phenomenon, much progress has been made in development of efficient screening techniques and in breeding for higher levels of resistance. We have made progress in two areas: genotypes have been selected with (a) anatomically stronger stalks and (b) a different physiological response to moisture stress. These latter types do not become predisposed to stalk rot by moisture stress as easily as common sorghum. By selecting within early-generation breeding material in multiple nurseries with variable stress and yield potential, progress has been made in combining good levels of stay-green with wide adaptation and good yield potential. Use of the visual leaf-plant death or stay-green rating is recommended as a very efficient selection method. Breeding and selection for stalk and root rot resistance should be done in a total-performance program, with strong emphasis on yield potential, adaptation, maturity, and other traits such as insect and disease resistance.

Future Research Needs

1. Research on the physiological and/or biochemical basis of the stay-green trait.
2. Determination of the physiological basis for preflowering drought tolerance, and how it differs from postflowering tolerance (stay-green). Such information is needed when breeding for charcoal rot resistance as a part of total performance.
3. More widespread use of currently available knowledge of screening and selection procedures and techniques by breeders and pathologists to breed for improved charcoal rot resistance. Emphasis should be on field performance, utilizing the best possible controls over timing of stress and uniformity of soil moisture. The stay-green trait should be used as an efficient breeding tool to select for charcoal rot resistance.

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References

AL-TAYAR, F.A. 1974. Stalk strength measurements to predict field lodging in *Sorghum bicolor* (L) Moench. Ph.D. thesis, Texas A&M University, College Station, Texas, USA. l26 pp.

BASHFORD, L.L., MARANVILLE, J.W., WEEKS, S.A., and CAMPBELL, R. 1976. Mechanical properties affecting lodging of sorghum. Transactions of the American Society of Agricultural Engineers 19:962-966,

BLUM, A, SCHERTZ, K.F., TOLER, R.W., WELCH, R.I., ROSENOW, D.T., JOHNSON, J.W., and CLARK, L.E. 1978. Selection for drought avoidance in sorghum using aerial infrared photography. Agronomy Journal 70:472-477.

COLEMAN, O.H., and STOKES, I.E. 1958. The inheritance of weak stalk in sorgo. Agronomy Journal 50:119-120.

DODD, J.L 1977. A photosynthentic stress-translocation balance concept of com stalk rot. Pages 122-130 *in* Proceedings of the 32nd Annual Corn and Sorghum Research Conference (eds. H.D. Loden and D. Wilkinson). Washington, D.C., USA: American Seed Trade Association.

DODD, J.L. 1980. The photosynthetic stress-translocation balance concept of sorghum stalk rots. Pages 300-305 *In* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

DUNCAN, R.R., 1977. Characteristics and inheritance of nonsenescence in *Sorghum bicolor* (L.) Moench. Ph.D. thesis, Texas A&M University, College Station, Texas, USA. 70 pp.

EDMUNDS, L.K. 1964a. Combined relation of plant maturity, temperature, and soil moisture to charcoal stalk rot development in grain sorghum. Phytopathology 54:514-517.

EDMUNDS, L.K. 1964b. Use of Arizona climate to induce charcoal rot in grain sorghum. Plant Disease Reporter 48:300-302.

EDMUNDS, L.K., VOIGT, R.L., and CARASSO, F.M. 1965. Charcoal rot induction and development in the field in Arizona. Pages 47-50 *in* Proceedings of the Fourth Biennial Grain Sorghum Research and Utilization Conference, sponsored by the Grain Sorghum Producers' Association (GSPA) and the Sorghum Improvement Conference of North America, Lubbock, Texas. Available from GSPA, Abemathy, Texas, USA.

EDMUNDS, L.K., VOIGT, R.L., FREDERIKSEN. R.A, and DUNKLE, L.D. 1973. Root and stalk rot problems in the Great Plains, Pages 88-92 *in* Proceedings of the Eighth Biennial Grain Sorghum Research and Utilization Conference, sponsored by the Grain Sorghum Producers' Association (GSPA) and the Sorghum Improvement Conference of North America, Lubbock, Texas. Available from GSPA, Abemathy, Texas, USA.

EDMUNDS, L.K., and ZUMMO, N. 1975. Sorghum diseases in the United States and their control. U.S. Department of Agriculture Handbook No. 468. Washington, D.C., USA: U.S. Government Printing Office. 47 pp.

ESECHIE, H.A., MARANVILLE, J.W., and ROSS, W.M. 1977. Relationship of stalk morphology and chemical composition to lodging resistance in sorghum. Crop Science 17:609-612.

FOSTER, D.G., TEETES, G.L, JOHNSON, J.W., ROSENOW, D.T., and WARD, C.R. 1977. Field evaluation of resistance in sorghums to Banks grass mite. Crop Science 17:821-823.

FREDERIKSEN, R.A., CASTOR, L.L., and ROSENOW, D.T. 1982. Grain mold, small seed and head blight: the Fusarium connection. Pages 26-36 *in* Proceedings of the 37th Annual Corn and Sorghum Research Conference. Washington, D.C., USA: American Seed Trade Association.

FREDERIKSEN, R.A., and ROSENOW, D.T. 1971. Disease resistance in sorghum. Pages 71 -82 *in* Proceedings of the 26th Annual Corn and Sorghum Research Conference, Washington, D.C., USA: American Seed Trade Association.

FREDERIKSEN, R.A., and ROSENOW, D.T.1980. Breeding for disease resistance in sorghum. Pages 137-167 *in* Biology and breeding for resistance to arthropods and pathogens in agricultural plants (ed. M.K. Harris). Proceedings of the International Short Course in Host Plant Resistance, 22 July-4 Aug 1979, Texas A&M University, College Station, Texas, USA. Texas Agricultural Experiment Station MP-1451.605 pp.

FREDERIKSEN, R.A., ROSENOW, D.T., and WILSON, J.M. 1973. *Fusarium* head blight of sorghum in Texas. Pages 33-36 *in* Proceedings of the Eighth Grain Sorghum Research and Utilization Conference, Lubbock, Texas. Available from GSPA, Abemathy, Texas, USA.

FROWD, J.A. 1980. Sorghum stalk rots in West Africa. Pages 322-324 *in* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored jointly by Texas A&M University (USA) and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT. 469 pp.

HOFFMASTER, D.E., and TULLIS, E.C. 1944. Susceptibility of sorghum varieties to *Macrophomina* dry rot (charcoal rot). Plant Disease Reporter 28:1175-1184.

HSI, D.C.H. 1961. An effective technique for screening sorghum for resistance to charcoal rot. Phytopathology 51:340-341.

JOHNSON, D.L, DAVIDSON, A.D., and HEATHMAN, E.S. 1966. *Fusarium* root rot *of Sorghum vulgare.* Phytopathology 56:148 (abstract).

KATSANOS, R.A., and PAPPELIS, A.J. 1965. Seasonal trends in density and cell death in sorghum stalk tissue. Phytopathology 55:97-99.

MALM, N.R., and HSI, D.C.H. 1965. Charcoal rot studies in New Mexico. Pages 51 -53 *in* Proceedings of the Fourth Biennial Grain Sorghum Research and Utilization Conference, sponsored by the Grain Sorghum Producers' Association (GSPA) and the Sorghum Improvement Conference of North America, Lubbock, Texas. Available from GSPA, Abernathy, Texas, USA.

McBEE, G.G., WASKOM, R.M., III, MILLER, F.R., and CREELMAN, R.A. 1983. Effect of senescence and nonsenescence on carbohydrates in sorghum during late kernel maturity states. Crop Science 23: 372-376.

ODVODY, G.N., and DUNKLE, L.D. 1979. Charcoal stalk rot of sorghum: effect of environment on host-parasite relations. Phytopathology 69:250-254.

RAO, K.N., REDDY, V.S., WILLIAMS, R.J., and HOUSE, L.R. 1980. The ICRISAT charcoal rot resistance program. Pages 315-321 *in* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored by INTSORMIL, ICAR, and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

ROSENOW, D.T. 1972. Selection for lodging resistance in grain sorghum. Page 18 *in* Agronomy Abstracts. Madison, Wisconsin, USA: American Society of Agronomy.

ROSENOW, D.T. 1977. Breeding for lodging resistance in sorghum. Pages 171-185 *in* Proceedings of the 32nd Annual Corn and Sorghum Research Conference. Washington, D.C., USA: American Seed Trade Association.

ROSENOW, D.T. 1980. Stalk rot resistance breeding in Texas. Pages 306-314 *in* Sorghum Diseases, a World Review: Proceedings of the International Workshop on Sorghum Diseases, sponsored jointly by Texas A&M Uni-

versity (USA) and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

ROSENOW, D.T., and CLARK, L.E. 1982. Drought tolerance in sorghum. Pages 18-30 *in* Proceedings of the 36th Annual Corn and Sorghum Research Conference. Washington, D.C., USA: American Seed Trade Association.

ROSENOW, D.T., and CLARK, L.E. 1983. Use of post flowering drought tolerance in a sorghum breeding program. Page 119 *in* Proceedings of the 13th Biennial Grain Sorghum Research and Utilization Conference, sponsored by the Grain Sorghum Producers' Association (GSPA) and the Sorghum Improvement Conference of North America. Available from GSPA, Abernathy, Texas, USA.

ROSENOW, D.T., CLARK, L.E., and WOODFIN, C.A. 1981. Breeding for drought resistance in sorghum. Page 11 *in* Proceedings of the 12th Biennial Grain Sorghum Research and Utilization Conference, sponsored by the Grain Sorghum Producers' Association (GSPA) and the Sorghum Improvement Conference of North America. Available from GSPA, Abernathy, Texas, USA.

ROSENOW, D.T., CLARK, L.E., and WOODFIN, C.A. 1982. Screening for drought tolerance in sorghum using field nurseries. Pages 81 -82 *in* Agronomy Abstracts. Madison, Wisconsin, USA: American Society of Agronomy.

ROSENOW, D.T., and FREDERIKSEN, R.A. 1982. Breeding for disease resistance in sorghum. Pages 447-455 *in* Sorghum in the Eighties: Proceedings of the International Symposium on Sorghum, sponsored by INTSORMIL, ICAR, and ICRISAT. Patancheru, A.P. 502 324, India: ICRISAT.

ROSENOW, D.T., JOHNSON, J.W., FREDERIKSEN, R.A., and MILLER, F.R. 1977. Relationship of nonsenescence to lodging and charcoal rot in sorghum. Page 69 *in* Agronomy Abstracts. Madison, Wisconsin, USA: American Society of Agronomy.

ROSENOW, D.T.. QUISENBERRY, J.E., WENDT, C.W., and CLARK, L.E. 1983. Drought tolerant sorghum and cotton germplasm. Agricultural Water Management 7:207-222.

SCHERTZ, K.F., AL-TAYAR, F.A., and ROSENOW, D.T. 1978. Comparison of methods for evaluating stalk strength in sorghum. Crop Science 18:453-456.

SCHERTZ, K.F., and ROSENOW, D.T. 1977. Anatomical variation in stalk internodes of sorghum. Crop Science 17:628-631.

STEPHENS, J.C., MILLER, F.R., and ROSENOW, D.T. 1967. Conversion of alien sorghums to early combine genotypes. Crop Science 7:396.

TARR, S.A.J. 1962. Diseases of sorghum, sudan grass and broom corn. Kew, Surrey, U.K.: Commonwealth Mycological Institute. 380 pp.

TEETES, G.L., ROSENOW, D.T., FREDERIKSEN, R.A., and JOHNSON, J.W. 1973. The predisposing influence of greenbugs on charcoal rot of sorghum. Texas Agricultural Experiment Station, College Station, Texas, USA (PR-3173). 6 pp.

VOIGT, R.L., and EDMUNDS, L.K. 1970. Tolerance to charcoal rot in hybrid grain sorghum. Page 22 *in* Agronomy Abstracts. Madison, Wisconsin, USA: American Society of Agronomy.

WOODFIN, C.A., ROSENOW, D.T., CLARK, L.E., and JOHNSON, J.W. 1979. Differential response of sorghum cultivars to drought stress. Page 82 *in* Agronomy Abstracts. Madison, Wisconsin, USA: American Society of Agronomy.

ZUBER, M.S. 1973. Evaluation for progress in selection for stalk quality. Pages 110-122 *in* Proceedings of the 28th Annual Corn and Sorghum Research Conference (ed, D. Wilkinson). Washington, D.C., USA: American Seed Trade Association.

ZUMMO, N., and FREDERIKSEN, R.A. 1973. Head blight of sorghum in Mississippi. Page 37 *in* Proceedings of the Eigth Grain Sorghum Research and Utilization Conference, sponsored by the Grain Sorghum Producers' Association (GSPA) and Sorghum Improvement Conference of North America/Lubbock, Texas. Available from GSPA, Abernathy, Texas, USA.

Questions

Mughogho:
I was impressed by your presentation slide taken in the Sudan showing one of your nonsenescent lines looking green in a field where other lines had senesced and lodged. It would be useful to know if all the lines were of the same maturity group, since plant growth stage has an important bearing on plant water use and hence drought resistance and predisposition to stalk rots and lodging.

Rosenow:
There was a wide range in maturity in this breeding nursery, and the stay-green expression in this case was not related to maturity. We recognize the extreme importance of maturity in this expression, so we make comparisons only among genotypes of similar maturity.

Partridge:
Have any isolations been made previous to the early plant death expression to determine if there

are other organisms present in the stalk tissue that might potentially be involved?

Rosenow:
Dr. Frederiksen did some isolation work several years ago and consistently found organisms in roots at an early stage, but no work was done on isolation from stalks at this later stage.

Seetharama:
Is there any study where someone has compared the water use pattern of stay-green and other types of genotypes? Or has anybody compared their root characteristics? Is such a study useful?

Rosenow:
Yes, Dr. Charles Wendt has used the soil neutron probe technique to evaluate stay-green versus senescing types under both rainout shelters and field conditions. He found little or no difference in total water extracted or in the depth from which water was extracted. He found slight differences in rate of moisture extraction, with senescing types using water slower in early stages of growth, but continuing to use more water later in the season, relative to senescing genotypes. Also Dr. W.R. Jordan has studied the rooting and wafer use pattern of one stay-green line, SC56, and found that it had a deeper root system and utilized more water from deeper soil depths late in the season. Although these studies showed some differences, it seems that the visual evaluation is much easier with bigger differences. However, I believe studies on roots are essential to basic studies of drought tolerance, but I question if roots can be used efficiently in a screening technique at this time.

Seetharama:
What percentage of genotypes considered resistant to drought at terminal stage of growth are also stalk rot resistant?

Rosenow:
For charcoal rot resistance and the way we define postflowering drought tolerance, the relationship is essentially 100%.

Frederiksen:
Is the stay-green trait stable across locations?

Rosenow:
Yes— from Arizona, all over Texas, Sudan, and India.

Pande:
Under what cultural practices did you test stay-green material? If under irrigated, at what plant growth stage did you stop the irrigation? If rainfed, what was the total rainfall before planting and after planting, with respect to plant growth stage?

Rosenow:
We screen the plants primarily in nurseries in West Texas, where we fertilize well and irrigate well during early stages of growth. Irrigation is then withheld prior to flowering to allow moisture stress to develop during the grain-filling stage. We also do some evaluation under rainfed conditions in South and Central Texas, where rainfall is higher. In these areas, there is a rather deep, heavy clay soil, which is essentially full of water prior to flowering. The plants normally receive sufficient rainfall during the early season, with decreasing rainfall and higher temperatures as they approach maturity.

Pande:
Have you tested this material under different soil types with differences in water-holding capacity?

Rosenow:
Yes, from sandy to clay soils in West Texas to sandy and clay soils in Sudan, and the stay-green trait behaves consistently. A problem with the highly stay-green types in sandy soil is that stress often develops prior to flowering and greatly retards head development, which then does not allow sufficient sink development to produce stress during grain fill.

Breeding for Stalk Rot Resistance as a Component of Acceptable Agronomic Performance

A.B. Maunder*

Stalk Rots of the Arid Americas

At the time of sorghum hybrid introduction in the United States in 1956, the most serious disease was thought to be charcoal rot (causal agent, *Macrophomina phaseolina*). Although head smut (causal agent, *Sphacelotheca reiliana*) produced a serious yield loss in the Coastal Bend area of Texas, the stalk rots common to more arid and hot conditions (caused by *M. phaseolina, Fusarium moniliforme,* and other lesser organisms) were estimated to account for 4.5% of sorghum yield losses in the United States (USDA 1965). Also, *F. moniliforme* is by far the most serious causal agent of stalk rot on sorghums in Argentina. With these stalk rots responsible for half of the U.S. total disease loss (9%) during this period and with the early hybrids all known to be susceptible to charcoal rot, a breeding effort towards charcoal rot resistance seemed natural for a commercial research program affecting all U.S. sorghum acreage, the majority of which was being grown from the arid southwest (Arizona, New Mexico, Texas) northeast through the Great Plains to South Dakota,

In this paper I would like to specifically report on the Dekalb AgResearch, Inc., approach to stalk rots, with primary emphasis on charcoal rot. Anthracnose, although considered significant and allocated research funding, will not be considered because of its quite different environmental requirements.

Breeding Approach

Three steps are required to develop a disease-resistant hybrid: (1) finding a source of resistance with a useable level of heritability, (2) combining this resistance with other required crop traits, and (3) isolating parental lines that in hybrid combination maintain an acceptable level of resistance without sacrificing yield. The disproportionate number of resistance genes to yield genes, the latter sometimes estimated at nearly 5000, suggests an applied breeding approach quite different from a basic attempt to isolate a genetic source of resistance.

Since both charcoal rot and fusarium stalk rot are most likely to develop under heat- and moisture-stressed growing conditions and multiple locations are expensive, a dependable field screen is difficult to simulate. At one time the University of Arizona provided a screen where supplemental irrigation was the only water normally available during the growing season. However, temperatures there generally exceeded the 38°C said to be optimum for charcoal rot.

Charcoal Rot Resistance

Early Generations

We grew two nurseries in 1960, one at Lubbock, the other in eastern New Mexico, in an attempt to

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NOTE: This paper is based on the author's 28 years of experience in the commercial sorghum industry. Further information can be obtained directly from him.

International Crops Research Institute for the Semi-Arid Tropics. 1984. Sorghum Root and Stalk Rots, a Critical Review: Proceedings of the Consultative Group Discussion on Research Needs and Strategies for Control of Sorghum Root and Stalk Rot Diseases, 27 Nov - 2 Dec 1983, Bellagio, Italy. Patancheru, A.P. 502 324, India: ICRISAT.

improve the odds for adequate infection on 820 entries of inbreds and hybrids. The New Mexico nursery was on soil known to have a high natural buildup of *M. phaseolina.* At the time of booting, supplemental moisture was withheld on the Lubbock entries, which were grown at relatively high populations and high nitrogen levels.

Dr. D.C.H. Hsi of New Mexico State University made significant initial suggestions for these screenings and provided the pathogen isolates for use in 1960. The fungus was cultured on toothpicks coated with potato dextrose agar. Three distinct isolates were used, and their identity was maintained during the early generations. Sources of isolates varied, with our main objective being to utilize those with the most virulence. Toothpick inoculation of stalks was made at approximately 5 cm above the soil level 3-4 weeks after flowering. Eighteen readings for each of the 700 test entries averaged from 1.3 cm to 27.2 cm infection from the point of inoculation.

As seen in Table 1, conditions favored charcoal rot development at Lubbock compared to the New Mexico location. At Lubbock the homozygous material averaged 7.8 cm infection, compared to 11.9 cm for the hybrids. Generally the hybrids were more severely infected than the mean of the parents, and frequently more than the most susceptible parent, suggesting that susceptibility was dominant. Lines showing resistance were re-screened to eliminate escapes. Some entries were obviously resistant; others mechanically excluded the fungus through internal structural barriers (e.g., compressed nodes); and finally tolerance through the lodging resistance provided by a stiff stalk was another possible source of improvement.

The better lines were not only crossed in dialleles but also used as source material for pedigree-type breeding. New lines suggested that progress would be possible, but slow, because of escapes and the complexity of the inheritance. Three yellow endosperm derivatives were the basis for segregating populations with elite but susceptible inbreds until 1964, when New Mexico 31 was released by Malm and Hsi (1964).

Table 1. Frequency distribution of *Macrophomina phaseolina* growth from point of inoculation in the stalks of 700 sorghum genotypes in 1960 screenings at Lubbock, Texas, and Texico, New Mexico, USA.

Growth of fungus (cm)	No. of entries	
	Lubbock	Texico
0.0- 2.9	22	149
3.0- 5.9	123	218
6.0- 8.9	169	177
9.0-11.9	153	82
12.0-14.9	132	41
15.0-17.9	64	20
18.0-20.9	19	7
21.0-23.9	5	1
24.0-26.9	1	0
27.0-29.9	1	0

Hybrid Application

Male or restorer lines resistant to charcoal rot were developed ahead of male-steriles, both because of the loss of generations in going from B to A lines and also because the initial pedigrees with resistance were of an R or male type. Resistant male-steriles, while agronomically acceptable, always traces back to some R or male germplasm as the source of their resistance. The recurrent or predominant parentage of these new resistant females was of a kafir, milo-kafir, or U.S. x plant introduction derivative.

By 1970, or 11 summer generations into this program, an experimental hybrid, X-1486 (later to be designated C-42c), was recommended for production. This pedigree was our first charcoal-rot-"resistanf hybrid, with the male parent coming directly from cycle 1 of the resistant x susceptible populations. The sterile, a stiff-stalk line, traces to kafir-milo x a Nigerian yellow plant introduction. Charcoal rot readings showed a significant improvement compared to checks, and yield was acceptable (see Table 2). Both parents exhibited the stiff stalk trait, considerable drought resistance, and a degree of nonsenescence. Seed yield of the female, plant height, and more than normal upper-node breakage late in the season limited the

Table 2. Percent growth of *Macrophomina phaseolina* in the stalk and yield of sorghum hybrid C-42c compared to two check hybrids (1969-70) at Lubbock, Texas, USA.

Hybrid	Fungus growth (% of check)	Yield (% of check)
DEKALB C-42a	61	105
DEKALB E-57	32	103
RS-610	10	115

acceptance of this dryland hybrid. A pedigree using this female, however, was used quite extensively in Australia as C-42t.

Several hybrids with resistance in both parents demonstrated good resistance in natural and inoculated field tests, but failed to be competitive. X-635 for example, yielded 91.5%, 95.4%, and 96.5% of the checks over 3 years, with a quite acceptable charcoal rot level. DEKALB C-46, however, which had a charcoal-rot-resistant male and a drought-tolerant, stiff-stalked female, gave an equally low charcoal rot reading, but more importantly, under stress, it stood and yielded well. The drought aspects of this hybrid appeared to be due to (a) nontillering, (b) reduced transpiration, (c) osmoregulation, and/or (d) nonsenescence.

C-46 became available in 1982 as DK-46. this hybrid has resistance to greenbug biotypes C, D, and E and apparently has even better stalk quality and yield potential. The pollinator of DK-46 is a tropical x charcoal-rot-resistant derivative, with the hybrid showing outstanding stalk quality. Unfortunately, the male tends to be a specific combiner. In 1983, a year of record heat and drought, DK-46 saw its first year of sizeable commercial plantings and gave excellent performance. A big question remains, of course, as to whether it was selected for nonsenescence or charcoal rot resistance, but the critical measurement for stalk rot remains positive and includes resistance to fusarium stalk rot.

Other useful lines have come from the program, but after 16 years line development was reduced in 1975. The significance of nonsenescence emphasized the need for continual field testing under limited water levels. Also, milo types in hybrid combination showed the obvious advantages of the introduction of yellow endosperm germplasm into commercial hybrids. R.E. Karper and O.J. Webster probably accomplished as much or more with their initial yellow endosperm introductions and breeding as was gained at the time of hybridization in 1956.

Another commercial hybrid, DK-57, uses a male from this program converted to greenbug biotype C resistance. Finally, an additional charcoal-rot-resistant line is in advanced testing in hybrid combination, with especially good stress results.

Relationship to Drought Resistance

The quantitative nature of drought resistance superimposed on the complex physiological requirements of charcoal rot resistance suggests that more might be accomplished by breeding for drought resistance. Here at least yield would be an integral objective if drought resistance were related to dry matter production per unit of water. As the charcoal rot program, discussed previously, matured in line development, so in turn did a parallel program that screened germplasm for these drought traits: (a) diffusive resistance, (b) heat tolerance, (c) dormancy, and (d) root development. Dormancy refers to the plant remaining healthy but with limited or no growth.

The above screening suggested good heritability for all these traits, but no one alone gave enough drought resistance, and the yield level was not acceptable. Therefore the quantitative task of combining yield with drought resistance and stalk rot resistance suggested a population approach. These populations must contain known germplasm for the three objectives. In addition, a screening system that adequately evaluated yield, drought, and stalk rot was essential.

Our approach was based on recurrent selection, with germplasm containing lines known to have desirable components of drought resistance, lines from our charcoal rot program, stiff-stalk lines, and germplasm of known good combining ability. Initially we confined our program to the male or restorer side but used testers of known heat and drought tolerance on the female side, except for a later, stiff-stalked sterile which has now been dropped for reasons of hybrid maturity. This material was tested under both drought stress and irrigation, and initial testcrosses were evaluated in a similar fashion.

Evaluation in advanced replicated trials was conducted in the following environments: Lubbock dryland, Lubbock limited irrigation (20-25 cm), Southwest Kansas dryland, and South Central Nebraska favorable dryland. Most hybrids tended to have high location interaction, with either good drought or good optimum performance related to the check means. The second year of testing, however, emphasized those performing well across all moisture levels,

Stress in 1983 was more severe than in 1982, giving an excellent evaluation. In addition, plants in the third replication at Lubbock were inoculated with charcoal rot. Yield data from 1982 and 1983 will be regressed against test means to determine B-values. It is hoped that we can avoid extremes beyond $B < 0.90$ or > 1.10 when selecting new releases by this approach. An improvement in stalk

quality is also anticipated, both from the germplasm involved and from the natural field screening across environments, as well as from toothpick inoculations.

Relationship to Insect Resistance

Infestations of greenbug or mites are frequently associated with a heavy degree of lodging, besides causing reduced yield. Since these insects predispose the plant to various fungal organisms, the availability of hybrids resistant to sorghum greenbugs in 1976 significantly reduced lodging in the U.S. crop, just as the widespread use of yellow germplasm did in the 1960s, Additionally, insect resistance might:

a. allow "dormancy" to be a trait in drought resistance by keeping plants healthy during stress,

b. produce better control of stomatal response in reducing transpiration,

c. produce more photosynthetic activity by the resistant form, as with nonsenescent sorghum.

Unfortunately, although mites are obviously closely related to lodging problems, progress with resistance to Banks grass mite (*Oligonychus pratensis*) has been slow, A breeding program concerned with stalk rots must include insect resistance as an additional component of recombination and screening.

Fusarium Stalk Rot

At the time we began the charcoal rot program we reviewed an ongoing fusarium stalk rot program. A limited attempt at inoculation and subsequent readings suggested that we confine our effort to charcoal rot. We relied somewhat on previous research suggesting that varieties resistant to *M. phaseolina* often are resistant to *F. moniliforme.* Also, there was the suggestion that in our material the degree of resistance to charcoal rot was greater than to fusarium stalk rot.

The 1983 season gave us plenty of opportunity to see field differences in the level of resistance to *F. moniliforme* from Lubbock, Texas, to Nebraska. Often the two pathogens were found together, and we verified the existence of resistance to both in the same material.

Argentina, the second largest sorghum producer in the Western Hemisphere, experiences much more loss from *F. moniliforme* than from other stalk-rotting organisms. In Argentina the breeding program attempts to screen over nine environments a diverse group of hybrids (5000 in 1982-83). The most severe incidence of fusarium stalk rot in Argentina occurred in 1982-83 to the west and southwest of the sorghum belt, approximating 32-36° S, with much less effect in the warmer but more humid north.

An approach to be tested in 1983-84 in Argentina will allow us to choose a set of 35 hybrids known to be outstanding in performance but whose level of resistance to *F. moniliforme* is unknown. These will be grown at three hot-spot locations at both normal (200000 plants/ha) and heavy populations (400000 plants/ha) in replication. The high population should stress the plants enough to allow stalk quality in the presence of the organism to be scored. Also, we will be able to observe testcrosses with new material having a nonrecurrent parent with *F. moniliforme* resistance. Whereas susceptibility to *F. moniliforme* also appears to be dominant, we note considerable variation between hybrids, suggesting the potential for improvement through effective screenings of a range of hybrid genotypes, as well as through line development.

Genetic Variability and Charcoal Rot

Level and Type of Resistance

The range of inbred reaction to charcoal rot points out very clearly that genetic gain can be achieved through selection. For example, using three isolates of *M, phaseolina* we obtained the mean values shown in Table 3. Unfortunately hybrids

Table 3. Mycelial growth of three isolates of *Macrophomina phaseolina* in the stalks of three cultivars at Lubbock, Texas, USA.

Pedigree	Mycelial growth in stalk (cm)
Redbjne 60	31.7
New Mexico 31	1.3
Superior inbred	2.0

often do not reflect even partial dominance for resistance. With susceptibility dominant, perhaps it is not surprising to see overdominance being expressed. Certainly a hybrid with more water-use efficiency and a greater sink will be under more stress later in the season.

The first released hybrids with a heavy component of milo or kafir-milo derivatives for pollinators, and frequently with kafir-milo females, were extremely susceptible. With a recessive type resistance, breeding becomes twice as difficult since both parents must be resistant and also combine well for hybrid yield. The introduction of improved yellow endosperm germplasm during the 1960s not only added improved yield, drought resistance, and disease resistance, but also had a major impact on stalk quality. The healthier plants resulting from red x yellow and yellow x yellow crosses were much more resistant to charcoal rot.

Although not a charcoal-rot-resistant hybrid in the strict sense, DK-46 has generally stood better than other commercials when stressed at a similar physiological growth stage, as was clearly demonstrated through the stress of 1983. No doubt the nonsenescence and osmo-regulating ability of the hybrid is a big factor if we accept the premise that the health of the plant is all-important in its ability to resist the organism.

The breeder need only to add the stiff stalk trait to a hybrid to greatly improve the odds for a standing plant at harvest. This use of tolerance to stalk rot was first shown with E-57, a hybrid released in 1964 and still grown rather extensively, especially in Australia. Additional favorable traits of E-57 are "dormancy" and an improved level of nonsenescence.

Since the nodal tissue appears to temporarily slow down the upward movement of the organism, a hybrid based on short plant stature can be expected to have less relative internode damage. Efforts to incorporate a 4-dwarf parent, which shortens a hybrid and generally pleases the producer, have also been responsible for reducing stalk rot loss.

Advances In Drought Tolerance

Even if a "true" charcoal-rot-resistant hybrid failed to result from this extensive program, numerous successful inbreds that added a new dimension to drought resistance were developed. Aiming for luxurious early growth followed by stress during and after pollination has given us every opportunity to isolate nonsenescent germplasm. The second phase of this program will allow recurrent selection to increase the heterotic potential of the lines developed by this method. However, caution must be given to any assumption that inbreds selected for drought or stalk rot resistance can be expected to produce hybrids with more than average standability.

Basic Results

Although extremely sensitive to the environment the toothpick screening system is a useful tool to the breeder. Replication and screening over several years may be required to verify classification. During the early years we used no less than three isolates of *M. phaseolina* at all times. No correlation existed between virulence in culture and structural damage or growth within the plant. Actually, our weakest culture was the most virulent in the plant.

As pointed out previously, the shorter internodes of 4x3-dwarf hybrids can add a level of mechanical resistance. In 1967 some six inbreds isogenic for the Dw2 height gene were grown in the charcoal rot nursery with three sources of inoculum. The 3-dwarfs had 40% more measured charcoal rot development than the 4-dwarfs of a similar genetic background. Also, the 3-dwarfs had a higher percentage of lodging, as might be expected.

To gain some insight into the importance of drought resistance and, in part, to verify the importance of the waxy bloom found on most sorghum, we evaluated isogenic forms of three sorghum varieties in the 1971 charcoal rot nursery. Two replications with a combined total of 12 measurements of fungus growth favored the normal plants (Table 4).

Table 4. *Macrophomina phaseolina* growth in inoculated stalks of bloom and bloomless varieties of sorghum in 1971 charcoal rot nursery.

Variety	Bloom	Mycelial growth in stalk (cm)
Martin Bl	present	12.4
Martin bl	absent	19.6
Combine Kafir 60 Bl	present	11.4
Combine Kafir 60 bl	absent	14.4
Redbine Bl	present	14.2
Redbine bl	absent	18.6

The more extensive growth of the pathogen in the bloomless plants suggests that sorghums with little or no waxy coating may be more predisposed to infection by the organism. Since additional information verifies that bloomless sorghums are less drought tolerant, we can assume the additional stress encountered by these lines compared to normal varieties provides a more favorable environment for charcoal rot development.

If we can accept a threshold concept of physiological resistance, hybrids will then vary according to their drought tolerance. Additionally, morphological resistance and tolerance will contribute to a final standability classification.

Conclusions

Although charcoal rot is of less significance in the United States today than when hybrids first appeared, we will continue to face a high percentage of cropped areas grown under stress. While the initial yellow endosperm introductions seem to have made a significant contribution, future progress will also rely heavily on plant introductions. We need to determine the best approach for rapid and efficient improvement of adapted lines from such germplasm.

Once source material has been determined, the most practical approach to developing useable lines will be to combine this material with the two primary objectives of sorghum improvement in arid zones: improved yield and drought tolerance. Selection for the presence of a stiff stalk and drought tolerance, especially of the nonsenescent type, combined with high yield, will be more productive than breeding for charcoal rot resistance alone. On the contrary, excellent progress in drought resistance has been possible by screening for charcoal rot under the appropriate late-season stress conditions. Finally, the breeder has much less understanding of the mode of action of *F. moniliforme* than of *M. phaseolina.* The obvious severity of diseases caused by this organism in the major sorghum areas of the Americas suggests that equal attention be given *F. moniliforme.*

References

MALM, N.R., and HSI, D.C.H. 1964. New Mexico 31, a charcoal rot-resistant grain sorghum line. New Mexico Agricultural Experiment Station Research Report 93. Las Cruces, New Mexico, USA: New Mexico State University.

USDA. 1965. Losses in Agriculture. USDA Handbook 291. Washington, D.C., USA: U.S. Department of Agriculture. 10 pp.

Lodging, Stalk Rot, and Root Rot in Sorghum in Australia

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Summary

Most of the 600000 ha of grain sorghum in Australia is grown under dryland conditions, Water deficits during the growing season are common, and lodging associated with stress during grain filling is prevalent; this type of lodging is due to weakening and fracture of the stem base. Root lodging is of little significance. Stalk rots, predisposed by stress, are also common.

Research and observations in Australia support evidence from elsewhere that the source-sink relationships of the plant during grain filling greatly influence lodging and stalk rot development Physiological stress caused by a low source/sink ratio is a necessary condition for lodging and for the development of stalk rots.

The source-sink relationship is important in selection for resistance to lodging, and characters that affect this relationship—particularly grain yield and maturity—are considered when selecting for lodging resistance. Nonsenescence is closely correlated with lodging resistance and is widely used as, an indicator characteristic during selection. The use of a tropical environment is discussed.

Little direct selection is practiced for resistance to stalk rots, although evidence exists for genetic variation in resistance to Fusarium moniliforme *and* Macrophomina phaseolina.

Some recommendations for future research are made.

Approximately 600000 ha of grain sorghum are grown in Australia each year, with about one third in each of the northern New South Wales, southern Queensland, and central Queensland regions (Table 1 and Fig. 1). In addition, a large potential for increased production is beginning to be realized in the semi-arid tropics of north Queensland.

Almost all sorghum in Australia is grown under dryland conditions in areas averaging 500 to 700 mm annual rainfall. This is mainly summer rainfall, and it is unreliable both in total amount and distribution. Water deficits are therefore common, and the consequent lodging is a significant factor in sorghum production, particularly in central Queensland. It is also expected to be a serious problem in the potential cropping regions of northern Queensland. Lodging resistance is an essential characteristic of grain sorghums for these areas and is desirable in other parts of Australia. Root lodging is of little significance in Australia.

Although lodging and stalk rots are important problems in sorghum production, poor seedling

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Table 1. Sorghum production in Australia, 1981/82 season. (Source: Australian Bureau of Statistics.)

State/region	Area (ha)	Production (tonnes)	Yield (t/ha)
Queensland:			
Southern Queensland	257098	647418	2.518
Central Queensland	228425	330437	1.447
Northern Queensland	3621	4580	1.265
Total Queensland	489144	982435	2.008
New South Wales	152346	325689	2.140
Western Australia	4928	5270	1.069
Victoria	1537	2477	1.612
Total Australia [a]	648574	1316706	2.030

a. Includes small area and production from South Australia and the Northern Territory.

emergence and root rots are also frequently encountered. The major planting period in southern Queensland and northern New South Wales occurs in spring and early summer when soil temperatures are frequently low. Seedling pathogens are commonly associated with poor emergence and establishment under these conditions; replanting is often necessary. Root rots during crop development are widespread, and root systems are often severely diseased; their effects on yield are not known, however.

The Causes of Lodging

Sorghum crops grown under ideal conditions are still green when the grain is physiologically mature. Extensive death of leaf or stem tissues ("senescence") at this time can be regarded as a stress symptom.

The most important type of lodging in grain sorghum in Australia occurs after a water deficit during the grain-filling period. Plants senesce and then lodge due to stems breaking at or just above ground level. The stems are weakened by degradation of the pith and rind in the basal internodes, leaving unsupported vascular strands. Invasion by stalk-rotting fungi is common, but not universal, in lodged stalks.

The causes of death and lodging are not well understood, but three hypotheses can be proposed: (1) plants die as a direct result of water deficit, i.e., a physiological breakdown due to dehydration; (2) pathogens are the cause of death; or (3) death is due to an interaction between physiological stress and pathogens.

The Role of Physiological Stress in Lodging

It has been proposed that physiological stress per se results in rapid senescence and subsequent lodging. This stress can be generated when a large "sink" for photosynthetic assimilate, such as a rapidly growing organ (the grain), creates a high demand in relation to the assimilate supply (photosynthetic capacity). A crop is considered "source limited" if the sink is capable of growing larger when the assimilate supply is increased; it is "sink limited" if the sink does not respond in this way. If, for example, single-grain weight does not increase when the crop is thinned or panicles artificially reduced in size at anthesis, then the crop is said to be sink limited with respect to grain yield, whereas an increase in grain weight would imply a source limitation. Various factors can reduce assimilate supply, including water deficit, leaf removal, leaf disease, insect damage, nutrient deficiency or toxicity, and low light intensity. The "physiological stress" is thought to result in a shortage of available carbohydrate in the stem (Chamberlin 1978). Cell death occurs when the level is too low to support sufficient maintenance respiration. Pith disintegration then begins at the base of the plant and may extend upwards several internodes as conditions worsen.

There is good circumstantial evidence to support the hypothesis that high rates of senescence in response to water deficits are caused by the presence of a relatively large grain-filling sink. This was illustrated by Henzell and Gillieron (1973), who altered the source-sink relationship in two hybrids, Texas610 and DeKalb E57, and one inbred variety,

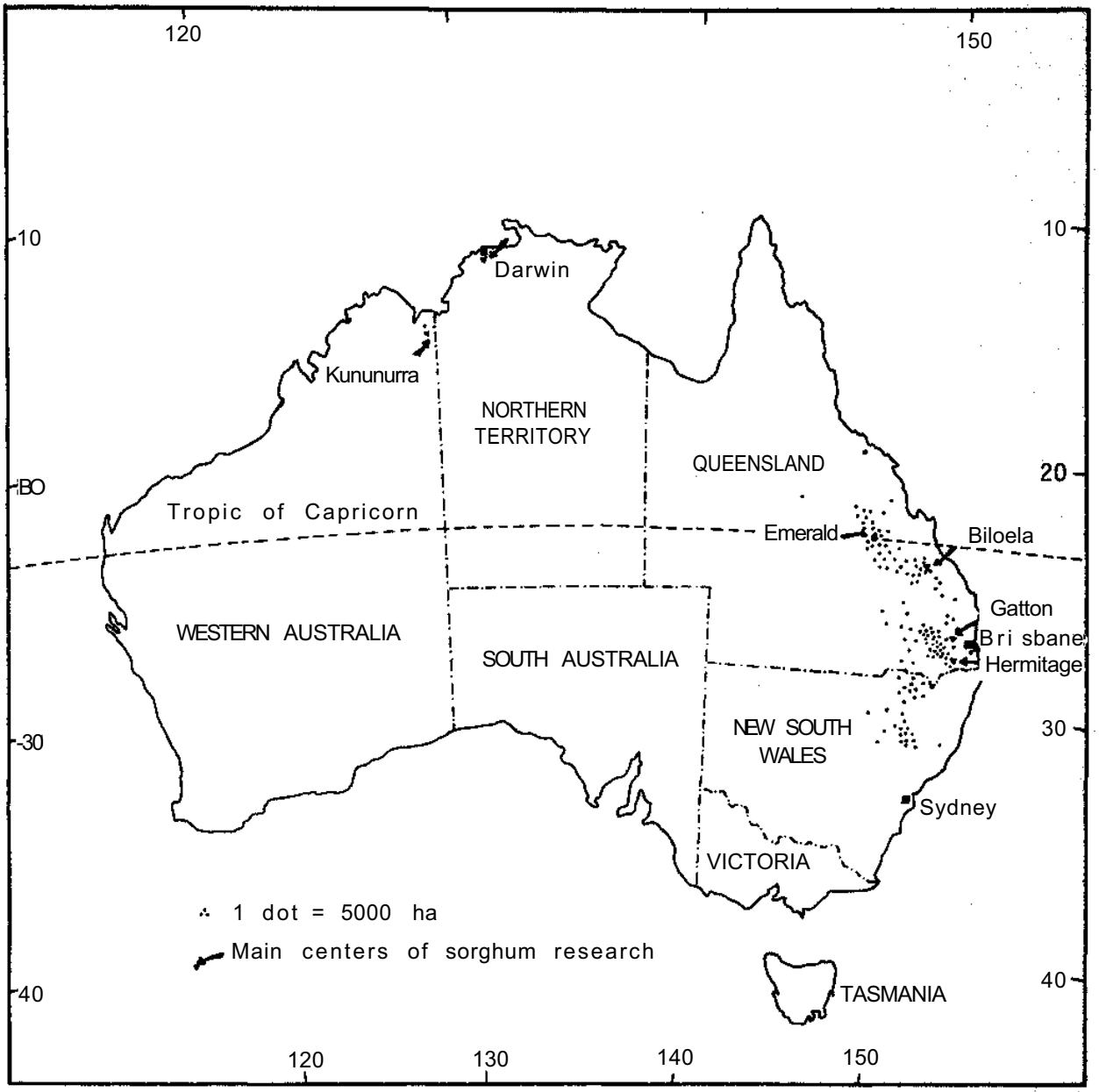
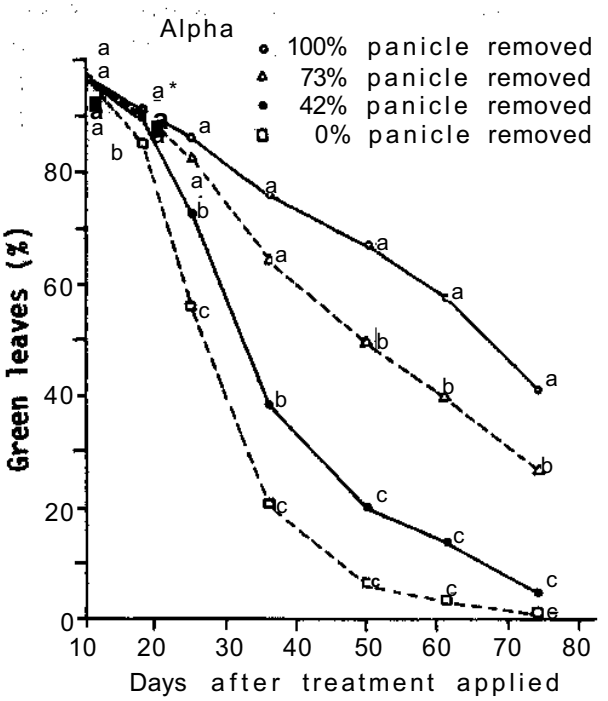


Figure 7. Grain sorghum production in Australia in 1981-82.

'Alpha,' by mechanically removing portions of their panicles at flowering. In each genotype, this reduction in sink size dramatically reduced the rate of leaf senescence in the presence of a water deficit. The relationship for 'Alpha' is shown in Figure 2. Charcoal rot caused by *Macrophomina phaseolina* (Tassi) Goid. was associated with some dead plants, but the majority showed no obvious symptoms of infection by any stalk-rotting fungus (Table 2). No isolations were made to determine the presence of pathogens. A.A. Done has also observed that nonf lowering plants senesce at a slow rate and do not lodge even after extended periods of drought stress.

The hypothesis that the source-sink relationship affects lodging is also supported by the association between maturity type and lodging. Early flowering is often associated with a higher senescence rate and therefore with susceptibility to lodging. This Is probably because early genotypes are likely to pro-



*At a particular date, points followed by the same letter are not significantly different ($P < 0.05$).

Figure 2. Effect of panicle treatment on the death of leaves for the inbred variety 'Alpha' in 1969-70. (Source: Henzell and Gillieron 1973.)

duce more grains per unit leaf area than later genotypes, This is at least partly due to a lower probability of water deficit at the time of panicle development in early genotypes and therefore less chance that a water deficit would inhibit panicle development. Even in the absence of water deficits during panicle development, competition for assimilate between developing leaves and panicle may be greater in late- than in early-flowering genotypes planted at moderate to high population densities. This results in a lower grain number to leaf area ratio in later genotypes.

It might be conjectured that later maturing genotypes would have a higher probability of encountering end-of-season water deficits and therefore a greater chance of lodging. Experience has shown, however, that the morphological and physiological characteristics associated with later maturity apparently contribute more to lodging resistance than phenological characteristics might be expected to contribute to susceptibility. This association between lodging resistance and late maturity can be seen in Tables 5,8,9, and 10, which are discussed later.

It is frequently observed that there is a negative correlation between days to flower and harvest index, as illustrated for four locations in Table 3. This observation supports the proposed explana-

Table 2. Percentage of dead plants and charcoal-rot-diseased plants (% of dead plants) for four panicle treatments for DeKalb E57 and Texas610 at Biloela, Queensland, Australia.

	DeKalb E57		Texas610	
Panicle	% dead plants	% rotted plants	%dead plants	% rotted plants
Full panicle	58	4	50	9
Two-thirds panicle	52	0	36	13
One-third panicle	1	0	0	0
No panicle	0	0	0	0

Table 3. Correlation between days to flower and harvest index in four experiments in Australia.

Test location	Correlation coefficient	No. of genotypes	Harvest index range
Kununurra (dry-season tropics restricted irrigation)	-0.63**	27	0.48-0.61
Hermitage (irrigated temperate)	-0.56	12	0.42-0.53
Hermitage (dryland temperate)	-0.87**	12	0.41-0.50
Dalby (dryland temperate)	-0.80	5	0.12-0.34

** $P<0.01$.

tion of the relationship between maturity and senescence rate. Plants with a high harvest index are more likely to have a low source/sink ratio and according to the hypothesis are therefore more likely to senesce when the source's capacity to fill the sink is reduced.

On the other hand, Brown (1978) has data that do not support this proposition. He found that the effect of adverse conditions during the panicle development was in the direction of enhancing panicle (sink) relative to leaf (source) development. However, plants in his study were grown in pots in a glasshouse, with stress usually being sudden and severe and applied during brief segments of the period of panicle development.

Because of the apparent importance of the source-sink relationship in determining senescence rate, it is of interest to consider the relationship for genotypes of known lodging resistance. Muchow and Wilson (1976) examined, under favorable growing conditions, the source-sink relationships in the grain yield of four hybrids: DeKalb E57, Pacific Goldfinger, Texas610SR (ATx3197/RTAM 422), and Texas626 (ATx3197/RTx415). DeKalb E57 is resistant to lodging, whereas the other three are very susceptible. Muchow and Wilson's analysis showed that the hybrids susceptible to lodging were source limited, whereas the resistant hybrid was partially limited by both source and sink—a finding that is consistant with our physiological hypothesis. The small number of genotypes limits general conclusions, and the source-sink relationship of particular genotypes is likely to vary with environmental stresses. Brown (1978) found, however, little if any evidence to suggest that source capacity was limiting grain yield in DeKalb E57 subjected to varying levels of water deficit during panicle development.

Done and Muchow (1983) found in an irrigated winter planting in northwest Australia that all genotypes (20 inbred lines and F_1 hybrids) except Ramada (a tall, late, sweet inbred) were source limiting for grain yield. The degree of limitation varied, but this did not appear to be related to the known lodging resistance of the genotypes. However, DeKalb E57 was less source limited than most genotypes.

It is also of interest to examine the hypothesis that lodging-susceptible genotypes, under conditions of photosynthate shortage, relocate dry matter from the stems to the developing grain. Such genotypes may also preferentially partition newly produced photosynthate to the grain (more so than under good conditions) to the detriment of the stem. Chamberlin (1978) examined this hypothesis using the lodging-resistant and lodging-susceptible hybrids DeKalb E57 and Texas610, respectively. He found very little evidence that a water shortage during grain filling resulted in increased mobilization of reserves assimilated prior to anthesis. There was also no indication that stress caused changes in the distribution patterns of current assimilates, favoring the grain at the expense of the stem. He concluded that "lodging seems rather to have resulted from the assimilate supply being too tow under conditions of water shortage during grain filling to provide the substrate for stem maintenance respiration." His results and conclusions strongly support the physiological hypothesis.

Chamberlin (1978) showed in glasshouse experiments that Texas610 lodged more than DeKalb E57, as has also been observed in the field. Although the causes were not clear, there were significant differences. Texas610 had a greater emphasis on grain production when assimilates were partitioned at the expense of the lower stem. Also there was a more gradual depletion of reserves from DeKalb E57 stems during grain filling. These reserves were higher in DeKalb E57 than in Texas610 at anthesis. As pointed out above, this pattern was not altered by water deficit in Chamberlin's test. Once again, however, the general conclusion from this work may be limited in that only two hybrids were tested and the plants were grown in pots in a glasshouse, resulting in a relatively rapid onset of stress.

As previously pointed out, Done and Muchow (1983) observed that while most genotypes were source limiting with respect to grain yield, the majority produced more dry matter during grain filling than was used to fill the grain. Stover dry matter yield at physiological maturity was higher than anthesis dry matter yield. In this experiment, grain yields were high (up to 7.6 t/ha), with littte senescence and no lodging at physiological maturity. Done found in another experiment that low grain yields associated with increased water deficit caused a reduction in the net amount of nongrain (surplus) dry matter produced during grain filling. Senescence and lodging occurred in the highly stressed treatments, but differences in dry matter partitioning could not be distinguished between genotypes susceptible and resistant to lodging. Failure to detect such an effect, however, could be attributed to large experimental errors and does not provide conclusive evidence for its absence.

It can therefore be conjectured that in a healthy, high-yielding sorghum crop, surplus dry matter will be produced during grain filling, and any reduction in this surplus could represent a "stress" situation resulting in senescence and lodging susceptibility. This argument can be extended to suggest that any attempt to genetically improve grain yields by using preanthesis assimilate or by increasing the proportion of assimilate partitioned to grain during grain filling would have the undesirable side effects of increased senescence and susceptibility to lodging.

The Role of Pathogens in Lodging

Stalk rots are often, but not always, associated with lodged stalks. Where stalk rot does occur, it is not clear what effect it has on grain yield and subsequent lodging.

In Australia, there are few reports of detailed surveys to identify the causes of lodging and its association with stalk rots and other factors. The pathogens involved with the characteristic symptoms of stalk rot have been examined, and it has been found that *M. phaseolina* is readily recovered from stalks with blackened piths typical of charcoal rot, while *Fusarium moniliforme* Sheldon is the predominant fungus from stalks with a very dark-red to deep-purple discoloration of the pith tissue (Burgess et al. 1981). These fungi are sometimes present in the same stalk. In addition, *Nigrospora sphaerica* (Sacc.) Mason can occasionally be recovered from discolored stalks, usually where one or both of the other pathogens are present (Mayers, unpublished data; Dodman, unpublished data; Trimboli1981).

Although these fungi are consistently associated with discolored stalks, they often cannot be recovered from lodged stalks that show no discoloration. In central Queensland, G.S. Purss (Department of Primary Industries, Brisbane, Queensland, Australia; personal communication) examined the stalks of a number of grain sorghum genotypes and found that although pith disintegration had occurred, *M. phaseolina* was rarely present and no other organism could consistently be recovered. Similarly, Henzell and Gillieron (1973) reported that charcoal rot was rarely seen in plants that were dead at the base. From surveys in New South Wales in 1978 and 1979, Trimboli (1981) reported that no fungi were isolated from non-lodged stalks exhibiting a hard, dry, brittle stalk syndrome (the frequency of occurrence of such stalks was not indicated). Severe water deficits occurred in 1982/83 in southern Queensland, and lodging was widespread. Most lodged stalks showed no discoloration, although the basal internodes were shrivelled and collapsed. *F. moniliforme* was the predominant fungus recovered, but it could be isolated from only about half of these stalks.

In contrast to these reports, Mayers found a high incidence of stalk rot in a detailed examination of a crop at Brookstead in southern Queensland in 1977/78. A sample of 1400 stalks was collected from randomly located quadrats and examined for symptoms of stalk rot. More than 80% of these stalks showed typical symptoms of invasion by *F. moniliforme,* and Mayers estimated that between 20 and 50% would have lodged before harvest.

Trimboli (1981) reported that *F. moniliforme* was the fungus most commonly found in lodged sorghum stalks in New South Wales. *M. phaseolina* and *N. sphaerica* were isolated much less frequently, and he concluded that they probably play a minor role in stalk rot development in the regions surveyed. However, there were indications that *M. phaseolina* occurred more often in areas of lighter soils. No information was provided on the amount of lodging or the proportion of lodged stalks with symptoms of stalk rot.

It is apparent from these observations that infection with stalk rot pathogens and symptoms of invasion are not always associated with lodging. However, the frequency of lodging with and without stalk rot and that of stalk rot without lodging has not been defined. Surveys should be conducted over several seasons to clarify the situation.

Recent research in Queensland by Mayers (from 1978-1981) and Dodman since 1981 has aimed at clarifying some of these issues by manipulating environmental conditions and inoculum levels of soilborne pathogens. The importance of soil moisture was studied in plots receiving adequate rainfall or irrigation and in plots where rainfall was excluded with plastic-covered shelters. The role of pathogens was examined in untreated plots and plots fumigated with the granular fumigant dazomet (tetrahydro-3,5-dimethyl-2H-1,3,5-thiadiazine-2-thione).

Our trials at Hermitage in southern Queensland in 1978/79 and 1980/81 showed that stalk rot developed only when the pathogen *F. moniliforme* was present in the soil and when this was accompanied by water deficits (Table 4). Fumigation

Table 4. The effect of moisture regime and soil fumigation on the incidence (%) of fusarium stalk rot In two sorghum hybrids at Hermitage, Queensland, in 1978/79.

		Moisture regime		
Soil treatment	Cultivar	Natural rainfall (low stress)	Rain excluded (high stress) [a]	Mean
Not fumigated:	Tropic	6.5	69.0	28.1
	DeKalb E57	4.0	33.0	
	Mean	5.2	51.0	
Fumigated:	Tropic	3.0	3.3	
	DeKalb E57	0.5	0.0	1.7
	Mean	1.7	1.6	
Mean		3.5	26.3	

a. Rainfall was excluded with a rain-out shelter erected 5 weeks after planting; symptoms of water deficit (wilting and senescence of leaves) developed soon after anthesis.

reduced both the inoculum level and the incidence of stalk rot even where a severe water deficit occurred, whereas no stalk rot developed in the absence of a water deficit. It was found that water deficits reduced grain yield by 36% through effects on both single grain weight and grain number. Stalk rot produced an additional yield loss of 6.5% by reductions in single grain weight. Where lodging occurs in commercial production, losses can be much greater due to harvesting problems.

In the 1981 /82 and 1982/83 growing seasons we extended our research to an examination of the susceptibility of a wide range of genotypes. Despite the imposition of severe water deficits through the use of rain-out shelters, disease development was lower than in the earlier work, particularly with the early-maturing genotypes. Although disease levels were low in 1982/83, the incidence of severe stalk rot in seven genotypes of medium maturity ranged from 0 to 40% (stalks with severe stalk rot have more than three diseased internodes). No relationship was found between reaction to stalk rot and a standability rating obtained from observations of hybrid-evaluation trials at many sites over several seasons.

Although water deficits are usually associated with lack of rainfall, the inability of plants to absorb soil moisture due to poor root systems is often a contributing factor. Root rots caused by fungal pathogens can develop soon after seed germination and continue to affect the roots during all stages of plant development. Severe destruction of crown (or prop) roots is often seen around anthesis and may restrict water uptake, even where soil moisture reserves are adequate for crop growth. Trimboli (1981) indicated that lesions on crown roots are initially small (0.5-4 mm), dark-red to purple, and usually restricted to one side of the cortex. These enlarge, girdling the root and extending along it for several centimeters. On large crown roots the cortex may eventually slough away and the necrosis may extend into the stele. He found that *F. moniliforme* and *Periconia circinata* (Mangin) Sacc. were the fungi most commonly recovered from such lesions. Similar observations were previously reported from Queensland (Mayers 1976).

Our research carried out in Queensland supports the hypothesis that pathogens invade stalk tissue that has been predisposed by a physiological stress. In Queensland the major cause of such stress is a plant moisture deficit associated with inadequate supplies of soil water and inability to absorb water because of root rots. At present it has not been resolved whether physiological stress or stalk rot is the main cause of lodging. It seems probable that there is one type of lodging associated with severe moisture deficits and little stalk rot, and another where there is a close association between physiological stress and pathogens.

Breeding for Resistance to Lodging and Stalk Rots

Variation in the Characters

Progress in selection for stalk rot and lodging resistance, or indeed any character, is dependent upon the presence of genetic variation for that

character. Fortunately, large genetic differences exist in sorghum for lodging caused by water deficits. The data in Table 5 illustrate the range of variation available. Unfortunately, most genotypes are susceptible. Table 6 lists some commonly used genotypes and their lodging reaction in Australia. Of these, KS19 is outstanding. It is a selection by W.M. Ross (University of Nebraska, USA) from a cross between CK-60 and Short Kaura made by O.J. Webster (Professor Emeritus, University of Arizona, Tucson, USA). KS19 is one parent in the pedigree of QL10, QL11, QL12, QL25, and QL27 (Table 6). We have not yet tested genotypes such as SC35-6, SC56-6, SC56-14, and SC599-6, reported as resistant in Texas (Rosenow 1977).

Information on resistance to stalk rots caused by *F. moniliforme* and *M. phaseolina* is less conclusive. Variation in stalk rot severity has been found, but the presence of inherent differences in resistance to the pathogen(s) has not been proved. Variations may have been caused by differences in the predisposing physiological stress because of differences in maturity. The data in Table 4 show that DeKalb E57, a lodging-resistant hybrid, had a lower level of disease than did Tropic, a lodging-susceptible hybrid. However, DeKalb E57 flowers earlier than Tropic and probably experienced less severe water deficits than Tropic in this test.

Table 5. Percentage of lodged plants, leaf senescence, grain yield, and days to flower of some grain sorghum genotypes at Hermitage, Queensland, in 1982/83.

Genotype	Lodged plants[a] (%)	Leaf nonsenescence[b]	Yield (t/ha)	Days to flower
NK150	85	9.7	3.85	68
Texas610SR	71	8.6	3.53	72
ATx624/RTx430	69	8.0	3.73	79
DK55	68	8.6	3.79	74
AKS4/KS19	22	8.0	3.39	73
ATx624/QL10	15	8.3	3.81	75
Goldrush	8	8.3	4.31	77
AKS4/QL12	6	7.7	3.97	75
DeKalb E57	5	5.7	3.84	82
Pride	2	7.0	5.16	79
A378/QL12	0	5.4	4.78	80
Dorado	0	6.0	3.90	84

a. Severe water deficits occurred during grain filling; lodged plants showed no visible symptoms of stalk rot.

b. Leaf nonsenescence ratings: 1 = all leaves green, 10 = all leaves dead. Ratings were made 105 days after planting.

Table 6. Lodging reaction in Australia of some sorghum genotypes, obtained over years and sites.

Genotype	Lodging reaction[a]	Genotype	Lodging reaction
KS19	R	RTx2536	VS
QL10	R	RTx430	VS
QL11	R	IS2816C(SC120C)	VS
QL12	R	IS12608C(SC108C)	VS
QL25	R	IS12664C(SC173C)	VS
QL27	R	RTx7000	VS
B399	MS	NM31	VS
SC170C-6-8-4	S	TAM428	VS
BTx3197	S	BTX3042	VS
RTx7078	VS	BTx378	VS
TAM422	VS	BTx622,623,624	VS

a. R = resistant; MS = moderately susceptible; S = susceptible,; VS = very susceptible.

Table 7. Percent incidence of macrophomina (M) and fusarium (F) stalk rot in nine sorghum hybrids grown over 2 years under water deficit in a rain-out shelter at Emerald, Queensland. (Source: G.D. Reefer and P.E. Mayers, Department of Primary Industries, Emerald, Queensland, Australia).

Hybrid	Trial 1		Trial 2		M/F resistance indicated[a]	Lodging resistance[b]
	M	F	M	F		
Goldrush	56	66	8	31		s
SM8	NA[c]	NA	31	39	R/S	R
AKS4/KS19	29	72	NA	NA	R/HS	HR
DeKalb E57	29	84	32	73	R/HS	HR
Sundowner	NA	NA	57	80	S/HS	R
DeKalb F64a	66	59	NA	NA	S/S	R
Texas610SR	84	52	68	65	HS/S	HS
Goldfinger	90	44	NA	NA	HS/S	HS
Dorado	90	41	65	54	HS/S	S

a. HR = highly resistant; R = resistant; S = susceptible; HS = highly susceptible.

b. Classification based on long-term field tests.

c. Not available.

G.D. Keefer (Department of Primary Industries, Emerald, Queensland, Australia) and Mayers have more positive evidence of the existence of *M. phaseolina* resistance genes since differences in disease incidence went across maturity classes. The data shown in Table 7 suggest that reaction of genotypes to the two pathogens may be inherited independently and that there may be a correlation between lodging resistance (as measured in long-term field tests) and resistance to *M. phaseolina,* but not to *F. moniliforme.*

Selection for the Characters

Our discussion on the causes of lodging and stalk rots clearly implicates the source-sink relationships of grain growth in their occurrence. It is essential that this be kept firmly in mind when selecting for resistance to them. For example, because of their apparent effect on the source-sink relationship, characters such as grain yield and maturity, particularly, must be considered. Plants with a high grain yield and early maturity are more likely to be source limited and therefore susceptible to the lodging and stalk rot syndrome. A consideration of lodging and stalk rot resistance alone would probably result in the selection of late-flowering, low-yielding genotypes. This would be particularly so if high grain yield was due to greater partitioning of dry matter to the grain (i.e., increased harvest index), rather than to an overall increase in the biological yield of the plant. Maturity differences are also important because of the influence they may have on severity of water deficits experienced by genotypes in different maturity classes.

Most breeding programs in Australia take a similar approach to evaluating lodging resistance. That is, hybrids rather than inbred lines are evaluated because the low grain yield of inbreds tends to make them lodging resistant. The genotypes under test are grown at a number of sites at which grain yield, maturity, nonsenescence, and lodging are measured if differences are expressed. Several sites are used to increase the chances of encountering lodging conditions. Then subjective selection is made for resistance to lodging within maturity and yield classes.

Often lodging does not occur, yet differences in rate of leaf and plant death (senescence) are expressed. Selection is then made for nonsenescence, again within maturity and grain yield classes. Evidence in Australia is similar to that reported by Rosenow (1977) in Texas: a significant positive association exists between nonsenescence and lodging resistance (Tables 8,9, and 10). This association is expected because plants die before they lodge, What is surprising is that the correlation coefficients are not higher. It seems other factors besides senescence rate are influencing lodging. Tables 8, 9, and 10 show the correlation matrices of lodging with some other factors, including yield, days to flower, and height at Hermitage in 1982 and 1983 and at Banana in

Table 8. Correlation coefficients between five characters in 81 hybrids at Hermitage, Queensland, in 1981/82.

Character	Grain yield	Days to flower	Non-senescence[a]	% lodging at 18 Feb 1983	% lodging at 28 Feb 1983
Grain yield					
Days to flower	-0.04				
Nonsenescence	-0.02	-0.82**			
% lodging on 18 Feb 1982	-0.18	-0.66**	0.56**		
% lodging on 28 Feb 1982	-0.16	-0.71**	0.64**	0.94**	

a. Leaf nonsenescence ratings: 1 = all leaves green, 10 = all leaves dead.
**P<001.

Table 9. Correlation coefficients between five characters in 72 hybrids at Hermitage, Queensland, in 1982/83.

Character	Grain yield	Days to flower	Non-senescence[a]	% lodging at 11 Feb 1983	% lodging at 18 Feb 1983
Grain yield					
Days to flower	-0.28				
Nonsenescence	0.10	-0.86**			
% lodging on 11 Feb 1983	-0.01	-0.36**	0.42**		
% lodging on 18 Feb 1983	-0.08	-0.46**	0.53**	0.96**	

a. Leaf nonsenescence ratings: 1 = all leaves green, 10 = all leaves dead.
**P<0.01.

Table 10. Correlation coefficients between five characters in 28 hybrids at Banana, Queensland, in 1979/80.

Character	Grain yield	Days to flower	Height	Non-senescence[a]	Lodging (%)
Grain yield					
Days to flower	-0.39*				
Height	0.19	0.07			
Nonsenescence	0.10	-0.18	-0.15		
% lodging	0.10	-0.50**	0.20	0.44*	

a. Leaf nonsenescence ratings; 1 = all leaves green, 10 = all leaves dead.
**P<0.01.
* P<0.05.

1980. Multiple regression analysis (lodging at 18 Feb 1983 and at 11 Feb 1982 was not included) indicated that the variation in lodging caused by other factors and nonsenescence was 55,30, and 50% at the three sites, respectively. The other factors certainly explain more of the variation in lodging than can be accounted for by nonsenescence alone, but there is still significant variation unaccounted for; this is an obvious area for research.

Because by far the most prevalent type of lodging in Australia is expressed only in plants that undergo water deficits during grain filling, some control over the environment is desirable. Rain-out shelters have been used to study specific aspects of lodging and stalk rots, but they are obviously of limited use in a breeding program. Very effective use has been made, however, of a dry-season (winter) planting in tropical Australia. This environment has an extremely predictable dry season, and lodging induced by water deficits can readily be achieved by manipulating irrigation.

Done, working in such an "abnormal" environment at the Kimberley Research Station near Kununurra in Western Australia, has found a high correlation (from 0.71 to 0.87) between the lodging scores during three seasons at Kununurra and the scores obtained in "normal" summer plantings in Queensland. This correlation was not necessarily

expected because of the different environmental conditions encountered in the winter dry season and in the summer of Queensland, the latter being the normal growing period for grain sorghum in Australia. Low grain yields are a feature of such out-of-season plantings with restricted irrigation, but it seems the source-sink relationships have not been altered substantially, as evidenced by harvest index values in excess of 0.50. Such an environment may in fact be superior to normal summer plantings in determining inherent differences in lodging resistance because maturity differences between genotypes are reduced by the short winter days. It is interesting that the correlation between lodging and days to flower in a test at Kununurra was only -0.27, whereas it is usually higher in the summer in Queensland (Tables 8, 9, and 10). The predictability of the environment at Kununurra is reflected in the high correlations (0.71-0.84) obtained between scores in different years. Done's results exclude particular genotypes that in some seasons fail to flower and do not senesce or exhibit stem lodging.

Very little direct screening for resistance to stalk rot pathogens is practiced in Australia. Some workers doubt its usefulness. Brengman and Dodman have had very limited success with toothpick inoculation with *M. phaseolina* because of maturity differences in genotypes and the unpredictable climate. Brian Hare (Pacific Seeds, Toowoomba, Queensland, Australia; personal communication) is attempting to establish a reliable procedure for screening for resistance to stalk-rotting pathogens. To this end he is looking at pathogenicity tests (to ensure organisms are causal agents), conditions for infection, and conditions in the plant for disease development.

Recommendations for Future Research

1. More information is needed on the cause(s) of genetic variation in lodging resistance and stalk rot resistance. Consideration should be given to how such information may be utilized in a breeding program. For example, differences in physiological responses to water deficits need to be examined. Differences in osmotic adjustment have been implicated by Wright (1981), and dry matter partitioning during grain filling may be of considerable importance.
2. More information is needed on the role of pathogens in the type of lodging caused by water deficits. For example, surveys could be conducted over several seasons to establish this role.
3. If it is established that pathogens are involved, then tests need to be devised for reliably identifying genetic differences in resistance.

References

BROWN, R.F. 1978. Environmental effects on panicle development in grain sorghum (*Sorghum bicolor* (L. Moench)). Ph.D. thesis, University of Queensland, Australia.

BURGESS, L.W., DODMAN, R.L., PONT, W., and MAYERS, P.E. 1981. Fusarium diseases of wheat, maize and grain sorghum in Eastern Australia. Pages 64-76 *in* Fusarium: diseases, biology and taxonomy (eds. P.E. Nelson, T.A. Toussoun, and R.J. Cook). University Park, PA 16802, USA: Pennsylvania State University Press.

CHAMBERLIN, R.J. 1978. The physiology of lodging of grain sorghum (*Sorghum bicolor* L Moench). Ph.D. thesis, University of Queensland, Australia.

DONE, A.A., and MUCHOW, R.C. [1983]. Yield limitations in a range of inbred and F_1 hybrid cultivars of *Sorghum bicolor* (L Moench). Australian Journal of Agricultural Research (in press).

HENZELL, R.G., and GILLIERON, W. 1973. Effect of partial and complete panicle removal on the rate of death of some *Sorghum bicolor* genotypes under moisture stress. Queensland Journal of Agricultural and Animal Sciences 30:291-299.

MAYERS, P.E. 1976. The first recordings of Milo disease and *Periconia circinata* on sorghums in Australia. Australian Plant Pathology Society Newsletter 5:59-60.

MUCHOW, R.C., and WILSON, G.L. 1976. Photosynthetic and storage limitations to yield in *Sorghum bicolor* (L Moench). Australian Journal of Agricultural Research 27:489-500.

ROSENOW, D.T. 1977. Breeding for lodging resistance in sorghum. Pages 171-185 *in* Proceedings of the 32nd Annual Corn and Sorghum Research Conference. Washington, D.C., USA: American Seed Trade Association.

TRIMBOLI, D.S. 1981. The fungi associated with stalk and root rot of grain sorghum in New South Wales. M.Sc. Agriculture thesis, University of Sydney, Australia.

WRIGHT, G.C. 1981. Adaptation of grain sorghum to drought stress. Ph.D. thesis, University of New England. Armidale, Australia.

Questions

Maranville:
I am confused about source/sink definition. What is source and what is sink? Can you quantify this, and what are the units? I don't think grains after physiological maturity can be a sink, so they couldn't be used as a factor in calculation. Do you agree or disagree? Why?

Henzell:
Join the club. However, we talk specifically about the source/sink ratio during grain filling. Under those conditions the sink is almost entirely the developing grain. A limited amount of experimentation suggests that most, if not all, the dry matter produced during grain filling ends up in the grain. The source we talk about is the nongrain part of the plant. In sorghum it appears as though the major component of this source is the leaves and not the stems, roots, glumes, etc., even under moisture stress conditions.

Odvody:
There seemed to be no pathogens isolated from dead stalks. Is it possible that root death due to pathogens was responsible, in part, for plant death, although there was no progression to stalk tissue?

Henzell:
Yes, it is possible. In many cases only stems were examined.

Odvody:
Based on your diagram of the physiological stress hypothesis, do you feel that all of the stalk and root rot occurrence is due to colonization of dead cells (i.e., strictly a saprophytic process)?

Henzell:
I don't really know. However, at least with *Fusarium moniliforme* infection the fact that anthocyanin production occurs would indicate to me that the cells are not entirely dead.

Control of Sorghum Root and Stalk Rots

Summary and Synthesis I

R.W. Schneider*

Biological and Cultural Control

Biological Control

For the purposes of this review, biological control is defined as the suppression of disease or inoculum density of the pathogen by an introduced biological agent. Two strategies for biological control may be employed: The agent must be effective against the pathogen apart from the host, or the agent must protect at the site of infection.

Apart from the Host

A review of the extensive literature on this aspect of biocontrol (Baker and Cook 1982, Cook and Baker 1983) indicates that there is no precedent for success in controlling soilborne plant pathogens except under highly artificial conditions. However, foliar pathogens are amenable to control by this strategy. The primary difference between these two classes of pathogens is that in the soil relatively high populations of the introduced agent must be maintained tor extended periods of time. Because the soil is well buffered with respect to abrupt changes in microbial constituents (Baker and Cook 1982), it is unlikely that an introduced agent will become established and maintain a population sufficiently large to eliminate target pathogens. Furthermore, stalk and root rots of sorghum are caused by several pathogens that are effective saprophytic competitors, and they may infect the plant anytime during the season (Reed et. al. 1983).

At the Site of Infection

In this case, high populations of the biocontrol agent need not be maintained. Rather, the agent must be capable of propagating itself as a root parasite or be an effective rhizosphere competitor.

There are numerous success stories with this type of biocontrol (Cook and Baker 1983). Seedling diseases are particularly amenable to control by this strategy because the seeds or other propagative material can be coated with the agent. The introduced organism need only colonize the emerging roots for a short period of time. Actinomycetes, *Trichoderma,* other fungi, and certain bacteria have been successfully tested against *Pythium, Fusarium, Rhizoctonia,* and other pathogens (Cook and Baker 1983).

In the case of root and stalk rot of sorghum, roots may become infected weeks or months after planting. This requires that the introduced agent must be able to grow with the developing root system tor an indefinite period of time. Weller (1983) recently demonstrated that an introduced pseudomonad, which was antagonistic to *Gaeumannomyces gra~minis on* wheat, colonized and displaced the native microflora from field-grown wheat roots for the duration of the season. This represents a signifi-

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cant advance toward the eventual use of biocontrol agents for soilborne plant pathogens.

Disease Suppressive Soils and Cultural Control

Suppressive Soils

Disease suppressive soils are those that suppress specific diseases even though the pathogen and susceptible host may be present (Schneider 1982). The suppressive agent(s) may be biological or physical/chemical.

Perhaps the most studied example of this phenomenon is take-all of wheat (Schippers and Gams 1979), In this case disease suppression has been attributed to, among other things, the competitive exclusion of virulent strains of the pathogen by avirulent strains, the development of populations of pseudomonads that suppress the pathogen, and changes in certain soil chemical characteristics that suppress disease development. Any one or all of these and other factors may be responsible for the decline of take-all. Elucidation of one or more mechanisms could lead to an effective disease control strategy in which disease suppression could be induced at will.

Such a phenomenon has not been described for root and stalk rots of sorghum. Perhaps these diseases are not amenable to suppression because of the diverse pathogens involved. However, pathologists must be alert to the possibility and investigate well-documented cases of disease remission over time or the lack of disease development in certain areas.

Cultural Control

Cultural control is defined as a reduction in disease incidence or severity by a specific cultural or agronomic practice, even though a susceptible host is used. Agronomic practices may include altered irrigation schedules the use of specific plant nutrients and forms of nitrogen, and crop rotations.

Ecofallow, as developed by Doupnik and Boosalis (1980) and Doupnik et al. (1975), is particularly relevant to root and stalk rot diseases of sorghum. A combination of reduced tillage and a specific crop rotation resulted in significantly less stalk rot in grain sorghum than with conventional agronomic practices. The cause of this reduction in disease incidence is thought to be related to physical and biological factors.

Breeding for Resistance

Programs, philosophies, and strategies in breeding for resistance to root and stalk rots were ably reviewed by authors in these proceedings. However, the difficulty in working with root and stalk rots of mature plants should be reemphasized. Not only is it impossible to grow a representative mature plant under controlled greenhouse conditions, but the task is made even more difficult by the fact that these diseases occur only in senescing plants.

The breeding program of Henzell et al. (these proceedings) takes account of this fact and incorporates novel means of measuring senescence potential. They also screen their entries under environmental conditions that accelerate senescence, namely high temperature and water deficits. Interestingly, they are more concerned with senescence-induced lodging than with root and stalk rot and consider the disease to be secondary to the physiological problem.

The physiology and biochemistry of senescence are not completely understood. Thus, factors that affect this process (Thomas and Stoddart 1980), including growth regulators, source:sink ratios, drought stress, and the environment, cannot be reproducibly imposed. Differences in senescence may account for the high variability over time and space in disease severity following inoculation of sorghum with various root- and stalk-rotting organisms.

Assimilate Partitioning

Another topic that has been discussed in these proceedings is assimilate partitioning, or harvest index (HI). It is generally accepted that domestication and selection of modern varieties of crop plants have not resulted in increased rates of CO_2 exchange per unit leaf area; rather increased yields have come primarily from changes in HI (Hanson 1979, Gifford and Evans 1981). This is known to be true for maize, sorghum, pearl millet, and numerous other crops. Therefore past improvements in yield have been derived largely by affecting the proportion of dry weight accumulated in the harvested organ. HI is thus genetically controlled. It appears that there is an optimum value for HI for any pro-

duction environment, and today's elite genotypes are often close to this optimum.

In the case of sorghum, roots and stalks may serve as sources of assimilates during periods of rapid grain fill or stress-induced decreases in rate of photosynthesis. Thus, the very process that may account for a high HI, namely accelerated senescence, also conditions the plant to susceptibility. Furthermore, stresses of various types, such as nutritional, water, and biotic, are known to accelerate the senescence process (Beevers 1976, Schneider and Pendery 1983, Thomas and Stoddart 1980).

As discussed by Rosenow and Henzell et al. in these proceedings, senescence and susceptibility may be inextricably related such that one would have to accept a slightly lower HI in order to maintain juvenility and resistance in the roots and stalks. Yet, in one recent review article (Evans and Wardlaw 1976) it was stated that assimilates remaining in the vegetative organs represent unused yield potential and should be diverted genetically to the grain. However, we know very little about the potential biological yield of wild relatives. Of course, the sacrifices one would be willing to make in terms of HI would depend on the area in which the crop is to be grown. Factors to be considered include the probability of a water deficit, anticipated availability of nutrients at the proper time, and pressure from other pests, which may induce senescence.

Because of the relationship between HI and senescence, several breeding programs have been developed to assess the quantity of reserve assimilates. A small-grains breeding program is now being implemented in which plants are chemically defoliated at a specific physiological age (Blum et al. 1983). Grain fill is then measured and compared to nondefoliated controls. A similar program is being used to screen for septoria leaf blotch in wheat (Zilberstein et al. 1984). Previous authors in these proceedings described other approaches. Thus, varieties can be selected for areas in which a high level of reserve assimilates can be made available during periods of accelerated senescence.

Perhaps a topic that should be explored in much greater depth is the potential for increasing biological yield. This could be done by measuring the CO_2 exchange rate in wild relatives and incorporating any superiority in this trait into cultivars with an acceptable HI. This probably represents a long-term breeding commitment, but the results should justify the effort.

Another area that may be worthy of investigation concerns the induction of senescence by exogenously applied growth regulators. This would provide a means of testing resistance during advanced stages of senescence without the complications of a nonreproducible adverse environment Promoters of senescence include abscisic acid and ethylene (Beevers 1976). Ethephon, a commercially available precursor of ethylene, can be sprayed on plants. This product is used to promote uniform ripening of fruits.

Chemical Control

Williams and Nickel (these proceedings) thoroughly reviewed the strategies and potential uses of traditional fungicides. However, in light of what we know about the relationships between senescence and susceptibility, and between drought stress and predisposition, it is worthwhile to examine other possibilities related to the use of chemicals that affect the host rather than the pathogen. This is particularly important with stalk rots because the pathogen is active in dead tissues (Pappelis, these proceedings), sites which may be inaccessible to fungicides.

One of the natural causes of physiological aging and senescence is a decreased supply of cytokin--s from the root to the shoot (Thomas and Stoddart 1980). Kinetin, one of the cytokinins, is the most effective senescence-retarding growth regulators known (Beevers 1976). Is it possible to apply such a compound to retard the onset of senescence and susceptibility? What would be the cost in terms of HI? There are many questions to be answered, but certainly this topic is deserving of a major research effort.

Furthermore, one of the metabolic breakdown products of benzimidazole fungicides is a kinetin-like compound that retards senescence (Wang et al. 1960). Thus, it may be possible to synthesize a compound that is both fungicidal and effective as a growth regulator.

Finally, work done with Cyclocel or CCC (2-chloroethyl trimethyl-ammonium chloride) should be mentioned. This material is used in the ornamentals industry to induce short, thick stems in shrubs. When applied to leaves of cereals, it causes the stalks to become shorter and thicker and thereby more resistant to breaking and lodging (Nilsson 1969). There are no reports of this material being used to control lodging associated with stalk rot of sorghum.

References

BAKER, K.F., and COOK, R.J. 1982. Biological control of plant pathogens. San Francisco, California, USA: W.H. Freeman. 433 pp.

BEEVERS, L. 1976. Senescence. Pages 771 -794 *in* Plant biochemistry (eds. J. Bonner and J.E Varner). New York, New York, USA: Academic Press. 925 pp.

BLUM, A., MAYER, J., and GOZLAN, G. 1983. Chemical desiccation of wheat plants as a simulator of post-anthesis stress: II. Relations to drought stress. Field Crops Research 6:147-155.

COOK, R.J., and BAKER, K.F. 1983. The nature and practice of biological control. St. Paul, Minnesota, USA: American Phytopathological Society. 550 pp.

DOUPNIK, B., and BOOSALIS, M.G. 1980. Ecofallow—a reduced tillage system—and plant diseases. Plant Disease 64:31-35.

DOUPNIK, G., BOOSALIS, M.G., WICKS, G.A., and SMIKA, D. 1975. Ecofallow reduces stalk rot in grain sorghum. Phytopathology 65:1021 -1022.

EVANS, L.T., and WARDLAW, I.F. 1976. Aspects of the comparative physiology of grain yield in cereals. Advances in Agronomy 28:301 -359.

GIFFORD, R.M., and EVANS, C.T. 1981. Photosynthesis, carbon partitioning and yield. Annual Review of Plant Physiology 32:485-509.

HANSON, A. 1979. Plant breeding and partitioning in cereals and grain legumes. Pages 18-23 *in* Partitioning of assimilates. Rockville, Maryland, USA: American Society of Plant Physiology. 23 pp.

NILSSON, H.E. 1969. Studies of root and foot rot diseases of cereals and grasses. Annals of the Agricultural College of Sweden 35:275-807.

REED, J.E., PARTRIDGE, J.E., and NORDQUIST, P.T. 1983. Fungal colonization of stalks and roots of grain sorghum during the growing season. Plant Disease 67:417-420.

SCHIPPERS, B., and GAMS, W. (eds.). 1979. Soil-borne plant pathogens. San Francisco, California, USA: Academic Press. 686 pp.

SCHNEIDER, R.W. (ed.). 1982. Suppressive soils and plant disease. St. Paul, Minnesota, USA: American Phytopathological Society. 88 pp.

SCHNEIDER, R.W., and PENDERY, W.E. 1983. Stalk rot of corn: Mechanism of predisposition by an early-season water stress. Phytopathology 73:863-871.

THOMAS, H., and STODDART, J.L. 1980. Leaf senescence. Annual Review of Plant Physiology 31:83-111.

WANG, D., HAO, M.S., and WAYGOOD, E.R. 1960. The effect of benzimidazole on the biosynthesis of chlorophyll. Biochemical Research Communications 2:92-101.

WELLER, D.M. 1983. Colonization of wheat roots by a fluorescent pseudomonad suppressive to take-all. Phytopathology 73:1548-1553.

ZILBERSTEIN, M., BLUM, A., and EYAL, Z. [1984.] Chemical desiccation of wheat plants as a simulator of post anthesis Septoria leaf blotch stress. Phytopathology 74:(in press).

Control of Sorghum Root and Stalk Rots

Summary and Synthesis II

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This commentary is based on the three breeders' papers presented by Drs Rosenow, Maunder, and Henzell.

The summary comments and generalizations we have read in these three papers represent extensive field experience spanning the past two decades in Australia, North America, and South America. In spite of the sharply contrasting environments in which these scientists have worked, there is a remarkable similarity in their breeding strategies, field screening, and selection criteria.

Major Points

All three breeders have had to wrestle with the following problems:

1. identification of what resistance is desired—charcoal rot, fusarium root rot, lodging, drought, or all at once;
2. lack of reliable field screening techniques for identifying sources and derivatives of heritable resistance;
3. identification of plant characters that either impart or indicate resistance.

The three authors are interested in breeding out yield limiters (stalk rots, lodging, or drought susceptibility) at the postfloral stage. Henzell reiterated several times that lodging ("stem collapse") is the ultimate effect of stalk rots and drought in Australia. Therefore he breeds directly for lodging resistance and thus indirectly for stalk rot resistance. Rosenow (personal communication, 1983) has come to the same conclusion. He no longer cuts stems to verify the presence of charcoal rot sclerotia. He takes lodging scores. Although Maunder places due importance on standability, he identifies separate sources of charcoal rot, fusarium root rot, and drought resistance. He takes charcoal rot measurements even on standing plants.

These authors agreed that reliable field screening is difficult. Consequently, all three breeders make multilocational plantings with large numbers of entries under a range of growing conditions. They are all interested in locations with disease or drought occurrence. In at least some of their nurseries they try to create a boom and bust situation with high plant populations, high fertility, and optimum irrigation, followed by postfloral heat and moisture stress. In Australia, an off-season location (at the Kimberley Research Station) has been found that, under irrigation manipulation, can accurately predict the lodging performance of main-season location nurseries. Excellent fusarium root rot "hot spots" have been identified in Argentina. Rosenow has made considerable progress by making lodging scores in nurseries left standing over winter.

Plant maturity differences can confound lodging and stalk rot response. If stress occurs too soon before or after flowering then stalk rots or stem collapse may not occur even in susceptible sorghums. To overcome the problem of plant maturity, these authors try to group their materials according to maturity and base their decisions on large numbers of multilocational observations.

Maunder and Rosenow make use of the tooth-

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pick method of charcoal rot inoculation in at least some replications of some nurseries. However, Henzell has found little success with that practice in Australia.

Nonsenescence was identified by all three authors to be the single most important plant character indicative of stalk rot, lodging, and postfloral drought resistance. Maunder and Rosenow relate nonsenescence closely with drought resistance. Henzell and Rosenow point out the significant association between nonsenescence and disease resistance. In the case of SC-599-6 nonsenescence is also linked with fusarium root rot resistance. Nonsenescence is used as a selection criterion per se by all three breeders.

The stiff stalk character is emphasized by Maunder and Henzell for both charcoal rot and lodging resistance. The lodging-resistant lines SC-56-6 and NSA 663 have been described by Rosenow as having an elastic stalk.

Short stature was related to lodging and stalk rot resistance. Maunder proposes that the shortened intemodes slow down stalk rot development in the stem.

Late maturity was also related to stalk rot and lodging resistance by Henzell and Rosenow. Henzell insists that the reason for late maturity resistance is due to a happy source-sink balance.

Henzell and his colleagues place considerable importance on the role of the source-sink equilibrium during grain fill. They propose that stem collapse during grain fill is due primarily to source limitations and that the problem of stalk rots and lodging can be best understood through source-sink dynamics.

Additional Information

Some recent observations of Malian local sorghums may shed additional light on the foregoing summary:

During the past 5 years we have made extensive multilocational observations of local varieties, introduced varieties and hybrids, and local x introduced hybrids. White-seeded exotic hybrids (U.S. and Indian) are generally susceptible to charcoal rot. Durra x exotic hybrids are highly susceptible and Guineense x exotic hybrids are highly resistant.

We have never seen a local Guineense or Guineense x exotic hybrid succumb to charcoal rot. The Guineenses are 3-5 m tall, relatively nonsenescent, and have elastic, dry stems. Under the microscope the cortex cells appear completely empty, In contrast, juicy- and intermediate-juicy-stem sorghums have cortex cells filled with sap.

It isclear that the Guineense stalk rot and lodging resistance is related neither to short stature nor to stiff stalk. Since their grain straw ratio is only about 20%, their resistance may be related to the favorable source-sink balance. However, their resistance may also be related to empty cortex cells. Without a readily available substrate, how can a stalk rot pathogen grow in the pith?

In juicy-stem sorghums, sudden changes of osmotic pressures in the sap during grain fill may cause cortex cell hemorrhage and stem collapse. That eventuality may be prevented by the absence of sap in the cortex cells.

Gaps in Knowledge and Research

1. Very little anatomical work has been done to clearly describe the stems and leaves of resistant vs susceptible varieties or senescent vs nonsenescent varieties. Schertz and Rosenow's article (1977) was a beginning, but only a beginning. These studies should be done with known separate sources of resistance to charcoal rot, fusarium root rot, and lodging. Parallel studies could trace the growth of stalk rot pathogens in stem tissues to accurately identify which tissues are affected.

2. The physiologists need to distinguish between cortex, vascular tissue, and sclerenchyma tissue in describing stem carbohydrate dynamics, so that pathogenic, physiological, and botanical descriptions of the stem can be coherently understood.

3. Nonsenescence needs to be more clearly defined and assessed. Many local Malian durras can show severe leaf firing and be rated highly senescent, yet they make immediate regrowth after late rains. On the other hand, CSH-5 (and 2077A hybrids in general) is nonsenescent when it does not succumb to charcoal rot.

4. A systematic screening of representative groups of the sorghum world collection is needed to identify separate and multiple sources of resistance to charcoal rot, fusarium root rot, lodging, and postfloral drought.

Possible Issues for Discussion

1. Should we breed for stalk rot resistance, lodging resistance, drought resistance, and yield in stepwise fashion or all at once?
2. If a source-sink equilibrium is essential for stalk rot and lodging resistance in stressed environments, are there yield limits for given plant statures and maturities?
3. Are all nonsenescent sorghums stalk rot, lodging, and drought resistant? If not, how does the nonsenescence of susceptible sorghums differ from the nonsenescence of resistant sorghums?

References

SCHERTZ, K.F., and ROSENOW, D.T. 1977. Anatomical variation in stalk internodes of sorghum. Crop Science 17: 628-631.

Discussion

Fungicides

Partridge:
There seems to be an interest in growth regulators, and when these are discussed in reference to chemical control, kinetin and IAA are often mentioned because these compounds are extremely lethal to cell protoplasts and callus tissues in sorghum.

Odvody:
Dr. Williams, since most of the systemic fungicides are acropetally transmitted, do you see any continuing problem in controlling diseases that occur in roots and stalks, and do you think that soil treatments for long-term control and seed treatments for short-term control will basically overcome that?

Williams:
You're probably right. This is a major difficulty. Granular fungicides with a slow release component can perhaps do a very good job in that regard.

Partridge:
What is the potential for using chemicals to study facets of stalk rot?

Williams:
There is plenty that we don't know, but we have fungicides that are specific to *Pythium.* So we could possibly use these chemicals to take apart the etiology and the interrelationships between the implicated fungi We could also use fungicides to try to focus on just when the critical infection occurs, like treating different plants at different times during their growth stage. There are several ways we could use these effective fungicides as research tools to fill gaps that still exist.

Frederiksen:
When we first had to diagnose *Pythium* from dead plants and dead roots without knowing what caused the disease, we used 10 kg/ha of a variety of selected fungicides. We found *Pythium* in the PCNB plots. We eliminated a series of other soil microflora and were able to select out the key pathogen. This approach might be useful in succession research and could give a better understanding of the role of specific organisms in these problems.

Biological and Cultural Control

Seetharama:
Dr. Doupnik, one of the things you suggested was to keep soil moisture at 80% of field capacity at flowering to control stalk rot. How widely can this be applied?

Doupnik:
Moisture conservation will reduce stalk rot incidence regardless of the location.

Rosenow:
You referenced the fact that plant population and not row spacing was important under your conditions. We have some data from Texas that are

somewhat to the contrary. We looked at narrow rows versus wide rows. At yield levels below 3000 kg/ha, we got poorer yield performance when plants were spaced equidistant than when they were planted in 1-meter rows. At low yield levels, plant spacing became important—not just plant population. At low yield levels they performed better in 1-meter rows than in solid equidistant plant spacings. At high yield levels under good moisture conditions, there was some advantage to equidistant spacing. With low yield levels under moisture stress, we were not taking moisture out of the lower soil depths. We had apparently enough competition of rooting in the upper surface that we were not using all the lower-profile moisture. The plants just collapsed and lodged when stress occurred. Recent data from Chillicothe, Texas, show a consistent advantage in the skip-row technique—1-meter rows with two rows planted and one row skipped—over solid plantings of 1 -meter rows with the same number of plants per hectare.

Clark:
To overcome high nitrogen, do you have any recommendations as to the form of N applied or when it is applied that might overcome disease incidence?

Doupnik:
No, we don't have any data. Purdue has some data on maize.

McBee:
From the standpoint of rotations and cultural control, to what extent has allelotrophy been considered along with the effects of pathogens? It has been shown to be a factor in rotations involving monocots and dicots. In sorghum and specific legume rotations, yields have been reduced significantly. Other legumes have no effect and there is a benefit from the residual nitrogen.

Doupnik:
This is a problem in continuous cropping, especially with the same genotype. Herbicide carryover and root exudates may be involved and confound the problem.

Schneider:
When there is residue on the soil surface and the sorghum comes up through this material, is the sorghum stunted for the first 4 weeks?

Doupnik:
There appears to be a slower growth rate and a smaller root system due to cooler soil temperatures.

Schneider:
J.M. Lynch in England [at Agricultural Research Council, Letcombe Laboratory, Oxford] worked on barley in crop residue, and he attributed the stunted plants to the anaerobic or cold-soil degradation of the residue, which results in the accumulation of anaerobic metabolic by-products, such as propionic and lactic acids. The plants are stunted only until these organic acids are metabolized and the soil warms up again.

Doupnik:
By the time the canopy develops, the plants simply do not look that different.

Eastin:
It's soil temperature as it relates to growth. We did some research on screening for plants that grow under cool temperatures. At 15°C the seed will germinate, but the plants don't grow very well and there's a big difference among genotypes. Respiration increases about 15% per degree from 15 to 30°C.

Mukuru:
Dr. Doupnik, you obtained good disease control, and lodging was reduced. Was this due to moisture conservation or because the organisms were reduced?

Doupnik:
We think it was due to soil moisture and soil temperature. Under the high residue, we were storing 7.6 cm more of soil moisture. During the growing season, it takes 22.9 cm of soil moisture to produce the first 62.8 kg/ha of sorghum grain. Every 2.5 cm above that, you can add about 628 kg/ha yield. So in the average years with 35.6-38.1 cm of rain, there's a potential of 3140-3768 kg of grain/ha. If you can conserve an extra 7.6 cm, you can gain an extra 1884 kg/ha. The heat reflection of sunlight off the residue may affect the physiological activity of the plant.

Pappelis:
In the no-till systems, the nodes stay alive at the base of the plants. This may be restricting pathogen penetration of the stalk.

Jordan:
Differences between the ecofallow and conventional systems in between-row soil-temperature can be 6-17°C based on your research. What about the effect of temperature directly in terms of heat stress on the root systems, particularly near the soil surface?

Doupnik:
Both temperature and moisture are important, and interactions may be involved.

Pappelis:
One study involving sandy soils and heat buildup on seedlings in a greenhouse showed a buildup of blight.

Schoeneweiss:
Reflected heat from media surfaces on succulent seedling parts is a problem. I don't know the mechanism involved, but with heat comes a disturbance in water relations—desiccation injury. This might occur on sorghum seedlings.

Pappelis:
Has anyone put a temperature probe on sorghum stalks or roots at the soil line to see how hot it is?

Seetharama:
I found no differences between stalk and soil temperatures, and the leaf sheath may have been a factor.

Schneider:
In a study involving fusarium hypocotyl rot in pine seedlings, soil surface temperature and temperature at the hypocotyl rose to 45°C, predisposing the plant to disease. Control methods included sprinkling, shading, and whitewashing. After 1 hour at cooler temperatures, the tissue was not susceptible.

McBee:
Temperatures in sorghum leaves do fluctuate during flowering and pollination. The temperature may rise during pollination.

Eastin:
Temperature effects must be evaluated on total growth. This may result in infection differences as the plant growth is suppressed under cool temperatures. What temperature has an effect on fungal growth or infection?

Schoeneweiss:
Considerable work on other host-pathogen systems has been reported. Heat shock treatments have been shown to alter the host's resistance. In soybean, leaves placed in a water bath at 50°C for 10 minutes were shown to predispose the host to *Phytophthoca megasperma.* The heat shock suppressed phytoalexin production, and after removal of the leaves from the heat treatment, phytoalexin production was resumed.

Eastin:
Cool soils and high moisture conditions will result in a reduction of plant growth, as it's more difficult for pathogens to invade roots in cool and wet soils.

Schoeneweiss:
The range of temperatures for growth is highly variable among pathogenic organisms. *Fusarium* is active at high temperature and *Verticillium* is more active at low temperature. Most will grow at a reasonable rate at moderate temperatures (18-20°C). At 15°C or below, they will grow more slowly.

Odvody:
The optimum temperature for *Macrophomina* is 35°C. This is not necessarily the best temperature under soil conditions, as it can't grow at lower temperatures due to soil microflora, host susceptibility, etc.

Maranville:
Mulching conserves heat during the warmer portions of the season, and mulched soils are warmer throughout the fall. This may create ideal conditions for disease development.

Breeding for Resistance

Pappelis:
Are breeding plots being grown under no-till or mulched conditions?

Eastin:
A limited amount is being grown under these conditions.

Rosenow:
We started using no-till plots in our dryland breeding program last year.

Doupnik:
Paul Nordquist routinely conducts screening pro-

grams under no-till conditions at the North Platte, Station near North Platte Nebraska. Variety testing is also conducted by Russell Moomaw under no-till at the Northeast Station near Concord, Nebraska.

Pappelis:
More corn [maize] borer tunnels were noted in maize plants grown under mulched conditions. No differences were noted in pith; however the rind characteristics of these plants were changed.

Rosenow:
In Texas, Southwestern corn [maize] borer populations are reduced by tillage that exposes the overwintering larvae to moisture and temperature fluctuations.

Partridge:
Dr. Maunder, are DeKalb-Pfizer's breeding programs designed for maximum yields or consistent yields?

Maunder:
Maximum yield is difficult to define. We strive for a continuity of maximum yield over several years. The hybrid C-46 was not a maximum-yield type but had a consistent yield over 5 to 10 years.

Partridge:
Are there different materials for different areas?

Maunder:
There are different breeding approaches for each target area. Each genotype will be tested in each environment. What works for one thing may not work for another.

Vidyabhushanam;
Are hybrids more susceptible than their inbred parents?

Maunder:
Hybrids may be more susceptible. In screening tests conducted during 1960-64, one parent was susceptible, and the resulting hybrid was more susceptible than the worst parent due to dominance for susceptibility. In DK-46, one parent is a charcoal-rot-resistant line, and the other parent is a nonsenescing line with a stiff stalk that is not susceptible. If one parent is really bad, the hybrid may be even worse. If one parent is good and the other carries charcoal rot resistance, you can have a good hybrid. It's not a really clear-cut inheritance where susceptibility is always dominant. It's'also very polygenic.

Vidyabhushanam:
If you want a resistant hybrid, I believe you should have resistance in both parents.

Maunder:
That would be optimum. But when you start requiring this, you have problems in bringing along the other favorable traits, and this is why we have gone to the intermediate x resistant approach. You're more likely to get more things you want in the end result. Anytime you have a new requirement, you double the effort to get what you want.

Rosenow:
We use a very stay-green, charcoal-rot-resistant parent on one side, and the other parent is a very high-yield-potential line with wide adaptation. The F_1 combines many of the good traits of both: This involves the dominant type of stay-green trait.

Vidyabhushanam:
These stay-green types are low yielders and sink-limiting.

Henzell:
They are not necessarily low-yielding. But they have a source-grain sink relationship such that the plant is nonsenescing. You can indeed have a high-yielding nonsenescing plant, but it likely would not be source limited.

Vidyabhushanam:
If the source is not limiting, can high yield be obtained?

Henzell:
High yields can be obtained, but the option of increasing grain yield via increasing harvest index would probably result in a plant that is relatively source limited and therefore senescent.

Mukuru:
I have looked at a number of Guineense and none of them have succumbed to charcoal rot. In crosses, the derivatives are not as resistant as the original parents. Of those lines converted in the U.S., are any resistant to charcoal rot?

Rosenow:
We have not looked at the charcoal rot resistance

of the converted Guineense. They don't have good lodging resistance. We don't really know whether they have the stay-green characteristic. It could be masked in them.

Scheuring:
I know it's expedient in breeding programs to go for lodging resistance and hope you can bring along everything else. In the long run we might make more progress by identifying different sources of charcoal rot resistance, recombining among these, and selecting against lodging at the end of the process.

Rosenow:
An effort was made this past year to select out early-maturing genotypes from converted materials that appeared to have drought tolerance.

Maunder:
The greatest asset to a breeding program is the utilization of the world sorghum germplasm collection, but to use it in the converted form can be a real disadvantage because you get one trait that is wanted and a lot that are not wanted.

Drought Resistance

Jordan:
What characteristics are best for different environments to promote drought resistance in association with stalk rot resistance?

Rosenow:
In the context of overall drought resistance—when I use the term drought resistance related to stalk rot resistance, I'm only talking about drought resistance at the late stage of grain fill—I don't know if the plant is avoiding drought due to efficient utilization of moisture. Soil water *extraction work* doesn't show big differences in amount of water extracted. Slight differences occur where nonsenescing types extract less water during early growth and greater proportions during later growth. The roots remaining active may be a key. There are no differences in genotypes in total amount of water extracted. There is no explanation of why one plant lives and the adjacent one dies.

Henzell:
The whole gamut of mechanisms involved with drought resistance are probably important in promoting resistance to stalk rot. Roots are a factor in drought avoidance. In drought tolerance, you want a plant that continues to operate at low leaf water potentials, and osmoregulation is important. E-57 is nonsenescent, lodging-resistant, and an osmoregulator, with resultant continuing photosynthesis and an actively growing root system. With an osmoregulation system, desiccation tolerance becomes important in the drought resistance mechanism. In limited surveys in Australia, lodged plants have been found to have pathogens present even though there were no symptoms. Other plants lacked the pathogens and had no symptoms, but they were lodged.

Maunder:
It's a very complex, quantitative problem. We have concentrated on roots too heavily. Our best root system is in a hybrid that requires irrigation. RS-610 and DK-46 have poor root systems but good drought tolerance: not using water excessively early in the season is a good trait. Tillering should be avoided. Osmoregulation may need more attention.

Jordan:
Do we have unique genetic variability for osmoregulation?

Henzell:
It is likely that every plant osmoregulates, but some are more efficient than others.

Mukuru:
We screened under moisture stress and selected the unscorched genotypes that recovered. We have selected against avoidance.

Seetharama:
Morphological and ecological considerations are more important in our drought resistance and stalk rot resistance programs, while physiological and biochemical factors are less important.

Stress Studies

Partridge:
Maize stalks lodged under greenhouse conditions without any internal parasites detected in the collapsed portion. It's also possible that sorghum will lodge under similar conditions if stressed severely and without pathogens being detected.

Pappelis:
Hydroponic units (gravel) can be used to measure stress factors under greenhouse conditions. Temperature, moisture gradients, herbicides, fertilizers, can all be measured.

Jordan:
Osmotic solutions to induce stress have been utilized in a useful manner. Carbowax used to induce stress is not a good choice, as water is still available. In a decreasing soil moisture situation, both the rate of transport of water and energy problems are important.

McBee:
Changes in alternating light from high to low with high and low temperature frequently produce desiccation and may result in collapse of plants. Plants differ slightly at different growth stages and the effects of nonsenescence and stalk rot need to be studied at each growth stage.

Charcoal Rot

Rosenow:
It appears that the local types grown in various countries possess charcoal rot tolerance. The introduced types from the U.S., ICRISAT, and other programs seemingly have an increased incidence of charcoal rot. Is this attributable to the high-yielding, photoperiod-insensitive, grain/straw ratios possessed by the introduced accessions, or do the local types possess resistance due to a different plant configuration and other factors?

Mukuru:
When the converted line was compared to the original in charcoal rot tests, the original line was resistant. When the photoperiod-insensitive line was crossed, the progeny were not resistant.

Rosenow:
Were the changes due to insensitivity per se or a complete change of the plant?

Scheuring:
We have a number of F_5s and F_6s from crosses between tall, late, local, and short, early exotic materials that are holding up well to charcoal rot pressure. The progeny are short and phenotypically quite different from the local parents.

Rosenow:
Would you agree that some local types may be resistant, while others may escape due to other mechanisms?

Scheuring:
The resistance of the Guineense parents is present and can be transferred. However, lateness might be one factor masking escapes. We often get a 2-week drought toward the end of the rainy season. That drought often occurs at or after flowering of the early lines but before the flowering of the lates. The earlier lines often collapse because they are at a vulnerable stage of development. The slower growing late sorghums seem to be less sensitive to drought even during the vulnerable grain fill stage.

Mughogho:
Local landraces need to be examined under controlled conditions to determine if they have charcoal rot resistance. There were reports of stalk rot problems on local landraces in West Africa.

Mukuru:
This appears to be something different, as there are various sorghum types in the areas that are susceptible. We haven't seen any local landraces of sorghum with charcoal rot.

Scheuring:
We must emphasize that local landraces are the topic and not other types.

Mughogho:
Some of these local landraces of the Guineense sorghums possess resistance to grain molds and, if they are resistant to charcoal rot, they would be most useful in breeding programs.

Group Discussions and Recommendations

Group A

Group B

Group C

Report of Group Discussions and Recommendations on Charcoal Rot, Fusarium Root and Stalk Rot, and Anthracnose Stalk Rot

Following the presentation and discussion of the four sets of background papers, participants were assigned to one of three groups A, B, and C. Each group consisted of nine persons. The nominal group technique, which provides a structured approach for considering specific problems and enhances productivity of conferences (Dalbecq et al. 1975), was used to arrive at recommendations on priority research areas on the three major root and stalk diseases: charcoal rot, fusarium root and stalk rot, and anthracnose stalk rot.

For each disease, the procedure followed involved five steps:

1 presentation of the problem in the form of a question; the questions presented to the three groups were:

   a. What information must be acquired before control of.....is possible?

   b. In the absence of constraints, what should be done to control.....?

   c. What are the major opportunities for interdisciplinary and collaborative research on.....?

2. recording of ideas generated by each member of the group (see Appendix at the end of this chapter);
3. discussion of each idea for clarification;
4. ranking (by vote) of the most important ideas as recommendations of each group;
5. presentation of each group's findings to a meeting of all participants, and steps 3 and 4 repeated in order to arrive at specific final recommendations for future research.

In accordance with the above procedure, this presents the priority research problems identified by groups and final recommendations of the whole workshop on the three diseases.

Charcoal Rot

Priority Research Problems Identified by Groups

Group A: ***What information must be acquired before control of charcoal rot is possible?***

1. Understand host-parasite-environment interactions.
2. Establish optimum parameters for uniform screening techniques.
3. Identify immune genera and new resistance sources for incorporation into varieties by classical or biotechnical methods,
4. Quantify stress and the plant's reaction as it affects pathogenesis.
5. Investigate the inheritance of resistance.
6. Identify plant traits responsible for resistance.
7. Determine chemical-physiological properties of plants that enhance disease.

Group B: ***In the absence of constraints what should be done to control charcoal rot?***

1. Collect, select, and screen germplasrn with emphasis on accessions from regions with high natural selective pressure.
2. Determine causes of genotype variation in

host plant resistance and lodging resistance with particular emphasis on physiological (e.g., source/sink relationships, osmoregulation, photosynthetic efficiency, living cell resistance) and anatomical aspects.

3. Develop more effective and relevant screening techniques.
4. Characterize the predisposing stress environment utilizing controlled stress techniques, e.g., hydroponics, gravel culture.
5. Study the biology of the pathogen, including the role of root exudates and its interaction with other organisms.
6. Study the genetic differences among charcoal rot resistance sources (inheritance) and their stability across environments (heritability).
7. Establish special interdisciplinary varietal development nurseries using present techniques,
8. Evaluate and utilize chemical and cultural practices to develop crop management systems for control.
9. Determine disease severity/crop loss relationships.
10. Examine the potential for biological control (e.g., suppressive soils).

Group C: ***What are the major opportunities for interdisciplinary and collaborative research on charcoal rot?***

1. Develop reliable, sound inoculation techniques for field evaluation of resistance.
2. Predict disease incidence and loss.
3. Investigate the relationship between *Macrophomina* and other organisms at soil-plant interface.
4. Identify mechanisms of physical and physiological resistance.
5. Determine the effect of genotype and environment interaction on disease, particularly temperature and water.
6. Study the genetics and stability of the nonsenescence trait.
7. Investigate the epidemiology and etiology of disease.

Final Recommendations for Priority Research Areas

1. Investigate host-parasite-environment interactions, with emphasis on temperature, moisture, and nutrient stress, and predisposing factors in disease development.
2. Develop more effective and relevant screening techniques for resistance.
3. Determine physical and physiological plant characteristics associated with resistance to the pathogen and to lodging.
4. Determine the relevance of the nonsenescent character to charcoal rot resistance and the stability of this trait across environments.
5. Collect, select, and screen germplasm for resistance, with emphasis on accessions from regions with high natural selection pressure.
6. Establish interdisciplinary nurseries for variety development.
7. Determine the inheritance and heritability of resistance.
8. Develop models to predict onset and development of the disease and resulting yield loss.
9. Identify and utilize exotic sources of resistance by classical or innovative methods (biotechnological/genetic engineering).
10. Evaluate and utilize chemicals (e.g., fungicides, plant growth regulators) and cultural practices to develop crop management systems for control.
11. Elucidate the biology and variability of the pathogen in its interaction with the host.
12. Elucidate the epidemiology of the disease.

Fusarium Root and Stalk Rot

Priority Research Problems Identified by Groups

Group A: ***What information must be acquired before control of fusarium root and stalk rot is possible?***

1. Determine the inheritance and heritability of resistance.
2. Identify immune genera and new sources of

resistance for incorporation of genetic material by classical or biotechnical methods.

3. Understand host-parasite-environment interactions for various geographical locations.
4. Identify plant traits conferring resistance.

Group B: *In the absence of constraints, what should be done to control fusarium root and stalk rot?*

1. Improve screening techniques for resistance, including the examination of development of the initial screening program using controlled water and heat stress.
2. Determine regionally the importance of fusarium disease(s) and the relative importance of the different *Fusarium* spp.
3. Determine causes of genotype variation in host plant resistance to lodging, with particular emphasis on physiological (e.g., source/sink relationship, assimilate partitioning, photosynthetic efficiency, etc.) and anatomical aspects.
4. Identify sorghum lines with relative resistance and susceptibility through a selected screening of the world collection.
5. Study host-parasite interactions, including predisposition and mechanisms of resistance.
6. Develop integrated management systems to control the problem.
7. Examine the potential for control with fungicides, plant growth regulators, and related chemicals.
8. Relate cell death patterns to susceptibility and resistance in roots and stalks, especially in senescing and nonsenescing (stay-green) genotypes.
9. Determine inheritance of different sources of resistance.
10. Determine root and stalk rot incidence and yield losses in no-till, conservative-till, and conventional tillage (include inoculation and pith condition rating when possible).

Group C: *What are the major opportunities for interdisciplinary and collaborative research on fusarium root and stalk rot?*

1. Investigate the epidemiology and etiology of the fusarium disease complex.
2. Determine environmental factors responsible for predisposition.
3. Determine the relationship of nonsenescence to resistance.
4. Investigate the effect of crop management factors in disease incidence.
5. Develop and standardize screening techniques.
6. Analyze interactions among *Fusarium* spp complex, host, and environment in disease.
7. Study the relationship between *Fusarium* spp and other root/stalk rot pathogens.

Final Recommendations for Priority Research Areas

1. Investigate host-parasite-environment interactions, with emphasis on temperature, moisture, and nutrient stress as predisposing factors in disease development.
2. Develop more effective and relevant screening techniques for resistance.
3. Identify physical and physiological plant characteristics associated with resistance to the pathogen(s) and to lodging.
4. Determine the regional importance of fusarium stalk rot and the relative importance of the different *Fusarium* spp in each region.
5. Determine the relationships between *Fusarium* spp and other root/stalk rot pathogens.
6. Determine the inheritance and heritability of resistance.
7. Evaluate and utilize chemicals (e.g., fungicides, plant growth regulators) and cultural practices to develop crop management systems for control.
8. Identify sorghum groups with relative resistance and susceptibility through selective screening of the world sorghum germplasm collection.
9. Identify and utilize exotic sources of resistance by classical or innovative methods (biotechnological/genetic engineering).
10. Relate cell death pattern to susceptibility and resistance in roots and stalks, especially in

senescing, and nonsenescing (stay-green) genotypes.

11. Elucidate the etiology and epidemiology of the disease.

Anthracnose

Priority Research Problems Identified by Groups

Group A: ***What information must be acquired before control of anthracnose is possible?***

1. Understand host-pathogen-environment interactions.
2. Determine the inheritance and heritability of anthracnose stalk rot resistance.
3. Incorporate improved sources of resistance using classical and biotechnical methods.
4. Determine the relative importance of grain and leaf anthracnose to stalk rot.
5. Determine the relative advantages of cultural control, chemical control, and biocontrol of anthracnose.

Group B: ***In the absence of constraints, what should be done to control anthracnose?***

1. Evaluate known sources of resistance worldwide and screen untested world collection items at known "hot spots''.
2. Determine mechanisms of resistance in a range of genotypes.
3. Determine race/genotype interactions in major sorghum-producing regions, using a set of common genotypes and regionally important additions.
4. Determine regionally the relative importance and virulence of the disease, including different phases of the disease.
5. Accelerate and coordinate breeding programs for resistance at strategic locations.
6. Evaluate and utilize chemical and cultural practices to develop crop management systems for control.

Group C: ***What are the major opportunities for interdisciplinary and collaborative research on anthracnose?***

1. Identify sources of resistance.
2. Analyse the relationship of stalk rot to other disease phases.
3. Evaluate collected isolates at containment facilities.
4. Compare the economic losses caused by anthracnose with the losses caused by other stalk rot pathogens.
5. Determine the role of seedborne inoculum in disease development and dissemination.
6. Identify the races of *Colletotrichum.*

Final Recommendations for Priority Research Areas

1. Investigate host-parasite-environment interactions.
2. Evaluate known sources of resistance worldwide and screen untested world collection items at known "hot spots."
3. Monitor pathogen variability using standard differential varieties and regionally important cultivars.
4. Determine the relationship of grain and foliar anthracnose to stalk rot.
5. Determine mechanisms of resistance.
6. Evaluate and utilize chemicals (e.g., fungicides, plant growth regulators), cultural practices and biological control to develop crop management systems for control.
7. Develop more effective and relevant screening techniques for resistance.
8. Determine the role of seedborne inoculum on disease development and dissemination.
9. Identify and utilize improved and exotic sources of resistance by classical or innovative methods (biotechnological/genetic engineering).
10. Determine the inheritance and heritability of resistance.

1.1. Compare the economic losses caused by anthracnose with losses caused by other stalk rot pathogens.

12. Elucidate the etiology and epidemiology of the disease.

13. Evaluate collected isolates at containment facilities to determine the variability of the pathogen.

Appendix: Problems Recorded by Groups (Step 2)

Charcoal Rot

Yield/Crop Loss

1. Apportion loss of yield caused by drought and disease.
2. Determine disease severity/crop loss relationships.
3. Develop a predictive model for losses.

Biology of *Macrophomina phaseolina*

1. Study fungal properties that induce disease symptoms.
2. Conduct field and laboratory studies of the fungus.
3. Evaluate the distribution of fungal propagules in the soil.
4. Investigate the rate of physiological change in *M. phaseolina.*
5. Determine the role of root exudates on the pathogen.
6. Study the role of root exudates in microbial ecology.
7. Assess kinetics of extracellular fungal enzymes in pathogenesis.
8. Study fungal succession in the disease complex.
9. Assess the rate of mutation in *M. phaseolina.*
10. Determine pathogen variability.

Associated Organisms

1. Determine nematode-pathogen-host interactions.
2. Obtain a better understanding of the epidemiology and etiology of *M. phaseolina* and its interaction with other microorganisms, particularly at the soil/plant interface.
3. Determine the role of other soil microorganisms associated with charcoal rot.

Epidemiology

1. Identify major predisposing factors in different cropping systems.
2. Quantify stress and the plant's reaction as it affects pathogenesis.
3. Determine the predisposing level and duration of water stress.
4. Determine time and location of host infection.
5. Determine the role of high temperature in disease development.
6. Study the chemical-physiological properties of plants that enhance disease.
7. Understand host-parasite-environment interaction.
8. Investigate seedborne dissemination of the pathogen.
9. Conduct a thorough in situ study of the biology of the pathogen.
10. Investigate the effect of plant architecture on disease development.
11. Determine the inoculum threshold.
12. Identify factors promoting infection and colonization.
13. Carry out studies of pathogenesis.
14. Determine time (development stage) of pathogenesis.
15. Determine the methods of penetration and establishment in the host.
16. Analyse the host maturity/susceptibility relationship.
17. Relate types and levels of carbohydrates in sorghum to environmental factors—i.e., stress and diseases.
18. Create gravel culture system(s) to study host / pathogen-stress interactions.
19. Develop accurate host-parasite models.
20. Model disease development (simulation).
21. Obtain a better description of the stress environment.

22. Understand response and adaptation to stress,
23. Quantify the stress level necessary for predisposition to infection.
24. Obtain a better understanding of the worldwide epidemiology and etiology of charcoal rot.
25. Study the interrelationship of heat/moisture stress and variety on disease.
26. Elucidate the epidemiology and biology of infection and disease development.
27. Understand the factors predisposing the plant to disease.
28. Gain a better understanding of epidemiology worldwide.
29. Analyse the pathogen's influence on plant metabolism.

Screening Techniques and Identification of Resistance

1. Develop reliable field screening techniques.
2. Formulate a uniform method of disease scoring.
3. Improve screening techniques for resistance.
4. Devise cell-culture or in vitro screening techniques based on mechanism of pathogenicity.
5. Establish optimum parameters for uniform screening techniques.
6. Screen all available genotypes for resistance.
7. Collect and screen germplasm from regions with high selection pressure.
8. Monitor uniform genotypes in screening nurseries.
9. Identify stable resistance sources.
10. Develop screening techniques, after establishing the causes of genetic variability in host resistance.
11. Study resistance to nonhosts.
12. Establish special interdisciplinary varietal development nurseries, using present techniques.
13. Systematize knowledge of unaffected plant genera for possible gene transfer.
14. Determine stability of charcoal rot resistance across environments.
15. Identify immune genera and new resistance for incorporation of genetic material by classical or biotechnical methods.

Plant Traits Associated with Resistance

1. Identify plant traits responsible for resistance.
2. Determine inheritance of the stay-green trait.
3. Understand the relationship of senescence and nonsenescence with stalk disease.
4. Study the genetics and stability of the nonsenescence character under drought stress.
5. Establish the relationship between nonsenescence, senescence, and cell death patterns in roots and stalks.
6. Relate the spread of the disease in the host to cell death patterns.
7. Determine the importance of root/stalk senescence.
8. Examine stay-green/ charcoal rot resistance multilocationally.

Nature of Resistance

1. Determine resistance mechanisms.
2. Determine the causes of genetic variability of resistance.
3. Analyse inheritance of resistance.
4. Study inheritance of genetic differences among resistance sources.
5. Study the physiology and biochemistry of resistance and pathogenecity.
6. Determine the role of osmoregulation in resistance and susceptibility.
7. Identify defense mechanisms of living cells.
8. Characterize all known sources of resistance (and susceptibility)—including stay-green and senescent types—anatomically, pathologically, physiologically, and biochemically.
9. Determine the relationship between resistance to *Macrophomina* and resistance to *Fusarium.*

10. Study the genetics of pathogenicity and pathogen variability.

Drought Tolerance and Disease Resistance

1. Study the physiological mechanisms involved in postflowering drought tolerance.
2. Improve the tolerance of the host to drought.
3. Correlate drought resistance/infection/disease.
4. Determine whether selection for drought resistance includes charcoal rot resistance.
5. Obtain a better understanding of the role of drought resistance, root and stalk senescence, and cropping systems on charcoal rot development.
6. Work towards improved environmental stress tolerance.

Chemical Control

1. Examine the potential for fungicidal and/or plant growth regulators or related chemicals as control components.
2. Determine the effects of antisenescence chemicals on host-pathogen interactions.

Biocontrol

1. Find biological control systems.
2. Study mechanisms of suppressive soils.
3. Develop crop management for disease control.
4. Determine whether rotation with nonhost crops reduces disease.
5. Determine host-parasite interactions under no-till, conservative-till, and conventional tillage.
6. Determine fertilizer effects on host response.
7. Carry out specific studies on nutrient interaction.
8. Study management influence on diseases.
9. Elucidate synergistic effects of genotype and management factors.
10. Determine the influence of soil and fertility factors.

Research Collaboration

1. Conduct studies in laboratory and field through teamwork of physiologists; pathologists, and breeders.
2. Aim for a better understanding of disease etiology by breeders and physiologists.
3. Stimulate fellow scientists to increase output.
4. Publish collaborative research jointly.
5. Obtain a better understanding of the disease complex.
6. Promote greater public and private support funding.
7. Determine how results could be useful internationally.
8. Establish collaborative research programs between INTSORMIL and ICRISAT.

Fusarium Root and Stalk Rot

Crop Loss

1. Clarify the relationships between disease and crop loss.
2. Develop methods to predict losses due to disease.
3. Estimate grain yield and quality reductions caused by fusarium stalk rots.
4. Determine root and stalk rot losses in no-till, conservative-till, and conventional tillage (include inoculation and pith condition ratings when possible).

Biology of the Pathogen

1. Identify the *Fusarium* spp in the disease complex.
2. Determine the role of the different *Fusarium* spp penetrating roots and causing disease.
3. Determine regionally the importance of fusarium disease(s) and relative importance of different *Fusarium* spp.

4. Specify the species of *Fusarium* important in causing root and stalk rot.
5. Examine the synergism hypothesis among *Fusarium* spp.
6. Outline the role of *Fusarium* spp in root and stalk senescence.
7. Determine whether the *Fusarium* spp/ sorghum stalk rot interactions follow the *Diplodia maydis* model.
8. Determine why *Fusarium moniliforme* appears to be inhibited in its systemic phase.
9. Analyze the interaction between the various *Fusarium* spp involved in the host tissue.
10. Determine the importance of seedborne *Fusarium* in the disease.
11. Gain a better understanding of the worldwide epidemiology and etiology of the *Fusarium* disease complex.
12. Undertake a multilocational and multicultivar study of fungal succession and systemicity.
13. Determine host differentials for species separation.

Pathogens

1. Determine the mechanisms of pathogenesis.
2. Describe in detail penetration, establishment, and spread of pathogens in living and dead cells of roots.
3. Determine relationships between plant stress and pathogenesis.
4. Correlate the association of quantity and quality of carbohydrates with severity of the disease.
5. Conduct physiological-pathological studies differentiating pathogenic and saprophytic attack.

Etiology and Epidemiology

1. Determine the "where" and "when" of primary infection.
2. Investigate the survival of inoculum in soil.
3. Study the epidemiology and etiology of the *Fusarium* spp involved.
4. Study host-parasite interactions, including predisposition and mechanisms of resistance.
5. Quantify host-pathogen-environment combinations favoring infection and pathogenesis.
6. Characterize environments in which the disease is a problem.
7. Quantify environmental conditions enhancing stalk and root rots.
8. Describe environmental conditions leading to stalk rot.
9. Determine the precise environment(s) necessary for infection in the field.
10. Determine which environmental factors predispose plants to the disease.
11. Understand the host-parasite-environment interactions for various geographic areas.
12. Describe the interaction between plant Stress and inoculum levels.
13. Identify factors responsible for predisposition to the disease.
14. Determine the effects of soil nutrients on disease development.
15. Understand host-parasite-environment interaction.
16. Determine the mechanisms for predisposition and disease resistance.
17. Determine the effects of plant injuries on fusarium root and stalk rot.
18. Study the host/parasite relationships in diverse sorghum areas.
19. Examine the occurrence and importance of the systemic phase.
20. Quantify the pathogen-host-soil-microflora-environment interaction for disease development.
21. Explore the relationship between root and stalk rots.
22. Compare sorghum and maize stalk rots caused by *Fusarium* spp.
23. Study the similarities between fusarium and charcoal rots.
24. Examine the relationships between fusarium and macrophomina root and stalk rot complexes.

25. Determine the interaction of other fungi with *Fusarium* spp.
26. Determine the relationship between *Fusarium* spp and other root and stalk rot pathogens.

Resistance Screening Techniques and Identification of Resistance

1. Develop effective screening techniques.
2. Devise screening techniques for all stages of plant growth.
3. Improve screening techniques for resistance, including the examination of an initial screening program using controlled water and heat stress.
4. Identify sorghum groups with relative resistance and susceptibility through a selected screening of the world collection.
5. Identify cultivars with specific reactions for use in standardized nurseries.
6. Identify sources of resistance.
7. Identify immune species and new sources of resistance for incorporation of genetic material by classical or biotechnical methods.
8. Determine the causes of genotype variation in host plant resistance, with particular emphasis on physiological and anatomical aspects.
9. Formulate scoring scales for measuring different kinds of damage.

Nature of Resistance

1. Study the inheritance and heritability of resistance.
2. Examine the genetics of host resistance.
3. Identify the physiological plant factors related to host resistance.
4. Determine the inheritance of several sources of resistance.
5. Determine whether free and glycocidic phenols are involved in resistance.
6. Investigate the genetics of resistance and susceptibility of stalk and root rots.
7. Determine the causes of genotypic variation in host plant resistance and lodging, with particular emphasis on physiological aspects (e.g., source-sink relationships, assimilate partitioning, photosynthetic efficiency) and anatomical aspects.
8. Define the relationships between grain mold resistance *(Fusarium)* and fusarium stalk and root rot resistance.
9. Identify plant traits conferring resistance.
10. Determine the relationship of senescence and nonsenescence to the resistance or susceptibility of the genotype to disease.
11. Relate cell death patterns to susceptibility and resistance in roots and stalks, especially in senescing and nonsenescing (stay-green) genotypes.
12. Undertake a comprehensive study of assimilate partitioning with respect to senescence, harvest index, susceptibility, and lodging.
13. Search for differences in photosynthetic efficiency.

Chemical Control

1. Examine potentials for control with fungicides, plant growth regulators, and related chemicals.
2. Investigate control of senescence with applied chemicals.

Crop Management and Disease

1. Develop integrated management systems to control the problem.
2. Develop cultural methods for disease management.
3. Study crop management factors influencing the disease.
4. Identify cultural practices that might alleviate the impact of the disease.
5. Define the role of cultural and location-specific factors on disease incidence in contrasting genotypes.
6. Determine the effects of row spacing and population on disease expression.

Anthracnose

Crop Loss

1. Explore the relationship between infection severity and yield loss by geographic areas.
2. Make separate assessments of yield losses due to infection of individual plant parts.

Biology

1. Describe the taxonomy of the pathogen and its relationship to other cereal anthracnoses.
2. Determine regionally the relative importance and virulence of the disease, including its different phases.
3. Study genetic variability of the pathogen.
4. Determine the race/genotype interactions in major sorghum-producing regions, using a set of common genotypes and regionally important genotypes.
5. Estimate the mutation rate of the pathogen.
6. Devise improved methods for identification of physiological races.
7. Identify predominant races of the pathogen by geographical area.
8. Identify physiological races of anthracnose.
9. Collectively evaluate isolates at a containment facility for pathogen variability.

Phases of the Disease

1. Elucidate the causes of different phases of anthracnose.
2. Explain the relationship of stalk rot to other phases of the disease,
3. Determine whether there is a relationship between anthracnose stalk rots of maize and sorghum.
4. Examine correlations of resistance to leaf, peduncle, and panicle phases.
5. Establish the relationship of grain and leaf anthracnose to stalk rot.

Epidemiology

1. Investigate the temperature/plant growth/ disease interaction.
2. Correlate plant nutrient balance with disease incidence.
3. Determine and quantify optimum environmental factors necessary for infection and pathogenesis.
4. Study host-pathogen-environment interaction.
5. Analyse environmental parameters in pathogenesis of anthracnose stalk rot.
6. Identify factors affecting inoculum survival in the soil.
7. Establish the relationship of seedborne inoculum to foliage infection, grain infection, and stalk rot.
8. Determine the importance of seedborne inoculum in epidemiology and seed exchange.
9. Study seedborne dissemination of new pathotypes.
10. Estimate the rate of growth of different phases of anthracnose in different cultivars under different environmental conditions.
11. Describe mechanisms of survival of the pathogen under natural conditions.
12. Determine how anthracnose spreads from leaves to stalk in senescent and nonsenescent genotypes, with emphasis on locating genotypes to resist this spread.
13. Examine the spread of the pathogen within the stalk.
14. Identify the mode of infection of leaf, grain, stalk, and root.
15. Determine which factors are favorable or unfavorable for the incidence of anthracnose and fusarium and charcoal rot.

Physiology of Host-Parasite Interaction

1. Conduct biochemical studies on host plant resistance.
2. Study the physiology of host response to infection.

3. Investigate the biochemistry of pathogenesis.
4. Relate the osmotic potential of the host plant to the degree of infection at different growth stages.

Control

1. Evaluate growth regulators as chemical controls.
2. Develop cost-effective chemicals for control.
3. Evolve methods for cultural control, chemical control, and biocontrol of anthracnose.
4. Evaluate and utilize chemicals and cultural practices to develop crop management systems for control.
5. Evaluate fungicides, plant growth regulators, and related chemicals as control agents.
6. Study the effect of conservation tillage on disease development.
7. Determine the role of preformed fungistatic compounds and phytoalexins and other postinfection resistance chemicals in resistance.
8. Study host disease reaction before and after treatment with ethylene.
9. Accelerate and coordinate breeding programs for resistance at strategic locations.

Screening Techniques and Identification of Resistance

1. Develop effective and relevant laboratory and field screening techniques.
2. Determine germplasm susceptibility/resistance to multiple disease.
3. Screen the world sorghum germplasm collection for resistance at locations where there are differences in pathogen variability.
4. Identify stable and durable anthracnose resistance for incorporation into improved cultivars.
5. Screen all known available sources of resistance.
6. Identify broad-spectrum resistance sources.
7. Determine the value of known sources of resistance for sorghum improvement programs.
8. Test multilocationally a carefully selected set of genotypes for stability of identified resistance.

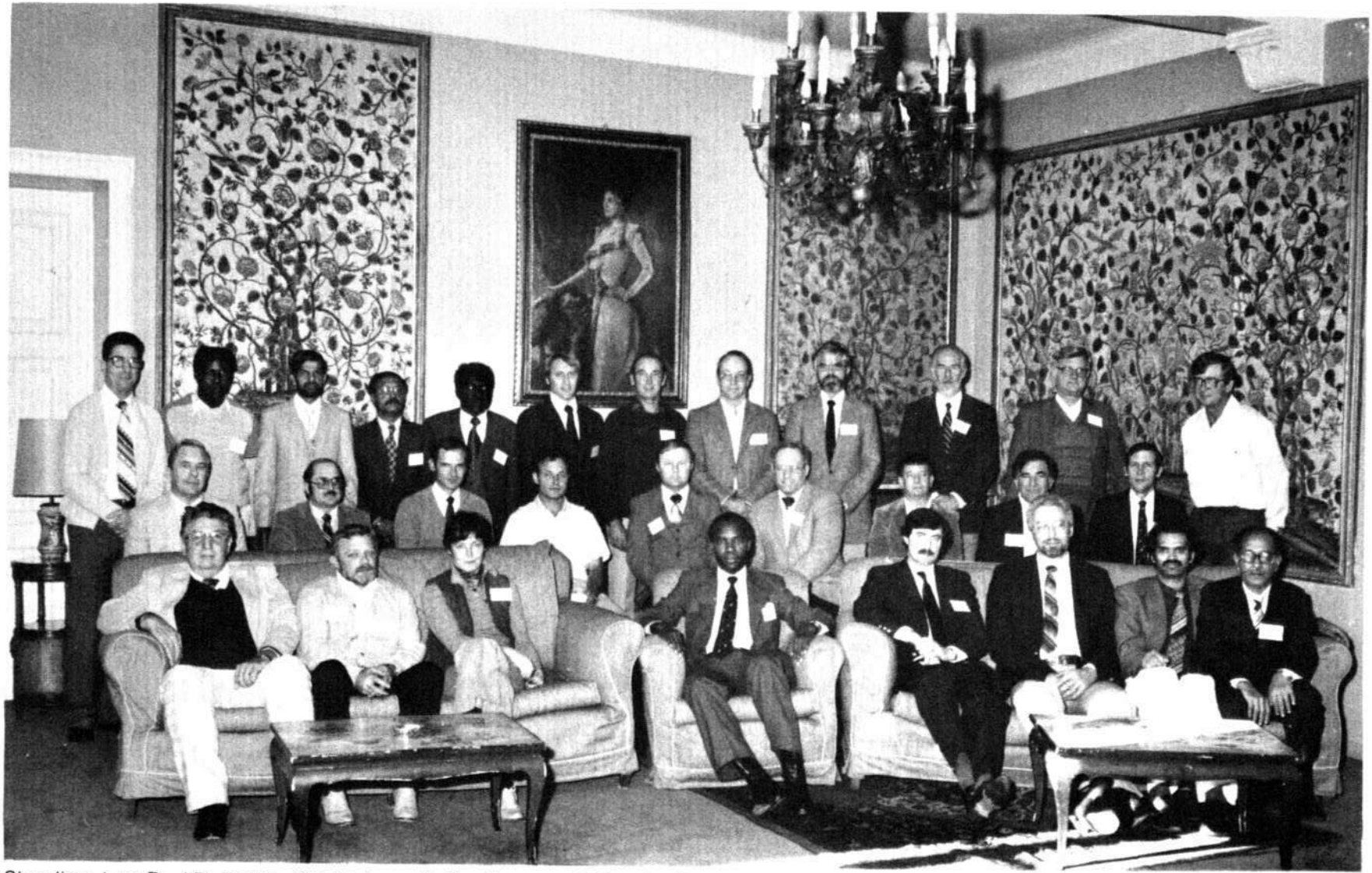
Nature of Resistance

1. Determine "r" for various cultivars or cultivar mixtures, and its value in reducing damage.
2. Study the genetics and stability of resistance.
3. Study the inheritance and heritability of anthracnose stalk rot resistance.
4. Analyze mechanism(s) of resistance related to stability of heritable character(s).
5. Identify plant factors conferring resistance.
6. Identify plant morphological factors affecting resistance.
7. Determine the mechanism of resistance in a range of genotypes, including preformed postinfection compounds.
8. Determine the relationship of senescence/nonsenescence in resistance.

References

DALBECQ, A.L., VAN DER VEN, A.H., and GUSTAFSON, A.H. 1975. Group techniques for program planning: a guide to nominal group and delphi processes, Glenview, Illinois, USA: Scott, Foreman.

Meeting Organization and Participants



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Cover:

S.C. Dalmacio, University of the Philippines at Los Banos: bacterial stalk rot

D.T. Rosenow: nonsenescent and senescent sorghum lines

R.J. Williams: stalk lodging

Frontispieces:

S. Pande: charcoal rot

N. Zummo: fusarium stalk rot and pokka boeng

G.N. Odvody: pythium root rot

R.A. Frederiksen: anthracnose stalk rot; acremonium wilt

G.N. Odvody: periconia root rot

LE. Claflin: nematode and nematode damage to sorghum

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