# Stalk Rots

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***Abstract***

*Stalk rots of sorghum are diseases of great destructive potential Rots caused by the fungi* Macrophomina phaseolina *and* Fusarium moniliforme *appear to be widely distributed stalk diseases of sorghum. Recently a vascular pathogen,* Acremonium strictum *that causes leaf and stalk death, has become important on sorghum.*

*Improved high-yielding varieties tend to be highly susceptible to these diseases. Losses vary from season to season and region to region. Grain losses exceeding 15% are not uncommon; as much as 60% can occur. Several fungi and bacteria are often associated in diseased roots and stalks, suggesting that stalk rot diseases are of complex etiology. Etiology and host resistance to charcoal rot, fusarium root and stalk rot, and acremonium wilt are discussed.*

## Introduction

Stalk rots are universally important and among the most destructive diseases of sorghum throughout the world. Development of stalk rots is favored by early grain-filling and a late-season stress. Postflowering stresses include various leaf diseases, excessive cloudiness, high plant densities, drought, hail damage, low K coupled with high N, root rots, and injury by root and stalk boring insects. Almost any factor that reduces photosynthate production by the sorghum plant favors stalk rot.

In most cases stalk rots follow root rots, and are caused by several fungi and bacteria that attack plants approaching maturity. A number of organisms often can be isolated from diseased roots and stalks; the well known causal agents are the fungi *Macrophomina phaseolina* (charcoal rot), *Fusarium moniliforme* (fusarium root and stalk rot complex), *Periconia circinata* (milo disease), *Pythium* spp (root and seedling rots), and *Colletotrichum graminicola* (anthracnose stalk rot).

M. *phaseolina* and *F. moniliforme* appear to be widely distributed in sorghum-growing areas. P. *circinata,* once thought to be restricted to USA, has been reported from Australia (Mayers 1976).

Recently *Acremonium strictum,* a vascular pathogen causing leaf and stalk death, has been recognized as an important disease in the Americas.

Root and stalk diseases reported by Tarr (1962) include pink root rot (*Pyrenochaeta terrestris),* southern sclerotial rot *(Sclerotium rolfsii),* and rhizoctonia stalk rot *(R. solani).* Bacteria, particularly *Erwinia* spp, have also been implicated as sporadically important causal agents of stalk rots in the Philippines (Karganilla and Exconde 1972), in India (Anahosur 1979), Nigeria (King 1973), and USA (Zummo 1969). Little is known about the etiology of these diseases and the topic is an obvious area that demands research.

Root and stalk rots are reported to cause crop losses, but data from experiments are limited. Losses vary from season to season and from region to region worldwide. Yield losses from 15 to 60% may occur on susceptible cultivars. Losses may be direct (due to poor grain filling) or indirect (due to stalk breakage or lodging).

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**Pande, S., and Karunakaz; R.I. 1992.** Stalk rots. Pages 219-234 *in* Sorghum and millets diseases: a second world review, (de Milliano, W.A.J., Frederiksen, R.A., and Bengston, G.D., eds). Patancheru, A.P. 502 324, India: International Crops Research Institute for the Semi-Arid Tropics. (CP 742).

Eighteen papers in the proceedings (Mughogho 1984) of the 1983 Consultative Group Discussion on Research Needs and Strategies for Control of Sorghum Root and Stalk Rot Diseases, Bellagio, Italy discussed in detail the physiological and environmental factors that influence these diseases and their control by fungicides, cultural practices, and host resistance. This review attempts to summarize the present state of knowledge and progress on basic aspects of charcoal rot and fusarium root and stalk rot, the two major root and stalk rot diseases of complex etiology.

Acremonium wilt has become an important disease of sorghum. Anthracnose stalk rot, another damaging disease of sorghum particularly in warm humid sorghum-growing areas, was reviewed by Ali (this publication). He discussed two other phases of the disease: foliar anthracnose and panicle and grain anthracnose phase.

## Charcoal Rot

Charcoal stalk rot of sorghum *[Sorghum bicolor* (L.) Moench] caused by the fungus *Macrophamina phaseolina* (Tassi Goid.), is a disease of great destructive potential when vigorously growing sorghums fill grain under drought stress (Edmunds 1964; Odvody and Dunkle 1979). Charcoal rot has been reported from all the ecologically diverse areas of sorghum culture in the tropics, subtropics, and temperate regions (Tarr 1962; ICRISAT 1980). In general, the worldwide distribution of the disease indicates its occurrence on many different soil types.

In diseased roots and stalks, M. *phaseolina* is often associated with other fungi, suggesting that the disease is of complex etiology (Mughogho and Pande 1984).

### Economic importance

The literature contains several reports on the destruction of sorghum crops by charcoal root and stalk rot, but sound and reliable quantitative data on yield losses are not available. Reports by Uppal et al. (1936) from Maharashtra state, India, and Harris (1962) from Kano, Nigeria, stated that charcoal rot caused "sufficient to considerable loss in yield.'' 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 and estimated yield losses up to 50%. Similarly "serious losses" in several states in USA were reported but quantitative data were not given (Leukel et al.1951).

In spite of the lack of such data, the destructive potential of charcoal rot in susceptible cultivars may be recognized in four ways: (1) loss in grain yield and quality due to plants smaller than normal and premature ripening; (2) poor crop stand due to seedling blight; (3) complete loss of yield of lodged plants where mechanical harvesting is practiced; destruction of lodged plants by termites or other animal pests before the grain or fodder is manually collected; and (4) loss in quality and quantity of fodder due to infection and destruction of the stalk.

Mughogho and Pande (1984) estimated grain yield losses of 23-64% in research sowings of the CSH 6 hybrid. Similarly Anahosur and Patil (1983) noted 15-55% loss in grain mass in their experiments at Dharwad, India. In these experiments, grain yield from plots subjected to drought was measured. Although drought alone must have contributed to some of the yield reduction, the combined effects of drought and charcoal rot that caused plants to lodge must have greatly increased the level of yield loss.

To separate the effect of drought from the combined effect of drought and charcoal rot on sorghum yield, we conducted an experiment during 1985 postrainy season at ICRISAT Center and at Dharwad. In this experiment the soil fungus flora was eliminated by methyl bromide gas fumigation of the test plots (500 g a.i. $m^{-2}$) before sowing. Control plots were not fumigated.

Natural charcoal rot infection was induced by continuously reducing soil moisture from postflowering to grain maturity (Mughogho and Pande 1984). We obtained 95-100% lodging and grain yield losses of 20% in nonfumigated plots of CSH 6 hybrid (Table 1). These data on grain yield losses clearly show the potential economic importance of the disease. There is still need for data, particularly from surveys in farmers' fields, on the various types of losses.

**Table 1. Lodging and yield of root and stalk rot-infected CSH 6 sorghum under continuously receding soil moisture and fumigated treatments at ICRISAT Center and at Dharwad, India, postrainy season 1985/86.**

| | ICRISAT Center | | | Dharwad | | |
|---|---|---|---|---|---|---|
| Treatment | Lodging; (%) | Yield [kg $(9m^2)^{-1}$] | 1000-grain mass (g) | Lodging (%) | Yield [kg$(9m^2)^{-1}$] | 1000-grain mass(g) |
| Fumigation[1] | 6.5 | 2.0 | 14.1 | 23.3 | 3.6 | 18.5 |
| No fumigation | 99.7 | 1.6 | 12.5 | 95.2 | 2.9 | 15.2 |
| SE | ±2.1 | ±0.1 | ±1.0 | ±6.8 | ±0.1 | ±0.7 |
| Yield loss (%)[2] | | 20 | 11.34 | | 19.44 | 17.83 |

1. Plots were fumigated with methyl bromide @ 500 g a.i. $(5\ m^2)^{-1}$
2. [(Fumigated - Nonfumigated)/fumigated] x 100.

## Symptoms

Symptoms of the disease include root discoloration, root rots, soft stalks and lodging of plants, premature drying of stalks, and poorly developed panicles with small, inferior-quality grains. Vascular bundles of infected roots and stalks are profusely covered with tiny charcoal-colored sclerotia (Uppal et al. 1936; Tarr 1962).

The most striking and usually the first indication of the disease is lodging of plants approaching maturity.

## Causal organism

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

## Fungal colonization

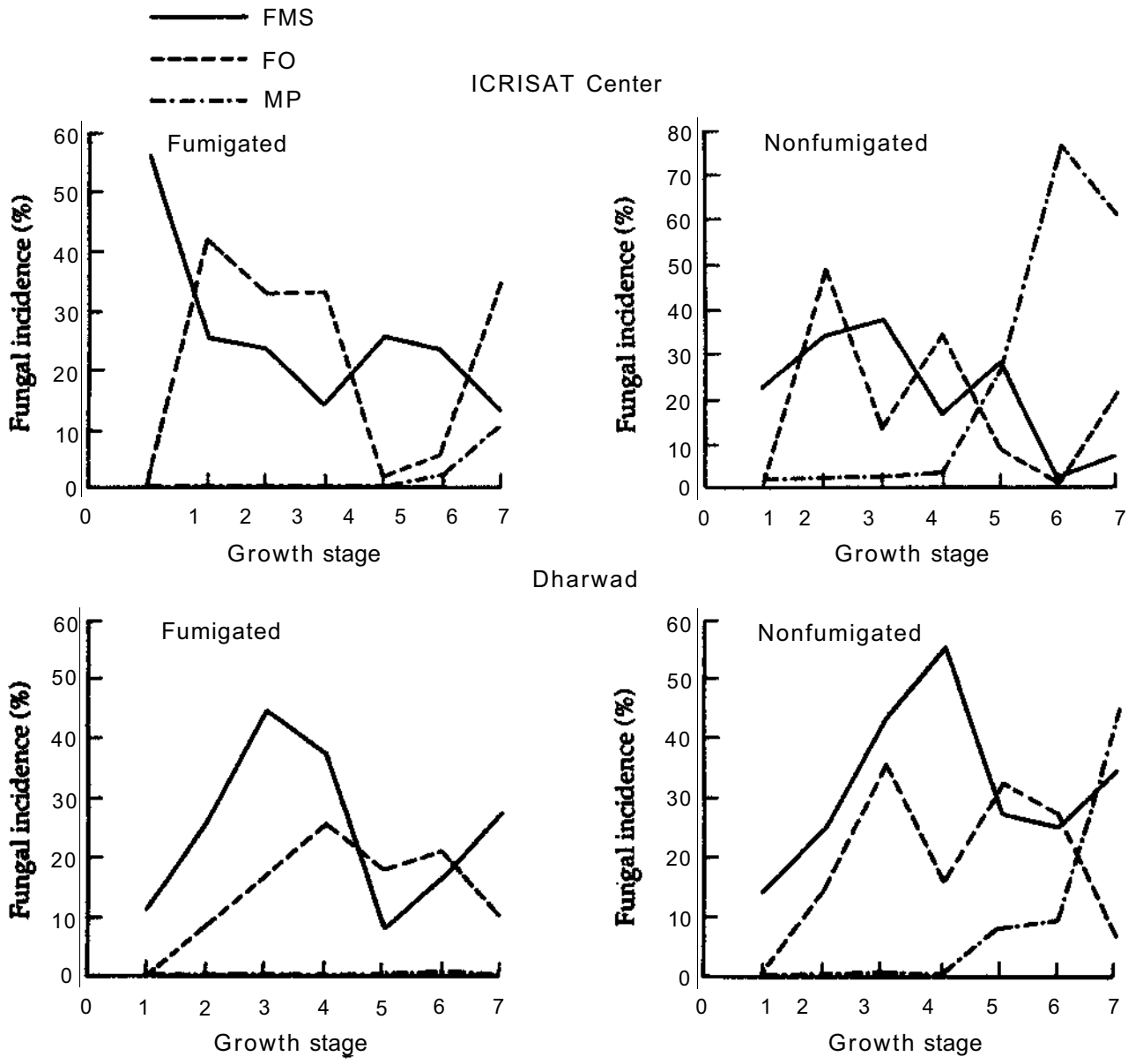
Sorghum roots may be infected by various fungi from the seedling stage until maturity. In diseased roots and stalks with conspicuous signs of charcoal rot, fungal isolations usually reveal the association of *M. phaseolina* with other fungi. In Argentina, where F. *moniliforme* was the predominant fungus isolated from diseased plants, 40% of the isolations were *M. phaseolina.* Others 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 predominate, M. *phaseolina* and *N. sphaerica* were also regularly isolated from these diseased roots and stalks (Trimboli and Bulges 1983).

Patridge et al. (1984) reported early colonization of sorghum roots by species of *Fusarium, Alternaria,* and *Epicoccum,* all common root inhabitants. Under certain external stress situations, the plant becomes quasidefenseless, and some of these fungi become pathogenic, causing stalk rot.

Recently we monitored fungal colonization of roots and stalks of susceptible hybrid CSH 6 at seven growth stages, from seedling to grain maturity (black-layer formation), in non-drought-stressed plots and in drought-stressed plots from onset of flowering to grain maturity. This was done by planting surface-sterilized pieces of roots, crown, and first internode on potato dextrose agar, Czapeck dox agar, and Meyer's medium. Six *Fusarium* species (F. *moniliforme,* F. *moniliforme* var *subglutinans,* F. *mon-*

*iliforme* var *intermedium, E solani, F. semitectum,* and *F. chlamydosporum), Macrophomina phaseolina, Rhizoctonia solani, Phoma sorghina, Exserohilum rostratum,* and *Trichoderma harzianum* were found to colonize sorghum toots and stems. Among these fungi we found F. *moniliforme* van *subglutinans and E oxysporum* to be early colonizers, increasing in abundance after the induction of drought stress from onset of flowering to maturity. M. *phaseolina* was not isolated until after the hard-dough growth stage and then only from drought-stressed plants (Fig. 1).

Data cited above show 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




FMS = F*usarium moniliforme* var. *subglutinans,* FO = *Fusarium oxysporum,* MP = *Macrophomina phaseolina*

1 = 5-6 leaf stage, 2 = Boot, 3 = Anthesis, 4 = Milk, 5 = Soft dough, 6 = Hard dough, 7 = Physiological maturity.

**Figure 1. Fungal incidence (%) at different growth stages of CSH 6 under fumigated and non-fumigated conditions.**

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. The pathological significance of this involvement of several fungi in root and stalk rot infection remains unknown, and calls for prompt detailed investigation.

## Variability and host range

M. *phaseolina* is highly variable in pathogenicity and mycological characteristics. Some isolates of the fungus are host specific (Hildebrand et al. 1945); others can attack a wide range of hosts (Holliday and Punithalingam 1970). Physiological races have been reported for isolates of some crops, such as jute (Ahmed and Ahmed 1969). Variability in cultural characteristics and pathogenicity of isolates from different parts of the same plant have been reported in soybean (Dhingra and Sinclair 1973). The fungus, a plurivorous pathogen, can affect 75 different plant families and about 400 plant species (Dhingra and Sinclair 1977).

Pathogen variation and physiological specialization of M. *phaseolina* are not known in charcoal rot of sorghum. It would be useful to know if sorghum is susceptible to isolates of the pathogen from other plant species and if physiological races exist among sorghum isolates of the pathogen.

## Biology and epidemiology

Most of our knowledge of the biology of M. *phaseolina* is derived from the 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. In this review only those aspects of the biology that influence the pathosystem will be discussed.

## Inoculum source and survival

At. *phaseolina* is a root-inhibiting fungus with little or no saprophytic growth in soil or in dead host cells of infected plants (Norton 1953; Edmunds 1964). It survives over seasons predominantly as small black sclerotia in diseased root and stem debris or, upon decay of the plant material in which they were formed, in soil. Inoculum density has a direct implication in disease management strategies.

## Drought stress and host colonization

Drought causes harmful physiological or metabolic changes in the plant. It reduces plant vigor; affected plants 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. His hypothesis implies that the interaction of drought stress and pathogens causes stalk rots and lodging. Direct evidence of this has been provided by Henzell et al. (1984).

Recent research (1984-86) in sorghum pathology at ICRISAT Center has further clarified the role of soilbome fungi in root and stalk rot disease. The role of pathogens was examined in nontreated plots and plots fumigated with either granular fumigant tetrahydro-3, 5-dimethyl-2H-1,3, 5-thiadiazine-2-thione or with methyl bromide gas. The importance of soil moisture was studied in plots receiving adequate irrigation up to physiological maturity and in plots where drought stress was created by withdrawing irrigation at the boot-leaf stage. Plant lodging in the plots irrigated until plants matured was low (3-18%), as compared to the drought-stressed treatments (27-100%). However, in the drought-stressed treatments, lodging (100%) was significantly higher in the nonfumigated than in the fumigated plots (Table 2).

Results of plant senescence and visible stem infection by M. *phaseolina* and *Fusarium* spp (especially F. *moniliforme* var *subglutinans)* followed the same pattern as that of plant lodging and soft stalks. Our results show that soil fumigation significantly reduced plant senescence and lodging in the drought-stressed plots, supporting the findings of Henzell et al. (1984) that soilbome root-infecting fungi (especially F. *moniliforme),*

**Table 2. Lodging, soft stalk, and yield of sorghum hybrid CSH 6 and sorghum variety E 36-1 under different treatment combinations of fumigation and drought stress at ICRISAT Center, India, postrainy season 1986/87.**

| | | CSH 6 | | | |
|---|---|---|---|---|---|
| Drought stress | Fumigation[2] | Lodging (%) | Soft stalk (%) | Plot yield [(kg(27m$^2$)$^{-1}$] | Grain mass (g> |
| Stress[1] | MB | 27.4 | 11.3 | 11.6 | 19.1 |
| | BAS | 50.1 | 19.1 | 13.1 | 21.0 |
| | NFC | 99.5 | 96.6 | 8.8 | 17.0 |
| | NF | 100.0 | 99.6 | 9.8 | 17.0 |
| Yield loss (%)[3] | | | | 20.0 | 15.0 |
| No stress[4] | MB | 2.9 | 0.8 | 14.2 | 23.0 |
| | BAS | 12.2 | 3.8 | 15.6 | 33.0 |
| | NFC | 16.4 | 9.3 | 3.7 | 24.0 |
| | NF | 17.6 | 3.7 | 14.1 | 22.0 |
| SE Fum x stress x cv | | ±1.79 | ±1.97 | ±0.86 | ±0.14 |
| | | E36-1 | | | |
| Drought stress | Fumigation | Lodging (%) | Soft stalk (%) | Plot yield [(kg(27m$^2$)$^{-1}$] | Grain mass (g) |
| Stress | MB | 0 | 0 | 12.9 | 27.0 |
| | BAS | 9.6 | 3.4 | 15.6 | 28.0 |
| | NFC | 8.9 | 6.5 | 13.2 | 26.0 |
| | NF | 10.4 | 5.2 | 12.9 | 26.0 |
| No stress | MB | 0 | 0 | 15.7 | 32.0 |
| | BAS | 0 | 0 | 17.0 | 32.0 |
| | NFC | 0 | 0 | 15.4 | 31.0 |
| | NF | 0 | 0 | 15.2 | 30.0 |
| SE Fum x stress x cv | | ± 1.7 | ±2.0 | ±0.86 | ±0.14 |

1. Stress = Irrigation stopped at final leaf stage
2. MB = methyl bromide, BAS = basamid granules, NFC = no fumigation, but covered, NF = no fumigation (control).
3. [(Average yield, fumigated plots - yield, nonfumigated plots)/average yield, fumigated plots] x 100.
4. No stress « irrigation continued until physiological maturity.

along with drought stress, play an important role in the root and stalk rot complex.

Henzell and Gullieron (1973) and Chamberlin (1978), on the other hand, hold the view that plant lodging under drought stress is purely physiological. Drought stress reduced assimilate supply to the lower part of the stalk for maintenance respiration. This results in senescence and disintegration of pith cells, and hence lodging. Drought stress alone can cause lodging without assistance from pathogens where inoculum is absent (Henzell et al. 1984). 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, in the absence of the pathogen, may be tolerated by the plant.

## Crop management

Crop management can influence soil moisture and in turn incidence and severity of root and stalk rots. Sorghums grown in close spacings show more charcoal rot than those in wider spacings. Differences were reported in the effect of plant densities on charcoal rot incidence, attributing increased disease incidence in higher plant populations to increased drought stress. In India, nitrogen fertilization adequate to maximize the yield potential of improved cultivars increased the severity of charcoal rot (Avadhani et al. 1979; Mote and Ramshe 1980).

A factorial experiment using line-source irrigation provided highly significant positive correlations between drought stress and plant density, but not N fertilization (Fig. 2). The effect of nitrogen in increasing charcoal rot may be due to its effect on shoot growth. Nitrogen promotes shoot growth, and partially restricts root development. A restricted root system reduces the ability of a plant to obtain moisture. At the same time its water needs increase, because of the increased foliage growth (Ayers 1978).

Practices that reduce pathogen inoculum and conserve soil water decrease the incidence of charcoal rot. Sorghum grown under wheat-sorghum-fallow rotation had 11% stalk rots, compared to 39% in conventional tillage (Doupnik and Boosalis 1975).

Sorghum growing in a mixed-cropping situation has been reported to suffer less charcoal rot damage than when growing as a sole crop (Khune et al. 1980).

## Control

Incidence of charcoal rot of sorghum can be minimized by maintaining soil moisture during the postflowering stage. This can be done by conservation tillage, seeding lower plant populations, selecting resistant sorghums, assuring good plant nutrition, and using fungicides and plant-growth regulators (such as antitranspirants). Host resistance is the most practical long-term solution for the control of charcoal rot.

## Host resistance

Four essential requirements for the identification and utilization of host resistance to charcoal rot have been discussed by Mughogho (1982). My objective is to review briefly techniques used to identify resistance, resistance sources, and factors associated with resistance. Rosenow (1980,1984), Maunder (1984), and Henzell et al. (1984) have comprehensively reviewed the breeding for host resistance to sorghum root and stalk rot complex.

## Resistance-screening technique

The procedure followed by most investigators to screen for charcoal rot resistance is essentially that reported by Edmunds et al. (1964). Sorghum is grown under irrigation in an environment favoring charcoal rot development. Drought stress is induced by withdrawing irrigation at selected stages of plant growth, and the stalks are inoculated by inserting mycelium- and sclerotia-infested toothpicks into holes punctured into the stalk just above the first node. Lodging, soft stalks, and distance the fungus travels upstem from the point of inoculation are measured to assess 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 natural infection begins in the roots and only later goes up to the stem. The level of disease development with toothpick inoculation is usually less than that occurring naturally; thus stalk inoculation is thought unsatisfactory for meaningful resistance assessment (Edmunds et al. 1964).

At ICRISAT Center we have successfully induced, without artificial inoculation, charcoal rot in field-grown susceptible sorghums. One method is to sow the crop just before the end of the rainy season so that it grows and matures under continuously receding soil moisture. This timing is similar to that of the postrainy season (rabi) crop in India; postrainy season sorghum suffers most from charcoal rot. A second method is to grow the crop, under irrigation, during the dry season and to withdraw irrigation at 50% flowering. In either method, charcoal incidence and severity varies according to location—probably due to soil type, level of drought stress, and the pathogen inoculum potential in the soil. Nevertheless, disease development in susceptible genotypes is sufficiently high for useful evaluation of test genotypes, but a reliable, effi-

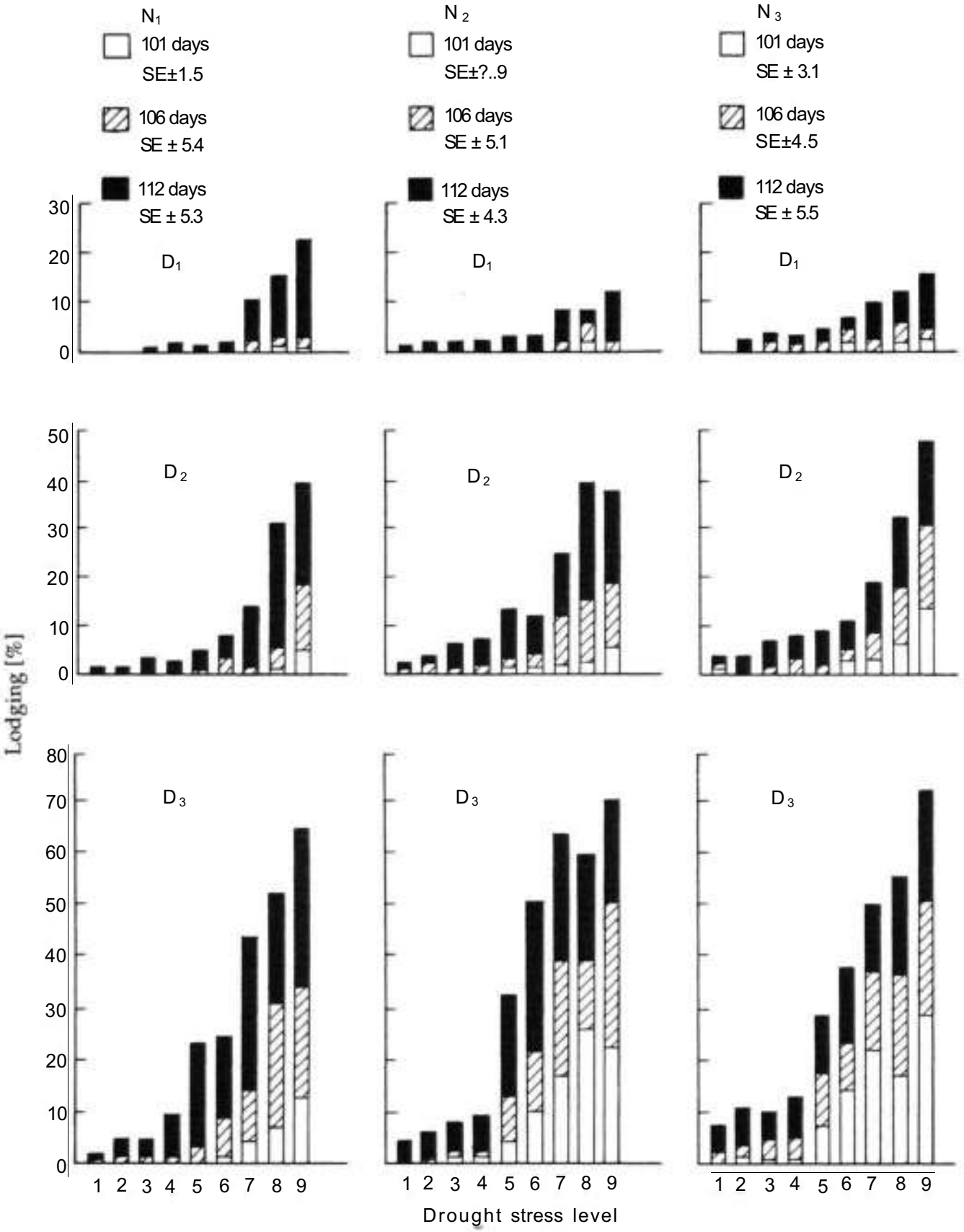



**Figure 2. Periodical lodging (%) in three nitrogen levels ($N_1$ = 20, $N_2$ = 60, and $N_3$ = 120 kg N $ha^{1}$), three plant densities ($D_1$ = 66 675, $D_2$ = 133 350 and $D_3$ = 266 700 plants $ha^{1}$) and nine drought stress levels [1-9, 1($S_1$) = nearest to LS and 9($S_9$) farthest from LS] created by LS at ICRISAT Center, Patancheru, during the 1981/82 postrainy season.**

dent, and epidemiologically sound resistance-screening technique for charcoal rot is yet to be developed.

### Plant characters associated with resistance

The plant character most promising to be positively correlated with charcoal rot resistance is nonsenescence, increasingly being used as a selection criterion. Rosenow (1980) reported significant correlations between nonsenescence, lodging resistance, and charcoal rot resistance in Texas, USA. Similar results were obtained by Mughogho and Pande (1984) at ICRISAT, India. However, they could not find a consistent and stable nonsenescent line among the genotypes tested. Recently we have tested a set of 47 lines (reported nonsenescent in trials in Australia, India, and Mali) at four locations in India (Patancheru, Dharwad, Nandyal, and Bijapur). Four lines from Australia (Q 101, Q 102, Q 103, and Q 104) retained 9-63% green leaf area and were free from root and stalk rot (Tables 3, 4). Stability of nonsenescence would most probably depend on the level of drought stress. Up to a

**Table 3. Time to 50% flowering, green leaf area, plot yield, and 1000-grain mass of four sorghum genotypes (rated as nonsenescents) at four locations in India, postrainy seasons 1985/86 and 1986/87.**

| Genotype | Location | Time to 50% flowering (days) | | Green leaf area (%) | | Plot yield [kg(3m$^2$)$^{-1}$] | | 1000-grain mass(g) | |
|---|---|---|---|---|---|---|---|---|---|
| | | 1985/86 | 1986/87 | 1985/86 | 1986/87 | 1985/86 | 1986/87 | 1985/86 | 1986/87 |
| Q101 | Patancheru | 69 | 59 | 17.5 | 45.5 | 1.4 | 2.3 | 26 | 28 |
| | Dharwad | 58 | 51 | 14.3 | 50.0 | 1.2 | 1.6 | 24 | 29 |
| | Nandyal | 54 | 59 | 40.8 | 30.0 | 0.7 | 1.5 | 17 | 22 |
| | Bijapur | 65 | .[1] | 10.7 | - | 0.6 | - | 19 | - |
| Q102 | Patancheru | 61 | 53 | 14.1 | 34.8 | 0.8 | 1.5 | 22 | 20 |
| | Dharwad | 56 | 51 | 14.3 | 29.2 | 1.1 | 1.7 | 21 | 25 |
| | Nandyal | 57 | 59 | 50.0 | 28.2 | 0.9 | 1.1 | 21 | 18 |
| | Bijapur | 63 | - | 8.7 | - | 0.7 | - | 18 | - |
| Q104 | Patancheru | 67 | 60 | 25.0 | 37.5 | 1.2 | 2.0 | 21 | 20 |
| | Dharwad | 59 | 55 | 17.9 | 38.9 | 1.3 | 1.7 | 20 | 22 |
| | Nandyal | 61 | 69 | 52.3 | 13.3 | 0.7 | 1.3 | 15 | 15 |
| | Bijapur | 65 | - | 9.9 | - | 0.6 | - | 20 | - |
| Q104 | Patancheru | 61 | 52 | 54.2 | 54.9 | 0.8 | 2.5 | 35 | 27 |
| | Dharwad | 57 | 52 | 32.2 | 63.3 | 0.9 | 2.2 | 35 | 36 |
| | Nandyal[2] | 58 | 57 | 63.3 | 15.0 | 1.1 | 1.2 | 213 | 19 |
| | Bijapur | 57 | - | 19.6 | - | 0.5 | - | 32.5 | - |
| Control | Patancheru | 62 | 51 | 0 | 0 | 1.4 | 3.9 | 23 | 27 |
| (CSH 6) | Dharwad | 53 | 46 | 4 | 6.3 | 1.7 | 2.0 | 27 | 22 |
| | Nandyal | 52 | 56 | 0 | 0 | 0.8 | 1.4 | 16 | 18 |
| | Bijapur | 63 | - | 0.2 | - | 0.9 | - | 17 | - |
| SE | Patancheru | ±2.1 | ±0.96 | ±4.2 | ±0.3 | ±1.63 | ±1.3 | ±1.3 | ±1.4 |
| | Dharwad | ±1.5 | ±0.61 | ±5.2 | ±4.53 | ±0.2 | ±0.17 | ±1.5 | ±0.8 |
| | Nandyal | ±2.4 | ±1.31 | ±5.9 | ±3.40 | ±0.1 | ±0.18 | ±1.2 | ±0.14 |
| | Bijapur | ±0.3 | - | ±2.4 | - | - | - | ±1.3 | - |

1. Poor emergence, experiment abandoned.

2. At Nandyal, Q104 was harvested 5 weeks after physiological maturity in 1986/87.

**Table 4. Lodging, soft stalk, nodes crossed, and root infection scores of four sorghum genotypes (rated as nonsenescents) grown at four locations in India, postrainy seasons 1985/86 and 1986/87.**

| Geno-type | Location | Lodging (%) | | Soft stalk (%) | | No. of nodes crossed | | Root infection score | |
|---|---|---|---|---|---|---|---|---|---|
| | | 1985/86 | 1986/87 | 1985/86 | 1986/87 | 1985/86 | 1986/87 | 1985/86 | 1986/87 |
| Q101 | Patancheru | 0 | 0 | 0 | 0 | 0 | 0 | 3.6 | 2.9 |
| | Dharwad | 0 | 0 | 0 | 0 | 0 | 0 | 3.0 | 2.1 |
| | Nandyal | 0 | 0 | 63 | 1.5 | 0.3 | 0.05 | 4.3 | 3.75 |
| | Bijapur | 0 | -[1] | 0 | - | 0 | - | 2.0 | - |
| Q102 | Patancheru | 0 | 0 | 0 | 0 | 0 | 0 | 3.3 | 1.8 |
| | Dharwad | 0 | 0 | 0 | 0 | 0 | 0 | 2.4 | 2.0 |
| | Nandyal | 2.5 | 0 | 3.8 | 1.5 | 0.2 | 0.05 | 4.3 | 2.95 |
| | Bijapur | 0 | - | 0 | - | 0 | - | 1.8 | - |
| Q103 | Patancheru | 0 | 0 | 0 | 0 | 0 | 0 | 3.1 | 2.3 |
| | Dharwad | 0 | 0 | 0 | 0 | 0 | 0 | 2.4 | 2.0 |
| | Nandyal | 0 | 2.5 | 0 | 935 | 0 | 0.25 | 3.7 | 3.35 |
| | Bijapur | 0 | - | 0 | - | 0 | - | 2.3 | - |
| Q104 | Patancheru | 0 | 0 | 0 | 0 | 0 | 0 | 2.72 | 2.3 |
| | Dharwad | 0 | 0 | 0 | 0 | 0 | 0 | 2.5 | 2.0 |
| | Nandyal | 1.8 | 6.85 | 1.3 | 60.72 | 0.1 | 2.9 | 4.7 | 4.5 |
| | Bijapur | 0 | - | 0 | - | 0 | 0 | 1.3 | - |
| Control (CSH 6) | Patancheru | 71 | 100 | 71 | 100 | 2.7 | 4.6 | 5.0 | 4.6 |
| | Dharwad | 73 | 100 | 73 | 100 | 3.1 | 5.0 | 4.9 | 5.0 |
| | Nandyal | 100 | 100 | 100 | 100 | 5.1 | 5.7 | 5.0 | 5.0 |
| | Bijapur | 100 | - | 0 | - | 0 | - | 5.0 | - |
| SE | Patancheru | ±75 | ±6.92 | ±7.4 | ±3.42 | ±0.3 | ±2.06 | ±0.4 | ±0.41 |
| | Dharwad | ±10.8 | ±5.59 | ±11.4 | ±7.47 | ±0.7 | ±0.20 | ±0.4 | ±0.33 |
| | Nandyal | ±4.9 | ±9.56 | ±5.6 | ±8.63 | ±0.4 | ±0.29 | ±0.3 | ±5.14 |
| | Bijapur | ±4.3 | - | ±5.4 | - | ±0.3 | - | ±0.4 | - |

1. Poor emergence, experiment abandoned.
2. At Nandyal, Q104 was harvested 5 weeks after physiological maturity in 1986/87.

specific stress level, a genotype would show stability in nonsenescence at several locations, but beyond that it may not Further research is needed to explain this.

## Resistance sources

Hoffmaster and Tullis (1944), in a most comprehensive testing program, screened 232 sorghum lines of diverse genetic background for 4 years at four locations. Although they found differences in stability to charcoal rot resistance in these lines, data showed no stability in performance of the lines from season to season. They concluded, "it is impossible, from the data available, to recommend certain varieties for localities in which macrophomina dry rot is a limiting factor."

In ICRISAT's charcoal rot research project we have also found inconsistencies in reaction to the disease by a large number of germplasm lines. This lack of stability is due to different levels of predisposition to the disease. However one line, E 36-1, has consistently shown resistance to lodging at several locations in 3 to 5 years of testing. Fungal isolations from roots and stalks showed presence of charcoal rot, but the infection was not severe enough to cause lodging (ICRISAT 1983). Rosenow (1980) identified 13 nonsenescent lines as good sources of resistance to charcoal rot. The stability of these lines in other countries where charcoal rot is a problem needs further evaluation. The need for stable

and better sources of resistance is obvious. Most of ICRISAT's sorghum germplasm collection (more than 20 000 lines) has not been screened, and it is conceivable that these (especially among lines from drought-prone areas) include lines resistant to charcoal rot. However, the priority should be to develop a reliable screening technique that can be used to distinguish resistance from susceptible lines under graded levels of drought stress.

## Fusarium Root and Stalk Rot

Fusarium root and stalk rot caused by the fungus *Fusarium moniliforme* has become an increasingly common stalk rot disease of sorghum in many areas of western Africa (Saccas 1954; Tarr 1962; Zummo 1980). In USA, 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). *Fusarium moniliforme* incites seed rots, seedling blights, and root and stalk rots of numerous crops, including maize, rice, millet, sudangrass, sugarcane, and sorghum (Bolle 1927,1928; Dickson 1956; Bourne 1961; Sheldon 1904; Ullstrup 1936; and Voorhies 1933). F. *moniliforme* affects sorghum plants at all growth stages. It causes diseases such as pokkah boeng.

### Etiology and symptoms

The fungus persists on plant residues that remain in the soil or rest on its surface. Mycelia, conidia, and (in its perfect state, *Gibberella fujikuroi),* ascospores may be produced on or in plants or the soil at any time during the growing season, and secondary infections of host tissue may occur when environmental conditions favor disease development.

Infection starts in the roots, typically involving the cortical and then the vascular tissues. Newly formed roots may exhibit distinct lesions. Older roots are often destroyed, leaving little plant anchorage. If root rot is extensive, the sorghum plants are often easily uprooted.

Fusarium stalk rot is usually accompanied by root damage. Under irrigation and heavy nitrogen fertilization/root damage may not change above-ground appearances before the stalk begins to rot. Stalk rot may reduce seed fill; grain weight losses may be as high as 60%. Fusarium stalk rot apparently requites some predisposing conditions for disease development as plants approach maturity, and is usually most damaging during cool wet weather following hot, dry weather.

Trimboli and Bulges (1983), in greenhouse trials, reproduced basal stalk rot and root rot on grain sorghum plants growing in *Fusarium moniliforme-iniected* soil at optimal soil moisture, then at flowering subjected the plants to a gradual development of severe drought stress between the flowering and the middough stages, followed by rewetting. Stalk rot did not develop, nor was root rot severe, in plants grown to maturity at optimal soil moisture, although many of these plants were infected by F. *moniliforme.* Stalk and root rot developed in the majority of stressed plants grown in soil initially noninfested but contaminated by F. *moniliforme* after sowing.

Fusarium stalk rot can usually be distinguished from charcoal rot by the less-pronounced pigmentation and disintegration of pith tissues and the slower rate of tissue damage by *fusarium.* Charcoal rot may destroy a field of sorghum in 2 or 3 days; Fusarium stalk rot may take 2 or 3 weeks. The appearance of sclerotia in the later stages of charcoal rot is confirmation of its identity.

## Acremonium Wilt

Acremonium wilt, caused by *Acremonium striatum* W. Gams (Syn. *Cephalosporium acremonium* Gord. Gams), has recently appeared in many sorghum-growing regions. *A. strictum* has a worldwide distribution in soil and the atmosphere (Domsch et al. 1980), but as an incitant of disease in sorghum, it has been reported from Argentina (Forbes and Crespo 1982), the Honduras (Wall et al. 1985), and USA (Frederiksen et al. 1980). A *Ceplialosporium* sp reported as the cause of sorghum wilt in Egypt (El-Shafey et al. 1979) was probably *A. strictum,* and the disease probably occurs in additional sorghum-growing countries (Frederiksen 1984). Recently the disease was reported in India (Bandyopadhyay et al. 1987). Similar disease symptoms on a few isolated sorghum plants were observed in the African countries of Lesotho, Zimbabwe, Malawi, Tanzania, Burkina Faso, Mali, and Zambia (UK.

Mughogho, R. Bandyopadhyay, and S. Pande, personal observations), but does not appear to be a serious disease on varieties currently grown by farmers. In the Honduras, however, the disease is important on local landraces (Wall et al. 1985). In maize, A *striatum* (= *Cephalosporium acremonium* Corda) causes wilting of mature plants, infects seedlings (Christensen and Wilcoxson 1966), and internally colonizes grain (Hesseltine and Bothast 1977).

## Symptoms

Symptoms of acremonium wilt include foliar desiccation and vascular discoloration of the lateral leaf veins. Initially only a portion of a leaf is affected, but as the disease progresses, large areas of wilted tissue develop on one axis of the leaf on either side of its midrib. Vascular plugging continues through the leaf sheath and into the vascular bundles of the stalk. In severely affected susceptible plants, the upper leaves, and the shoot die. Vascular browning in the stalk associated with foliar wilting is the most distinguishing symptom of the disease.

## Etiology

Natural infection probably begins in the leaf blades or leaf sheath and spreads through the vascular system. Infection and colonization through roots appear to be the exception (Frederiksen 1984). However, reports from Egypt suggest that the pathogen is soilborne and colonizes the roots prior to invading vascular tissue (El-Shafey and Refaat 1978). Frederiksen et al. (1981) reproduced disease symptoms experimentally through root dippings, soil amending, and hypodermic infections of conidia of *A. strictum* in sorghum whorls. Sorghum cultivars with field resistance (Redlan, Martin, and Wheatland) developed severe wilting when inoculated by these techniques. According to Salama (1979), wilting occurs commonly in regions of Egypt, similarly Frederiksen (1984) observed foliar infections in the farmer's field in the Nile delta near Numberia, Egypt. These authors hold the view that acremonium wilt is not a stalk rot, because *A. strictum* acts like a true vascular parasite. However, stalk-rotting fungi often develop in wilted plants; in this respect *A. strictum* acts as a predisposing agent.

Greenhouse studies of several inoculation methods for the establishment of infection and the disease's systemic colonization of the host plants and transmission through seed were reported by Bandyopadhyay et al. (1987). In pots containing the susceptible cultivar IS 18442, the soil was drenched on the 10th day post-emergence with a conidial suspension of *A. strictum;* more than 70% of the plants became diseased. Disease symptoms in the greenhouse were similar to those in the field, and suggested that root injury was not essential for entry of *A. strictum.* In these experiments, it was possible to isolate A. *strictum* from any part of the infected plants, from roots to grains, confirming systemic colonization of the host. Infected seeds produced diseased plants when sown in autoclaved soil, but the actual potential of seed transmission under field conditions remains to be studied.

## Host resistance

Most sorghum varieties appear to be resistant to acremonium wilt, or the pathogen develops so slowly that it does not cause serious losses. However, tropically adapted sorghums derived from IS 12610 are super susceptible to acremonium wilt. Elite inbreds such as ATx 623 and ATx 625 and many hybrids produced with these inbreds are unusually susceptible in the Honduras (Frederiksen 1984).

## Research Recommendations

1. Crop losses. Quantitative crop-loss data of root and stalk rot diseases on sorghum in farmers' fields are limited or nonexistent It is recommended that systematic crop-loss surveys be conducted in farmers' fields in areas where these diseases are economically important
2. Pathogenesis. Knowledge of host-pathogen environment interactions is scanty. There is need to investigate pathogenesis, with emphasis on temperature, moisture, and nutritional stress as predisposing factors in charcoal rot, fusarium root and stalk rot and acremonium wilt diseases of sorghum.

3. Associated pathogens. Interactions among the different pathogens involved in charcoal rot, different *Fusarium* species in fusarium root and stalk rot complex, and relations between *Acremonium strictum* and stalk rotting and lodging needs detailed study.
4. Reliable field screening. Development of a reliable field-screening technique for these diseases is essential for success in breeding for resistance.
5. Plant-character and disease-resistance relationships. Determine physical and physiological plant characteristics associated with resistance to the pathogen and to lodging. Study the physiological basis of nonsenescence, its stability under different environmental conditions, and its relationship to charcoal rot and fusarium root and stalk rot resistance.

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