SORGHUM DISEASES
IN THE UNITED STATES
AND THEIR CONTROL

Agriculture Handbook No. 468

Agricultural Research Service
UNITED STATES DEPARTMENT OF AGRICULTURE
in cooperation with Kansas and Mississippi
Agricultural Experiment Stations
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**Courtesy Photographs—**

W. Willis, Department of Plant Pathology, Kansas State University, Manhattan. Figures 4B, 4C, 9D, 10, 11, 12, 14A, 15, 18A, and 18B.

R. Frederiksen, Department of Plant Sciences, Texas A&M University, College Station. Figures 4A, 5A, 5B, and 9A.

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SORGHUM DISEASES
IN THE UNITED STATES
AND THEIR CONTROL

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IMPORTANCE OF SORGHUMS IN U.S. AGRICULTURE

Sorghum is one of the least known of our major food crops. Many people know sorghum as a source of syrup which uses a small part of the crop. The bulk of the crop is grown for livestock feed. Sorghums are perennials in frost-free climates. In the United States, however, all except johnsongrass normally winterkill; therefore, they generally are treated as annuals. Some sorghums, such as kafirs, hegari, and most late-maturing sorghums, have excellent drought resistance because they tend to become dormant when moisture becomes deficient. Such dormancy, however, is not typical among feteritas, kaoliangs, milos, and most early-maturing sorghums. Because of drought tolerance, sorghums are grown most extensively in the southern half of the Great Plains. Considerable acreage is planted to sorghum in adjoining States on soils considered too risky for corn. Significant acreage also is grown in several southwestern and southeastern States.

In the United States, four general classes (41) of sorghums generally are recognized:

1. Grain sorghums (*Sorghum bicolor* (L.) Moench.).—These have relatively large kernels that are readily separated from the glumes. The stalk juice has little or no sweetness. Grains are borne on relatively large, heavy panicles, and they are red, brown, yellow, or white. Parentage of most present-day grain sorghum hybrids is from one or more groups referred to as milo, kafir, feterita, hegari, shallu, and durra. Short stature required for combine harvesting has been accomplished by using one or more of four major dwarfing genes (51). Taller types such as early hegari and blackhull kafir are grown mainly for silage. Sorghum grain is used extensively in fattening beef at commercial feed

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1 Italic numbers in parentheses refer to Literature Cited, p. 42.
lots. The poultry industry, as it is known today, would not exist without sorghum grain as a major part of feed rations. Considerable sorghum grain also is fed to hogs.

2. Sweet sorghums (S. bicolor).—These have in common tall, juicy sweet stalks. Kernels usually are smaller than those of grain sorghums and are more difficult to separate from the glumes. Sweet sorghums are grown mainly for stalk juice extracted to produce syrup or sugar. Some such varieties as Atlas and Tracy are used extensively for silage and sometimes for forage.

3. Grass sorghums.—Compared with grain and sweet sorghums, grass sorghums have slender stems, narrow leaves, numerous tillers, and small seeds fully enclosed by glumes. Nearly all types have lax, open panicles. Sudangrass (S. sundanense (Piper) Staph.), including sudangrass-sorghum hybrids, is the only type cultivated extensively. It is used for pasture, greenchop, and hay. Johnsongrass (S. halpense (L.) Pers.) is cultivated to some extent for hay and pasture, but it thrives primarily as a weed in row crops, right-of-ways, and wastelands. Johnsongrass differs from most other sorghums by having rhizomes that maintain it as a perennial in relatively cold climates.

4. Broomcorns (S. bicolor).—Panicles of broomcorn sorghums are characterized by an extremely short rachis and long rays or branches. The kernels are small and mostly enclosed in long, ellipsoid glumes. With the seed removed, the long panicle branches are a material source in industrial and domestic brooms. Use of synthetic fibers and mechanical cleaning machines, however, has reduced demand for broomcorn fibers considerably. Less than 20,000 tons are produced annually (73).

Sorghum was introduced from Africa to various parts of the western hemisphere during the 17th and 18th centuries (41). The culture of broomcorn for domestic use was a considerable industry in colonial times. Systematic varietal introductions apparently began with culture of sorghum for syrup. Those sorghums were used by early settlers as a source of sweetening or "long sugar." Chinese Amber was introduced from France in 1853, and it was followed, four years later, by fifteen additional sweet sorghums from South Africa. Use of sweet sorghums as a source of sweetening by pioneers led to use of sorghums as forage.

Commercial grain sorghum production followed introduction of two durras from Egypt in 1874, two kafirs from South Africa in 1876, shallu from India about 1870, and milo from East Africa via Columbia in 1879. Other important introductions were pink kafir from South Africa about 1905, and feterita and hegari from the Sudan in 1906 and 1908, respectively. Blackhull kafir of uncertain origin was being grown after 1890.
From those and other introductions, grain sorghum was improved by selection during the first part of this century. Several widely adapted varieties were available by 1920 when a record crop of more than 87 million bushels was produced on 4 million acres \( (68) \). Prolonged drought and insect and disease problems prevented a repeat of that record for 20 years. Then World War II required increased acreages in food crops and sorghum production increased rapidly. In the postwar period, combine harvesting, multiple-row planting equipment, and improved tillage and cultivation machinery made continued large acreages as cash crops feasible. The excellent yield response of sorghum to ample soil moisture and high soil nitrogen levels resulted in much larger acreages of the crop on irrigated and summer-fallowed land.

Concurrently, varieties continued to be improved through hybridization and selection. Such varieties, together with the better cultural operations, set the stage for the production of \( F_1 \) hybrids, which were made possible by discovery in 1952 of cytoplasmic male sterility \( (63) \). Hybrids now yield more than 700 million bushels annually on about 14 million acres \( (69) \). As a feed grain, sorghum is second only to corn in total production. This led to the development of the Great Plains as a major meat-producing area, the South as a major poultry-producing area, and an important export industry.

Sudangrass was introduced from the Sudan in 1909 \( (41) \). In recent years, sudangrass has gained added importance as a pollinator of cytoplasmic male-sterile sorghums to produce sorghum-sudangrass hybrids.

Johnsongrass was introduced from Turkey in 1830 as a forage crop \( (40) \). It has spread throughout most areas where sorghum is grown and has become a major weed pest. In addition, johnsongrass has become implicated as a carryover host for numerous sorghum diseases, especially maize dwarf mosaic and its vectors.

**IMPORTANT OF SORGHUM DISEASES**

It is an irony of agriculture that as cultivation of a given crop species becomes intensified, also its vulnerability increases to pests and diseases. The period of introduction of most original sorghum varieties, 1853-1906, was marked by few disease problems. As sorghum production increased during the 1920's, *Periconia* root rot, which is extremely destructive to the milo varieties, and kernel smut became important diseases. During the prolonged periods of hot, dry weather of the next decade, weak neck and stalk rots aroused concern. Under intensified cropping during World War II, seed rot, damping-off, and seedling blight became important. Losses from many of those diseases
were reduced by resistant varieties, seed treatment, and cultural practices.

The development of grain sorghum hybrids not only brought increased production but also increased disease problems. Soon after large acreages were planted to newly developed hybrids, head smut became a serious problem, especially in southern Texas (53). Resistance was available by 1964 (56), but a new race appeared in 1966 (24). Maize dwarf mosaic (MDM) also appeared in 1966, and became epidemic on the High Plains in 1967 (57). The threat of MDM was intensified in 1968 with appearance of a new greenbug biotype. This is a more efficient vector of the virus (MDMV) than is the corn-leaf aphid, which heretofore had been the principal vector (10, 29). Sorghum downy mildew was first observed in 1961 in southern Texas and it has since spread to several other States (26). In 1970, the possibility of a new strain of *Periconia circinata* virulent to many sorghum hybrids was reported from Arizona (72).

Although available information on losses from diseases is not totally accurate, those losses rank with losses from weeds and insects in reducing sorghum yields. The most recent estimate of annual reduction in yields from diseases was 9 percent (34). Like most crops, sorghum is subject to some diseases that are inconspicuous. For example, disease damage to roots and stalks particularly is often overlooked or blamed on unfavorable weather or poor soil conditions.

The purpose of this handbook is to make information on sorghum diseases more readily available, to promote better appreciation for the existence and prevalence of diseases, and to show the need for continued development of safeguards against the losses diseases may cause.

**THE PHENOMENON OF DISEASES**

Diseases of sorghums, like those of other crops, vary in severity from year to year and from one locality or field to another, depending on environment, causal organism(s), and host resistance. Misconceptions of the nature of disease often arise because the interdependency of those factors is not adequately considered in disease development.

Environment is critical to disease development, to a degree that varies considerably among individual diseases. In general, soil temperature and moisture are most critical for soilborne pathogens, which attack roots and stalks or infect young shoots before they emerge. Air temperature, moisture, wind, and light are most critical for airborne pathogens, which attack foliage, stalks, and inflorescences.
No disease will occur, however, if the causal organism or pathogen—a virus, bacterium, fungus, or nematode—is absent, even though the environment is favorable for disease development and the host is susceptible. The causal organisms may vary in virulence as much as the hosts they attack vary in relative resistance. Some pathogens, especially those attacking roots and stalks, have little or no specificity, and they attack many plant species in addition to sorghum. Others, such as smut fungi, are highly specific and attack only certain varieties of sorghum. Most have intermediate specificity and attack several grasses, only corn and sorghums, or only certain types of sorghum. As the intimacy between parasites and host increases, so usually does the specificity of both for each other.

Resistance or susceptibility of various sorghum genotypes to a given pathogen often reflects the genetic nature of virulence in the pathogen. For example, the first resistance chosen to control the common race of the head smut fungus was imparted by a single dominant gene (56). For such a pathogen as *Sphacelotheca reiliana*, however, that type of resistance can be overcome simply by buildup of a race that can attack the new resistance. Such resistance is used when the pathogen has little capacity for producing new strains or when quick (although temporary) control is needed (71). For such a disease as charcoal rot, on the other hand, such resistance seems unlikely because the pathogen involved has little specificity for plant species or varieties it attacks. Developing resistance to such a pathogen is difficult because of little chance of finding resistance that is simply inherited. If, however, it is possible to separate out genotypes that tolerate such a nonspecific pathogen, such tolerance probably would be durable and would justify the extra time and effort required to obtain it.

**COEXISTENCE WITH DISEASE PROBLEMS**

Sorghum probably always will have diseases. It is not feasible to eradicate them, but rather to minimize disease losses by discrete control methods requiring cooperation of those most directly concerned with sorghum production—producers, seed companies, pesticide manufacturers, and extension and research personnel.

Producers are in the best position to monitor crop health and report any decline or abnormality, as soon as observed, to county agents and experiment stations. The relative importance of disease problems could be evaluated much better by those responsible for developing control measures if they were aware of such observations. Adequate records kept by the producer on his crop culture would help pathologists assess a disease situation developing in a given area or over a period of time.
Availability of such information could do much toward aligning priorities for research programs directed at disease control.

Appraising crop health regularly is important. Every producer should keep abreast of what diseases are potentially most dangerous in his area and know how to recognize symptoms. This handbook should help producers, associated private industries, and extension personnel to recognize symptoms and relative potentials of various diseases that can cause crop losses.

Control of sorghum diseases depends heavily on development of disease-resistant genotypes, using simple and multigenic resistances available in many breeding nurseries and in a world collection of sorghum germplasm (51). Usually, resistances first are identified in nurseries established at locations where each disease can develop naturally. When incidence of a given disease is high among susceptible genotypes in such nurseries, it is relatively simple to identify and preserve the resistant genotypes. If, however, using natural infection to identify resistances is either too slow or too unreliable, it may be necessary to inoculate artificially and to produce epidemics under controlled conditions. Artificial inoculation is particularly effective if reactions to a particular disease can be obtained from seedlings to minimize time and space requirements.

Incorporating identified resistances is a final step in developing resistant hybrids or varieties. When a single disease is involved, resistances controlled by one or a few genes can be incorporated by simple crossing and backcrossing. When incorporation involves resistance to several diseases or multiple-gene resistances to few diseases into one genotype, other methods must be used. Currently, one method is random-mating populations, where by use of suitable male-steriles, resistances inherent in many selections are brought together in single genotypes (58).

Cultural control methods are used when resistance to disease is unavailable or incomplete. New strains of highly specific pathogens that can reproduce rapidly or pathogens having little or no specificity for sorghum cultivars may require that type of control (71). For example, one should avoid growing (and incorporating into the soil) varieties supersusceptible to such diseases as downy mildew and head smut, which are initiated each season by soilborne spores. Destroying diseased plant refuse may reduce losses from some foliage pathogens if fields are isolated sufficiently. Little is known of the effects of modern cultural practices on incidence of root and stalk rots; but it has been suspected that increased incidence of those diseases may relate to some cultural practices, especially heavy applications of nitrogen. Sometimes such weeds as johnsongrass and such insects as aphids are so involved in
spreading disease that they must be controlled, even though their direct damage is minimal.

Chemical control of disease in sorghum is not feasible, except by seed-treatment for such diseases as the kernel smuts, seed-rot, damping-off, and seedling blight. Before offering seed for sale, most seed industries routinely apply, by the slurry method, captan or thiram-type fungicides. Development of new systemic fungicides having long-lasting protection, however, may lead to other practical means of chemical control.

**SEED AND SEEDLING DISEASES**

Young sorghum plants are much more delicate than corn and also grow more slowly. It takes at least 10 days to establish permanent, adventitious roots (25). Until then, anchorage and nutrients must be obtained by a relatively diminutive and transitory primary root system and a few adventitious roots arising from the coleoptile node. Under less than optimum growing conditions, that root system is highly vulnerable to soilborne pathogens.

Sorghum mortality during the first month after planting continues to be a problem in many areas. Unlike most cereal seed, sorghum seed showing high viability (90 percent) in standard germination tests often exhibits drastically reduced germination (50-70 percent) in the field. Although soil crusting or surface desiccation sometimes can be blamed, seedborne or soilborne fungi that attack seed or seedling parts usually are responsible. Cold rains, poor aeration, competition with weeds, and toxic plant residues can offset seed treatment given as protection against those fungi. Resulting failure to get good stands often leads to excessively high planting rates, which in turn can introduce other such problems as stress and stress-related diseases at later stages of growth.

**Preharvest and Postharvest Problems**

Even before harvest, sorghum seeds are highly susceptible to infection with airborne field fungi (5). Although most of those fungi do not survive storage and act directly as pathogens of the next crop, they may alter or damage the seed so that other fungi can become pathogenic. Because sorghum seeds are largely exposed and closely grouped on large panicles, they provide an ideal site for fungal growth, especially if humid conditions prevail as the grain matures. Honeydew, and natural plant foods on seed surfaces further enhance fungal growth. If the moisture content of seeds is above 18 percent, numerous fungi may penetrate pericarps and invade endosperms.
**Alternaria**, the most common field fungus that attacks seed in the Great Plains, may be found as early as soft dough, and it is prevalent (70 to 90 percent) at later stages of seed development. Infestation often is severe under cool, wet conditions, when seed surfaces become blackened with sporulating fungus. *Cladosporium* also is quite prevalent (30-50 percent) in seeds at harvest. Other fungi frequently isolated (5-10 percent) include *Fusarium*, *Chaetomium*, *Curvularia*, and *Helminthosporium*.

Under such farm practices as high-moisture harvesting and reconstituting grain for cattle feed, some field fungi can damage seed severely after harvest. Three genera of field fungi found in sorghum—*Alternaria*, *Cladosporium*, and *Fusarium*—are known to produce toxic metabolites in feedstuffs. When seed is stored at 18 to 24 percent moisture content, growth of *Fusarium* increases. Storing such seed at temperatures below 50° F, however, will largely prevent growth of those fungi.

Such airborne fungi as *Aspergillus glaucus* Lk. ex Fr., *A. flavus* Lk. ex Fr., and *A. candidus* Lk. can attack seed stored at a moisture content lower than 18 percent (8). Normally, those fungi contaminate the seed as it is put into the storage bin. Mechanical damage to the seedcoat during harvesting and storing increases chances for fungal invasion. Stored seed also may be attacked when the minimum moisture content is 15.5 percent during the first 90 days, or when the moisture content remains above 14.5 percent during the first 150 days. When embryos become infected, grain deteriorates rapidly from seed grade to feed grade.

**Postplanting Problems**

Some fungi may act directly as seedborne or soilborne pathogens of the next crop. Some seed rots even before germination. *Aspergillus niger* V. Tiegh. and *Rhizopus nigricans* Ehr. most commonly cause seed rot, especially when inferior seed is subjected to cold, wet soils soon after being planted (37). At low temperatures, those fungi sometimes cause seedling blight. *Penicillium oxalicum* Currie & Thom, a seedborne and soilborne fungus, has caused seed rot at 60° F and severe seedling blight at 70° to 80°; oxalic acid produced by that pathogen is believed to cause young leaves to discolor, wilt, and die. *Fusarium* species, mostly *F. moniliforme* Sheld., probably causes the greatest plant damage, often by destroying small root branches and causing red to black lesions of various sizes and shapes on large roots. Plants appear stunted and often show pigmentation with anthocyanins characteristic of the sorghum genotype.

*Pythium* spp., mostly *P. arrhenomanes* Drechs. and *P. debaryanum*
Hesse, are soilborne fungi that cause damping-off and seedling blight, especially in cold, wet soils. Characteristically they cause roots to become flaccid and necrotic, converting root cortical tissues to a mushy consistency. Typically, infected roots produce less red pigment in reaction to damage from *Pythium* than in reaction to that from *Fusarium*. Pigmentation of aboveground parts, however, may occur as in other seedling diseases or from cold injury alone.

**FOLIAGE DISEASES**

Except for virus diseases, severe damage to foliage from diseases is most likely to occur under prolonged humid conditions. Sorghums grown for forage or sugar are most directly affected by leaf damage. Grain sorghums usually can sustain considerable damage to foliage without yield losses, unless that damage occurs among upper leaves at the time the grain is filling.

**Insecticide Injury**

Some sorghum varieties are susceptible to injury by insecticides, especially those applied to cotton crops: methyl parathion, toxaphene, and some organic phosphates. Drift from airplanes applying such insecticides on cotton or other crops may cause widespread injury to sorghum less than one-half mile from treated fields. Within 24 hours after the chemical is applied, insecticide injury appears as irregular circular or elliptical water-soaked spots on any part of the leaves. Those spots dry out and turn reddish-to-blackish purple on the margins within 72 hours (fig. 1).

When a large quantity of insecticide is applied to a susceptible variety, large portions of the leaves may be destroyed. Sorghum plants generally outgrow the injury and new growth will not be affected. Repeated applications, however, of insecticides may severely stunt or kill plants of susceptible varieties. Insecticide injury often may be identified by noting, where leaves cross one another, a band free from spotting on the lower or protected leaf.

Varieties of grain sorghum, sweet sorghum, and forage sorghum having high resistance to insecticides are available. If sorghum is planted near cotton or other fields where airplane applications of insecticides are made, varieties should be used with known resistance to insecticides.

**Virus Diseases**

Several viruses have been isolated from sorghum, but until the development of maize dwarf mosaic (MDM), none were considered
economically important. In the South, mosaic incited by the sugarcane mosaic virus (SCMV) for many years has been present on corn, sorghum, and broomcorn (4). The disease generally has been confined to areas where from year to year that virus is harbored in infected sugarcane plants (*Saccharum officinarum* L.). Before 1964, except for two reports (1, 33) of single infected plants, sugarcane mosaic had not been found on or transmitted to johnsongrass.

Since 1964, MDM, a similar mosaic disease of corn and sorghum, has become widespread in the United States where sugarcane has not been grown (74). The incitant virus, maize dwarf mosaic virus (MDMV), readily infects johnsongrass and numerous other grasses (20). Where found on johnsongrass, MDMV also may be found on sorghum. In the field, MDM and sugarcane mosaic are indistinguishable on sorghum. The viruses causing both diseases are transmitted by aphids in a styletborne, nonpersistent manner (49). Although the viruses can be transmitted readily by several species of aphids, the corn leaf aphid (*Rhopalosiphum maidis* Fitch) and the greenbug (*Schizaphis graminum* Rond.) currently are the most important. Both aphids commonly are found on sorghum and johnsongrass. It has been suggested that MDMV probably arose as a mutant of SCMV in an area where the latter is endemic in native grasses or in sugarcane growing in proximity to johnsongrass (61). Considerable experimental evidence supports the belief that MDMV and SCMV are closely related.

On sorghum, the mosaic symptom caused by both viruses normally
is most evident on the upper two or three leaves as an irregular mottling of dark- and light-green areas often interspersed with longitudinal white or light-yellow streaks, (fig. 2, A). In sorghums that carry genes for red pigmentation, mottling in infected plants, especially on abnormally cool nights, may be replaced by a red-leaf symptom—usually elongated stripes with necrotic centers and reddish margins (62, 76) (fig. 2, B). Rarely, round to irregular spots may appear. The red-leaf symptom may be confused with advanced symptoms of bacterial diseases.

In mosaic-affected plants, however, lesions never appear water-soaked nor do they exhibit exudate as in some bacterial diseases. Plant reaction to the disease varies considerably, depending on time of infection. Plants infected as seedlings are more severely affected than plants infected after 5 or 6 weeks of growth. In the most severe cases, plants may die. Also, growth may be stunted, flowering delayed, and the plants may fail to head or set seed.

When johnsongrass, which serves to overwinter both MDMV and its vectors, breaks dormancy in the spring, aphids feed on it and acquire the virus from infected plants. The aphids then move to nearby crop plants and transmit the virus to them. After feeding on infected plants, aphids may retain infective virus up to 20 minutes (49). Buildup and migration of aphids from field to field can result in extensive spread of the disease in a relatively short time.

Different isolates or strains of both the sugarcane mosaic and the

Figure 2.—Maize dwarf mosaic: A, typical green and chlorotic mottle on leaves; B, pigmented-necrotic reaction on leaves.
maize dwarf mosaic viruses vary considerably (77). Strains that have become localized in certain areas are mutants. Even within a given area there may be several variants. Those different variants have appeared so rapidly, becoming endemic in certain areas, that new strains probably will continue. Currently, no sorghum varieties can resist all virus infection. Some varieties, however, possess high tolerance to mosaics and when infected, even at an early stage of growth, can produce acceptable yields of grain and stalks. Some varieties show enough resistance to infection to permit (despite some stunted, infected plants) satisfactory yields from plants remaining uninfected. Also, there is evidence that some sorghum breeding lines may be resistant to particular mosaic strains.

**Bacterial Leaf Diseases**

Three bacterial leaf diseases—bacterial stripe, bacterial streak, and bacterial spot—are found on sorghum (13). Though conspicuous on the plants, these diseases generally do little damage in relation to the leaf area affected. Bacterial leaf diseases are most prevalent during warm, humid weather. Bacteria responsible for those diseases are carried over from season to season on seed, on infected plant material, in the soil, or on johnsongrass. In view of improved seed technology and seed-certification programs of the various States, it is unlikely that those diseases are spread through commercial seed-distribution channels. All three pathogens are spread by wind, wind-splashed rain, insects, and cultivation when foliage is wet. Pigments characteristic of host genotypes are produced by infected tissue.

Bacterial leaf diseases are controlled by using clean seed, crop rotation, and clean cultivation to destroy plant residues. Varieties differ in susceptibility to the diseases, but distinctly resistant varieties have not been developed.

**Bacterial stripe.**—Incited by *Pseudomonas andropogoni* (E. F. Sm.) Stapp and found wherever sorghum is grown, bacterial stripe disease is characterized by tan-brick-red to dark-purplish-red stripes. These generally are restricted to the interveinal areas of apical portions of the lower leaves (fig. 3). Ranging from less than one inch long to the length of leaf blades, the stripes sometimes fuse so that large areas of the leaf are affected. A slime or bacterial exudate may be found on the underside of affected portions of the leaves and along the leaf margins. The shape of the stripes generally is the same on all varieties, but the color varies. Some varieties exhibit a dark or purplish-blackish-red reaction, whereas others always show a tan reaction.

**Bacterial streak.**—Bacterial streak, incited by *Xanthomonas holcicola* (E. C. Elliot) Standard Burkh., occurs on sorghum leaves and
Johnsongrass as narrow, water-soaked, translucent streaks about 1/8 inch wide by 1 to 6 inches long. Those streaks may develop on plants anytime between the seedling stage and near maturity. At first no color is evident except the light-yellow, beadlike drops of exudate standing out on the young streaks. Later, narrow, red-brown margins or blotches appear in the streaks; after a few more days, the streaks are red throughout and no longer appear water-soaked or translucent. Parts of the streaks may broaden into elongated oval spots with tan centers and narrow, red margins. When numerous, the streaks may join to form long, irregular areas covering much of the leaf blade. At that advanced stage, dead tissue with dark, narrow margins forms between the reddish-brown streaks, and the bacterial exudate has dried to thin white- or cream-colored scales.

Bacterial spot.—Incited by *Pseudomonas syringae* van Hall, bacterial spot attacks sorghum leaves, broomcorn, sudangrass, johnsongrass, pearl-millet, foxtail millet, and corn. On sorghum the spots appear first on the lower leaves, and infection gradually spreads to the uppermost leaves as the plants approach maturity. The spots may occur on any part of the leaf, usually are circular to irregularly elliptical, and are from one-twenty-fifth to one-third inch in diameter. Appearing dark green and water-soaked at first, they turn red in a few hours, then soon become dry and light-colored in the center (retaining a red border). Most small lesions are red throughout, having tiny, somewhat sunken centers, but the color bordering the lesions varies somewhat in different varieties. For example, lesions on shallu are dark brown instead of red. Frequently the spots are so numerous that they unite into large diseased areas and the whole leaf dies.
Downy Mildews

Three downy mildews—sorghum downy mildew, green ear, and crazy top—affect sorghums and corn in the United States. Caused by fungi that are obligate parasites, their sexually produced spores (oospores) remain viable in the soil for many years. All are regarded as indigenous to tropical Asia or Africa and probably were introduced to this country with seed. Although each disease has distinctive symptoms, all may incite hypertrophy or grotesque growth patterns of plant parts, especially of inflorescences.

*Sorghum downy mildew.*—The most important of the downy mildews, sorghum downy mildew is incited by *Sclerospora sorghi* Weston and Uppal and is relatively new in the United States. First observed in 1961 in Texas, it did not cause serious damage until 1967 (21). Its known distribution now extends west to Arizona, north to Kansas, and east to Tennessee and Georgia (26). Although the distribution still is limited, the potential for further spread and damage is considerable. That potential includes corn in the Corn Belt (22). Once the fungus is introduced and established in a given area, eliminating it is unlikely.

The most conspicuous symptom of sorghum downy mildew is the appearance of vivid green and white stripes on the leaves in late spring or early summer (fig. 4, A). Whole leaves may become chlorotic, or they may show just one or two narrow green stripes. The chlorotic tissue results from systemic infection, either from soilborne oospores or sometimes from heavy infection by conidia (asexually produced spores). Systemically infected plants usually fail to head. If heads are exerted, they are small, compact, or club-shaped and have little or no seed set.

Close examination of the underside of chlorotic leaves during humid weather will reveal presence of white "down" made up of conidia (fig. 4, B) and structures (conidiophores) on which they are borne (fig. 5, A). Conidia usually are produced only at night when free moisture is on leaf surfaces for several hours. They are produced abundantly when long dew periods are associated with cool nights (65°-70° F)—a new crop every day, with one leaf supporting the production of billions of conidia.

Conidia, spread by wind, initiate local infections on healthy leaves (4, C). Viable conidia may be carried several thousand feet from where they are produced, but most remain within a few inches or feet of the infected leaves. Local infections from conidia first appear on the leaves as small, rectangular, chlorotic spots, which rapidly become pigmented and necrotic. Conidia also are produced by those local infections.
Secondary spread of the disease is possible under certain conditions resulting in whole leaves becoming affected and causing a general blight of a crop. In the South where conditions do not favor conidial infections until late in the season, their direct effect on plant

Figure 4.—Sorghum downy mildew: A, chlorotic stripes associated with advanced systemic infection of leaves; B, “down” on lower leaf surface; C, local lesions from infections by airborne conidia; D, shredding of leaves with chlorotic stripes.
development is negligible. If conidial infection occurs early enough in the growing season, unexpanded leaves may be infected systemically.

Oospores develop only in systemically infected tissue (fig. 5, B). They are released to the soil when that tissue is shredded by winds or is incorporated into the soil by cultivation (fig. 4, D). When sorghum is planted in a field infested with oospores, young seedlings become infected at or soon after germination (32). The infected seedlings that emerge show a chlorotic mottle, which is similar to, though usually distinguished from, mosaic caused by a virus with the appearance of down when free moisture is present. Those plants that do not die within the first few weeks generally survive as systemically infected plants to repeat the disease cycle.

Susceptibility to sorghum downy mildew varies considerably among sorghum types (23). In general, sudangrass is highly susceptible. Most feterita and broomcorn varieties are relatively susceptible. Sweet sorghums, kafirs, milos, and hegari tend to be resistant. Most grain sorghum hybrids with yellow endosperm parentage are quite suscep-
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tible. Sorghum downy mildew is partly controlled by using resistant varieties wherever the disease occurs. In such areas, grain sorghum should not be planted after sudangrass. If possible, cultivating sudangrasses and sorghum-sudan hybrids should be avoided where sorghum downy mildew is a problem. Seed production, especially for sudangrasses, and sudangrass-forage hybrids, should be conducted in areas remote from those where the disease occurs. Resistant sweet sorghum varieties and grain sorghum hybrids currently are available, and they should be used where the disease is prevalent.

*Green ear disease.*—Green ear, incited by *Sclerospora graminicola* (Sacc.) Schroet., occurs on sorghum only during extended periods of extremely high humidity. *S. graminicola* produces “down” on the leaves similar to that produced by *S. sorghi*, but it is named after a condition in which the head is transformed (or partially transformed) into a loose, green head composed of a mass of small, twisted leaves. Generally of little economic importance on grain sorghum, the disease occasionally severely affects fields of sweet sorghum and broomcorn.

*Crazy top.*—Crazy top, incited by *Sclerophthora macrospora* (Sacc.) Thirum; Shaw and Naras., occurs sporadically on sorghum, corn, and many grasses in the United States (59). It generally occurs only when soil becomes flooded or waterlogged from heavy rains or irrigation before the seedlings emerge and become established. Saturated soil apparently is necessary for germination of oospores, which then release infective zoospores (motile). *S. macrospora* produces abundant oospores on sorghum, but only rarely produces conidia (fig. 6). There generally is no secondary spread of the disease as in sorghum downy mildew.

Sometimes young sorghum plants exhibit mottling of upper leaves that closely resembles mosaic of virus-infected plants. The absence of “down” can make early diagnosis of this disease in the field difficult. Infected plants eventually develop thick, stiff, twisted, or curled leaves (with puckered surfaces and finally with long yellow-tan-brown stripes), which usually do not shred as do leaves of plants affected with sorghum downy mildew or green ear disease (fig. 7, A). Infected plants often fail to head, and the heads that are produced may be barren or show proliferation of floral structures (fig. 7, B). Oospores produced in deformed leaves or heads serve to perpetuate the disease from one year to the next.

Attempts to control crazy top may not be warranted, but poorly drained areas should be avoided where the disease is troublesome. So far, the disease has caused little trouble in irrigated fields, but irrigating too soon after planting could cause problems. Grain sorghum hybrids vary in susceptibility to the disease and some are quite resistant.
**Helminthosporium Leaf Blights**

For many years, northern leaf blight (incited by *Helminthosporium turcicum* Pass.) and southern leaf blight (incited by *H. maydis* Nisikado and Miyake) have been known on corn and sorghums in the humid areas of the United States. Those diseases are much more damaging to corn than to sorghum; although, under proper environmental conditions, they can severely damage some of the sorghums, including sudangrasses. Isolates of *H. turcicum* that attack corn, however, generally are not pathogenic on sorghum.

**Northern leaf blight.**—This leaf blight appears on the leaves of sorghum plants as elliptical, grayish or tan spots several inches long. Seedlings may be infected and in severe cases many die. Under favorable conditions, those spots enlarge sufficiently to kill large parts of the leaves, which then wither so much that plants may appear to suffer from frost injury. The centers of the spots usually are grayish to straw-color and, depending on the sorghum variety, have reddish-purple
or tan borders. During humid weather the fungus sporulates readily on those leaf areas, and the spores are spread by wind. Those spores, after overwintering on diseased plant refuse left on the ground after harvest, serve as primary inoculum the next growing season. The fungus also attacks johnsongrass, which in some areas serves to carry over the disease from year to year.

Grain and sweet sorghum varieties have satisfactory resistance to northern leaf blight. Kafir and kafir hybrids are somewhat resistant. Piper and Tift sudangrass are relatively resistant if the inoculum potential is not overwhelmingly great. Minimum tillage practices are questionable if the disease is a problem; refuse should be plowed under in the autumn soon after harvesting the crop. This disease can predispose plants to stalk rots by other pathogens. In the humid areas of the South where it has been a problem, most varieties will sustain some leaf blight.

Figure 7.—Crazy top downy mildew: A, foliar symptoms; B, proliferated inflorescence.
Southern leaf blight.—Southern leaf blight generally can be distinguished from northern leaf blight by the similar but smaller lesions, or spots, on the leaves. Those angular lesions, sometimes limited by the leaf veins, range in width from small flecks to 1 1/2 inches. Occasionally, lesions merge to cover large areas of the leaves.

Southern leaf blight is not as damaging to sorghum as is northern leaf blight. During the southern leaf blight epiphytotic of corn in 1970, almost all sorghums showed slight infection. *H. maydis* does not sporulate readily on sorghum nor does it infect or sporulate readily on johnsongrass. On corn, however, the fungus sporulates profusely and in addition, produces abundant sexual spores (*Cochliobolus heterotrophus* (Drechsler)Drechsler). The sexual cycle in which those spores are produced facilitates segregation of possible strains virulent to sorghum.

Anthracnose and Red Rot

Anthracnose and red rot are two phases of one of the most destructive diseases of sorghums in warm, humid regions of the United States. The disease is incited by the fungus *Colletotrichum graminicolum* (Ces.) G. W. Wils., which also attacks corn, johnsongrass, and sudangrasses. Anthracnose and red rot generally are most prevalent in areas where periods of high humidity alternate with relatively dry periods.

The anthracnose phase, which may occur at any stage of plant development, usually appears on the leaves of susceptible varieties in midsummer as small circular-to-elliptical spots one-eighth to one-fourth inch in diameter. The spots are well defined and, depending on the variety affected, they may be tan, orange, red, or blackish purple. Under conditions of high humidity the spots increase in number and enlarge to cover much of the leaf area.

Midrib infection commonly occurs as elliptical-to-elongate, discolored lesions, which frequently coalesce to cover the entire length of the midrib (fig. 8, A). On many of those spots, a blackish growth (visible to the naked eye) examined under magnification is seen made up of short, stiff hairs (setae) that are part of the fruiting bodies of the fungus. Anthracnose may defoliate sorghum plants markedly, reducing growth and further development. In severe cases, plants of susceptible varieties may die before reaching maturity. On broomcorn, anthracnose causes production of poor heads, resulting in appreciably reduced quality and value of the brushes. The sugar content of sweet sorghum plants severely affected with leaf anthracnose is reduced markedly.

The red rot phase primarily affects stalks of mature sorghum plants. Spores of the fungus produced on the leaves in the anthracnose phase, when washed downward behind leaf sheaths by rain, germinate; the
fungus, which enters the stalks at any time after the jointing stage, rots the stalk's interior. Splitting diseased stalks lengthwise reveals discolored areas interspersed with white, giving a marbled appearance throughout the affected portions (fig. 8, B). Stalk lesions are prevalent on the peduncle and internodes of the upper half of the plant.

Figure 8.—Anthracnose: A, foliar phase; B, red rot phase; C, resistant and susceptible selections in screening test.
Symptoms similar to those described for *Fusarium* head blight may develop in panicles. Depending on the variety affected, the discolored areas range from tan to purplish red. Diseased stalks frequently break near the middle of the stalk or just below the seed head. Diseased but unbroken stalks often produce small heads, sometimes with abnormally small seeds. Badly rotted stalks of sweet sorghum yield less juice than do healthy stalks, and infected stalks produce a reddish, poor-quality syrup.

The fungus overwinters in the soil, on plant refuse, on johnsongrass, and on susceptible weeds. In addition, the fungus may be carried on the seed. Fortunately, sorghum varieties resistant to both phases of the disease are available (fig. 8, C). All new sweet sorghum varieties are highly resistant to anthracnose and red rot. The broomcorns as a rule are susceptible, but they are grown in areas where the disease generally is not prevalent and losses generally are minimal. Using clean seed, destroying plant refuse from previous susceptible crops, and controlling susceptible weeds also will help to control the disease.

**Gray Leaf Spot**

Gray leaf, or angular leaf spot of sorghum—incited by *Cercospora sorghi* Ell. and Ev.—is a conspicuous though rather unimportant disease. The fungus attacks all forms of sorghum, corn, johnsongrass, and numerous species of wild and cultivated grasses. The disease begins as small, circular-to-elliptical, usually dark-purple or red spots. Later, the centers may become tan or brown, and the spots elongate (10 to 30 mm long by 2 or 3 mm wide) until they appear angular (being limited somewhat by the leaf veins). As the spots enlarge, grayish mycelium of the fungus covers them copiously (fig. 9, A). The fungus produces abundant conidia. During periods of high humidity, leaves may be covered with the fungus over large areas. Although all grain sorghum, sweet sorghum, and broomcorn varieties are susceptible to gray leaf spot, the disease generally occurs late in the growing season, after the crop is mature; little loss results. If abnormally cool, humid weather persists in midseason, however, considerable damage may occur.

No sorghum varieties are available that resist gray leaf spot. But some varieties seem to tolerate the disease better than others do.

**Zonate Leaf Spot**

Zonate leaf spot, incited by the fungus *Gleocercospora sorghi* Bain and Edgerton, is common on sorghums (as well as on corn, johnsongrass, sugarcane, and other grasses) in the humid regions of the South. The disease is conspicuous on sorghum leaves as circular, reddish-purple
bands alternating with tan- or straw-color areas, which give a concentric, or zonate, pattern with irregular borders (fig. 9, B). The spots sometimes occur in semicircular patterns along the margins of the leaves, or they may occur in circular form on other parts. Ranging in diameter from a fraction of an inch in early stages to several inches in later stages, the spots may cover the entire width of the leaf. Both leaf blades and sheaths can become infected.

Figure 9.—A, Gray leaf spot; B, zonate leaf spot; C, rough leaf spot; D, sooty stripe, young lesions on leaves.
In warm, wet weather, pink to salmon-color, gelatinous spore masses form above leaf pores (stomata). High incidence of the disease on plants in the seedling stage may result in severe defoliation, even death, of infected plants. Abundant spotting on leaves of older plants may cause premature destruction of foliage and poorly filled seed. In sweet sorghum, the disease may reduce yield, as well as sugar content of stalk juice. The fungus also may reduce markedly the amount of forage produced by sorghum-sudan hybrids.

The fungus overwinters in the form of sclerotia that are formed readily within the dead tissue of old leaf lesions as small raised bodies in lines parallel to the veins. The sclerotia germinate in the spring and give rise to spores (conidia) that infect the next crop \((11)\). The fungus also may be carried on the seed.

Highly resistant varieties are not available. Breeding lines with considerable mature tissue and high field resistance, however, are being identified and incorporated into desirable types. Likelihood of severe losses from this disease can be reduced by use of clean seed, and by crop rotation and clean cultivation to destroy residues of susceptible host crops.

**Rough Spot**

Rough spot, incited by the fungus *Ascochyta sorghina* Sacc., is prevalent throughout the humid areas of the Southeast. In its late stages this conspicuous leaf disease is easy to identify by the sandpaperlike roughness caused by hard, black, raised fungal fruiting bodies (pycnidia) that develop on lesion surfaces. The disease begins near the ends of leaves as small, circular-to-oblong, light-colored spots with well-defined margins. As the spots enlarge, they grow together to form large blotches (fig. 9, C). As the leaves mature, the pycnidia may fall off or be washed from the leaves by rain, so that only large areas of necrotic tissue remain. The lesions then may resemble those of leaf blight but differ in that they have no well-defined margin or halo.

Rough spot attacks grain sorghum, sweet sorghum, sudangrass, and johnsongrass. The disease, most severe in fields where sorghum or sudangrass is grown for several successive seasons, is spread when spores oozing out of pycnidia during wet weather are dispersed by wind and wind-splashed rain. The fungus is carried from year to year on crop residues of sorghum and sudangrass.

Losses because of rough spot generally are minor. Some grain sorghum and sweet sorghum varieties have high resistance. Damage from this disease can be reduced by crop rotation and clean cultivation to destroy plant residues.
Sooty Stripe

Sooty stripe, incited by *Ramulispora sorghi* (Ell. & Ev.) Olive and Lefebvre, commonly is found on leaves and sheaths of sorghum, sudangrass, johnsongrass, and broomcorn. Oldest leaves usually are attacked first and most extensively. The disease first appears as small, water-soaked spots that may be colored with host pigment. Those spots enlarge and lengthen rapidly, and affected leaves often turn bright yellow (fig. 9, D). Eventually, black sclerotia form in abundance on the lesion surfaces; hence the name sooty stripe. Heavy gray sporulation by the fungus often precedes the forming of sclerotia. Broad, yellow margins distinguish sooty stripe from leaf blight; sooty stripe's sclerotia (irregular and tends to stick to the fingers like soot) distinguish it from rough spot (which has pycnidia). The fungus lives from one season to the next on crop residues.

Most sorghum varieties will sustain some damage from sooty stripe, and, generally, they are not affected early enough in the season to reduce yields. Sorghum varieties completely resistant to sooty stripe currently are not available. Clean cultivation and destruction of crop refuse, along with crop rotation and seed treatment, will aid in controlling the disease.

ROOT AND STALK DISEASES

On sorghum grown under adverse conditions (or damaged by cultivation) on dry or irrigated soils, root rot incited by species of *Fusarium* (especially *F. moniliforme*) may occur anytime from seed germination to plant maturity. *Periconia circinata*, which caused milo disease many years ago, and *Pythium spp.* are closely associated with a root/stalk rot complex presently causing problems among grain sorghums, especially in irrigated fields on the High Plains and in Arizona, California, and New Mexico.

Stalk rots may follow root rot or certain combinations of environmental and plant-development sequences that predispose plants to attack by stalk-rotting pathogens. In the Great Plains, charcoal rot, incited by *Sclerotium bataticola*, and *Fusarium* stalk rot, incited by *F. moniliforme*, are the most serious stalk diseases. In humid regions of the South, red rot, incited by *Colletotrichum graminicolum*, and pokkah boeng, incited by *F. moniliforme var. subglutinans* commonly are found.

Stalk diseases in grain sorghum usually are caused by soilborne fungi that become moderately to highly pathogenic under certain environmental conditions. Those conditions may be extremely complex,
involving several factors that must either coincide or occur in a precise succession before stalk rot can occur.

**Periconia Root Rot, or Milo Disease**

*Periconia circinata* (Mang.) Sacc. caused serious root rot of milo sorghums in the 1920’s and 1930’s (35) (fig. 10). It was first noted in 1924, near Chillicothe, Tex.; 2 years later it was observed at Garden City, Kans.; and within a decade it was so widespread in major sorghum-producing areas that it was a limiting factor in producing milo sorghums (18). Fortunately, resistance was found, and losses from the disease soon were minimized (48). *Periconia* root rot reappeared briefly in the early 1960’s because some of the first grain sorghum hybrids were not totally resistant. In 1970, *P. circinata* was found closely associated with a root rot problem of grain sorghum near Yuma, Ariz. That problem is most severe among late-planted fields. In 1971, the fungus was found closely associated with roots of mature grain sorghum lodged among irrigated fields on the High Plains.

Fungus populations in the soil apparently must reach a certain threshold before the disease becomes important. A quantitative relationship also exists between the level of soil infestation and the rate or extent of disease development. Highly susceptible varieties grown on heavily infested soils show root damage, especially of cortical and vascular tissue, which usually causes severe stunting and floral inhibition.

Root damage is progressively greater and occurs earlier as the level of soil infestation increases or as the planting date is postponed. Damage may range from early mortality of seedlings to mildly affected plants whose heads produce somewhat underdeveloped seed. *P. circinata* produces toxins, which by themselves can produce all symptoms of the disease in absence of the fungus in susceptible sorghums (60).

The extent of wilting, rolling, yellowing, and firing of leaves is proportional to the amount of tissue damaged by toxins in the lower parts of the plant. Anthocyanin, a red pigment, normally is produced by *Periconia*-infected tissues, particularly in the lower internodes, crown, and large secondary or buttress roots (fig. 11). Using resistant varieties and hybrids control the disease. Resistance to *Periconia* root rot must be monitored closely in producing new varieties or hybrids because the genes for disease resistance are incompletely dominant (3). Resistance may be diminished if genes for susceptibility are introduced into the makeup of new hybrids.
Pythium Root Rot

Before *Periconia circinata* was shown to cause milo disease, *P. arrhenomanes* was regarded as the primary pathogen (18). In 1972, a *Pythium* sp. was found consistently related to the extent of root rot among sorghum hybrids in the Texas Panhandle (17). Isolations of that fungus proved highly pathogenic on sorghum seedlings in greenhouse tests, and similar to the root discoloration observed among nearly mature plants in the field, they caused roots to turn gray to black.
Figure 11.—*Periconia* root and crown rot.

Although root rot severity was directly related to lodging observed among hybrids, there was no indication of clear-cut resistance.

**Weak Neck**

The term "weak neck" is restricted to physiological collapse of peduncles resulting from inherent plant characteristics and environmental stresses in combine types of grain sorghum (28) (fig. 12). The term has been extended, however, to include peduncle damage caused by predominantly saprophytic bacteria and fungi that grow in water, honeydew, and plant exudates accumulating between leaf sheaths and culm.

The disease was first reported in 1938 in Kansas. Weak neck is particularly serious among plants not harvested before culms are dead or dried out. Long peduncles and heavy grain accentuate vulnerability to peduncle breakover, whether plants are dead or living, especially if windy conditions prevail.

Satisfactory control has been achieved by developing sorghums in which grain ripens before culm mortality.

**Fusarium Stalk Rot**

*Fusarium* stalk rot, incited by *Fusarium moniliforme* Sheld., has become increasingly common in recent years as a root/stalk rot pathogen of sorghum. *F. moniliforme* attacks many grasses, but corn
and sorghum are preferred hosts. The disease generally is found in the same areas where charcoal rot occurs, particularly on the High Plains from Texas to Kansas (fig. 13). Like charcoal rot, *Fusarium* stalk rot apparently requires some predisposing conditions for disease development as plants approach maturity. Unlike charcoal rot, however, *Fusarium* stalk rot usually is most damaging during cool, wet weather following hot, dry weather.

*Fusarium* stalk rot usually is accompanied by extensive root damage. Under irrigation and heavy nitrogen fertilization, root damage may not cause any noticeable change in the crop's aboveground appearance.

Figure 12.—Lodged peduncles typical of weak neck in grain sorghum.
before the stalks begin to rot. Stalk rot may reduce seed filling, resulting in seed weight losses as high as 60 percent.

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

_Fusarium_ stalk rot usually can be distinguished from charcoal rot because of its less pronounced pigmentation and disintegration of pith tissues and slower rot rate (fig. 14, A). Where charcoal rot may destroy a field of sorghum in 2 or 3 days, _Fusarium_ stalk rot may take 2 or 3 weeks.

Coincident with increased _Fusarium_ problems are several cultural practices suspected of contributing to the increase of the disease. Those cultural practices include maximum tillage, high nitrogen fertilization, high plant populations, and continuous cropping. The fungus persists in the soil, on crop refuse, and on johnsongrass or other weed hosts.

The dramatic buildup of aphid populations (most notably the new greenbug biotype C and corn leaf aphid), generalized infection of sorghums with MDMV following spread of both weed host johnsongrass and those aphids, and possibly added stress from mites are coincident...
with increased *Fusarium* problems. Corn infected with MDMV is attacked more readily by root rotting pathogens than is virus-free corn (66). This situation also may be true for sorghum. Possibly root damage by *Periconia circinata* or *Pythium spp.* may be a predisposing factor to *Fusarium* stalk rot (42).

Sorghum varieties completely resistant to *Fusarium* root and stalk rot are not available. Avoiding conditions that may predispose the crop to the disease may help reduce losses.

**Pokkah Boeng, or Twisted Top**

Pokkah boeng, or twisted top, is incited by the soilborne fungus, *Fusarium moniliforme* var. *subglutinans*, which occurs in all sorghum areas where high humidity is prevalent. Although the disease may be conspicuous on some sorghum varieties, losses usually are small.

Pokkah boeng is characterized by deformed or discolored leaves near the top of the plant (fig. 14, B). In some cases, the leaves become so wrinkled they are unable to unfold properly, resulting in a plant 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 often resemble that of the mosaic symptom caused by virus.

Pokkah boeng can be differentiated from mosaic by its wrinkled-leaf bases and numerous small transverse cuts in the leaf margins. Sometimes the disease causes stalks to bend, and sometimes the stalks display "knife-cut" symptoms (narrow, uniform, transverse cuts in the rind that give the impression the tissue has been removed with a sharp knife). Because they are covered by the leaf sheaths, those lesions may not be apparent when pokkah boeng leaf symptoms are present. Under conditions of such physical stress as in windstorms, the stalks may break along the "knife-cut" lesions.

During prolonged wet weather, *F. m.* var. *subglutinans* grows upward on the outside of sorghum stalks, and the fungus temporarily may become established behind the leaf sheath or in the whorl. Metabolites produced by the fungus incite distortions in the plants. Later, when wet weather subsides, the fungus dries and the plant resumes normal growth. There is some evidence that the fungus may be transmitted by seed. The fungus also affects corn, johnsongrass, sugarcane, broomcorn, and sudangrass.

**Charcoal Rot**

Charcoal rot, incited by *Sclerotium bataticola* Taub., is a serious disease of dryland grain sorghum. It can occur during seed development wherever and whenever the crop is subjected to low soil moisture and
high temperature. The disease, therefore, is more widespread in some years and localities than in others.

The first plants affected generally are those in areas where drainage is best, such as on terrace crowns, knolls, or soils underlain by coarse sand or gravel. Soil moisture, prebloom rainfall, tillage method, crop sequence, plant spacing, and soil fertility also may influence patterns of disease development in individual fields. Entire plantings often succumb simultaneously.

Relative susceptibility of varieties apparently is influenced by such factors as drought resistance, heat tolerance, time required for seed development, and amount of seed produced (12, 14). Among varieties, conditions required for predisposition to charcoal rot vary; some varieties are more likely than others to succumb to the disease under similar stress conditions (15).

Charcoal rot, which advances within the stalk from the crown upward, is typified by three successive stages as shown in figure 14, C: (1) General water-soaking of pith tissues; (2) intense pigmentation (red to black) of affected tissues; (3) host mortality, followed by fading of color, drying of affected tissues, and formation of sclerotia on the vascular remnants (16) (fig. 15). Affected plants then lodge and tissues within the stalk appear shredded. Under conditions favoring disease development, all three stages may be completed within a few days. If stress conditions are interrupted, the disease process may be inhibited or retarded, resulting in incomplete or incidental tissue damage. Sclerotial formation, physical collapse of stalk tissues, and invasion by other micro-organisms then may follow. Diagnosing the cause of damage in such cases is difficult.

Sweet sorghums and many forage sorghums are highly resistant to charcoal rot, but controlling charcoal rot in the grain sorghums has been difficult. Under borderline stress conditions, lessening the likelihood of stress during seed development may help. This can be done by spacing plants as far apart as practical, avoiding light or extremely well-drained soils and fallowing or green-manuring every 2 or 3 years. If stress conditions are severe, however, such practices may not be effective because they tend to promote vegetative growth, thereby intensifying the plant’s moisture demands during its late developmental stages.

Growing varieties resistant to predisposing stress conditions may be the best way to control charcoal rot. Using grain sorghum varieties known to resist charcoal rot (30, 39) to develop hybrids should reduce losses from the disease.
Figure 14.—A, *Fusarium* stalk rot; *B*, pokkah boeng; *C*, charcoal rot, showing progressive stages in tissue pigmentation and disintegration which occur before sclerotial production.
Diseases of the inflorescence concern especially producers of grain sorghum; severe damage to the heads also lessens the value of sorghums grown for silage and forage. Before seed treatment was practiced, covered kernel smut was the most widespread and serious disease of grain sorghum. Loose kernel smut remains essentially a curiosity, but head smut (which has not been controlled by seed treatment) continues to exact losses in certain areas where susceptible hybrids are grown. Reduced head and seed size may result from direct attack of
inflorescences or indirectly from diseases attacking foliage, roots, and stalks.

**Maize Dwarf Mosaic Head Blight**

Under certain conditions, seed of plants infected with maize dwarf mosaic virus (MDMV) at or near heading may fail to fill properly (50). When that happens, symptoms of virus infection may be absent among vegetative plant parts, and excessive shrinkage of ripening seed may be the first indication of crop damage. Commonly both normal and underdeveloped seed occur together in the same spikelet or panicle branch. The small seed may be restricted to one side of a head or follow patterns of anthesis. Affected seeds are lightweight, usually have chalky endosperms, shatter readily, and are attacked by such fungi as *Alternaria* more readily than are normal seeds in the same head. Preceding abnormal seed shrinkage, pigmented, necrotic lesions commonly appear on branches of panicles (fig. 16, A and B). That symptom is most likely to develop under conditions similar to those associated with the red-leaf symptom. The problem has been most acute among grain sorghums on the High Plains, where such conditions commonly occur as plants approach maturity. Similar symptoms can occur from *Fusarium* in the humid South. Small seed resulting directly

![Figure 16.—Maize dwarf mosaic head blight: A, Pigmented-necrotic reaction in head; B, head with seed removed to show pigmented necrotic lesions on panicle branches.](image-url)
from root or stalk rots or from drought or insects sometimes resembles that resulting from MDM, except that necrosis of panicle branches is lacking.

**Fusarium Head Blight**

In the humid South, head blight incited by *Fusarium moniliforme* may be a limiting factor in grain sorghum production. The disease is characterized by death of several to all florets in seed heads (fig. 17, A). When the disease is severe, the entire seed head may be covered with a copious cream to pinkish-tan fungal growth. If the panicle is split lengthwise, a red-brown-black discoloration is evident in the upper portion of the peduncle and extends into the branches of the head. Sometimes the discoloration may extend throughout the peduncle and into the upper internodes of the stalk, in which case the rind also may be discolored (fig. 17, B). In severe cases, extensive breakover of peduncles may occur. Patterns of penetration and infection by the fungus have not been elucidated fully. Mycelium of the fungus could grow up along the outside of the stalk on waxy bloom or the fungus could arrive in the head as airborne conidia. Penetration probably occurs through cracks or insect wounds in the rind of the peduncle, rachis, or panicle branches. Presently, there are some grain sorghum breeding lines which show excellent resistance to *Fusarium* head mold, and hopefully those lines can be used in developing future hybrids.

**Figure 17.—Fusarium head blight:** A, peduncle exterior symptoms; B, head mold symptoms.
Smut Diseases

Fungi that cause smut diseases of sorghum are highly developed, closely related parasites. Interspecific hybrids between all combinations of *Sphacelotheca sorghi* (Link) Clinton (covered kernel smut), *S. cruenta* (Kuhn) Potter (loose kernel smut), and *S. reiliana* (Kuhn) Clinton (head smut) have been produced experimentally (54, 55, 67, 70). All are known to initiate meristem infections during seedling growth stages.

Those infections then are carried in apical meristems of floral primordia as the plant develops, causing negligible damage to vegetative plant parts. Just before exsertion of heads, the smut fungi grow rapidly in developing floral parts until space and nutrients are exhausted; then sori (fungal fruiting bodies) develop.

During sorus development, vegetative fungal growth is converted to dark-colored, diploid spores (teliospores) contained en masse within a fungal-host membrane (peridium). Those spores become soilborne or seedborne and give rise to haploid spores (basidiospores). Complementary sex factors direct discriminate pairing of basidiospores before initiating new infections. Kernel smut fungi are borne on seed and are controlled easily by treating seed with captan or thiram-type fungicides. Host resistance and sanitation are used to control head smut.

**Covered kernel smut.**—In the United States where most sorghum seed is treated, covered kernel smut, incited by *Sphacelotheca sorghi*, has become relatively rare. Sori develop in place of the ovary or stamens, or both, of individual florets, which then may support a single sorus in place of the ovary (or fused sori in place of both ovary and stamens) (fig. 18, A). Fused sori often are lobed according to the number of floral parts included. The peridia of sori have various thicknesses; some rupture easily, but others persist until ruptured by threshing operations. By contaminating seed during threshing, spores pass from one season to the next. Soilborne spores are not considered important in the disease cycle.

Environment will affect incidence of infection (46). Smut incidence decreases when seed is planted in progressively warmer soils (from 60° to 90° F). Smut incidence also is lower when seed is planted in wet soils. Seedlings planted in warm, moist soils may emerge so rapidly they escape infection (31).

Presence or absence of sori in heads of main stalks, axillary branches, or tillers, depends on patterns of early infection (45). If the uppermost cells of the apical meristem are not invaded, main heads may escape infection. Likewise, secondary heads will reflect incidence of infection in other cells of the apical meristem.

The number and distribution of smutted florets in a panicle varies, and sometimes all florets may be smutted. Sori also may be restricted
Many variations result from infection by different races of *S. sorghi*. When infected by race 1, some sorghum varieties exhibit blasted panicles with no sori or only a few underdeveloped sori. Normal sori versus blasting is controlled by a single pair of alleles; the gene controlling normal sorus development is dominant (7). The gene for resistance to race 1 of *S. sorghi* is epistatic to both reactions (6). Height, internode number, stalk diameter, and leaf width may be reduced (though rarely) in some varieties infected by *S. sorghi* (47).

*S. sorghi* has demonstrated considerable capacity for physiological specialization. At least five distinct races have been identified in the United States (65). Efficacy of treating seed to control kernel smut has removed pressures for using host resistance. Resistance has been found
to exist as single-factor pairs to each of races 1, 2, and 3, and the genes involved appear linked. Although treating seed with captan or thiram fungicides is standard, certain systemic fungicides also offer promising means of control.

Loose kernel smut.—Except for a few differences, most aspects of loose smut, incited by *Sphacelotheca cruenta*, are similar to those described for covered smut. Normally, seedlings are infected as in covered smut, but localized infection has been demonstrated on floral parts from airborne spores (19). Peridia of sori rupture soon after the host exserts heads. Most spores, when released before harvest, reveal prominent, long columellae containing deformed floret remains (fig. 18, B). Glumes usually are elongated; other floral parts also may be proliferated, especially when sori are absent. Pistils and stamens of a floret are more likely replaced by a single sorus, although sometimes there are multiple sori, fused only at the base. Sori commonly develop on the rachis and its branches, sometimes on the glumes, and occasionally on the pedicles and stalks. Stunting of host plants is more easily detected in loose smut than in covered smut. Two physiologic races of *S. cruenta* have been identified in the United States (44). A third race, usually referred to as *S. holci* Jackson, is especially pathogenic to johnsongrass and sudangrass (38).

Head smut.—Head smut, incited by *Sphacelotheca reiliana* (Kühn) Clint., often causes serious losses in sorghum. The soilborne pathogen cannot be controlled by seed treatment. When grain sorghum hybrids were introduced in the United States, the disease became a serious problem in grain sorghum production, especially in southern Texas (53).

In head smut, part or all of the panicle becomes incorporated into a single sorus (fig. 19, A and B). Parts of an infected panicle not included in a sorus usually show blasting or proliferation of individual florets (fig. 19, C). Sori also may develop on foliage and culms in some sweet sorghums and sudangrass varieties (fig. 19, D). The sorus has a thick, whitish peridium that ruptures readily after exsertion, and the black mass of teliospores gradually falls out.

Naturally released teliospores of *S. reiliana* germinate sparingly (fig. 20); immature spores, however, may germinate readily (64). When land is cropped continuously in susceptible sorghums, disease incidence and soilborne inoculum from the previous crop become sufficient to cause a high incidence of disease. Cool, dry soils favor infection (9).

Infection patterns in the apical meristem determine the type of sorus produced in one or more heads of a plant (75). If the entire floral primordium becomes infected, a single sorus replaces the panicle. When mycelium of early infection is scattered by elongation of cells in a floral
Figure 19.—Head smut; A, young sorus; B, mature sorus; C, sterility often manifested in head smut; D, sorus formation on leaves, a relatively rare manifestation.
primordium, various proportions of the panicle may be replaced by the mature sorus. Infection localized in the base of the apical meristem may result in a blasted or proliferated head that is free of any sorus formation. Failure in sorus production is most common in terminal heads of the main culm. Sorus formation usually is most extensive in panicles of side branches and tillers.

Physiologic specialization occurs in *S. reiliana*. At least two distinct forms are known—one infects corn; the other, sorghum. Cross inoculating those forms usually is unsuccessful (52). At least three races of *S. reiliana* exist in the United States (2, 43): A race that attacks most

Figure 20.—Germinated teliospores of *Sphacelotheca reiliana.*
grain sorghum hybrids grown before 1965; a race (in California) pathogenic to Early Hegari SA 281, a variety resistant to most isolates of *S. reiliana* (27); and a race which is virulent to resistance derived from Tx 09 Combine White Feterita (24).

In head smut, epicotyls become infected after they have grown beyond the zone protected by seed-treatment fungicides (36). Resistant varieties and hybrids must be used to control the disease. Several grain sorghum hybrids resistant to one or more races of the pathogen are available. Most sweet sorghum varieties are highly resistant.

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