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Efforts to control rice diseases in the tropics depend for their success on recognizing the disease symptoms and applying effective controls. This pamphlet, intended primarily for use by trainees at the International Rice Research Institute, presents general information on 15 important diseases of rice in the tropics. For each disease it discusses the history and geographic distribution, damage, symptoms (including photos), causal organisms, disease cycle, effect of environmental conditions, varietal resistance, and control measures. A list of all known rice diseases is included to indicate the scope of the rice disease problem. The fifteen diseases discussed in this handbook are tungro, grassy stunt, yellow dwarf, bacterial blight, bacterial leaf streak, rice blast, sheath blight, stem rot, brown leaf spot, narrow brown leaf spot, bakanae disease, false smut, sheath rot, white tip, and stem nematode. More details on rice diseases may be found in Rice Diseases, published by the Commonwealth Mycological Institute, Surrey, England, and in Rice Virus Diseases, published by the International Rice Research Institute.

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A handbook of rice diseases in the tropics

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FOREWORD

This pamphlet contains some general information on the important diseases of rice in the tropics. It is intended primarily for use by trainees at the International Rice Research Institute.

For each of 15 common rice diseases, it briefly discusses the history and distribution, damage, symptoms, causal organism, disease cycle, effect of environmental conditions, varietal resistance, and control measures. Illustrations are provided to aid in identifying the diseases. A list of all known rice diseases is included at the end of the pamphlet to indicate the scope of rice disease problems.

More details on these and other diseases may be found in *Rice Diseases*, published by the Commonwealth Mycological Institute, Surrey, England, and in *Rice Virus Diseases* published by IRRI.

TUNGRO

Tungro was first recognized as a distinct virus in 1963 in the Philippines. It is probably the same disease that affected rice in the Philippines in 1940 or earlier, but which had been erroneously identified as "stunt" or "dwarf" disease. Penyakit merah (red disease), which had been known in Malaysia since 1938 and which was considered a physiological disease, was identified as tungro in 1965. The *mentek* disease of Indonesia is probably tungro, too. Mentek has been considered a physiological disorder since 1859. Because resistant varieties are widely used, mentek causes little damage now. In Thailand, tungro, or yellow-orange leaf disease, as it is called locally, was first found in 1964. It has spread widely in subsequent years. Tungro is also commonly found in Bangladesh, in West Bengal, India, bordering Bangladesh, and in several other parts of India.

Tungro is one of the most widely distributed and most destructive rice diseases in tropical Asia. F.B. Serrano estimated the loss in the Philippines due to "accep na pula" or stunt disease

(believed to be tungro) during the 1940's at 30 percent or 1.4 million metric tons annually. An outbreak of tungro in 1971 affected hundreds of thousand hectares in Central Luzon, Bicol, and other areas in the Philippines. In the northern part of West Malaysia, thousands of hectares are damaged each year by penyakit merah. Mentek disease affected 30,000 to 50,000 hectares of rice in Java in the 1930's. In Thailand, tungro severely damaged half of the 660,000 hectares that were infected in 1966.

Symptoms. Tungro stunts rice plants and changes the leaf color to shades of yellow or orange. The symptoms differ with variety, environmental condition, age of plants, and strain of the virus. Stunting is severe on susceptible varieties, slight on more resistant ones. Infected plants usually have somewhat fewer tillers than healthy plants. The degree of stunting decreases the older the plants are at the time of infection. When infection occurs late, no symptoms appear, but secondary growth from stubble after harvest may show the symptoms.

Yellowing starts from the tip of the leaf and may extend to the lower part of the leaf blade. Often only the upper portion is discolored. Young leaves may appear mottled and old leaves may have rusty specks of various sizes. On moderately resistant varieties, the discoloration of infected plants is conspicuous at early stages of growth but it may gradually disappear at later stages. When the discoloration disappears, the plant is said to recover from the disease.

Three strains of tungro virus have been found, "S," "M," and "T," which produce distinct symptoms on certain rice varieties. On FK 135 and Acheh the "S" strain produces chlorotic stripes while the "M" strain produces mottling. On Taichung Native 1, IR5, IR8, and IR22, the "T" strain produces narrower leaf blades than normal. The "T" strain does not stunt the plant as much as the "S" or "M" strains. It also produces stripe symptoms on FK 135.

Diseased leaves contain relatively large quantities of starch; they turn black or dark brown when treated with iodine after the chlorophyll has been removed by boiling in alcohol.

From left, healthy plant, plant infected by the "M" strain of tungro virus, plant infected by the "S" strain of tungro virus.

Vector and transmission. The principal vector of the tungro is *Nephotettix virescens*, the green rice leafhopper. *N. nigropictus*, *N. parvus*. *N. malayanus*, and *Recilia dorsalis* also transmit the virus, but at a lower rate.

About 85 percent of *N. virescens* individuals are active transmitters. The insect can acquire the virus if it feeds on an infected plant for at least 30 minutes and it can infect healthy plants by feeding on them for a least 15 minutes. The latent period in the plants is 6 to 9 days. The virus has no apparent incubation period in the insect. The insect may transmit the virus "instantly"—in 2 hours from acquiring the virus. It retains the virus for not more than 5 days, however. After this period, the insect is no longer infective unless it reacquires the virus. The virus is thus "nonpersistent" in the insect, but the insect can reacquire the virus repeatedly.

The other species are less efficient, having lower percentages of active individuals that transmit the tungro virus, 4 to 8% for *R. dorsalis*, 0 to 45% for *N. nigropictus*, 6 to 8% for *N. parvus*, and 30 to 48% for *N. malayanus*. Besides, rice is not the principal host of the three species of *Nephotettix*.

The virus is not transmitted through eggs, seed, or soil, nor by mechanical means.

Disease cycle. Since the insects must repeatedly acquire the virus from diseased plants to be continuously infective, the size of the infective insect population may vary greatly, depending upon the amount of virus reserve. Removing diseased plants from the field may reduce the sources of virus.

The virus causes severe damage only in areas where the host plants and the insect vector multiply the year round. In areas such as Central Thailand, where rice is grown once a year and where there is a long dry and warm period, alternative hosts, especially wild rice, are probable sources of inoculum when no rice is growing. Stubble of infected plants from the previous season that ratoons is also a source of inoculum. Grassy weeds, such as *Eleusine indica*, *Echinochloa colonum*, *Echinochloa crusgalli*, *Ischaemum rogosum*, and *Dactyloctenium aegyptium* may be infected occasionally.

As with all virus diseases, the incidence of tungro disease correlates with the population of the insect vector.

Varietal resistance. Under a standard artificial inoculation test, tungro may infect 95 percent of Taichung Native 1 plants, compared with only 4 percent of the plants of Pankhari 203, one of the most resistant varieties. At IRRI, over 60 varieties have been found to be resistant, that is, to have less than 30 percent infection. Several other varieties have also been found resistant in India. Such resistant varieties have very slight infection under normal field conditions. A few varieties have less than 10 percent infection under artificial inoculation: Pankhari 203, Habigonj D.W. 8, Red Rice, Tsou Yuen, Adday local (sel.), Basmati 37, and PI 184675-4.

Several commercial varieties such as Tjeremas, Bengawan, Peta, Intan, and Sigadis, were developed in Indonesia for resistance to mentek even though the cause of mentek was not known. These varieties are also resistant to penyakit merah in Malaysia, yellow-orange disease in Thailand, and tungro virus in the Philippines.

Varieties such as Pankhari 203, ASD 7, Ptb 18, DK1, and AC 435 are resistant to the vector. Independent genes control resistance to the virus and to the vector. Varieties resistant to the tungro virus may not be resistant to the insect or vice versa. Researchers use both types of resistance for breeding.

Chemical control. Researchers in several countries have tried to control the virus diseases by eliminating the insect vector. In general, the results are satisfactory in mild cases but unsatisfactory in severe cases. Most insecticides kill the insects several hours after application. By then the insects have transmitted the virus to the plants.

In trials at IRRI, soaking the seeds and dipping the seedlings in carbofuran (Furadan) solution protected the seedlings from infection for about 30 days. Insects were killed almost instantly (in a few minutes) when they fed on the treated seedlings. Since the plant is protected in the seedling stage, when it is most vulnerable to infection, this method may be practical though costly.

GRASSY STUNT

Grassy stunt disease was first noticed in 1962 at the IRRI experimental farm and in farmers' fields in the Philippines. Researchers established in 1964 that *Nilaparvata lugens,* commonly known as the rice brown planthopper, transmits the disease. Grassy stunt also occurs in Ceylon, Thailand, India, Indonesia, and elsewhere. The rice rosette disease reported from the Philippines in 1966 is apparently identical to grassy stunt.

Dr. E. Shikata of Hokkaido University found that diseased tissues contain mycoplasma-like bodies that may cause grassy stunt as they do in yellow dwarf disease.

The disease has caused severe damage at the IRRI farm and in a few localities in the Philippines where intensive and overlapping rice croppings occur. But generally grassy stunt appears only sporadically in the field. When it infects young seedlings, the plants produce essentially no grain.

Symptoms. The disease is characterized by severe stunting, excessive tillering, and an erect growth habit. The leaves are short, narrow, yellowish green, and covered with numerous rusty spots that form irregular blotches at later stages. When infection occurs at later stages of plant growth, diseased new tillers appear with normal-looking tillers in the same hill.

Grassy stunt resembles yellow dwarf disease, but leaves infected with grassy stunt are greener than those infected with yellow dwarf. Rusty spots are usually not seen in yellow dwarf. The numerous tillers give the hill a fan-like or grassy appearance. The plants live until the crop matures but produce no panicles or only a few empty ones.

Vector and transmission. About 20 to 40 percent of the individuals of *N. lugens* can transmit the virus. The males and females, long-winged and short-winged forms, light-brown and dark-brown forms all have the same ability to transmit the virus. The insects become infective after feeding on diseased plants for 30 minutes. The incubation period of the virus in the insect is usually 10 to 11 days but it may vary from 5 to 28 days. Inoculation

lants infected with grassy stunt (left) and yellow dwarf (middle) in
›mparison with a healthy plant of the same age.

feeding of 5 to 10 minutes induces a small percentage of infection. Up to 24 hours, longer feeding time increases the percentage of infection. The incubation period in plants is 10 to 20 days. Insects that have acquired the virus are infective throughout their lives. Although they may not transmit the disease every day, most of them do intermittently 2 out of 3 days. The virus is not transmitted through seed or egg.

Disease cycle. The development of grassy stunt disease in fields depends largely on the presence of infected plants (source of inoculum). In fields where no disease is present, few infective insects can be collected, but in diseased areas the percentage of infective individuals is high. Non-infective insects, however, are potential transmitters. The brown planthopper multiples quickly under favorable conditions so the disease incidence may also increase rapidly. The long-winged form which is able to travel widely can spread the disease faster than the short-winged form.

Varietal resistance. Thousands of varieties and hybrid lines and species of wild rice have been tested but none have been found highly resistant except a strain of *Oryza nivara.* Its resistance to grassy stunt, which is controlled by a single pair of dominant genes, has been transferred to lines that have good agronomic traits.

Several varieties are resistant to the vector, the brown planthopper--for example, Mudgo, ASD 7, Ptb 18, Kayama, and MGL 2. ASD 7 is also resistant to the green leafhopper.

YELLOW DWARF

Yellow dwarf disease was observed in Japan in the early 1900's. Experiments in 1950 confirmed a suggestion in 1943 that the leafhopper *Nephotettix cincticeps* transmitted the disease. The disease recently has been found throughout tropical Asia.

Yellow dwarf usually occurs sporadically in fields. It rarely causes severe damage. Probably because of change in cropping pattern, however, yellow dwarf has recently become a serious problem in Taiwan, where it has been known since 1932. It has

also caused considerable damage in Japan. Japanese workers suspect that the disease is caused by a mycoplasma-like organism.

The characteristic symptoms are general chlorosis, pronounced stunting, and excessive tillering. The chlorotic leaves are uniformly pale green or pale yellow. Plants infected early may die prematurely, but usually they survive until maturity. They produce very poor panicles or none at all. Plants infected late may not show any symptoms, but characteristic symptoms appear on ratoon plants grown from stubble.

Yellow dwarf disease is transmitted by *N. cincticeps*, *N. virescens*, and *N. nigropictus*. *N. cincticeps* is common in Japan, the other two species are common in the tropics. Most leafhoppers acquire the causal agent (virus or mycoplasma-like organism) by feeding on diseased plants for 1 to 3 hours. The minimum acquisition feeding periods are 10 minutes for *N. cincticeps* and 30 minutes for *N. virescens*. The incubation period in the insect is 20 to 39 days, most often 25 to 30 days. The minimum inoculation feeding period is 1 minute for *N. cincticeps* and 3 minutes for *N. virescens*. About half the infective individuals of both species can infect healthy plants within 1 hour of feeding. The latent period in the plants varies from 1 month at high temperature to 3 months at low temperature. Once infective, the insects can transmit the disease to healthy plants daily throughout their lives. The causal agent is not transmitted through the eggs of either species.

Because the incubation and latent periods are relatively long, the cycle of transmission is slow. In seriously diseased fields, the percentage of infective leafhoppers late in the season can be extremely high—80 percent has been recorded in Japan. In the tropics, heavily infected ratoon plants often account for many late infections.

The disease also infects the wild grasses, *Alopecurus aequalis* and *Glyceria acutiflora*. The causal organism is carried from one season to another by the insects or the grass hosts.

A new testing program for varietal resistance is under way in Japan and Taiwan because of the increasing importance of the disease. The known resistant varieties are Saitama Mochi 10, Kagura Mochi, Mangetsu Mochi, Shinano Mochi 3 (all glutinous rice), and Tetep.

BACTERIAL BLIGHT

Bacterial leaf blight was first observed in Japan in 1884. From 1908 to 1910 it was common in southwest Japan. Since 1950 the disease has spread remarkably. By 1960 it was found throughout Japan, except Hokkaido. In Indonesia, "kresek" disease, which kills young plants completely, was reported in 1950. In 1964 kresek was found to be a severe form of bacterial leaf blight. In India bacterial blight was first reported in 1959. Little attention was paid to it until 1963 when a severe outbreak occurred in Bihar.

In Japan and other temperate regions where the disease infects mainly the leaves, it is called bacterial leaf blight. In the tropics, it is usually called bacterial blight because it often kills entire young plants.

Bacterial blight occurs in all countries of tropical Asia. Some reports indicate its presence in Africa. It is not known in North America or South America. In Japan, 300,000 to 400,000 hectares were damaged by the disease annually in recent years. Yield losses in severely diseased fields range from 20 to 30 percent, and occasionally up to 50 percent. In the tropics, particularly in the Philippines and Indonesia, the losses are reportedly higher than in Japan, though little data is available. Pot experiments show that losses can range from 70 to 80 percent in severely infected susceptible varieties, and up to 40 percent in moderately susceptible varieties. In India, perhaps millions of hectares are infected. Yield losses have been as high as 60 percent in some states.

The kresek type of the disease causes very heavy losses. When the disease appears on mature plants, usually at or after the maximum tillering or booting stage, it does not affect the number of ears or number of spikelets. It causes poorly developed grains, increases underdeveloped grains, reduces the weight of grains, and causes improper maturation and broken grains. The earlier the disease attacks the greater the damage. The disease also affects the chemical composition of the grains—because maturity is incomplete, the soluble nitrogen decreases and crude protein increases.

Bacterial blight infection. The two leaves on the left show the undulating margins of early lesions. The leaves on the right show distinct diseased and healthy areas and various degree of infection.

Symptoms. Lesions usually start as water-soaked-looking stripes along the margins of the upper parts of leaf blades. The lesions enlarge and turn yellow within a few days. The areas near the healthy parts of the blade look water-soaked at the early stage and later become distinct from the healthy part. Lesions may start at one edge or at both edges of the leaves. As the disease advances, the lesions may cover the entire blade, turning it white and later grayish from the growth of saprophytic fungi. On susceptible varieties lesions extend to the leaf sheath and may reach its lower ends.

In severely diseased fields, grains may be infected. Discolored spots appear on the glumes surrounded by areas which look water-soaked. The spots are conspicuous on young and green grains. In mature grains, they are gray or yellowish white.

Two other types of symptoms occur only in the tropics: kresek, characterized by withering of leaves and entire young plants, and the production of pale-yellow leaves in older plants.

Kresek symptoms may appear 2 to 3 weeks after transplanting. The infected leaves become grayish green and begin to roll along the midrib. The common practice, in the tropics, of cutting off the tips of the leaves before the seedlings are transplanted provides easy entry for the bacterium. Broken roots also serve as points of entry. An early indication of the presence of the bacterium is a green water-soaked-looking area just beneath the cut surface which soon turns grayish green. Then the entire leaf, including the leaf sheath, rolls and withers. The bacterium spreads through the vascular system to the growing point of the young plant and infects the base of other leaves. The entire plant dies.

Another symptom of bacterial blight in the tropics is the production of pale-yellow leaves in older plants. While old leaves are normal green, the youngest leaf is uniformly pale yellow to whitish, or a broad greenish-yellow stripe may appear on the leaf blade. Little further growth of the infected tiller occurs.

The leaf symptoms of bacterial blight are often confused with some physiological conditions which cause the leaf to turn yellow. The yellowing of the diseased leaf, however, is not symmetrical on both sides of the leaf blade as it is when a physiological disorder exists. There is a distinct margin between the diseased and healthy

parts of the leaf. In physiological disorders, the color change is gradual. If a few diseased leaves are cut at the lower ends of the lesion and the cut ends are placed in clean water in a test tube or in a glass, bacterial ooze streams from the cut ends into the water in a few minutes. The ooze in a small piece of diseased leaf can be observed under a microscope, unless the tissue has been dead for long.

Kresek symptoms sometimes are mistaken for damage from stem borers. But when the lower end of the young plant infected with kresek is cut and squeezed between the fingers, a yellow bacterial slime oozes out.

Causal organism. Bacterial blight is caused by *Xanthomonas oryzae* (Uyeda et Ishiyama) Dowson. The bacterium is a short rod, about 1 to 2 microns x 0.8 to 1.0 microns with a flagellum at one end. The cells are surrounded by mucous capsules and are joined in an aggregated mass. The organism is easily cultured in masses on media but difficult to grow from single cells.

The bacterium is attacked by bacteriophages (viruses) which are common in fields. Each kind of bacteriophage can attack special strains of the bacterium.

The bacterium has many strains that differ in ability (virulence) to infect rice plants. Some strains are weak, causing only small lesions; others are virulent, causing large lesions. Strains in tropical countries are usually much more virulent than those in temperate areas like Japan. Some strains from India, Ceylon, and Burma are more virulent on certain varieties than those from other tropical countries.

Disease cycle. The bacterium enters the plant through water pores along the edges of the leaf and through injuries in roots or leaves. It does not enter through the stomata. Bacterial blight is primarily a vascular disease: the bacterial cells move along the vascular tissues, causing wilting.

As soon as young lesions develop, the bacterial cells ooze from the lesions whenever dew or rain occurs. In the early morning drops of dew on the lesions are milky owing to the presence of bacterial cells. The drops dry to become minute beads that easily

fall into the paddy water. Rainstorms and typhoons speed the spread of the organism and of the disease. Irrigation water also carries the organism from field to field.

Seed-borne bacterial cells are not important in spreading the disease. Under high temperature in the tropics, bacterial cells on or in the grains and leaves die in a few weeks.

The organism can survive in the absence of rice plants. It may infect a few grasses that serve as alternate hosts. Or, it may form microcolonies in the rhizosphere of non-host grasses.

Effect of environmental conditions. High temperature favors development of the disease. In one experiment, kresek symptoms appeared within 10 days at about 30 C while none were observed after 40 days at 20 C. Lesions enlarged and leaves died faster at higher temperatures.

Many experiments have shown that high rates of nitrogen fertilizer increase the incidence of the disease, possibly because luxuriant growth of rice plants favors the spread of the organism. But levels of nitrogen do not seem related to the size of individual lesions. Phosphorus and potassium deficiency and excess silica and magnesium also increase disease incidence.

Varietal resistance. More than 8,700 varieties have been screened for resistance against Philippines strains of the bacterial blight organism and 24 showed good resistance to all virulent strains. When the 24 varieties were tested in India, however, several were moderately susceptible or susceptible to a few strains. In further tests in Hawaii with bacterial strains from 10 Asian countries, several strains from Ceylon and Burma were virulent to some varieties. The varieties resistant to most Asian strains were BJ1, TKM 6, Wase Aikoku 3, Sigadis, Malagkit Sungsong, Nagkayat, and Lacrosse x Zenith-Nira. Resistance may be built up by crossing two varieties that are resistant to different strains. Among the newly developed varieties, IR20, IR22, and Syntha (Indonesia) are resistant in the Philippines.

Chemical control. Many chemicals, including several antibiotic compounds, have been tried for control of the disease. None

seem to have any practical use, however. TF-130, a systemic compound developed in Japan, controls the disease quite well when used as soil treatment and as a spray, but its effect is short-lived. Moreover, the bacterium easily develops resistance to the chemical. The usefulness of the chemical may therefore be limited. The chemical is not produced now because of residue problems.

BACTERIAL LEAF STREAK

Bacterial leaf streak was first called bacterial stripe by O.A. Reinking who found it in the Philippines in 1918. It was not studied in detail, however. Several people erroneously referred to it as bacterial blight. In 1957 it was distinguished from bacterial blight in South China and called bacterial leaf streak. The disease is common in tropical Asia but is not known in Japan or other parts of the world. Under favorable climatic conditions the disease may cause as much leaf damage as bacterial blight. No definite figures on losses are available, however.

Fine translucent streaks between the veins of the leaf are the first symptoms. The lesions enlarge lengthwise although they may also advance over larger veins laterally. Bacterial exudates appear on the surface of lesions under humid conditions and then dry up to form numerous minute round particles on the lesions. Old lesions become light brown. On very susceptible varieties a yellow halo appears around the lesions. At late stages of plant growth, entire leaves turn brown from numerous infections. These leaves die and then are bleached to grayish white by saprophytic organisms. At this stage the disease is indistinguishable from bacterial blight.

The bacterium was originally called *Xanthomonas oryzicole* Fang et al. Then the name *X. translucens* f.sp. *oryzae* was used. A more logical name is *X. translucens* f.sp. *orizicola* (Fang et al.) Bradbury. The organism is a short rod, about 1.2 microns x 0.3 x 0.5 microns, single, occasionally paired, without spores and capsules, motile by a single polar flagellum. The bacteria may be cultured on media which have been used for *X. oryzae*.

Bacterial leaf streak showing long, narrow lesions.

Like *X. oryzae*, the bacterial cells are attacked by many kinds of bacteriophages.

Many strains of the bacterium differ in pathogenicity. Virulent strains cause longer streaks.

The bacterium enters the leaves through the stomata and wounds. It mainly infects the parenchyma cells but does not enter the vascular systems. Unlike bacterial blight, bacterial streak is not systemic. When the leaves are wet, exudates from the lesions spread to other portions of the leaf, to other leaves, and to other plants. Because of the copious exudates, the organisms may spread quickly through an entire field. Irrigation water also carries the bacterial cells. Rainstorms and typhoons favor the spread of the disease. High temperature favors lesion development.

The bacterium infects most wild rice species as well as a few grasses.

Many of the over 1,200 varieties that have been tested with virulent strains of the organism were found resistant. Resistance has also been found among wild rice species. It should not be difficult to develop resistant new varieties. Other methods of control have not been studied in detail.

RICE BLAST

Rice blast is probably the earliest recorded and most widespread disease of rice. A book published in China in 1637, described a rice malady that resembled the blast disease. It was believed that heat absorbed by the grain during drying under the hot sun caused the disease. That is perhaps the origin of the strange term "rice fever disease," which is extensively used in Chinese and Japanese literature. The disease was recorded in Japan in 1704; in Italy, it was reported in 1828 as "brusone" disease which is believed to be the blast disease. In the U.S.A., the disease was reported in 1906. The disease was first recorded in India in 1913.

Seventy countries have reported the presence of blast. The disease occurs wherever rice is grown commercially except that no damage from the disease has ever been reported in California,

U.S.A., or South Australia. These areas have special climatic conditions.

Blast is often considered the most serious rice disease because it spreads rapidly and is highly destructive under favorable conditions. It may infect plants at any stage of growth. Depending upon weather and varietal resistance, plants may have only a few leaf spots or may be killed completely. Two phases of the disease are most pronounced: leaf blast, which occurs from infection between the seedling and maximum tillering stages, and panicle blast, which occurs from infection after flowering.

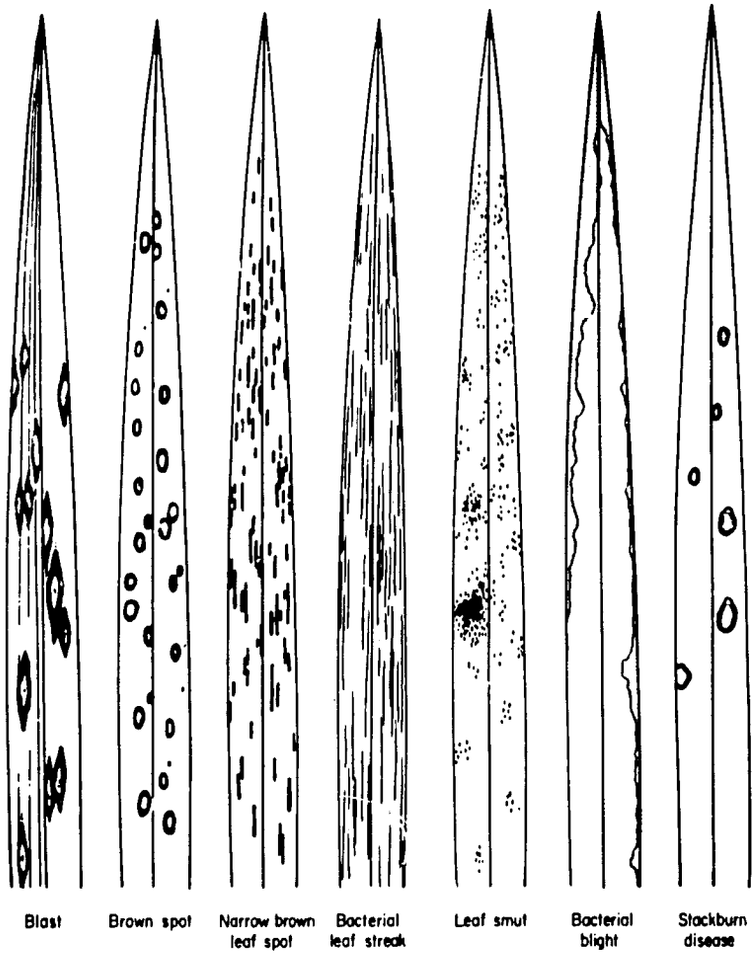
Leaf blast stunts the plants and reduces the number of mature panicles, the weight of individual grains, and the weight and quality of brown rice. Few exact figures on losses exist, however. In Japan in 1953, an epidemic year, yield losses reached about 800,000 tons. In spite of the extensive use of chemicals, annual losses from 1953 to 1960 varied from 1.4 percent to 7.3 percent, with an average of 3 percent of the national production. In 1962, leaf blast occurred on 865,000 hectares, of which 847,000 hectares were sprayed with chemicals against blast, and panicle blast developed on 721,000 hectares of the 909,000 hectares sprayed with chemicals.

Several workers have attempted to correlate the percentages of panicle blast with yield losses. Formulas, such as $y = 0.69X + 2.8$ (y is percentage loss, and X is percentage incidence of panicle blast), have been used in Japan. Various studies in India found that 1 percent panicle blast reduced yield by as little as 0.4 percent or 1.4 percent to as much as 17.4 percent. Besides percentage, time and degree of infection greatly affected the yield loss.

Symptoms. The fungus produces spots or lesions on leaves, nodes, and parts of the panicles and the grains, but seldom on the leaf sheath.

Typical leaf spots are elliptical and somewhat pointed at both ends. The margins are usually brown to reddish brown and the center is often gray or whitish. Both the shape and color of the spots vary, however, depending upon the environmental conditions, age of the spots, and varietal susceptibility. The spots

Typical spindle-shaped lesions of rice blast on leaves.



Typical lesions of common leaf-spot diseases of rice.

begin as small, water-soaked, whitish, grayish, or bluish dots on susceptible varieties. They enlarge quickly under moist conditions. Fully developed spots reach a length of 1 to 1.5 cm and a width of 0.3 to 0.5 cm with brown margins. Highly resistant varieties have no lesions or only pinhead-sized brown specks. Varieties with intermediate reaction have small, round or short-elliptical lesions, a few millimeters long with a small necrotic center and a brown border. A brown color indicates that the lesion has stopped enlarging because of varietal resistance or unfavorable conditions. When numerous spots occur on a leaf it soon dies. Plants attacked between the seedling and maximum tillering stages are often completely killed, leaf spots of the blast disease are sometimes confused with those of other leaf spot diseases.

When the node is infected, the sheath pulvinus rots and turns black, and when dry it often breaks apart, remaining connected to the straw by the nodal septum only. All parts above the infected node die.

Infection may occur in any part of the panicle, producing brown lesions. It often occurs near the panicle base, causing "rotten neck" or "neck rot" symptoms, and the stem below the panicles usually snaps. Panicle branches and glumes also are attacked.

Causal organism. Blast is caused by *Pyricularia oryzae* Cav. There has been much confusion and discussion about the correct name. In most of the published literature the generic name is erroneously spelled *Piricularia.*

No perfect stage of the fungus has ever been found. The conidiophores are produced in clusters with a few to many conidiophores per cluster. The conidiophores are septate, slightly thickened, and dark-colored at the base. One to 20 conidia may be found on the tip of each conidiophore. The conidia are pear-shaped, two-septate, and measure about 20 to 26 microns x 8 to 10 microns, depending on the isolate and where the fungus is grown. They germinate from the apical or basal cells, or from both, less frequently from the middle cell.

Many pathogenic races of the fungus have been identified. Various sets of differential varieties have been used in various

countries. An international set now in wide use consists of eight varieties: Raminad Str. 3, Zenith, NP 125, Usen, Dular, Kanto 51, Sha-tiao-tsao(s), and Caloro.

At IRRI we recently found that not only do pathogenic races exist in nature but also that single spores or single cells (hyphal tips) may also produce many new races. The fungus therefore has very variable pathogenicity. The pathogenicity pattern of a race based upon one test is only a partial and transitory phenomenon. A single spore may produce as many as 50 races when 100 to 200 single-spore progeny are tested. This is an unusual feature of the fungus.

Two toxins have been isolated from the filtrate of the fungus culture and severely diseased plants: picolinic acid and an unknown compound called "piricularin." The toxins inhibit both the growth of rice seedlings and the germination of the conidia of the fungus.

Disease cycle. The conidia of *P. oryzae* germinate in 4 hours and invade the host cells in 8 to 10 hours when conditions are favorable. Lesions begin to appear in 4 days and a crop of spores is produced in 6 to 7 days. A large lesion (the type that appears on susceptible varieties) produces 2,000 to 6,000 conidia each night for about 2 weeks. Each small lesion (intermediate type) produces 50 to 300 conidia for about 5 to 7 days. Most conidia are released at night in the presence of dew or rain. The disease cycle is short and most damage is caused by secondary infections.

Air can carry the conidia for long distances. Conidia have been trapped at 2,000 meters above the ground. Most conidia, however, are near the ground at the level of plants. In a blast nursery, 4,000 or more conidia have been collected in each liter of air in 1 day.

In the tropics, conidia may be collected from the air throughout the year, though the population is low during the dry season. In temperate regions, the fungus overwinters in straw piles and on grain. This may also happen in the tropics.

Whether grasses serve as hosts is controversial. Some researchers have shown that *P. oryzae* can infect grasses, others have found that it cannot. The change in pathogenicity of the

races used in these experiments may partly explain the disagreement.

Effect of environmental conditions. Climatic conditions and the nutritional status of the host plants greatly affect the development of blast disease. Since the temperature in the tropics is near the optimum range for the causal organism, moisture is the main climatic influence on the development of blast disease. Both the release and germination of spores require the presence of free water. High humidity favors development of lesions. The disease is more destructive in areas with more rainfall, although the duration and frequency of rainfall are as important as the amount of rainfall, if not more. Heavy rainstorms or typhoons are unfavorable for the development of the disease. Close planting or heavy application of nitrogen fertilizers favor the disease because thick growth raises the humidity of the microclimate. Relatively cool nights favor the formation and duration of dew.

Many experiments and observations have shown that a high nitrogen supply increases the incidence and severity of blast, particularly when quick-acting fertilizers, such as ammonium sulfate, are applied at high rates all at one time. Split applications usually lessen the effect. The severity of blast is also greater in soils with low fertilizer-holding capacity, such as sandy or shallow soil, while it is less in clay or deep soils. The accumulation of soluble nitrogen in heavily fertilized plants favors the growth of the fungus.

Several early experiments showed that potassium reduced infection. Later, however, it was found that in soil with adequate potassium the addition of potassium increases incidence of the disease.

Plants high in silica content often are less infected by blast and increased application of silica increase resistance to blast. Blast causes more damage on upland rice than on lowland rice, possibly because more silica accumulates in lowland soils and because of more favorable microclimate.

Varietal resistance. Hundreds of pathogenic races of *P. oryzae* occur. In the Philippines, about 200 races have been identified.

Varietal resistance is best measured by the number of races to which a variety is resistant. Highly resistant varieties are not infected by most races. Even the most susceptible varieties are resistant to a few races.

In one season at one locality, a group (say, several dozens) of races develop. The conidia population of each race may differ, that is the conidia of some races may be more numerous than the conidia of other races. In a different season at the same locality, different races may be present although certain races tend to be present from season to season. Within a season, the group of races present at one locality is usually quite different from the group present at another locality. A susceptible variety will be infected in most seasons or in most localities. A moderately resistant variety sometimes escapes infection because among the races present in a given locality or season, few or none may be pathogenic to it. But in other seasons or localities the same variety is likely to be infected. A highly resistant variety rarely becomes infected because only a few of the hundreds of races are pathogenic to it.

In a test of 8,200 varieties in the IRRI blast nursery, 1,400 (17%) were rated highly resistant. But after several subsequent tests, only 75 of the 1,400 varieties could still be rated resistant. That shows that the races present from season to season are different. Similarly, races in one locality often differ from those in other localities. This phenomenon is most obvious in susceptible varieties from temperate regions that are resistant in the tropics and vice versa.

The largest test for varietal resistance to blast is the international uniform blast nurseries program (IBN). Between 1963 and 1972, tests at 56 test stations in 26 countries identified many resistant varieties. A partial listing of varieties showing a high degree of resistance to blast from the results of IBN is given below. These varieties are believed to be the best sources of resistance for breeding programs in all countries.

The resistance of these varieties is stable. That means that the few races that can infect these varieties cause little damage. These few races produce, in their progenies, many new races, but only few of the progeny races can infect these varieties. This is contrary

Partial list of the most resistant varieties selected from international uniform blast nurseries.

GROUP I	
Tetep	Ram Tulasi
Nang chet cuc	Ram Tulasi (Sel)
C-46-15	Thava Lakkanan Ptb 9
Tadukan	Macan Tago
Trang cut L. 11	Ahmee Puthe
Pah leuad 111	Ca 435/6/5/1
H-5	DNJ-60
R-67	Ca 902/b/2/2
CI 7787	R-67
MekeoWhite	N-12
D-25-4	Pah Leuad 29-8-11
M-302	T 23
Padang Trengganu 22	DZ-193
Ta-poo-cho-z	Pi-4
Amritsari HR 22	Ca 902/b/3/3/1
GROUP II †	
C-46-15	DL-5
Mamoriaka	Andi from N. Pokhara
Carreon	Ca 902/b/2/1/
Huan-sen-go	T 9
Dissi Hatif	T 3
M 320	Pi-3
Colombia 2	W.R.C. No. 4
Basmati T3	T 1
J-519	PI 184675-4
Unblatuzi Valley Sugar Co.	S20 J.K.W.

* 280 tests † 190 tests

to the conventional concept that when a new race develops, the resistance of a variety breaks down completely. "Horizontal" resistance, in its restricted sense, has not yet been demonstrated in rice varieties.

Blast nurseries are most useful for testing varietal resistance or for screening breeding lines for blast resistance. Repeated tests must however be conducted in several localities in a country or internationally to determine the degree of resistance of varieties.

SHEATH BLIGHT

Sheath blight was reported in Japan about 1910, and in the Philippines in 1918. From 1930 to 1940 it was reported in Sri Lanka, China, and India, and a similar disease was reported in the U.S.A. It now occurs in most rice growing countries.

In Japan in recent years, sheath blight infected about 120,000 to 190,000 hectares resulting in a loss of about 24,000 to 38,000 tons of rice annually. In one study in Japan, fungicide treatment increased the yield by 1.6% when 5% of the hills in a field were infected, by 6.4 to 7.1% when 50% of the hills were infected, and by 8.9 to 10.1% when all hills were infected. When the disease reaches the flag leaves, loss is estimated at 25 percent.

In the tropics, when infection occurs, it is not unusual to find most leaves killed by maturing time. The yield losses in the tropics are greater than in Japan. The destructiveness of the disease in the tropics tends to increase with the use of more fertilizer and the use of the modern varieties. The new varieties produce more tillers which raise the humidity of the plant layer, creating favorable conditions for the disease.

Symptoms. Sheath blight causes spots or lesions, mostly on the leaf sheath. The lesions may extend to the leaf blade if conditions are favorable. The spots first appear near the waterline in fields. They are greenish gray, elliptical or oval, and about 1 cm long. They enlarge and lengthen to 2 or 3 cm and become grayish white with brown margins. Brown sclerotia, which are easily detached, form on these spots. Under humid conditions, the mycelium of the fungus may grow upward to infect other leaf sheaths and leaf blades touching each other. In severe infections, all leaves of a plant may be blighted.

Causal organism. The fungus is generally referred to as *Hypochnus sasakii*, *Corticium sasakii*, *Pellicularia filamentosa*, *P. sasakii*, and other names. The imperfect stage is *Rhizoctonia solani* and according to recent studies, the perfect stage should be called *Thanatephorus cucumeris*.

The fungus grows readily on common media. The hyphae

Tillers infected with sheath blight showing characteristic lesions on the sheath and sclerotia formed on the lesions.

are colorless when young, yellowish brown when old. Many sclerotia form on media; they are brown or dark brown, more or less globose, flattened on the lower side. Both the mycelium and the sclerotia vary in form, size, number, and rate of growth. The perfect stage produces basidia, 10 to 15 microns x 7 to 9 microns, and basidiophores, 8 to 11 microns x 5 to 6.5 microns.

The fungus grows over a wide range of temperature. The optimum temperature is relatively high, about 28 to 30 C. Some strains grow well at 35 C. The size and number of sclerotia on media apparently depend on the nitrogen source.

Strains of the fungus differ in pathogenicity. Strains with poor mycelial growth often are less pathogenic.

Disease cycle. The sclerotia of the fungus can survive in soil from several months to 1 or 2 years, depending upon temperature and moisture conditions. While the soil is being prepared for rice planting, the sclerotia float on water and infect plants with which they come in contact. Mycelium grows rapidly on or inside the tissues, upward as well as sidewise, initiating secondary spots. In turn sclerotia are produced on the spots. The mycelium enters the plant through the stomata or it penetrates directly through the cuticles. From the ordinary mycelium, a special type of mycelium, short and much branched, is produced which causes infection.

Effect of environmental conditions. Many reports have shown that the disease is especially destructive under highly humid and warm temperature. While temperature in the plant layer runs parallel to the daily air temperature, humidity is greatly affected by the thickness of stand. Close planting and heavy fertilization tend to increase the incidence of the disease. For the same reason the disease is usually observed in the field after the plants have reached maximum tillering stage—when high humidity can be maintained in the microclimate.

High rates of nitrogen fertilizer also make the tissues more susceptible to the disease. More potassium fertilizer, on the other hand, seems to reduce the disease incidence.

The intensity of primary infection in fields is closely related

to the number of sclerotia in contact with plants. The subsequent disease development is greatly affected by environmental conditions and susceptibility of plants.

Varietal resistance. Two years of field tests in Taiwan showed that a resistant variety lost 30 percent of its leaves while a susceptible variety lost 90 to 100 percent. It seems difficult to obtain highly resistant varieties, though no systematic, large-scale screening for resistant varieties has been done. Recent studies at IRRI suggest that plants at late stage of growth (flowering) are more susceptible than at early stage and varieties resistant in seedling test may become susceptible at late stage or vice versa.

Some resistant varieties found in Taiwan are Ching-kao-chan, Chung-lin-chung, Pingtung-sung-su 2391, Zenith, and Kamrose. Among the varieties tested at IRRI, DV112, 46 Palman, Ta-poo-cho-z, Mehran, Kam Bau Ngan, Dular, several hybrid lines of IR1103, IR1330, IR442, and a few others are resistant.

Chemical control. Chemicals are used to control sheath blight in Japan. Organic arsine compounds are the most effective, particularly methylarsine sulfide and urbacid. They are applied two times at about 50 ppm. Adding a small amount of iron, such as ferric chloride or ferric sulfate, reduces the phytotoxicity of the organic arsines. An antibiotic compound, Polyoxin, has also been developed in Japan for controlling the disease. Several chemicals tested at IRRI also showed effective control.

STEM ROT

Stem rot was reported in Japan in 1910, in India in 1913, in Sri Lanka in 1920, in Vietnam and the U.S.A. in 1921, and in the Philippines in 1924. The fungus, however, was described earlier. The sclerotial stage, *Sclerotium oryzae,* was reported in 1876; and the conidial stage, *Helminthosporium sigmoideum,* in 1889. E. C. Tullis in 1932 found that the two were the same fungus at different stages. He also found that its perfect stage, *Leptosphaeria salvinii,* was the same fungus and discovered a similar

Stem rot. Numerous small, black sclerotia inside the stubble.

fungus causing the same disease called *Helminthosporium sigmoideum* var. *irregulare*. Recently, however, workers in the U.S.A. found that the perfect stage is *Magnaporthe salvinii* (Catt.) Krause and Webster.

Stem rot has also been found in Bulgaria and Italy in Europe; in Kenya, Malagasy, and Mozambique in Africa, and in Brazil, Colombia, and Guyana in Latin America. It is present in all countries of Southeast Asia, in almost every field where rice has been grown for many years.

Early reports indicated heavy losses from stem rot. As much as 50% yield loss has been reported in Vietnam, 30 to 80% in the Philippines, 18 to 56% in India, and 10% in the U.S.A. In Japan 51,000 to 122,000 hectares are infected annually and yield losses amount to 16,000 to 35,000 tons. On the other hand, although the fungus is endemic and present in virtually all of Italy, it seldom causes appreciable damage unless the infected plants are weather-beaten.

IRRI studies in the Philippines show that the stem rot fungus is a wound parasite. The damage may be caused by a combination of insect damage, lodging, and other injuries which facilitate the occurrence of the disease. The disease was present in all 299 samples collected from all parts of the Philippines, and 67 percent of the 120,000 tillers examined contained the fungi. Two fungi sometimes occur on the same hill.

Symptoms. Stem rot starts with a small, blackish, irregular lesion on the outer leaf sheath near the water line at the later growth stages of the rice plant. The lesion enlarges as the disease advances. The fungi penetrate the inner leaf sheath and finally the leaf sheath rots and sclerotia form. Further advance of the fungi causes infection on the stem (culm). One or two internodes of the stem rot and collapse, leaving only the epidermis and vascular bundles. These infected stems lodge. Small black sclerotia dot the inside of split internodes. The next internodes may be completely healthy. The presence of the sclerotia usually makes diagnosis easy and positive.

Occasionally in the tropics, the disease appears on young

transplanted seedlings. Outer leaf sheaths rot at the water line, causing the leaves above them to die.

Causal organisms. Two closely related fungi cause stem rot. *Leptosphaeria salvinii* or *Magnaporthe salvinii* is the perfect stage of *Helminthosporium sigmoideum*, but very rarely found. Only conidial stages are known for *H. sigmoideum* var. *irregulare*.

The sclerotia of *H. sigmoideum* are spherical, or nearly so, black at maturity, smooth surfaced, 180 to 820 microns, mostly 230 to 270 microns. The conidia are fusiform, slightly curved, four-celled; intercalary cells are brown while terminal cells are almost colorless, mostly 11 to 12 microns x 34 to 40 microns. The perithecia are dark, globose, 202 to 481 microns, with a short beak. The asci are narrowly clavate, mostly 103 to 125 microns x 13.5 microns. Ascospores are fusiform, four-celled, somewhat curved, 38 to 53 microns x 7 to 8 microns.

The two organisms differ morphologically. *H. sigmoideum* produces spherical, shiny, smooth, slightly larger sclerotia, mostly on the surface of plant tissues or agar; *H. sigmoideum* var. *irregulare* has irregular, dull, rough, and smaller sclerotia, mostly embedded in the tissues or agar. The former has more aerial mycelium. The conidia of the latter often have long appendages and are not always four-celled.

Both fungi are readily cultured on media and produce abundant sclerotia. *H. sigmoideum* produces pigments in medium, but *H. sigmoideum* var. *irregulare* does not. Little is known about the pathogenic strains and variability of the two fungi.

Disease cycle. In the field, sclerotia are mostly distributed in the upper 5 to 10 cm of the soil. These sclerotia float on the water during plowing, puddling, weeding, and other operations. Those in contact with the leaf sheath produce appressoria or infection cushions and may start infection.

The pathogenicity and mode of infection have not been studied critically. By floating the sclerotia on the water some workers successfully inoculated the plants while others failed. Recent studies at IRRI showed that infection takes place readily if a wound is present while no infection occurs without a wound.

That perhaps explains the inconsistency of results obtained by earlier workers. The fungi are mainly aggressive wound pirates; on susceptible varieties an internode may be rotted completely in 10 days.

After harvest, the fungus continues to grow on stubble, producing large quantities of sclerotia. Irrigation water may carry the sclerotia to other fields.

Effect of environmental conditions. High nitrogen levels increase stem rot but potassium reduces the harmful effect of excessive nitrogen. Phosphorus also stimulates the disease but with less effect. Silica inhibits the disease. In one study the disease incidence was two or three times higher on plants infested by stem borers than on those not infested. We believe that any injuries, such as from insects or lodging, induce infection.

Varietal resistance. Varieties differ greatly in resistance to stem rot. In the same inoculation experiment, the internode of a susceptible variety may completely rot in 10 days while that of a resistant variety may have only a brown lesion less than 1 cm long.

Early reports showed that variety Dudshar of Bengal was highly resistant, Hondurawela (Ceylon) was free from the disease, while others were severely attacked. Ramai, Elonelon, Arabon, and Kinaturay of the Philippines have promising degrees of resistance. A resistant variety, Raminad Str. 3, was developed by crossing Ramai and Inadhica. Bozu, Onsen, Span Jap., Mubo Aikoku 22, and others were resistant in the U.S.A. Gimbozu was highly resistant and strains of Shinriki and Sembon were usually resistant in Japan. Only a few hundred varieties have been tested at IRRI. Most had an intermediate reaction. The most resistant varieties tested are Seri Raja, Lembu Basah, Machang, Raminad Str. 3, and BPI-76. Dular is very susceptible.

Control. No chemical has been useful for control of stem rot. Burning rice stubble and infected straw, when practical, may reduce the availability of inoculum. The use of resistant or nonlodging varieties are basic approaches in controlling the disease.

BROWN LEAF SPOT

J. Breda de Haan first described the brown leaf spot fungus in 1900 and called it *Helminthosporium oryzae*. About the same time the disease was being studied in Japan. Since then many studies have been conducted in other countries. Since 1950 extensive studies have been made in Japan in connection with a physiological disorder called *akiochi*.

Brown leaf spot has been found in all rice-growing countries in the Western Hemisphere, in Asia, and in Africa. In Southeast Asia, however, it is seldom found in normal, good soil but is common in soils with nutritional deficiencies or in poorly drained soils where uptake of nutrients is hindered.

Early studies reported heavy damage from the disease through seedling blight. The most dramatic report about the disease was that it contributed significantly to the Bengal famine in 1942, having caused 50 to 90 percent yield losses. The disease is now known to be associated with unbalanced or poor soil. Brown leaf spot adds to the plant damage caused by poor soil.

Symptoms. The most conspicuous symptoms of brown leaf spot appear on the leaves and the glumes. The disease has been called "sesame leaf spot disease" in Chinese and Japanese literature. The typical spots on leaves are oval, about the size and shape of sesame seeds. They are relatively uniform and fairly evenly distributed over the leaf surface. They are brown with a gray or whitish center when fully developed. Young or undeveloped spots are small, circular, and appear as dark-brown or purplish-brown dots. On very susceptible varieties the spots may be larger, up to 1 cm long. Sometimes numerous spots occur and the leaves wither.

On glumes, black or dark-brown spots appear. In severe cases, the spots may cover the entire glumes. Under favorable climatic conditions, dark-brown conidiophores and conidia develop on these spots, giving them a velvet-like appearance.

The leaf spots may be confused with leaf spots of physiological origin. Fully developed spots with gray centers are the only diagnostic symptoms in the field.

Typical symptoms of brown leaf spot are oval, relatively uniform spots.

Causal organism. The causal organism was first called *Helminthosporium oryzae*, the imperfect stage. The perfect stage, discovered in 1927, was named *Ophiobolus miyabeanus*. Later it was changed to *Cochliobolus miyabeanus* (Ito et Kuribayashi) Drechsler ex Dastur.

The conidiophores are simple, brownish, nearly colorless at the tips, 150 to 600 microns x 2 to 8 microns. Conidia are brown, 35 to 170 microns x 11 to 17 microns, septate. Large conidia contain as many as 13 septa, are more or less cylindrical, slightly curved, wider at the middle or somewhat below the middle, the distal portion tapering toward the hemispherical apex.

The perithecia are globose to depressed globose, 560 to 950 microns x 368 to 377 microns. The asci are cylindrical to long, fusiform, 142 to 235 microns x 21 to 36 microns. The ascospores are filamentous or long-cylindrical, coiled together, 6- to 15-septate, 250 to 468 microns x 6 to 9 microns.

The fungus grows over a wide range of temperatures on different kinds of media.

Disease cycle. The conidia usually germinate by germ tubes from apical and basal cells, sometimes from other cells. The germ tube is covered with a mucilaginous sheath which adheres to the leaf surface and an appressorium is formed at the tip. Infection pegs form under the appressoria and penetrate directly into the epidermis. The germ tubes may enter the leaf tissue through the stomata without forming appressoria. The mycelium penetrates and kills the cells, causing brown spots to appear.

The fungus reproduces on the spots and is disseminated by air current. Few or no spores develop on minute spots. A small number of conidia are produced on small spots while a large number develop quickly on big spots. Studies in India show that the air-borne conidia population over rice fields is high from October to January. These air-borne conidia are responsible for secondary infections.

In infected tissues the fungus lives long; a life span of 3 years has been recorded in Japan. Its longevity, of course, depends on the temperature and moisture conditions. Under high temperature and humidity the fungus does not live more than a few months.

Most primary infections start from diseased seeds. Coleoptiles and sometimes roots are infected. Leaf spots are mostly from secondary infections.

By artificial inoculation, several grasses can be infected by the fungus, but they seldom are infected under natural conditions.

Effect of environmental conditions. Brown spot disease has been associated with soils that are deficient in nutritional elements and with reduced soils in which toxic substances have accumulated.

Brown leaf spot disease and akiochi disease occur together when silica, potassium, manganese, or magnesium is deficient or where hydrogen sulfide is evolved, causing root rot. Brown spot disease also attacks the plants when nitrogen deficiency is induced at late growth stages. Plants are less susceptible, however, when soils have low phosphorus levels.

Hydrogen sulfide inhibits the absorption of nutrients and water. Akiochi and brown spot occur because H_2S selectively inhibits uptake of nutrients and disturbs the nutrient balance. Harmful organic acids such as butyric and acetic acids affect the plant in the same way as H_2S.

These studies seem to show that the brown spot disease is brought about directly or indirectly by deficiency of one or more nutritional elements, and often brown spot and akiochi are actually inseparable. Brown spot disease has been used as an index of akiochi. Thus it is difficult to distinguish between the losses due to brown spot and those due to soil conditions.

Some experiments have also shown that in plants grown in dry soil the disease is more severe than in plants in moist or wet soils. Too high or too low water temperature and high air temperature selectively inhibit nutrient absorption, and thus induce brown spot.

Varietal resistance. Experiments have been conducted in many countries to determine varietal resistance. Most of them, however, involve relatively few varieties. In one study in India, Dakar Nagra, 273-22, 5-34-33, Patnai 23, Kalma 219, and Nagra 41-14 were resistant. In another study in India Ch 13, Ch 45, T-141,

T-498-2A, Co 20, BAM-10, T-988m, T-2112, T-2118, and T-960 were resistant. Hainan 217 and Chiu Tiu Chiu, Kukubozu, Diakokuwase, Yamahaku, Hinomaru, and Norin 17 were resistant in Japan. In the U.S.A. the variety CI 9515 was found highly resistant. It was crossed with Texas Patna, TP4-9, Century Patna, and Bluebonnet. Several lines were selected but the yields were low. Variety Dawn, released in 1966, is moderately resistant to brown spot disease.

Chemical control. Treating seed with chemicals and field spraying to control brown spot has been widely tried. Seed treatment may be beneficial if the seed is harvested from infected fields. Since the disease is associated with poor soil, even if it is controlled, the plants will not grow normally. The value of leaf spray is doubtful.

NARROW BROWN LEAF SPOT

Narrow brown leaf spot disease was first described in Japan in 1910. It is widely distributed in Asia, North America, and Africa. During the 1930's and 1940's, narrow brown leaf spot was an important disease in the U.S.A. A 40-percent loss from the disease was reported in Surinam in 1953-54. In Asia narrow brown leaf spot is important on very susceptible varieties.

Narrow brown leaf spot produces short, linear, brown lesions or spots, mostly on leaf blades. It may also occur on the sheath, pedicels, and glumes. The lesions are about 2 to 10 mm long, about 1 mm wide, narrow, short, and dark brown on resistant varieties but wide and light brown on susceptible ones. Numerous lesions may appear at later stages of growth.

The conidial stage of the fungus is *Cercospora oryzae* Miyake. Conidiophores emerge from the stomata; they are dark at the base, paler at the apex, have three or more septa and are 88 to 140 microns x 4.5 microns. Conidia are cylindrical to clavate, 3- to 10-septate, 206 x 5 microns.

The perfect stage is *Sphaerulina oryzina* Hara described in 1918. It was not known as the perfect stage of *C. oryzae* until 1948. Perithecia are globose or nearly so, black, 60 to 80 microns

Narrow brown leaf spot.

in diameter. Asci are cylindrical or club-shaped, 50 to 60 microns x 10 to 13 microns. Ascospores are spindle-shaped, more or less straight, three-septate, hyaline, 20 to 23 microns x 4 to 5 microns.

The fungus grows on bean agar and rice straw agar, producing abundant conidia on the latter.

Studies in the U.S.A. with eight differential varieties revealed that the fungus has several pathogenic races.

The fungus enters the host tissue through the stomata, becomes established in the parenchyma, and spreads longitudinally. When fully developed, conidiophores are produced through the stomatal openings. Other information on the disease cycle in the tropics is lacking.

Many varieties have been found resistant in tests in the U.S.A.: Rexoro, Fortuna, Nira, Iola, CI 461, and 4440. Some varieties were resistant to certain races but susceptible to others. Asahi and Kamrose were resistant to all races in 1950. Several resistant varieties have been developed: Delrex (Rexoro x Delitus), Sunbonnet (Rexoro x Fortuna), Selection 44C507, and Toro.

BAKANAE DISEASE

Bakanae disease has been known in Japan since 1828. E. Kurosawa in 1926 demonstrated the hypertropic or "bakanae" effect of the fungus on its hosts. This unique phenomenon led to the isolation of gibberellin and other growth-regulating substances in 1935.

Bakanae disease is widely distributed in all rice-growing areas. It was called "white stalk" in China and "man rice" in the Philippines. It has been reported from British Guiana and other countries.

In general, severe damage is rare except in specific localities or seasons. But losses as high as 20 percent in Hokkaido, Japan, 15 percent in Uttar Pradesh, India, and 3.7 to 14.7 percent in Northern and Central Thailand have been reported in some years.

Symptoms. The most conspicuous and common is the "bakanae" symptom, an abnormal elongation of the infected plants in the

seedbed or the field. Infected seedlings are several centimeters taller than normal plants and are thin and yellowish green. Infected plants are scattered throughout the seedbed. Most severely infected seedlings die before transplanting, some die afterwards.

In the field, infected plants have tall, lanky tillers bearing pale-green flag leaves above the general level of the crop. These plants have few tillers and the leaves die one after another. Those surviving until maturity bear empty panicles. A whitish or pink fungus growth may appear on the lower portion of the dying plants.

Infection may also stunt some plants instead of causing them to elongate. Environmental conditions affect the balance of growth hormones that an infected plant produces, so the disease can cause either stunting or elongation.

Causal organism. The fungus has both the conidial stage and the perfect stage. The conidial stage is a *Fusarium,* the perfect stage, *Gibberella.* Several names have been applied to the fungus: *F. heterosporium, Lisea fujikuroi,* and *G. fujikuroi.* According to present conventional classification, it is called *Gibberella (Fusarium) moniliforme* (Sheld.) Winel.

The perithecia are dark blue, spherical or egg-shaped, 250 to 330 microns x 220 to 280 microns; asci are more or less cylindrical, 90 to 102 microns x 7 to 9 microns; ascospores have one septum, 15 x 5.2 microns.

Two types of conidia are produced. Microconidia are agglutinated in chins, yellowish to rosy white, one-celled or two-celled, and fusiform or egg-shaped. Macroconidia are awl-shaped, sickle-shaped, or almost straight, narrowed at both ends, buff or salmon-orange in mass, mostly three- to five-septate, variable in size from 12 to 22 microns x 2.2 to 3.5 microns for the one-septate conidia to 61 to 82 microns x 2.7 to 4.5 microns for the five-septate conidia.

The fungus can be grown on many media. producing fusaric acid and gibberellin depending on the conditions under which it is grown. When more fusaric acid is produced the infected seedlings are stunted; when more gibberellin is produced the plants elongate abnormally.

Disease cycle. The disease is seed-borne. Seeds may be infected at flowering time. Severely infected grains are discolored, sometimes pink due to the presence of conidia masses. Healthy-looking seeds may also carry the fungus which infects the seedlings at the very early stages of germination. Infection is milder if the seeds are pregerminated 3 days before sowing in infected soil than if pregerminated for only 1 or 2 days. Sowing dry seeds results in a higher percentage of infection than sowing pregerminated seeds. Mycelium and microconidia are systemic in the plants. They grow upward, and are produced later on the dead tissues. The fungus on seeds may live for a long time.

Bakanae disease is also said to be soil-borne. But the fungus seems to be short-lived in the soil in the tropics. Experiments show that 90 percent infection from soil may be reduced to 1 percent after 3 months.

The fungus produces conidia on infected plant parts. The copious production of conidia on dead culms during the later part of the growing season coincides with flowering and maturation of the crop. The conidia infect the seeds.

The application of nitrogen has been reported to stimulate the development of the disease.

Varietal resistance. Varieties differ greatly in resistance, though no large-scale test has been made. Shiroka, Akage 3, and Kairyo Mochi 1 were resistant in Japan. In tests in India, Wateribune had 1.1% infection, Aryan, 1.9%, and GEB 24, 4.8%, while Co 13 had 95.6%, Tinnevelly Anarkomban, 95.1%, and Gobikar, 93.5%.

Control. Seed treatment with organic mercury compounds or other fungicides, in dust form or in solution, effectively controls the disease.

FALSE SMUT

False smut (or green smut) was first described in ancient Chinese literature. Its presence was believed to indicate a good crop year. This belief is still common in countries of Southeast Asia. The fungus was named *Ustilago virens* by M.C. Cooke in 1878 based

on a specimen from India. Several studies followed and other names have been used. The perfect stage, *Ustilaginoidea virens*, was discovered in 1934 in Japan.

Most countries in Asia, Latin America, and Africa have reported the presence of the disease. The disease usually causes little damage, except in small areas under special conditions. O.A. Reinking mentioned severe infection in the Philippines in 1918, and L.N. Seth reported an epidemic in Burma in 1935.

The symptoms are conspicuous. The fungus transforms individual grains of the panicle into yellow or greenish velvety spore balls which are small at first and 1 cm or longer at later stages. At early stages, the spore balls are covered by a membrane, which bursts with further growth. The color of the ball changes from orange to yellowish green to greenish black. Usually only a few grains in a panicle are infected.

The fungus is called *Ustilaginoidea virens* (Cke.) Tak. The asexual stage is the smut ball on plants. The smut balls mature from outside to inside. The outer layer consists of maturing spores and inner layers are mycelia in different stages of development. Chlamydospores are spherical to elliptical, warty, olivaceous, 3 to 5 microns x 4 to 6 microns. Young spores are smaller, lighter colored, and almost smooth. The chlamydospores germinate to produce very small, ovoid conidia.

Some smut balls develop one to four sclerotia in the center. After overwintering, the sclerotia produce stalked stromata in the next summer and fall which contain perithecia. The perithecia are flask-shaped, each containing about 300 asci. The asci are cylindrical, 180 to 220 microns x 4 microns. The ascospores are hyaline, filiform, 120 to 180 microns x 0.5 to 1 micron.

In temperate regions, the fungus survives the winter through sclerotia as well as through chlamydospores. Ascospores produced on the overwintered sclerotia apparently start primary infections. Chlamydospores are important in secondary infection which is a major part of the disease cycle.

Field observations in Japan indicate that infections occur at the booting stage of rice plants. Histological studies in India show that two types of infection may occur. One is at early flowering where the ovary is destroyed, while the style, stigmas,

False smut showing individual grains turned into yellowish green smut balls.

and anther lobes remain intact and are ultimately buried in the sclerotium. The second type of infection takes place when the grains are maturing. Spores in contact with the endosperm through glumes grow, changing the endosperm into smut balls.

Chlamydospores are air-borne but do not free themselves from the ball easily because of the presence of sticky material.

The disease also infects other species of *Oryza* and maize *(Zea mays)*.

High moisture favors the development of the disease. Conditions favoring vegetative growth of the rice plant also stimulate the disease.

Little is known about varietal resistance to false smut. Certain varieties are more frequently attacked than others planted in the same field. The disease usually does not warrant special control measures.

SHEATH ROT

Sheath rot, first described in Taiwan in 1922, is found in Japan and all countries of Southeast Asia. Little is known about the exact damage it causes though in some fields 10 to 25 percent of the tillers may occasionally be infected.

The rot occurs on the uppermost leaf sheaths enclosing the young panicles. Oblong or irregular spots develop into a lesion 0.5 to 1.5 cm long, with a brown margin and a gray center, or the lesion may be grayish brown throughout. The lesion enlarge and often merge. They may cover most of the leaf sheath. The young panicles remain in the sheath or emerge only partly. A whitish powdery growth of the fungus may be found on the panicle inside the sheath. The young panicles may rot completely.

The causal organism is *Acrocylindrium oryzae* Sawada. The mycelium and conidiophores are inconspicuous. The conidia are hyaline, smooth, single-celled, cylindrical, and 4 to 9 microns x 1.2 microns.

In Southeast Asia diseased plants are usually associated with stem borers or other causes of injury to the lower portion of the stem. Such injuries keep the panicles from emerging normally.

Panicles attacked by sheath rot.

This observation suggests that the fungus mainly attacks panicles that do not emerge normally.

WHITE TIP (NEMATODE)

White tip was first discovered in Japan in 1915 and was called "black grain disease." The following year, a similar disease was found on Italian millet, also in Japan. In 1944, the disease was renamed "nematode heart blight" and the nematode was identified as *Aphelenchoides oryzae* Yokoo in 1948. The disease was first reported in India in 1936 and was called "light ears." In the U.S.A. it was noticed in 1935 and was attributed to iron or magnesium deficiency. In 1949 it was found to be caused by a nematode similar to the nematode disease in Japan. The disease was called "white tip."

White tip disease is also found in Bangladesh, Indonesia, Philippines, Sri Lanka, Taiwan, and Thailand in Asia; Cuba, El Salvador, and Mexico in Latin America; Malagasy, Sierra Leone, and Senegal in Africa; and in Australia and Italy.

The losses vary from one country to another. In Japan estimated loss ranges from 10 to 35 percent annually, in Taiwan, 29 to 46 percent, and in the U.S.A., 40 to 50 percent in severely infected fields.

Symptoms. The common name of the disease, "white tip," is based on the characteristic symptom: the leaf tip becomes chlorotic or white. Later, this area turns brownish and breaks off. Affected panicles have high sterility, distorted glumes, and small and distorted kernels. The infected leaves are dark green. Diseased plants also produce tillers at higher nodes.

The causal organism. The white tip nematode, once referred to as *Aphelenchoides oryzae* Yokoo (1948), was found later to be identical to *A. besseyi* Christie (1942). The female nematode ranges from 0.62 to 0.88 mm long, the male from 0.44 to 0.72 mm.

To examine the nematode in infected materials, hulls and seeds are separated and soaked in warm water (about 50 ml) for

White tip.

12 to 24 hours. Folded young leaves are cut into small pieces and soaked in warm water. Some nematodes emerge a few hours after soaking but most of them take 12 to 24 hours.

Disease cycle and host range. The nematode survives in seeds for 8 to 24 months in the U.S.A., and even up to 3 years in Japan. The worm is an ecto-parasite occuring within the young folded leaves in the early stages, entering the spikelets after ear formation and mostly remaining within the glumes at maturity of the grains. The nematodes can be found mostly on the inner surface of the hulls and some on kernels. Some may be found also in the inner surface of the leaf sheaths. Few nematodes remain in seeds at germination. The population of nematodes increases as the plant grows older. They move in paddy soil from hill to hill.

Infection may start from infected seeds. Irrigation water containing nematodes or diseased husks also spreads infection.

The nematodes can be found on a wide range of hosts such as *Setaria virides, S. italica, Panicum sanguinale, Cyperus iria, C. polystachyus,* and *Imperata cylindrica.* It also occurs on millet, strawberries, Vanda orchids, chrysanthemums, African violet, and ramie.

The nematode can infect rice seedlings more easily in submerged soil than in moist soil. High temperature tends to reduce the number of nematodes present in the grains.

Varietal resistance. Some varieties become infested but do not produce symptoms. Among these varieties are Norin No. 8, Asa-Hi, Asahi No. 1, Nankai, and Tozan Nos. 36, 37, 38, and 58. Norin Nos. 6, 37, and 38, Norin-mochi No. 5, Saikai No. 37, and Nakate Ginbozu are resistant in Japan. In the U.S.A. the varieties found resistant are Blue Bonnet, Blue Bonnet 50, Asahi, Century 53, Century Patna 231, Fortuna, Improved Blue Bonnet, Nira, Rexoro, Texas Patna, Toro, and TP 49.

Control. Treating seed for 15 minutes with hot water (52 to 53 C) will reduce infection without injuring seeds. The nematodes in infested seeds may be killed by presoaking the seeds in cool water for 8 to 12 hours, preheating them in water at 55 C for 15 seconds,

and then treating them at 50 C for 15 minutes and in cool water for 5 minutes, and finally drying them.

Infested seeds may also be fumigated with methyl bromide at 410 g/25 m^3 for 6 hours. This treatment injures some varieties, however. Other chemical treatments are (1) 25 percent parathion dust, 50 percent Systox on carbon dust at 28 g/kg, (2) soaking the seeds for 12 hours in 1:1000 mercuric chloride solution, (3) Aagrano dust at 28 g/kg, (4) soaking the seeds for 24 hours in 1:200 to 1:400 of a 20-percent emulsion of ethyl thiocyanate (reported to completely control the white tip nematode), or (5) dipping rice seeds for 24 hours in 1:100 to 1:500 concentrations of active ingredient of either methyl thiocyanate or butyl thiocyanate.

In localities commonly affected by the nematode, planting earlier than usual may reduce or prevent infection in the field.

STEM NEMATODE

Stem nematode was first found in 1913 by E.J. Butler in what is now Bangladesh, where it was called *Ufra* or *Dak Pora*. Later it was found in Burma and named *akhet-pet*. It was also found in most paddy tracts in Uttar Pradesh, India. In 1956, the disease was observed in Bulacan province, Philippines, and more recently, in a small area of Quezon province. It was found in South Thailand in 1963 and caused serious damage on the high plateaus in Malagasy in 1965 and 1966.

As early as 1916 about 2.5 million hectares of rice in the north of the Bay of Bengal and east of the Ganges river were infected. In many instances the plants produced so little grain that harvest was not attempted. In 1953, stem nematode caused up to 50 percent damage in infested areas in India. In Thailand, about 500 hectares were severely damaged, causing 20 to 90 percent yield loss in 1962.

Symptoms. The most noticeable symptoms are twisted culms and empty grains. If panicles are attacked, the stems may become black and withered and grain heads may fail to form or wither.

Panicles infected with stem nematodes.

Grain heads, if formed, are twisted and deformed and contain only shrivelled grains. Culms may show brown areas near the nodes. Plants may be severely stunted. In the seedling stage the entire leaf may be twisted or severely malformed. Sometimes the base of the young leaves becomes wrinkled after the whitish-green splash patterns appear.

Causal organism. In 1913 E.J. Butler named the nematode *Tylenchus angustus*. Later it was changed to *Anguillulina angusta* in 1932 and finally, in 1936, to its present name, *Ditylenchus angustus* (Butler) Filipjev. The female nematodes are about 0.7 to 1.23 mm long and the males, 0.6 to 1.1 mm.

Disease cycle. The nematode is an obligate ecto-parasite. Infestations occur in seedlings. When air humidity rises the active worms climb up and enter the tender growing tissues where they suck the sap of the epidermal cells with their stylets. As the plant grows, the worms move up to the younger tissues. The nematodes feed on the young ear, the leaf sheath, and the young leaf blades. At panicle formation the nematodes collect and feed at the base and above the top of the stem nodes.

When ripe the host plant dries up and the nematodes coil up and pass into a resting condition. When placed in water the worms uncoil, become active, and move easily. Under desiccation the power to resume motility has been shown to remain for over 6 months and uncoiling for 15 months. Thus, the worm may easily survive from one infective period to the next. Infection takes place from nematodes left in the stubble of the previous crop.

Conditions for development. High humidity during the growing and ripening season favors the organism. The nematodes remain dormant during the dry season when no rice is planted. Infection, which takes place mostly at 28 to 30 C, is slightly reduced at 32 to 33 C or at 27 to 28 C, and is greatly reduced below 12.5 C.

Varietal resistance and host range. Little is known about varietal resistance to the organism. Among four Thai varieties, Khao-tah-oo appears to be somewhat resistant while Khao-teh-haeng 17 is

susceptible. Other varieties which show some resistance are Ginga and Norin No. 22.

The nematode can infect several species of *Oryza*, such as *O. sativa* var. *fatua*, *O. glaberrima*, *O. cubensis*, *O. officinalis*, *O. meyeriana*, *O. latifolia*, *O. eichingeri*, *O. alta*, and *O. minuta*. It cannot infect maize (*Zea mays*) or many grass species, however.

Control. Removal or destruction of the stubble in the infested field reduces the inoculum. Plowing the field after burning the stubble, then fallowing and drying for 2 to 3 months also helps reduce the nematode population.

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Appendix

DISEASES OF RICE

Virus diseases and their vectors (or methods of transmission)

Dwarf	*Nephotettix cincticeps* (Uhler)
	N. nigropictus (Stal.) (Motsch.)
	Recilia dorsalis (Motsch.)
Stripe	*Laodelphax striatellus* (Fallen)
	Unkanodes sapporonus (Mats.)
	Ribautodelphax albifascia (Mats.)
Yellow dwarf (Mycoplasma-like organism?)	*Nephotettix virescens* (Distant)
	N. cincticeps (Uhler)
	N. nigropictus (Stal.)
Black-streaked dwarf	*Laodelphax striatellus* (Fallen)
	Unkanodes sapporonus (Mats.)
	Ribautodelphax albifascia (Mats.)
Hoja blanca	*Sogatodes oryzicola* (Muir)
	S. cubanus (Crawf.)
Tungro (penyakit merah, yellow-orange leaf, mentek)	*Nephotettix virescens* (Distant)
	N. nigropictus (Stal.)
	N. parvus Ishihara et Kawase
	N. malayanus Ishihara et Kawase
	Recilia dorsalis (Motsch.)
Transitory yellowing	*Nephotettix nigropictus* (Motsch.)
	N. cincticeps (Uhler)
Orange leaf (mycoplasma like organism?)	*Recilia dorsalis* (Motsch.)
Grassy stunt	*Nilaparvata lugens* (Stal.)
Yellow mottle	*Sesselia pusilla* Gerstaeker; mechanical transmission
Mosaic	mechanical transmission
Necrosis mosaic	soil transmission

Bacterial diseases and their causal agents

Bacterial (leaf) blight	*Xanthomonas oryzae* (Uyeda et Ishiyama) Dowson
Bacterial leaf streak	*Xanthomonas translucens* f.sp. *oryzicola* (Fang et al.) Bradbury

Bacterial sheath (brown) rot (Bacterial bruzone diseases)	*Pseudomonas oryzicola* Klement *Erwinia carotovora* (Jones) Holland
Bacterial sheath (brown) stripe	*Pseudomonas panici* (Elliot) Goto et Okabe
Black rot and other bacterial diseases of rice grain	*Xanthomonas itoana* (Tochina) Dowson *X. cinnamona* (Miyake et Tsunoda) Muko *Bacterium atroviridegenum* Miyake et Tsunoda

Fungus diseases of foliage, and their causal agents

Blast	*Pyricularia oryzae* Cav.
Brown leaf spot	*Cochliobolus miyabeanus* (Ito et Kuribayashi) Dreschler ex Dastur *(Helminthosporium oryzae* Breda de Haan)
Downy mildew	*Sclerophthora macrospora* (Sacc.) Thirum., et Nares
Narrow brown leaf spot	*Sphaerulina oryzina* Hara *(Cercospora oryzae* Miyake)
White leaf streak	*Ramularia oryzae* Deighton and Shaw
Leaf smut	*Entyloma oryzae* Sydow
Stackburn diseases	*Trichoconis padwickii* Ganguly
Leaf scald	*Rhynchosporium oryzae* Hashioka et Yokogi
Collar rot	*Ascochyta oryzae* Cattaneo
Rust	*Puccinia graminis* P. f. *oryzae* Frag. *Uromyces coronatus* Miyabe et Nishid ex Deitel

Fungus diseases of the stem, leaf sheath, and roots, and their causal agents

Stem rot	*Leptosphaeria salvinii* Catt. [= *Magnaporthe salvanii* (Catt.) Krause and Webster?] *(Helminthosporium sigmoideum* Cav.*)* *Helminthosporium sigmoideum* Cav. var. *irregulare* Cralley et Tullis
Bakanae disease	*Gibberella moniliforme* (Sheld.) Winel.
Sheath blight	*Thanatephorus cucumeris* (Frank) Donk
Sheath spot	*Rhizoctonia oryzae* Ryker and Gooch

Other sclerotial diseases on leaf sheath	*Sclerotium oryzae-sativae* Sawada
	S. oryzicola Nakata and Kawamura
	S. fumigatum Nakata
Sheath rot	*Acrocylindrium oryzae* Sawada
Sheath net-blotch	*Cylindrocladium scoparium* Morgan
Crown sheath rot	*Ophiobolus oryzinus* Sacc.
	O. oryzae Miyake
Sheath blotch	*Pyrenochaeta oryzae* Shirai ex Miyake
Witch weed	*Striga lutea* (Lour.)
	S. harmonthica (Benth.)

Fungus diseases of seedlings, and their causal agents

Seedling damping off	*Fusarium* spp.
	Pythium spp.
	Achiya spp.
	Pythiomorpha sp.
Seedling blight	*Sclerotium rolfsii* Sacc.

Fungus diseases of the grain and inflorescence, and their causal agents

False smut (green smut)	*Ustilaginoidea virens* (Cke.) Tak.
Kernel smut	*Neovossia barclayana* (Bref.) Sacc. & Syd.
'Udbatta' diseases	*Ephallis oryzae* Syd.
Black kernel	*Curvularia* spp.
Minute leaf and grain spot	*Nigrospora* spp.
Glume blight	*Phyllosticta (Phoma) glumarum* (Ellis et Tracy) Miyake
Scab	*Gibberella zeae* (Schw.) Petch. (*Fusarium graminoum* Schw.
Red blotch of grain	*Epidoccum neglectum* Desmagier
Speckled blotch	*Septoria* spp.
Discolored grains	various fungi

Nematodes of rice

Bud and leaf nematode (white tip)	*Aphelenchoides besseyi* Christie
Stem nematode (Ufra or Dak Pora)	*Ditylenchus angustus* (Butler) Filipjev

Root nematode	*Hirschmanniella oryzae* Luc et Goodey
Root-knot nematodes	*Meloidogyne incognita* var. *acrita* Chitwood
Cyst nematode	*Heterodera oryzae* Luc. et Briz.
Stunt and other parasitic nematodes	*Tylenchorhynchus martini* Fielding
	Pratylenchus spp.
	Helicotylenchus spp.
	Hoplolaimus spp.
	Criconemoides spp.
	Xiphinema spp.

Physiological diseases

Akiochi
Akagare
Bronzing
Straighthead