Chapter 4. Grain diseases

Various organisms can infect rice grains before or after harvest, causing grain discoloration externally or internally. The extent of this depends on season, location, type of organism, and the time when the grains are infected, either in the field or in storage. There are two groups of fungi that are specifically responsible for grain diseases that cause discoloration. One group is known as field fungi and the other as storage fungi or storage molds. Some of the field fungi that may affect both the leaf sheath and grain infect the rice plant while it is still growing in the field before harvest. These fungi are saprophytes or weak pathogens, which are also known as opportunistic pathogens that attack the crop when it is under stress. A "true" pathogen of rice grains attacks the grains similar to the pathogens infecting other organs of the plant. Storage fungi, which cause grain discoloration of rice solely during storage, are an important component of postharvest problems. They are not included here. This online resource covers fungi causing grain diseases mostly categorized as field fungi. Some of the bacteria that may be involved in grain or sheath discoloration are collectively termed the "sheath rot complex" (Cottyn et al 1996a,b) and are discussed in the chapters on bacterial diseases in Section 2.

The majority of rice fungal pathogens are seedborne. Not many true grain diseases have been reported in the literature. "True" here means that the grain is the primary organ of the rice plant grown in the field that the fungus attacks and also referred to by Ou (1985) as "diseases of grains and inflorescence." The first of the three major grain diseases discussed in this chapter is false smut, the pathogen of which is not seedborne. Kernel smut and "udbatta" are the other two diseases of the grain in which the pathogens are seedborne. Other diseases, such as glume blight, scab, black kernel, red blotch of grain, and ear blight, are all grain diseases caused by seedborne pathogens, but are minor in importance in terms of their effect on rice production. They are mentioned briefly in the discussion partly because of this reason and partly because little research has been done on them. Although grain discoloration is also listed in this group and included in Ou (1985), in reality, it is a complex problem. Grain discoloration is more associated with modern rice production because of the lack of photoperiod sensitivity among most modern varieties.

The causal agents are numerous, including many fungi and bacteria and also some physiological disorders of rice plants. Grain discoloration may also be manifestations of infection by opportunistic pathogens after rice stink bug (*Oebalus pugnax* F.) infestation. Among the microorganisms isolated from discolored seeds are *Bipolaris oryzae, Sarocladium oryzae*, and *Cercospora oryzae*, the primary target organs of which are other parts of the rice plant. In the USA, a panicle problem known as "peck", a syndrome of floral abortion, was once associated by farmers with *B. oryzae*, but later proven to be false (Marchetti and Petersen 1984). Instead, *B. oryzae* was partly associated with kernel discoloration; the major cause of which is related to "kernel blight" of unknown etiology and rice stink bug feeding was the major factor in kernel blight (Marchetti and Petersen 1984).

In addition, other microorganisms, including *Culvuria oryzae* and many *Fusarium* spp. (for some of these fungi, see Mew and Gonzales 2002), detected from rice seed are both field and storage fungi. They do not cause distinctive symptoms that can be easily diagnosed on the grain and on other plant parts such as the culm, especially the leaf sheath. We will mention some of these but will focus discussion on only a few. As mentioned earlier, most of the grain diseases are of minor importance with the exception of false smut and, to some extent, kernel smut.

In recent years, because rice crop production has intensified, false smut has become more prevalent in irrigated rice with increased intensity in hybrid rice. Likewise, the intensity of kernel smut has also increased in hybrid rice because of the expansion of its cultivation, both inside and outside of China. Seeds of hybrid rice can be heavily infected with *Tilletia horrida*. IRRI's Seed Health Unit has found that more than 80% of the seed lots of hybrid rice are attacked by *T. horrida*.

References

Cottyn, B., van Outryve, M. F., Cerez, M. T., De Cleene, M., Swings, J., Mew, T. W. 1996a. Bacterial diseases of rice. II. Characterization of pathogenic bacteria associated with sheath rot complex and grain discoloration of rice in the Philippines. Plant Dis. 80:429-437.

Cottyn, B., Cerez, M. T., van Outryve, M. F., Barroga, J., Swings, J., Mew, T. W. 1996b. Bacterial diseases of rice. I. Pathogenic bacteria associated with sheath rot complex and grain discoloration of rice in the Philippines. Plant Dis. 80:438-445.

Marchetti, M. A., Petersen, H. D. 1984. The role of Bipolaris oryzae in floral abortion and kernel discoloration of rice. Plant Dis. 68:288-291.

Mew, T. W., Gonzales, P. 2002. A handbook of rice seedborne fungi. Los Baños (Philippines): International Rice Research Institute, and Enfield, N. H. (USA): Science Publishers, Inc. 83 p.

Ou, S. H. 1985. Rice diseases. 2nd ed. Kew, Surrey (England): Commonwealth Mycological Institute, CABI. 380 p.

1. Kernel smut (KSm)

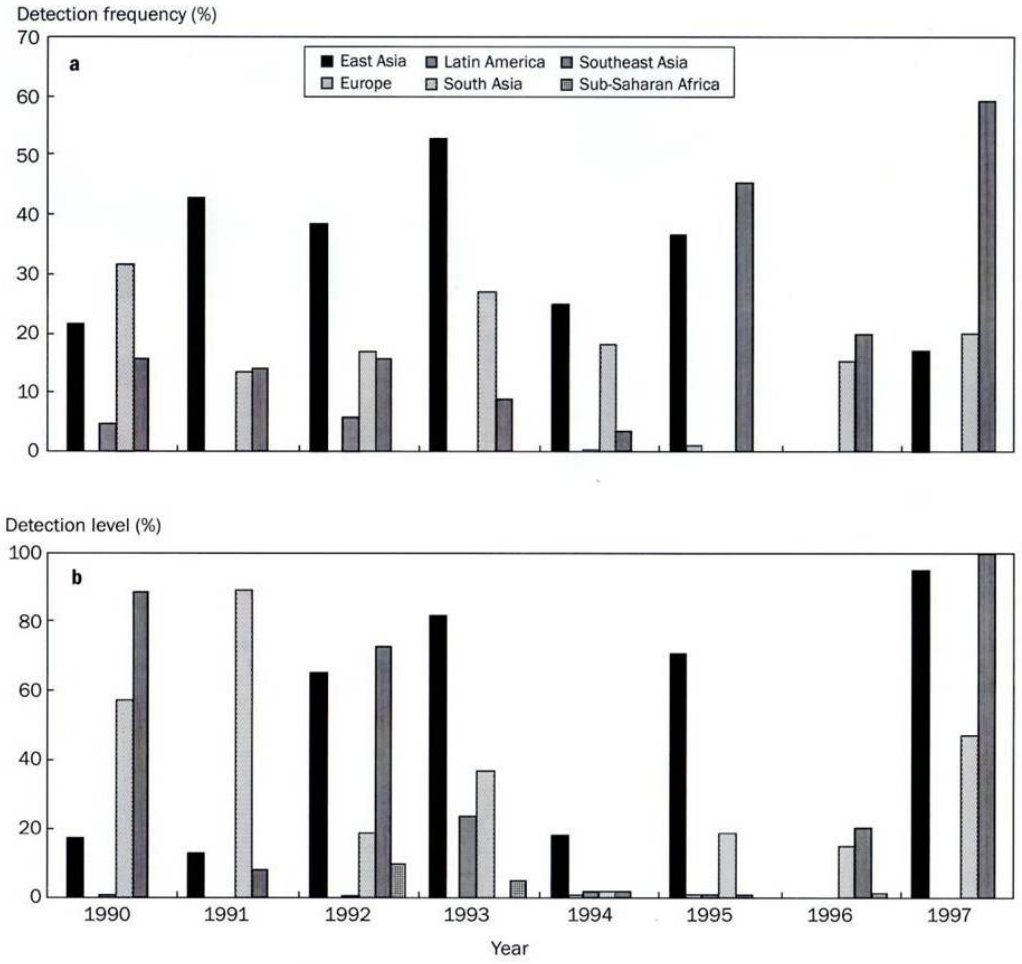
KSm (also known as caryopsis smut, black smut, or grain smut), is a "true" smut (in contrast to false smut) caused by the fungal pathogen *Tilletia horrida*, which affects both crop yield and grain quality. The disease was first reported by Takahashi in Japan in 1886 (Ou 1985). By the early 1900s, KSm had been detected in most rice-growing countries in Asia, including China, India, Indonesia, the Philippines, and Thailand (Ou 1985). It was first reported in 1898 in the United States (Anderson 1899) in rice plants grown in Georgetown, South Carolina. It was speculated that kernel smut was probably introduced in the USA through rice seeds imported from Japan (Anderson 1899). Today, KSm occurs throughout Asia, Australia, Europe, South America, and Africa.

KSm is persistently found in rice seed but is minor in importance throughout rice-growing countries. It occurs more frequently in indica than in japonica rice and is intermediate in medium-grain rice (Ou 1985, Biswas 2003). It is unclear whether the level of occurrence on these different rice types is related to the susceptibility of the rice varieties or to morphological characteristics. KSm has been consistently observed and detected on hybrid rice (Mew et al 1988). Its importance is not so much on the loss in rice yield but on the seedborne nature of its causal pathogen. Although only a few grains in each panicle may be affected, its presence in rice seed is a general concern in the production of healthy rice seed.

It affects milling quality more than grain yield. Rice containing more than 3% bunted kernels is graded as "smutty" in the USA. (USDA 2005), and is penalized at milling (Gravois and Bernhardt 2000) because of the staining effect of kernel smut on milled rice and the fetid smell due to trimethylamine (Biswas 2003). In recent years, high disease incidence in fields of hybrid rice seed production has been reported in China with incidence as high as 100%. During a regular year, kernel smut incidence can still be 25% (Tian and Li 1994). According to Biswas (2003), 35% of the seeds produced in 1980 by the National Seed Corporation of India had a heavy incidence of kernel smut in seed multiplication plots in Orissa. Consequently, seeds produced in this area were rejected for distribution in the country.

In seed health testing conducted by the Seed Health Unit at IRRI, hybrid rice seed tends to show a higher frequency of seed infection than on seeds of inbred rice (Mew and Gonzales 2002; **KSm Figure 1**). Sometimes, 100% of hybrid rice seeds can be infected, perhaps due to floret structure, as discussed later in this section.

1.1. Symptoms



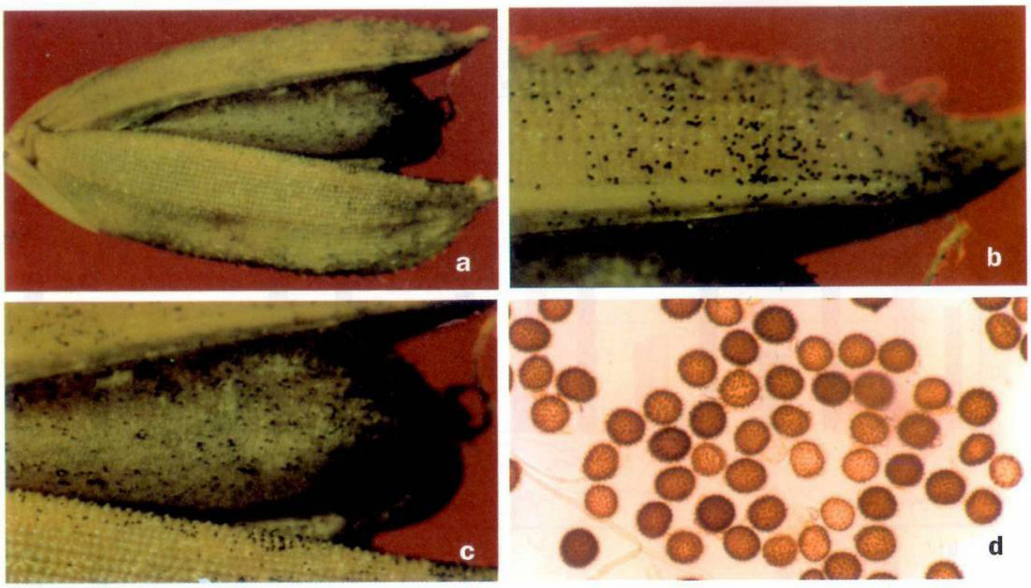
KSm Fig. 1. Detection frequency (a) and level (b) of *T. horrida* from imported, untreated seed at the Seed Health Unit of the International Rice Research Institute, 1990-97. Source: Mew and Gonzales (2002).

KSm is often found in the field at or shortly before maturity of the rice crop. Only a few grains are infected, with various degrees of infection. Normally, infected grains show minute black pustules or streaks bursting through the glumes. In severe cases, the entire grain is replaced by a powdery black mass. Infected grains may be detected by their dull or slightly grayish color externally before the glumes burst open with the black mass of spores.

1.2. Causal organism

The causal fungus was originally named *Tilletia horrida* by Takahashi (1886) in Japan. Anderson (1899) in the USA wrongly renamed it *Tilletia corona* in 1899 (Ou 1985). Padwick and Khan (1944) placed the fungus in the genus *Neovossia* and renamed it *N. horrida* (Tak.) Padwick & Khan. Later, Fisher (1953) retained the genus *Tilletia* and changed the name back to *T. horrida* Tak. Tullis and Johnson (1952) retained the genus *Tilletia* and designated the fungus *T. barclayana* (Bref.) Sacc. & Syd. This name has been generally used but many scientists thought the name *T. horrida* should be retained (Whitney 1989, Carris et al 2006). This name is used here. No matter what the name, the fungus belongs to *Basidiomycetes,* class *Teliomycetes*, order *Ustillaginales*, and family *Tilletiaceae*.

The teliospores are 15-17 µm in size. They are pulverulent, black in mass, globose to irregularly rounded, sometimes with a short hyaline appendage, and 18-23 µm in diameter. The spore surface is covered with conspicuous spines, which are hyaline to slightly colored, pointed at the apex, irregular polygonal at the base, and 2.5 to 4.0 µm in length. The teliospores germinate to form a promycelium, also known as the basidium, which emerges through the ruptured wall of the teliospore and either immediately produces basidiospores or elongates to over 500 µm in length. Elongating basidia form retraction septa, confining the cytoplasm to the apical region (Carris et al 2006). A terminal whorl of 10 to 150 primary basidiospores, also known as primary sporidia, is formed. The primary sporidia do not fuse. From the primary sporidia, two types of secondary sporidia are formed, an allantoid type (15-22 x 5 µm) and a filiform type (35-41 x 2 µm). The allantoid sporidia are the primary infective unit of *T. horrida*.



:Sm Fig. 2. Habit character of *T. horrida* on (a) whole seed (16X), (b) palea (40X), and (c) coty-edon and portion of lemma (40X). In (d), the teliospores are shown in a photomicrograph (40X). Source: Mew and Gonzales (2002).

1.3. Host range

The host range of *T. horrida*, besides rice, includes wild rice *O. barthii, Pennisetum alopecuroides, P. glaucam, P. sarosum,* and *P. orientale* var. *triflorum.* The pathogen is also reported to occur on several genera of plants, such as *Brachiaria, Digitaria, Echinochloa*, and *Panicum*

(Ou 1985, Biswas 2003). However, most of the reported hosts other than rice have not been fully established in careful experiments, such as those based on isolation of the fungus from plants of natural infection and with a completed cross-inoculation test. Despite these shortcomings, the information is useful for students to carry out field observations, and as a guide to make surveys of a particular fungal pathogen in related rice ecosystems.

1.4. Disease cycle

The components of the disease cycle are fragmented. There is little research on the disease and its pathogen.

1.4.1. Primary inocula. The teliospores are the resting spore structure for overwintering or between crop seasons. The primary inoculum is the primary sporidia derived from the germination of the teliospores. The teliospores of *T. horrida* float to the surface of irrigation water in rice fields and germinate after rice is planted (Whitney and Frederiksen 1975). Sporidia and hyphae of *T. horrida* may be like those of *T. indica* and could either become established in the crop canopy (Bains and Dhaliwal 1990) or form a dense mat on the surface of the rice paddy water (Templeton et al 1960, Webster 2005). The primary sporidia are formed on tips of promycelium and may also bud on plant surfaces of rice or weeds (Huang et al 2000). Most of the secondary sporidia are produced by epiphytic budding. They are of allantoid types and considered the important source of inoculum in the field.

The significant linkage of the infection process is to ensure that ample teliospores are germinating at the heading stage of the host plant, the rice crop, so that infection is achieved. If this does not happen, the infection cannot take place. It is mysterious in nature as how this is timed so accurately between the pathogen and the host to continue the infection process.

There appears to be a "safety mechanism" to make sure that the teliospores germinate at the right time, i.e., when the host plant is at the most susceptible stage (Carris et al 2006). The actual penetration process of *T. horrida* into the host tissue is not known but is postulated as follows: "the sporidia land on the feathery stigma and penetrate through the style to the chalazas end of the ovary" (proposed by Sing and Pavgi 1973, as cited by Carris et al 2006). The fungus causes local infection of the florets by sporidia but not the leaves of the seedlings as postulated by Chowdhury (1946) and confirmed by Templeton (1961) who showed that the sporidia infected the opening flowers and later that the developing grains became smutted.

There is no information on whether the teliospores of *T. horrida* can spread through air and can be carried by air currents for long-distance dispersal. However, it is known that kernel smut fungus can spread via infected seed, which is perhaps the most effective mechanism by which *T. horrida* has "traveled" around the world throughout most rice-growing regions. The teliospores are released from smutted grains before or during harvest, thus, contaminating healthy kernels, crop debris, and soil. Seedborne teliospores are the most important source of overwintering or off-season resting body, although there were reports of soilborne inoculum of teliospores (Biswas 2003).

The survival of *T. horrida* as soilborne inoculum remains uncertain based on the available information. The teliospores may survive in the field until the next planting season and then follow an infection process on rice grains as described earlier. The fungus belongs to the so-called "nonsystemic infecting bunt fungi" (Carris et al 2006), where infection in early rice crop growth would unlikely result in the kernel. Teliospores are dormant on rice seeds for months and sometimes for more than a year, pointing to the fact that *T. horrida* is a monocyclic pathogen and is unlikely to produce a secondary inoculum for secondary infection.

1.5. Control measures

Because *T. horrida* is seedborne and seed-transmitted, the use of healthy and clean seed for planting is an important means for preventing the spread of kernel smut to other regions or new areas. Seed treatment is also an effective means to control the disease in the field. Most rice-importing countries require certification that seeds are free of KSm fungus. Seed selection using 10% salt water that can separate infected seed from healthy seed is an old method to minimize most seed-borne or grain-infected diseases. It is still the most effective measure to control seedborne diseases, including KSm. Seed selection is feasible for small landholding farmers but, for farmers with larger holdings, it may have to be integrated into a routine farm operation and management procedure.

1.5.1. Varietal resistance. There are no convincing data from research that can demonstrate varietal resistance to *T. horrida*. Nevertheless, there seems to be a relationship between the floral structure and the disease incidence which might as well be an escaping mechanism or disease avoidance. Long-grain rice varieties are more susceptible than the short-grain types, the medium-grain types being intermediary in reaction (Biswas 2003 as cited by Carris et al 2006).

Similarly, a difference between inbred and hybrid rice varieties is evident through seed health testing (Mew and Gonzales 2002) and also through careful examination of the flowering structure of the inbred and hybrid rice (Mew et al 1988). The florets of cytoplasmic-male sterile (CMS) lines remain open and the stigma remains receptive for a longer period during the anthesis stage compared with inbred lines. Moreover, CMS lines have a wider opening angle of the lemma, and their anthesis duration is longer; thus the exposed stigma become more vulnerable to infection. Consequently, the pathogen, which infects only the floral parts of the host, causes increased KSm disease (Pan et al 1995, Tian and Li 1994). However, there is no evidence to suggest that such susceptibility of CMS lines is associated with the cytoplasm. Breeding of a hybrid for "avoidance" of KSm that focuses on the floral structure of the parents may be feasible.

References

Anderson, A. P. 1899. A new *Tilletia* parasitic on *Oryza sativa*. L. Bot. Gaz. 27:467-567.

Bains, S. S., Dhaliwal, H. S. 1990. Production of *Neovossia indica* sporidia on host and non host plants. Plant Soil 126:85-89.

Biswas, A. 2003. Kernel smut disease of rice: current status and future challenges. Environ. Ecol. 21:336-351.

Carris, L. M., Castlebury, L. A,. Goates, B. J. 2006. Nonsystemic bunt fungi – *Tilletia indica* and *T. borrida*: A review of history, systematics, and biology. Annu. Rev. Phytopathol. 44:113-133.

Chowdhury, S. 1946. Mode of transmission of the bunt of rice. Cur. Sci. 15:111.

Fisher, G. W. 1953. Manual of the North American smut fungi. New York; Ronald Press. 343 p.

Gravois, K. A., Bernhardt, J. L. 2000. Heritability and genotype x environment interactions for discolored rice kernels. Crop Sci. 40:314-318.

Huang, F., Pan, X., Cheng, K., Wang, Y., Zhang C., Liu, X. 2000. Studies of the epiphytic budding of secondary sporidia of Nevossia horrida causing rice kernek smut on the plant surfaces. P1-74. In G. H. Zhou and H. f. Li (ed.) Proc. 1st Asian Conf. on Plant Path., Beijing, China.

Mew, T. W., Gonzales, P. 2002. A handbook of rice seedborne fungi. Los Baños (Philippines): International Rice Research Institute, and Enfield, N. H. (USA): Science Publishers, Inc. 83 p.

Mew, T. W., Wang, F. M., Wu, J. T., Lin, K. R., Khush, G. S. 1988. Disease and insect resistance in hybrid rice. In: Hybrid rice. Los Baños (Philippines): International Rice Research Institute. p 189-200.

Ou, S. H. 1985. Rice diseases. 2nd ed. Kew, Surrey (England): Commonwealth Mycological Institute, CABI. 380 p.

Padwick, G. W., Khan, A. 1944. Notes on Indian fungi. II. Mycological papers No. 10. 17 p.

Pan, X., Cheng, K., Huang, F., Wang, Y., Zhang, C., Lin, K. 1995. Studies on source of inoculum of kernel smut and its occurrence on hybrid rice. Acta Phytopath. Sin. 22:243-250.

Singh, R. A., Pavgi, M. S. 1973. Artificial culture and chlamydospore development of *Neovossia horrida*. Nova Hedwigia 24:487-491.

Takahashi, Y. 1886. On *Ustilago virens* Cooke and a new species of *Tilletia* parasitic on rice plant. Bot. Mag. Tokyo 10:16-20.

Templeton, G. E., Johnston, T. H., and Henry, S. E. 1960. Kernel smut of rice. Arkansas Farm Res. 9(6):10.

Templeton, G. E. 1961. Local infection of rice florets by the rice kernel smut organism, *Tilletia horrida*. Phytopathology 51:130-131.

Tian, J. R., Li, X. K. 1994. Managing diseases and insect pests of hybrid rice in China. In: S. S. Virmani (ed.), Hybrid rice technology – new developments and future prospects. Los Baños (Philippines): International Rice Research Institute. p 115-122.

Tullis, E. C., Johnson, A. G. 1952. Synonymy of *Tilletia horrida and Neovossia barclayana. Mycologia* 44:773-788.

USDA (United States Department of Agriculture). 2005. United States Standards for Rice. Washington, DC: USDA.

Webster, R. K. 2005. Being at the rice place, at the right time, for the right reason – plant pathology. Annu. Rev. Phytopathol. 43:1-24.

Whitney, N. G., Frederiksen, R. A. 1975. Kernel smut of rice. Tex. Agric. Exp. Stn. Misc. Publ. 1231.

Whitney, N. G. 1989. Taxonomy of the fungus causing kernel smut of rice. Mycologia 81:468-471.

2. Udbatta Disease (UbD)

"Udbatta" was first described by Sydow (1914) in India. In Karnataka, UbD is common in farmers' fields where in the local Kannada language, "udbatta" means "incense sticks," so called because the panicles of infected plants are compacted taking a cylindrical rod shape looking like udbatta or incense sticks. Interestingly, the name of the disease in Chinese is also "incense sticks" (Tai and Siang 1948). It is also known as "false ergot" in other countries. So far, its distribution is limited to a few countries and it has not been reported to occur widely in most rice-growing regions. UbD seems to be more closely associated with rainfed rice in low-production environments (Govindu 1969). Even in these areas, it occurs sporadically. Thus, there is little information about the disease's importance, ecology, and epidemiology.

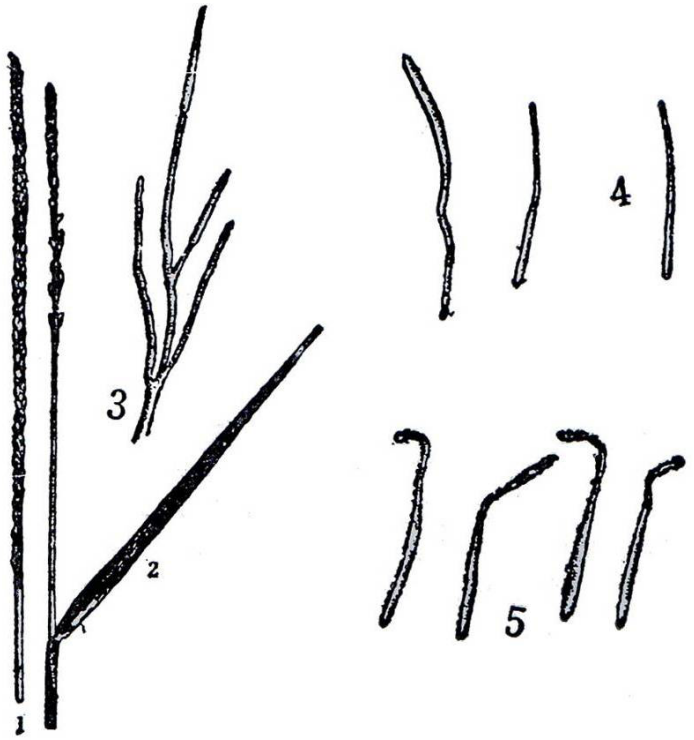
2.1. Symptoms

UbD usually begins before panicle emergence, while the infected panicle is still embedded in the sheath and matted together by mycelium of the fungus. The symptoms, therefore, do not become apparent until heading when infected plants produce erect spikes instead of normal inflorescences (Mohanty 1964). The panicles emerge as single cylindrical rods covered with white mycelium (**UbD Figure 1**). The infected panicles eventually become dark and mummified, sclerotium-like. No grain develops on infected panicles. The flag leaf and the sheath of infected tillers are sometimes slightly distorted. The flag leaf and upper leaves may appear silvery. Infected plants may also be stunted (Mohanty 1976).

2.2. Causal organism

The causal fungus of UbD belonging to Ascomycota is *Balansia oryzae-sativae* Hashioka (anamorph *Ephelis oryzae* Syd.) and is related to ergot fungi.

Sydow (1914) described the causal organism in its anamorphic state as *Ephelis*



UbD Fig. 1. Panicles emerge as single cylindrical rods covered with white mycelium: (1-2) infected panicles and leaf, (3) conidiophore, (4) conidia, (5) germinating conidia. Source: Tai and Siang (1948).

oryzae Sydow and the description detailed by Ou (1985) is reproduced here. The fungus produces hard, greyish-black stroma, which occupy the whole length and surrounding the inflorescence. Black and slightly convex pycnedia are immersed in the stroma and finally more or less exposed. Spores are needle-shaped, hyaline, 20-35 x 1 µm. Tai and Siang (1948) gave another description in which sporochia are black, cupulate or convex, roundish, measuring 1-1.5 mm in diameter, conidiophores branched, hyaline, 57-85 x 0.8-1.4 µm, conidia hyaline, acicular, aseptate, straight or curved 12-22 x 1.2-1.5 µ m.

Narasimhan and Thirumalachar (1943) obtained immature teleomorphic stromata in sand culture from diseased rice grains and referred to the fungus as *Balansia oryzae* without a formal description of the sexual stage. Deighton (1956) found the Balansia (teleomorph) state on rice and several wild grasses in Sierra Leone. Hashioka (1971) described the teleomorph from herbarium specimens under the name *Balansia oryzae-sativae* Hashioka and this name is currently accepted for usage (Booth 1979). The descriptions of Hashioka (1971) follow.

Stromata on mummified spikelets, hemispherical, capitate, gregarious, 0.67-1.2 µm in diameter with a black, coarsely papillate surface, yellowish-brown to white interior, arising from a mummified spikelet upon which the conidiamata developed. Ascomata with perithecia embedded in periphery stromatal head, perithecia rounded, ovoid to pyriform,

125-200 x 85-100 µm diameter Asci cylindrical, 92-120 x 6 µm with a rounded, thickened apex, an attenuated base, eight-spored. Ascospores filiform, nonseptate, straight or curved, 12-27 x ca. 1 µm in width.

Acervuli on surface of mummified inflorescence emerging from leaf sheath; when wet, appearing gelatinous, cupulate or convex fructification, 1-1.5 mm diameter bearing a palisade of conidiophores. Conidiophores terminating in narrow conidiogenous cells that proliferate percurrently to form a mass of filiform to acicular, hyaline conidia,13-35 x 1-2 µm.

2.3. Host range

The fungus attacks several grass weeds, but it is unknown whether these hosts also serve as a source of inoculum.

2.4. Disease cycle

The disease cycle of UbD has not been characterized. However, seed inoculation with the spores of the pathogen produces the typical disease symptoms (Mohanty 1977). Planting seeds harvested from diseased fields appears to produce more infected panicles (Ou 1985), suggesting the systemic nature of the disease (Tai and Siang 1948, Booth 1979). The production of conidia in the diseases heads and leaf blades (Mohanty 1964) led to the belief that the pathogen is airborne and can tolerate dryness. The fungus is internally seedborne and seed inoculation expressed the disease in the inflorescence (Mohanty 1964). Besides, all the tillers are usually infected and the fungus has been shown to persist in the base of rice plants and infect the ratoon crop indicating the systemic nature of the disease (Mohanty 1964, 1979). The asymptomatic development of the fungus from seed infection to symptom expression in the ear heads lends support to the biotrophic nature of parasitism.Thus, infected seed is a major source of primary inoculum for initial infection.

2.5. Control measures

No control measure has been suggested or needed. Assuming the pathogen is seedborne, the best option is to avoid using seed stock originating from diseased fields for planting.

References

Booth, C. 1979. *Balansia oryzae-sativa* even. CMI Descriptions of Pathogenic Fungi and Bacteria No. 640, 2 pp.

Deighton, F. C. 1956. Diseases of cultivated and other economic plants in Serra Leone. Government of Sierra Leone. 76 p.

Govindu, C.H. 1969. Occurrence of *Ephelis* on rice variety IR8 and cotton grass in India. Plant Dis. Rep. 53:360.

Hashioka, Y, 1971. Rice diseases in the world - VIII. Diseases due to Hypocreales, Ascomycetes (fungal diseases - 5). Riso 20:235-258.

Mohanty, N. N. 1964. Studies on "Udbatta" disease of rice. Indian Phytopathol. 17:308-316.

Mohanty, N. N. 1976. Influence of some meteorological factors on the development of Udbatta disease of rice. Indian J Agric Sci.46:399-403.

Mohanty, N. N. 1977. Mode of infection of "Ubdatta" disease of rice. Riso 26:243-247.

Mohanty, N. N. 1979. Seedborne nature and systemic character of infection of Udbatta disease of rice. Riso, 28:133-139.

Narasimhan, M. J., Thirumlachar, M. J. 1943. Preliminary note on the perfect stage of *Ephelis oryzae* Syd. (*Balansia oryzae* (Syd.) comb. nov.) Curr. Sci. 12:276.

Ou, S. H. 1985. Rice diseases. 2nd ed. Kew, Surrey (England): Commonwealth Mycological Institute, CABI. 380 p.

Sydow, H. 1914. Beitrage zur Kenntnis der Pilzflora des Sudlichen Ostindiens II. Ann. Mycol. 12:484-490.

Tai, F. L., Siang, W. N. 1948. "I-chu-hsing" disease of rice caused by *Ephelis oryzae* Sydow in Yunnan. Acta Agric 1:125-131.

3. Glume blight (GlB)

GlB was first found in the USA and Japan but it is now widely reported in many rice-growing countries in Asia, the Americas, and Africa (Ou 1985). It is a minor disease. If infection occurs early and under favorable conditions, grains may not form. Late infection may result in partly filled grains or discolored and brittle grains. Thus, like other grain diseases except false smut, in combination with other grain infections, GlB may cause some losses in rice yield.

3.1. Symptoms

Symptoms are variable and usually occur on the glumes 2 to 3 weeks following the emergence of the panicle, that is, from the time of heading to maturity. Initially, the lesions are small (pinhead size) and brown in color, which gradually enlarge, becoming whitish with bleached centers bearing numerous small black dots of pycnidia. The lesions usually have a distinct brown margin. When panicle emergence coincides with rainy days, the disease appears as reddish brown blotches on the glumes. Infection, depending on the stage of panicle emergence and grain filling and maturation, may result in grain sterility or in grain weight reduction (Ou 1985, Webster and Gunnell 1992).

3.2. Causal organism

A pokeweed isolate of *P. sorghina* has been shown to produce six nonspecific epoxydon group of toxins in culture capable of causing necrosis on host tissues (Rabie et al 1975, Venkatasubbaiah 1992). There are indications that some of these toxins affect human and animal health and as a seed colonizer in rice, it raises concern. However, as the pathogen has a very wide host range, direct evidence for the production of these toxins in rice needs careful further examination.

GlB is caused by *Phoma sorghina* (Sacc.) Boerema, Dorenbosch & Van Kesteren, which has been reported in rice under different names, including *Phoma glumarum* Ellis & Tracy, *Phoma insidiosa* Tassi, *Phyllosticta glumarum* (Ellis & Tracy) Miyake, *Phyllosticta glumicola* (Speg.) Hara, *Phyllosticta oryzina* Padwick, and *Phyllosticta sorghina* Sacc. (Ou 1985).

Pycnidia of the fungus are dark brown to black, irregularly globose with a short neck, measuring 80-200 µm in diameter. When moistened, conidia are extruded from the pycnidia in tendrils, germinate in free moisture, and invade the glumes. Conidia are hyaline, biguttulate or without guttules, ovoid to ellipsoid, and mostly measured 4-7 x 2-3.5 µm. Single chlamydospores and dictyochlamydospores are formed. The teleomorph of *P. sorghina* is supposed to be either *Leptosphaeria sacchari* Breda de Haan, (according to Index Fungorum) or *Didymella holci* (Tehon) Arx (according to MycoBank), both leaf pathogens of grasses belonging to Pleosporales (Von Arx 1987).

3.3. Host range

P. sorghima, with seed- and soilborne natures, is a facultative phytopathogen and a cosmopolitan fungus occurring in a variety of plants (Aveskamp et al 2008, Boerema et al 2004). The fungus exhibits variable aggressiveness when inoculated on the same rice cultivar (De Souza 1988).

3.4. Yield loss and host resistance

The disease affects the grain filling. Panicle weight losses in three different fields (cv. IAC25) during an epidemic in Brazil (1979-80) were 41, 25, and 45%, resulting in head yield losses from 0-14% showing its potential to cause losses in grain weight and quality (Prabhu and Bedendo 1988). Of a limited number of Brazilian cultivars evaluated under artificial inoculation, Limeria and Iguape Redondo have been found to be highly resistant varieties to GlB (Prabhu and Bedendo 1988).

3.5. Control measures

The fungus is seedborne and commonly detected in seed. As it likely causes damage in the rainy season, the best control option is seed health management and using clean seed for planting.

References

Aveskamp, M. M., De Gruyter J., Crous, P. W. 2008. Biology and recent developments in the systematics of Phoma, a complex genus of major quarantine significance. Fungal Diversity 31:1–18.

Boerema, G. H., De Gruyter, J., Noordeloos, M.E., Hamers, M. E. C. 2004. *Phoma* Identification Manual: Differentiation of Specific and Infra-Specific Taxa in Culture. CABI Publishing, Wallingford (UK).

De Souza, N., Zambolim, L., Thiebaut! J. T. L. 1988. Variabilidade de isolados de *Phoma sorghina* em arroz. Pesq.Agropec.Bras. 23:1139-1141.

Ou, S. H. 1985. Rice diseases. 2nd ed. Kew, Surrey (England): Commonwealth Mycological Institute, CABI. 380 p.

Prabhu, A. S., Bedendo, I. P. 1988. Flume blight of rice in Brazil: Etiology, varietal reaction and loss estimates. Trop. Pest Manag. 34:85-88.

Rabie, C. J., Van Rensburg, S. J., Van Der Watt, J. J., Lubben, A. 1975. Onyalai – the possible involvement of a mycotoxin produced by *Phoma sorghina* in the aetiology. South Afr. Med. J. 57:1647-1650.

Venkatasubbaiah, P., Van Dyke, C., Chilton, W. 1992. Phytotoxic metabolites of *Phoma sorghina*, a new foliar pathogen of pokeweed. Mycologia 84:715-723.

Von Arx, J. A. 1987. Plant pathogenic fungi. Nova Hedwigia Beihefte 87:1-288.

Webster, R. K.; Gunnell, P. S. 1992. Compendium of Rice Diseases. APS Press, St. Paul, Minnesota.

4. Grain discoloration (GrD)

Today, grain discoloration occurs in most rice-producing countries. Grain discoloration syndrome has a complex etiology involving microbial pathogens, viruses, nematodes, insect infestations, and chemical injuries (Pizzatti and Cortesi 2008). Different names are given and various causal agents have been reported; yet, there is little information on the causal organisms. In tropical Asia, GrD is often known to farmers as "dirty panicle" and oc-

curs more frequently in the rainy season than in dry season. Seed discoloration, in addition to reducing market value, affects seed germinability, seedling emergence, and plant vigor besides causing human and animal health concerns when mycotoxins are involved.

The situation on grain discoloration seems to be worsening since farmers are now growing more photoperiod-insensitive rice varieties, which are not bound by seasons. Changes in plant type and in culture methods have not come without a cost. However, the overall benefit to farmers is greater than with the absence of these changes. Scientists need to anticipate the cost that farmers have to pay for these changes. Increasing food production by planting high-yielding, photoperiod-insensitive rice varieties often enables the rice plant to set flowers during the rainy season, especially in the later crop growth stages. At heading stage, the rice plant is most vulnerable to attack by grain-feeding insects and by many weak fungal and bacterial rice pathogens, resulting in grain discoloration. The cause of GrD is not limited to any individual pathogen but to a combination of various organisms, including the pathogens causing brown spot, rice scab, red blotch, black rot, and stackburn and an array of other opportunistic bacterial pathogens. Insects feeding on rice grains often predispose the grains to invasion by other weak pathogens.

Other pathogens have been detected more often than before, even though the distinctive symptom caused by that organism may differ somewhat from the textbook. Kernel smut caused by *Tilletia horrida* best exemplifies this. Although detection of kernel smut is higher in hybrid rice than in normal rice seed, it is often detected together with other fungi associated with grain discoloration. Its occurrence in hybrid rice is due to the structure of the florets in the inflorescence where the stigma is longer than the stamen and the considerable longer opening time of the inflorescence than in inbred rice (Pan et al 1995, Tian and Li 1994). This morphological change in the rice plant favors seed production but may cause more infection by microorganisms that otherwise do not succeed in landing on the grains or floral parts to cause infection.

4.1. Symptoms

Discoloration may appear externally or internally or both on the kernels. On glumes, the discoloration varies depending on the organisms involved and the degree of infection. In severe cases, discoloration may appear on all the kernels of the panicles. In less severe incidences, discoloration occurs on only a few kernels, mostly in the mid-portion of the panicles. Some kernels are distinctly pale and grayish in the centers, while others have blackish blotches. Still, there are distinct black dots on normal and bleached areas of the glumes. Thus, in the literature, certain lesions that appear on the grains have been identified based on a specific pathogen (**GrD Table 1**). In reality, many microorganisms may cause similar types of lesions or grain discoloration. When the leaf sheath is diseased, it usually shows discoloration, as can be seen with "sheath rot" incidence, which Cottyn et al (1996a,b) categorized as the "sheath rot complex." Cottyn et al (1996a,b) conducted comprehensive isolations and analyses of the bacteria involved in the complex. The relation of these bacteria to the occurrence of various fungi is obscure; some are antagonistic while others are synergistic.

4.2. Causal organism

Many fungi and bacteria have been isolated from discolored seed as well as from flag leaf sheath tissues. Based on the frequency of detection, amount of seed infection, and disease occurrence in the field, seedborne fungal pathogens can be categorized into four groups (**GrD Table 2**). The fungi in group 1 with high detection frequency often show low disease

GrD Table 1. Some fungi known to cause discoloration in rice seed. Source: Mew et al (1989).

Name of discoloration/	
Brown spot	Bipolaris oryzae
	Helminthosporium sativum
Grain spot	Nigrospora spp.
Kernel smut	Tilletia horridea
Black kernel	Curvularia spp.
Pink; stackburn	Alternaria padwickii
Brown and black	Sarocladium oryzae Alternaria tenuis Alternaria padwickii Fusarium solani Phoma spp.
Discolored spots	Leptocorisa oratorius Epicoccum spp.

Note: Grain discoloration exclusively refers to the grain harvested in the field, not in storage.

potential. In contrast, fungi in group 4 with low detection frequency possess high disease potential. The high frequency of detection and the level of infection appear to bear no relationship to the occurrence of GrD in the field. Again, grain discoloration and the many pathogens associated with it must be assessed in the context of the rice production ecosystem, cultural types, and rice varieties with their actual management. Information is lacking on which to base a logical assessment between organisms isolated from seed and the amount and types of discoloration observed in the field. Fungi may be categorized into two broad groups, namely, field fungi and storage fungi. Storage fungi may cause discoloration of rice seed in storage but it is unknown whether they are also involved in grain discoloration of a field crop. Field fungi may be weak or opportunistic pathogens.

Table GrD 3 provides a list of fungi that are frequently detected in discolored rice seed. Clearly, they cause minimal disease problems in the field.

The field fungi consist of *Sarocladium oryzae* and *Bipolaris oryzae*, intermediate in detection frequency and known to cause grain discoloration. Other fungi such as *Curvularia* spp., *Fusarium* spp., and *Dreschleria oryzae* are also high in detection frequency and known to be associated with GrD. Italian researchers (Cortesi et al 2008) have attributed the GrD and panicle sterility to bacterial etiology, particularly of seedborne bacteria. In an extensive study, Zeigler and Alvarez (1990) isolated several pathogenic (95 strains) and nonpathogenic (21 strains) pseudomonads from diseased (grain or sheath rot, 'dirty panicle', and 'manchado de grano') rice grains from 22 different countries. Of these, with symptoms resembling those caused by sheath rot and brown spot, four different *Pseudomonas* spp. (*P. fuscovaginae, P. avenue, P. syringae* pv. *syringae*, and *P. glumae*) have been identified.

4. Disease cycle

Because of syndrome complexity and the variable causal agents, individual symptoms are traditionally related to specific causal agents. However, with this approach, potential interactions between various organisms and the host tissues in certain environments may be neglected. Added to this complexity is the colonization of some weak pathogens on injury caused by insect pests on the grains, making it impossible to pinpoint the disease

GrD Table 2. Frequency of detection of selected fungal pathogens associated with untreated rice seedlots (1991-95).

	Detection frequency (%)[ab]						
Pathogen	1991	1992	1993	1994	1995	Mean	Groups
Curvularia spp.	91.7 (8.3)	89.9 (17.5)	97.5 (15.0)	90.0 (7.5)	92.3 (10.8)	92.3 (11.8)	I
Alternaria padwickii	93.9 (22.9)	93.1 (10.1)	96.1 (14.8)	85.9 (9.7)	89.6 (19.3)	91.7 (15.4)	I
Sarocladium oryzae	27.8 (2.0)	50.9 (2.7)	57.4 (4.9)	15.2 (2.0)	28.9 (4.0)	36.0 (3.1)	II
Fusarium moniliforme	39.6 (1.2)	38.4 (3.5)	14.0 (2.0)	28.8 (1.4)	32.8 (1.7)	30.7 (2.0)	II
Bipolaris oryzae	77.4 (5.8)	55.4 (7.2)	75.4 (3.5)	74.2 (13.9)	49.3 (6.4)	66.3 (7.4)	I
Tilletia barclayana	14.4 (42.0)	17.0 (14.1)	12.0 (10.4)	4.6 (17.8)	11.0 (13.0)	11.8 (19.5)	III
Magnaporthe oryzae	3.5 (2.8)	10.7 (2.1)	2.0 (1.0)	1.6 (6.2)	11.9 (3.8)	5.9 (3.2)	IV

Note: Other seedborne fungi following into the different groups are: Microdochium ourzae (or Gerlachia oryzae), Cercopsore oryzae, and Pinatubo oryzae
in Group III.

[a] Based on a total of 26,755 seedlots.
[b] Figures in parentheses denote infection frequency.

GrD Table 3. Incidence of different fungal pathogens from 1,832 seedlots grown at IRRI in 1987 without seed treatment. The seed samples for seed health processes were done at Seed Health Unit, 1987.

Fungal pathogens	Affected seedlots (%)	Detected range (%)	Mean value (%)
Bipolarisoryzae	5	1.0 - 16.0	1.4
Fusarium moniliforme	3.3	1.0 - 5.0	1.2
Gerlachia oryzae	10.1	1.0 - 17.0	2.9
Sarocladium oryzae	21.9	1.0 - 48.0	4.9
Magnarporthe oryzae	0.1	1.0 - 2.0	1.5
Alternaria padwickii	30.6	1.0 - 82.0	17.5
Curvularia spp.	27.8	1.0 - 91.0	7.9
Tilletia horridea	0.4	1.0 - 51.0	15.7

cycle of GrD per se. If a disease cycle is involved in such a large number of microorganisms, it may not be feasible to pinpoint individual types of lesions or grain discoloration associated with certain microorganisms. Thus, the disease cycle of GrD should be treated collectively. All these microorganisms associated with the disease are seedborne. Rice farmers use seed saved from their harvest for the next crop planting; thus, the intensity of grain discoloration continues to be magnified.

Except for rice viruses and most of the nematodes save one, among the pathogens attacking rice, a majority of the rice fungal and bacterial pathogens are seedborne. Although seed as a source of primary inoculum may vary drastically on the pathogens it carries, the crop may be exposed to colonization by a large number of microorganisms during the reproductive stage of rice in the most favorable environment (the rainy season). This is a consequence of using modern rice varieties most of which lack photoperiod sensitivity.

4.4. Control measures

The best option in managing grain discoloration is to encourage farmers to use clean seed for planting. In the past, seed health has been considered only in the context of plant quarantine regulations. The importance of some seedborne pathogens cannot and should not be ignored for plant quarantine purposes, germplasm exchange, and seed trading. Several seedborne pathogens have quarantine importance in seed movement and in exchange or rice trading. The pathogen infects seed, which is the most important vehicle for movement and dissemination of pathogens, such as *Tilletia horrida* (causal organism of kernel smut) and *Aphelenchoides besseyi* (causal nematode of white-tip). Both diseases can be properly managed and minimized in the field by using healthy or clean seed for planting.

References

Cortesi, P., Bartoli, F., C. Pizzatti, C., Bertocchi, D., Schaad, N. W. 2008. Panicle sterility and grain discolouration: New and emerging bacterial diseases of rice in Italy. In: M'Barek Fatmi, et al, editors. *Pseudomonas syringae* Pathovars and Related Pathogens – Identification, Epidemiology, and Genomics, Part VI. Springer Netherlands. Doi: 10.1007/978-1-4020-6901-7_40. p 391-411,

Cottyn, B., Van Outryve, M. F., Cerez, M. T., De Cleene, M., Swings, J., Mew, T. W. 1996a. Bacterial diseases of rice. II. Characterization of pathogenic bacteria associated with sheath rot complex and grain discoloration of rice in the Philippines. Plant Dis. 80:429-437.

Cottyn, B., Cerez, M. T., van Outryve, M. F., Barroga, J., Swings, J., Mew, T. W. 1996b. Bacterial diseases of rice. I. Pathogenic bacteria associated with sheath rot complex and grain discoloration of rice in the Philippines. Plant Dis. 80:438-445.

Mew, T. W., Merca, S. D., Gergon, D. M., Huelma, C. C. 1989. Movement of germplasm and sanitation procedure adopted to meet phytosanitary reguirements. In proceedings, "Introduction of germplasm and plant quarantine prodedures". Published by ASEAN Plant Quarantine Centre and Testing Institute, Malaysia. p 163-175.

Pan, X., Cheng, K., Huang, F., Wang, Y., Zhang, C., Lin, K. 1995. Studies on source of inoculum of kernel smut and its occurrence on hybrid rice. Acta Phytopathol. Sin. 22:243-250.

Pizzatti, C., Cortesi, P. 2008. Effect of chemicals, nitrogen, time of sowing and panicle brown spot epidemics on rice grain discoloration in Italy. J. Plant Pathol. 90:197-209.

Tian, J. R., Li, X. K. 1994. Managing diseases and insect pests of hybrid rice in China. In: S. S. Virmani, editor. Hybrid rice technology – new developments and future prospects. Los Baños (Philippines): International Rice Research Institute. p 115-122.

Zeigler, R. S., Alvarez, E. 1990. Characteristics of *Pseudomonas* spp. causing grain discoloration and sheath rot of rice, and associated pseudomonads epiphytes. Plant Dis. 74:917-922.

5. Scab (ScB)

ScB is one of the first recorded diseases of rice and has been reported in most rice-growing countries (Ou 1985). The disease is not a serious constraint to rice production but the pathogen is one of the commonly detected fungi in rice seed. Research reveals that the pathogen belongs to the group of *Fusarium* spp. that produces mycotoxins harmful to animals and humans (Desjardins et al 2000). More recent data indicate that, although the mycotoxin-producing strains are equally effective in infecting wheat and rice, no mycotoxin has been detected in rice (Goswami and Kistler 2005).

The same fungus seems to infect rice, wheat, and maize and was first reported in rice in Italy with a different name (Cattaneo 1879). Infected grains may produce weak seedlings or, in more severe cases, the seed may not germinate.

5.1. Symptoms

The fungus initially causes bleached lesions or discoloration of the glumes. The affected area becomes yellow or salmon to carmine with the production of sporodochia and conidial masses of the fungus. Blue-black perithecia may also be present on infected glumes. Infected grains are light, shrunken, and brittle and may be sterile. The fungus has also been reported to infect the node of rice tillers, causing black rot and resulting in the breaking of the stem (Ou 1985).

5.2. Causal organism

Scab, also known as head blight, is caused by *Fusarium graminearum* Schwabe (teleomorph *Gibberella zeae* (Schweinitz) Petch). Recent work has shown that nine distinct cryptic species exist within the *F. graminearum* complex. These species have been described and named (O'Donnell et al 2004), with *F. asiaticum* (formerly *F. graminearum* lineage 6) being the most common cause of ScB in certain regions of China and other parts of Asia. Hence, ScB of rice is assumed to be caused by *F. asiaticum*.

The perithecia of this fungus are superficial, blue-black, ovoid, and measuring at 140-250 µm in diameter. Asci are clavate, measuring at 60–85 x 8-11 µm, and usually contain eight distichous or obliquely monostichous ascospores. Ascospores are hyaline to light brown, fusoid, somewhat curved, three-septate at maturity, measuring at 19-24 x 3-4 µm. Macroconidia are straight to moderately sickle-shaped, thick-walled, with a distinct foot-shaped basal cell and a short, tapered apical cell, three- to seven-septate, measuring at 35-62 x 2.5-3 µm. Globose chlamydospores may be present. Microconidia are absent. The fungus produces a distinct carmine pigment and white to tan to yellow floccose mycelium on potato-dextrose agar (Lee 1992).

6.3. Disease cycle

The fungus is seedborne but can be found in soil and on crop residues. ScB affects wheat, barley, and other small grains, both in temperate and semitropical areas (Goswami and Kistler 2004). *F. graminearum* (syn: *F. asiaticum*) has also been isolated from cultivated wild rice (*Zizania Palestrina* L.) widely used in rice mixtures and in other foodstuffs (Nyvall et al 1995). Rice is most susceptible to infection at the flowering stage but less susceptible from milky stage to grain development and onward. *Gibberella zeae* also causes scab on wheat.

F. asiaticum isolates from rice and wheat have been shown to be pathogenic to both crops when inoculated on wheat spikes and rice kernels in a glasshouse assay (Gomes et al 2015). Members of *F. graminearum* including *F. asiaticum* are known to produce mycotoxins belonging to the group trichothecens (Miller et al 1991) and these are of concern to animal and human health (Peraica et al 1999).

Although strains of the fungus from different small grains can attack rice, no mycotoxin have been detected from infected rice florets under greenhouse conditions (Goswami and Kistler 2005). In support of this, a recent study (Gomes et al 2015) asserts that although majority of isolates of *F. asiaticum* obtained from rice grains produced the mycotoxin in rice-based substrate *in vitro* with a small proportion of isolates not producing the toxin, the mycotoxin was not produced in rice kernels from inoculated panicles.

5.4. Control measures

Using clean seed for planting may be the best control measure for this disease.

References

Cattaneo, A. 1879. Contributo allo studio dei miceti che nascono sulle painticelle di riso. Archivio del Laboratorio di Botanica Crittogamico, Universita di Pavia 2-3:117-128.

Desjardins, A. E., Manandhar, H. K., Plattner, R. O., Manandhar, G. G., Poling, S. M., and Maragos, C. M. 2000. *Fusarium* species from Nepalese rice and production of mycotoxins and gibberellic acid by selected species. Appl. Environ. Microbiol. 66:1020-1025.

Gomes, L. B., Ward, T. J., Badiale-Furlong, E. and Del Ponte, E. M. 2015. Species composition, toxigenic potential and pathogenicity of *Fusarium graminearum* species complex isolates from southern Brazilian rice. Plant Pathology. doi: 10.1111/ppa.12332.

Goswami, R. S., and Kistler, H. C. 2004. Heading for a disaster: *Fusarium graminearum* on cereal crops. Mol. Plant Pathol. 5:515-525.

Goswami, R. S. and Kistler, H. C. 2005. Pathogenicity and *in planta* mycotoxin accumulation among members of the *Fusarium graminearum* species complex on wheat and rice. Phytopathology 95:1397-1404.

Lee, N. F. 1992. Scab. In: R. K. Webster and P. S. Gunnell, editors. Compendium of Rice Diseases. APS Press, St. Paul, Minnesota.

Miller, J. D., Greenhalgh, R., Wang, Y., and Lu, M. 1991. Trichothecene chemotypes of three *Fusarium* species. Mycologia 83:121-130.

Nyvall, R. F., Porter, R. A., Percick, J. A. 1995. First report of scab on cultivated wild rice in Minnesota. Plant Dis. 79:82.

O'Donnell, K., Ward, T. J., Geiser, D. M., Kistler, H. C., Aoki, T. 2004. Genealogical concordance between the mating types locus and seven other nuclear genes support formal recognition of nine phylogenically distinct species within the *Fusarium graminearum* clade. Fungal Genet. Biol. 41:600-623.

Ou, S. H. 1985. Rice diseases. 2nd ed. Kew, Surrey (England): Commonwealth Mycological Institute, CABI. 380 p.

Peraica, M., Radic, B., Lucic, A., and Pavlovic, M. 1999. Toxic effects of mycotoxins in humans. Bull. World Health Org. 77:754-766.

6. Ear blight (EB)

EB, also known as "panicle blight", "pecky rice", or "ear discoloration," refers to a disease complex showing discoloration or blight of the panicles. This disease has been reported in many rice-growing countries in South and Southeast Asia. The disease complex involves several fungi. The etiology of "panicle blight" occurring in USA has been established to be caused by two bacterial pathogens, *Burkholderia glumae* (Kurita and Tabei) and *B. gladioli* (Zopf) Yabuuchi et al (Nandakumar et al. 2009). It has come to be known as "bacterial panicle blight".

6.1. Symptoms

Symptoms vary depending on the fungi involved, environmental conditions, plant age and nutritional status, and cultivar susceptibility. In general, EB is characterized by discoloration and/or blight of either the entire panicle or a portion of the panicle, including the neck, brachia, internodes, nodes, and spikelets. The discoloration may be defined as brown to dark streak or it may appear as spreading. The grain of affected panicles often develops poorly or aborts.

6.2. Causal organism

Many fungi have been associated with EB, including some causing major foliar diseases (Atkins 1974, Lee 1992, Ou 1985). Fungi that have been cited include *Bipolaris oryzae* (Breda de Haan) Shoemaker, *Cercospora oryzae* Miyake, *Curvularia lunata* (Wakk.) Boedijn, *Fusarium nivale* (Fr.) Ces., *Fusarium* spp., *Alternaria oryzae* K. Hara, *Phoma* spp., *Gerlachia oryzae* (Hashioka & Yokogi) W. Gams, *Nakataea sigmoidea* (Cavara) K. Hara, *Cladosporium* spp., *Epicoccum* spp., and *Nigrospora* spp. In reality, this disease is perhaps part of the "sheath rot complex" in which the causal agents have been focused solely on the fungal flora and not on the bacterial community reported by Cottyn et al (1996a,b). To expand the syndrome, this is also part of the grain discoloration commonly observed on modern rice lacking photoperiod sensitivity and grown in the rainy season as previously discussed (for more details, see Grain discoloration above).

6.3. Disease cycle

Because the causal agents involve numerous fungi and bacteria, the presence of these pathogens, directly or indirectly and in various combinations, influences EB incidence and severity. However, it has been observed that EB is more likely to occur in late-planted fields or in fields deficient in nitrogen, potassium, iron, manganese, and magnesium (Lee 1992). Heavy rainfall during heading and extreme temperatures during maturation increase its severity.

6.4. Control measures

The disease can best be prevented by following recommended control procedures for the major leaf pathogens involved. Rice planting should be timed to mature before cool weather begins and avoid ear emergence and early grain filling stage during rainy periods. Fields should have a proper balance of nutrients sufficient for normal plant development. Fungicides have been reported to provide some control.

References

Atkins, J.G. 1974. Rice Diseases of the Americas: A Review of the Literature. Agr. Handbook No. 448, ARS, USDA, Washington, D.C. p 55-58.

Cottyn, B., Van Outryve, M. F., Cerez, M. T., De Cleene, M., Swings, J., Mew, T. W. 1996a. Bacterial diseases of rice. II. Characterization of pathogenic bacteria associated with sheath rot complex and grain discoloration of rice in the Philippines. Plant Dis. 80:429-437.

Cottyn, B., Cerez, M. T., van Outryve, M. F., Barroga, J., Swings, J., Mew, T. W. 1996b. Bacterial diseases of rice. I. Pathogenic bacteria associated with sheath rot complex and grain discoloration of rice in the Philippines. Plant Dis. 80:438-445.

Lee, N. F. 1992. Ear blight. In: R. K. Webster and P. S. Gunnell, editors. Compendium of Rice Diseases. APS Press, St. Paul, Minnesota.

Nandakumar, R. M., Shahjahan, A. K. M., Yuan, X. L., Dickstein, E. R., Groth, D. E., Clark, C. A., Cartwright, R. D., Rish, M. C., 2009. *Burkholderia glumae* and *B. gladioli* cause bacterial panicle blight in rice in the southern United States. Plant Dis. 93:896-915.

Ou, S. H. 1985. Rice diseases. 2nd ed. Kew, Surrey (England): Commonwealth Mycological Institute, CABI. 380 p.

7. False smut (to come)