Major seed-borne disease of cereals (wheat, barley, rice, etc.)

Wheat Diseases

Wheat (*Triticum aestivum* L.) is one of the major cereal crops cultivated worldwide and is not only a staple food but also has increasing global demand for wheat based products. The world acreage under wheat crop is 222.28 mha with production of 727.2 mt with average yield of 3254 Kg/h. After China, India is the second leading producer of wheat in the world.

The major impact of seed-borne diseases in wheat is not only the yield reduction but also deterioration in market quality of grains. Infected wheat seeds are the carrier of pathogens for long distance dissemination. Detection and management of seed-borne diseases through quality-control programmes that monitor seeds from harvest to purchase, marketing and sowing in the field are essential to ensure high-quality, pathogen-free and genetically pure seed. Major seed-borne diseases of wheat are Karnal bunt, *Fusarium* head blight, loose smut and spot blotch (Table).

Table: Major seed-borne diseases of wheat.

Disease	Pathogen
Karnal bunt	*Tilletia indica*
Head (ear) blight or scab	*Fusarium* spp.
Loose smut	*Ustilago nuda* var. *tritici*
Spot blotch	*Bipolaris sorokiniana*
Alternaria leaf blight	*Alternaria triticina*
Tundu or ear cockle or yellow ear rot disease	*Clavibacter tritici* and *Anguina tritici*

Karnal Bunt

The disease is known by various names such as new bunt, Karnal bunt and partial bunt. The Karnal bunt is native to South Asia and was detected in experimental wheats grown at the Regional Station of IARI at Karnal, Haryana in 1930. The occurrence of Karnal bunt is sporadic in nature but becomes serious in epidemic years and has caused substantial losses to wheat crop estimated loss up to 20% in a number of wheat cultivars.

Symptoms

Karnal bunt pathogen infects wheat at the flowering stage prior to seed formation, hence the symptoms are visible only when the grains have fully developed in ear heads. In a stool all the ear heads are not affected and also all the grains in a spike are not infected. A careful examination of individual ear reveals bunt infection in the field. In the standing wheat crop, the infected spike can be detected by the shiny silvery black spikelets, with glumes spreading apart and swollen ovaries. The spikes of infected plants generally are reduced in length and in number of Spikelets.

The pathogen converts the infected ovary into a sorus where a mass of dark brown coloured teliospores are produced. Infection of grains varies from small sori to completely bunted seeds. Small sori are generally developed in longitudinal furrow, leaving the dorsal side of the seed and endosperm unaffected. If the host is highly susceptible, the whole endosperm material may be converted into a large sorus, and seed looks hollow leaving only the pericarp and the aleurone layer. The infected ears emit a fishy odour due to trimethylamine, a volatile compound produced due to pathogenesis. In such cases the shriveled embryo is dead.

Pathogen

The pathogen of Karnal bunt is caused by the Basidiomycete fungus *Tilletia indica*. Teliospores of *T. indica* are dark brown to black, globose to sub-globose in shape having hyaline sheath of 2–4 μm thickness and measure 22–49 μm in size, average being 35 μm in diameter. The spores are globose to elongate, yellowish sterile cells which have smooth wall. They are smaller in size (15–28 μm) than normal teliospores. Fresh teliospores have a period of dormancy and dormancy period of 1–6 months is needed prior to spore germination. The highest germination occurs with year-old teliospores.

Disease Cycle

The pathogen survives in the form of teliospores which fall in the soil during harvesting of the crop. The spores adhere to the surface of healthy seed and act as contaminant. The spores on the seed create problem only when they are transmitted through the seed and deposited on the soil surface where infection occurs. The spores remain viable for a number of years in the soil lying 15 and 25 cm deep. Obviously, soil-borne inoculum is the primary source of annual recurrence of the disease. Normally, the resting spores buried in the soil germinate from the middle of February to middle of March when soil temperature and moisture are suitable. The germinated spores produce a germtube (promycelium) to come up to the soil level bearing tuft of 110–185 sickle-shaped primary sporidia which they in turn produce two types of secondary sporidia. Occasionally, the air-borne allantoid sporidia get lodged on spike at anthesis and germinate on the glume surface, and the fungus becomes partially systemic in rachis and the rachilla. Subsequently, the hyphae spread to the adjacent florets and spikelets around the infection site. Hyphae of the fungus then grow through the base of glume into sub-ovarian tissue and enter the pericarp through the funiculus. Further growth of hyphae is entirely within the pericarp.

These hyphae become sporogenous produce teliospores in wheat seed to cause Karnal bunt infection in the next crop season.

Management

- Grow resistant cultivars
- Crop rotation following non-cultivation of wheat crop for two consecutive years
- Apply seed dressing fungicides

Loose smut of wheat

Loose smut has a wide distribution and can occur anywhere wheat is produced.

Symptoms

Mild symptoms may be present prior to heading, including yellowish leaf streaks and stiff, dark green leaves. Affected plants head out early, producing sterile heads with clumped, sooty olive-black spores in place of healthy glumes and kernels. Spores are not enclosed by the seed coat, so are quickly dispersed by rain or wind after emergence. After spores disperse, only a bare rachis remains with a few fragments of glumes or awns. These spores infect other wheat plants at flowering, causing seed infection. Infected seed appear healthy. The best time to scout is after heading.

Pathogen

The disease is caused by the basidiomycete fungus *Ustilago tritici*. The fungus is seedborne, and unless infected seed is planted, no disease will occur in a field. New infections are favored by humid, cool weather 15-22 ^{o}C during flowering.

Disease life cycle

The fungus lies dormant within the embryo of infected seed. When the infected seed germinates, the fungus grows within the developing shoot, eventually reaching the ear primordia. The fungus develops within the young ear, eventually replacing spikelets with masses of fungal spores. Usually, a mass of black fungal spores replaces grain, affecting all or part of the ears. This leaves only the bare remains of the ear rachis. Because the blackened ears are obvious, the disease appears to be very severe, even at very low incidence levels. Spore release occurs from infected ears as they emerge. These are spread by wind to open flowers. Once spores germinate, the fungus grows in the developing grain site.

Management

- Use disease free seed
- Use a disease resistant variety
- Treat the wheat seed with systemic fungicides like carboxin and tebuconazole.

Rice diseases

Rice blast

This disease that it has been ranked among the most important plant diseases of them all. Other grasses, including crabgrass, are also infected by the pathogen.

Symptoms

The symptoms of rice blast include lesions that can be found on all parts of the plant, including leaves, leaf collars, necks, panicles, pedicels, and seeds. A recent report shows that even roots can become infected. However, the most common and diagnostic symptom, diamond shaped lesions, of rice blast occur on the leaves.

On rice leaves, lesions may initially appear gray-green and water-soaked with a darker green border and they expand rapidly to several centimeters in length. On susceptible cultivars, older lesions often become light tan in color with necrotic borders. On resistant cultivars, lesions often remain small in size (1-2 mm) and brown to dark brown in color. Symptoms of infection of the collars consist of a general area of necrosis at the union of the two tissues. Collar infections can kill the entire leaf and may extend a few millimeters into and around the sheath. The fungus may produce spores on these lesions. Necks are often infected at the node by the rice blast fungus and infection leads to a condition called rotten neck or neck blast. Infection of the necks can be very destructive, causing failure of the seeds to fill (a condition called blanking) or causing the entire panicle to fall over as if rotted. The rice blast fungus can also infect the panicles as the seeds form. Lesions can be found on the panicle branches, spikes, and spikelets. The lesions are often gray brown discolorations of the branches of the panicle, and, over time, the branches may break at the lesion. he fungus has often been isolated from the pedicels of the seeds. Seeds are not produced when pedicels become infected, a condition called blanking. Symptoms of rice blast on seeds themselves consist of brown spots, blotches (Figure 8), and occasionally the classic diamond-shaped lesion often seen on leaves. The process and the time during which infection of seeds by spores of the pathogen occurs has not been fully described but recent information shows that the fungus can infect seeds by infecting the florets as they mature into seeds, and it is believed that this is the main way seed infection develops.

Pathogen

Rice blast is caused by the Ascomycete fungus called *Magnaporthe oryzae* (formerly *Magnaporthe grisea*) which is the sexual, or teleomorphic, stage of the rice blast pathogen. The asexual stage of *Magnaporthe oryzae* is described

by the name *Pyricularia oryzae* (formerly called *P. grisea*) and it is the most common spore form of the fungus. These spores, called conidia, are produced abundantly on lesions.

Disease cycle

The overwintering sources of spores that comprise the primary inoculum consist of grasses, volunteer plants, infested refuse, and infested seed on the soil surface after mechanical planting. Spores produced as the primary inoculum on the overwintering tissues produce the initial infections on young seedlings when the spores that are deposited on leaves, germinate and invade leaf tissues. Lesions on the young seedlings appear within a few days after infection. These secondary lesions produce more spores and these spores are readily wind disseminated to nearby healthy leaf tissues. The secondary cycles can be repeated many times during the growing season, with the potential for very high amounts of disease within the crop.

The amount of disease at the end of the vegetative phase of the growing season influences the amount of disease during the reproductive phase. Spores produced near the end of the growing season may infect the collar of the flag leaf producing symptoms called collar rot. They may also infect the neck when it emerges from the infected collar upon which the head will be supported to produce a condition called rotten neck or neck blast.

Disease Management

- Crop rotation is one simple and effective technique that is highly recommended
- Grow resistant varieties
- Use of chemical fungicides to control the disease, either as seed treatment and/or foliar application.