DISEASES OF FIELD CROPS AND THEIR MANAGEMENT

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The Disease Triangle

Environment
Host
Pathogen
Plant disease triangle

- Environment (Favourable)
- Pathogen (virulent)
- Host (Susceptible)

Disease
Plant disease triangle

- Environment (Unfavorable)
- No Disease
- Pathogen (virulent)
- Host (Susceptible)
Plant disease triangle

Environment (Favorable)

No Disease

Pathogen (Avirulent)

Non-Host (Immune)
Disease Tetrahedron

Man

Pathogen

Environment

Host
Disease Development

Pathogens (virulent)
• Fungi
• Viruses
• Bacteria
• Phytoplasma
• Nematodes
• Parasitic higher plant.

Host – susceptible

Environment-favourable
Disease Development
-The classic disease triangle establishes the conditions for disease development, i.e. the interaction of a susceptible host, a virulent pathogen and a favorable environment.
Plant Pathogens

**Fungi:**
- 10,000 - 15,000

**Viruses:**
- ~1,000

**Nematodes:**
- Several hundred species

**Bacteria:**
- ~100 species

**Fungi:**
- 10,000 - 15,000
Assessment of losses

• at least 10-30% of global food production

• representing a threat to food security

Disease Management

• Two basic measures
  - Preventive (prior to infection)
  - Curative (after infection)

• Four approaches
  - Cultural/physical practices
  - Host resistance
  - Biological control
  - Chemical control

IDM
Important diseases of wheat (*Triticum aestivum* L.)

**Rusts of Wheat** - caused by the fungus *Puccinia*
- Stem or black rust - *Puccinia graminis* f. sp. *tritici*
- Brown rust or leaf rust - *Puccinia triticina* (Syn. *P. recondita*) and
- Yellow or stripe rust - *Puccinia striiformis* f. sp. *tritici.*

**Smuts of Wheat**

**Bunts** - caused by the smut fungus *Tilletia*
- Hill bunt or Common bunt or stinking smut or covered smut - two very closely related fungi, *Tilletia tritici* (syn. *Tilletia caries*) and *T. laevis* (syn. *T. foetida*).
- Karnal or Partial bunt - *Tilletia indica* (Syn. *Neovossia indica*)
- Loose smut - *Ustilago nuda* (Syn. *U. segetum*)
- Flag smut - *Urocystis agropyri*

**Powdery mildew of wheat** - *Blumeria graminis* (DC.) Speer

**Tundu disease of wheat** - *Calvibacter tritici* associated with ear cockle disease (caused by the nematode *Anguina tritici*).
• CLASSIFICATION:
  – Kingdom: Mycota
  • Division: Eumycota
    – Subdivision: Basidiomycotina
• *Puccinia* is an obligate parasite.
• It is a heteroecious parasite.
• It’s life cycle on Wheat and on Barberry.
• Wheat is a primary host.
• Barberry is a secondary or alternate host.
Puccinia Life Cycle
Puccinia Life Cycle
Puccinia Uredo-sporangium

Uredospore

Mycelium
Puccinia Uredo-sporangium-Wheat
Stage III: Telia bearing teliospores $(n+n>2n)$
Puccinia Teluto sporangium

Telutospores
Germinated Teluto spore and formation of Basidium & Basidio spores
Infection of *Puccinia* on Barberry leaf. Lower and Upper sides.
Stage I: Aecia bearing aeciospores (n+n)
Puccinia - Polymorphic fungi

- Polymorphism: Various types of spores produced during life cycle according to hosts' life cycle and climatic condition.
- Known as Polymorphism.
- Fungi shows polymorphism known as Polymorphic fungi.
*Berberis vulgaris*: dicot

**Stage 0**: Spermagonium
**Stage I**: Aecium

*Wheat* (*Triticum aestivum* L.): Monocot

**Stage II**: Uredium
**Stage III**: Telium
**Stage IV**: Basidium
Puccinia Life Cycle
Stem or black rust of wheat

DISEASE: Stem rust (black rust or cereal rusts)
PATHOGEN: *Puccinia graminis f. sp. tritici*
HOSTS: Wheat, barley and common barberry (*Berberis, Mahoberberis*, and *Mahonia* spp.)
On wheat and other grass hosts:

- After 7 to 15 days infection
- Elongated to irregular shaped pustules (uredinia) of brick-red uredinia spores break through the epidermis and develop on the upper leaf surfaces, leaf sheaths, glumes and awns.
Microscopically, these red spores are covered with fine spines.
The pustules may be abundant and produced on both leaf surfaces and stems of host plants.
Later in the season, pustules of black teliospores (telia) begin to appear in infected host plants. Microscopically, teliospores are two celled and thick walled.
Pre disposing (environmental) factors

- **Warm-humid** weather conditions with intermittent rains.
- Hot days 25-30°C and mild nights (15-20°C)
- Leaf wetness from rain or dew
Pathogenesis
Disease Cycle and Epidemiology
DISEASE: Leaf or Brown rust

PATHOGEN: *Puccinia triticina* (Syn. *Puccinia recondita f. sp. tritici*)

HOSTS: Wheat, barley and common barberry (*Berberis, Mahoberberis, and Mahonia* spp.)
Symptoms and Signs

Scattered oval to circular small brown pustules (rusty-red uredospores) containing a powdery mass of orange to red-orange spores develop on the upper leaf surfaces, leaf sheaths, glumes and awns.
Black pustules form as the plant approaches maturity (Telia). Microscopically, teliospores are two celled and thick walled.
Pre-disposing (environmental) factors

- **Warm-humid** weather conditions with intermittent rains.
- Hot days 25-30°C and mild nights (15-20°C)
- Leaf wetness from rain or dew
Life and disease cycles for *Puccinia tricinia*
DISEASE: Stripe or Yellow rust
PATHOGEN: *Puccinia striiformis f. sp. tritici*
HOSTS: Wheat
Symptoms and Signs

The first sign of stripe rust is the appearance of yellow streaks (pre-pustules), followed by small, bright yellow, elongated uredial pustules arranged in conspicuous rows on the leaves, leaf sheaths, glumes and awns.
Mature pustules will break epidermis and release yellow-orange masses of urediniospores.
Black pustules form as the plant approaches maturity (Telia).

Teliospores of stripe rust (\textit{Puccinia striiformis}) (400x)
Pre-disposing (environmental) factors

- **Cool - humid** weather conditions with intermittent rains.
- Cooler climates (10-16ºC)
- Leaf wetness from rain or dew
- Heavy dew or intermittent rains can accelerate the spread of the disease
Yellow rust life cycle

**SUMMER**
- Pustules erupt releasing uredospores
- Disease spread by wind dispersal
- Late in the season black telia form on leaves
- Teliospores produce basidia and basidiospores
  (No alternate host known)

**SPRING**
- Early infection of plants by wind-borne uredospores

**AUTUMN**
- Over-winters on volunteer plants
Stages of rusts in wheat

- Pycnial stage (stage 0)
- Basidiospores (n) (stage IV)
- Germination & meiosis
- Teliospores 2n
- Karyogamy (n + n) 2n
- Primary host (wheat)
- Telial stage (n + n) (stage III)
- Urediniospores (n + n)
- Epidemic
- Aecial stage (stage I)
- Aeciospores (n + n)
- Alternate host meadow rue Thalictrum speciosissimum
- Plasmogamy (n + n)
- Urediniospores
Rusts can produce up to five spores types during their life cycle

0- **Pycniospores** (Spermatia)-Haploid gametes in heterothallic rusts.

I- **Aeciospores** - non-repeating dikaryotic vegetative spores

II- **Urediniospores** - repeating dikaryotic vegetative spores. These spores are referred to as the repeating stage because they can cause auto-infection (re-infect the same host from which the spores were borne). These spores are red/orange and are a characteristic sign of rust fungus infection.

III- **Teliospores** - Diploid spores that produce basidia and are the survival stage of life cycle

IV- **Basidiospores** - stem from basidia. Haploid spores which infect the alternate host.
Mehta from 1920 to 1950 studied the perpetuation of rusts in more detail and proved that the

- Barberis species found in India do not play any role in the perpetuation of stem rust in India.
- Similarly it has been proved that Thalictrum species occurring in the hills are also not playing any role in the perpetuation of stem rust.
•Rusts over summer in cooler climates of hills on self-sown wheat plants, ratoon tillers and also on summer crop grown in Nilgiri and Palini hills in south India. The fixed path between Nilgiri and Palini hills to Narmada and Tapti river belt is known as Puccinia path.
Initially, Mehta had shown the movement of leaf rust both from north and south Indian hills. Later, Joshi and his team supported this view and further demonstrated that leaf rust (\emph{P. recondita}) spreads both from southern and northern hills. It is introduced from Nilgiri and Palini hills and is established in the plains of Karnataka and Tamil Nadu in South India. The rust population from southern foci moves northwards towards Maharashtra and Madhya Pradesh. The spread of leaf rust over the Indo-Gangetic plains is predominantly from the warmer north-eastern region and is influenced by the number of rainfall during winter month.
• Stripe rust (P. striiformis) is a major problem only in cooler parts of the country especially north and north-western region. Its infection in south, central and eastern parts remains isolated and seldom become a serious threat to wheat. In north India, the inoculum moves from northern hills and get established in the plains of Punjab, Haryana and western Uttar Pradesh. In the foot hills and northern Indian plains stripe rust spread much faster by uredospores than leaf rust due to favourable cool temperature but the spread after February is checked due to rise in temperature and this time the telial stage is developed which does not play any role in the spread of the disease.
Management of rusts of wheat

Prevention, early warning and rapid response

- Using resistant cultivars and early intervention are the key principles of controlling wheat rust diseases.

- **Ug99** is a virulent strain of *stem rust* which emerged in *Uganda in 1998-1999*. Most of the wheat varieties are susceptible to this strain.

- FAO in collaboration with CIMMYT provide policy and technical support to the concerned countries to combat this threat in the context of the Borlaug Global Rust Initiative.
Cultural practices

- Grow early maturing resistant wheat varieties.
- Appropriate spacing and fertilizer (N) application.

Chemical control

- Prophylactic sprays of Mancozeb (Dithane Z-78) @ 0.25%
- Fungicides that inhibit the synthesis of sterols [i.e., sterol biosynthesis inhibitors-SBIs] like Tilt (Propioconazole) 25 EC @ 0.1% at 15 days intervals.
The black powdery spores (Teliospores) are typical of smut diseases, which also includes bunt.
Loose Smut of Wheat

DISEASE:  Loose Smut

PATHOGEN:  *Ustilago nuda var. tritici*. (Syn. *U. segetum var. tritici*)

HOSTS:  Wheat, barley and common
Classification:
Phylum: Basidiomycota
Class: Ustilaginomycetes
Sub-class: Ustilaginomycetidae
Order: Ustilaginales
Family: Ustilaginaceae
Causal organism: Ustilago tritici (Persoon) Rostrup (1890)
(Obs. syn. Ustilago nuda var. tritici Schaffnit)
(Syn. Ustilago segetum var. tritici (Pers.) Brunaud (1878))
(Mathur and Cunfer 1993, and Wikipedia)
Symptoms and Signs

Every ear of the affected plants is converted into a black mass of spores and no grains are formed. The infected heads emerge from the boot one to three days earlier than those of healthy plants. As a rule, all the glumes (chaff) and grain in a smutted head are completely transformed into black powder.
This sooty mass is composed almost entirely of millions of teliospores. The spores are quickly dispersed by the wind during the flowering period of normal heads, and by harvest only an erect bare rachis remains. All of the floral parts of the head, except for the rachis and pericarp membrane, are invaded by mycelium of the fungus and converted into loose aggregates of smut spores (teliospores).
Predisposing Factors

• Wind
• Moderate rains
• Cool temperatures (16-22 degrees Celsius)
Disease Cycle of Loose Smut

Loose smut

*Ustilago nuda f.sp tritici*

Mycelium follows growing point of wheat plant

Mycelium invades the grain sites

Grain sites replaced by masses of teliospores

Teliospores land on flowers of healthy plants and infect developing grain

Mycelium invades part of embryo in seed

Mycelium invades young seedlings
USTILAGO NUDA (Flugbrand der Gerste)

USTILAGO TRITICI (Flugbrand an Weizen)

**interzelluläres** Wachstum im Meristem

**intrazelluläres** Wachstum im Keimling

Körner und Spelzen werden zersetzt

Brandsporen keimen auf Narbe und Bilden eine Basidie

dikaryontisches Myzel infiziert via Griffel oder Ovarwand den Embryo

Überwinterung im Embryo
Management of Loose Smut of Wheat

- **Disease control:**
  The disease is internally seed borne and as such spray of fungicides is not effective in controlling the disease.

- **Seed treatment**

- **Use of resistant varieties:**
  The most successful method is the use of resistant varieties.

- **Crop rotation:**
  Crop rotation at suitable intervals is also effective in disease control.
Seed treatment

(a) Hot water treatment:
The seeds are first soaked in water for five hours at 20°C, the water is drained off and then they are treated with hot water at 49°C for about a minute and finally with hot water at 52°C for 11 minutes. Immediately after the hot water treatment, the seeds are cooled off by dipping in cold water and dried. The dormant mycelium inside the seed dies off by this treatment.

(b) Use of systemic fungicides:
Several fungicides like carboxin, vitavax and benlate @ 2.0 g/Kg seed and Propioconazole @ 0.1% are used for seed treatment to reduce the pathogen infectivity. A combination of vitavax with thiram is very effective for disease control.
Hill Bunt of Wheat

DISEASE: Hill Bunt or Stinking Smut or Common Bunt

PATHOGEN: *Tilletia caries* and *Tilletia foetida*.

HOSTS: Wheat
Symptoms and Signs

Plants with common bunt may be moderately stunted but infected plants cannot be easily recognized until near maturity and even then it is seldom conspicuous.
After initial infection, the entire kernel is converted into a sorus consisting of a dark brown to black mass of teliospores covered by a modified periderm, which is thin and papery. The sorus is light to dark brown and is called a bunt ball.
Symptoms and Signs

The bunted heads are slender, bluish-green and may stay greener longer than healthy heads. The bunt balls change to a dull gray-brown at maturity, at which they become conspicuous. The fragile covering of the bunt balls are ruptured at harvest, producing clouds of spores. The spores have a fishy odor (trimethylamine)
Disease cycle of hill bunt

- Infectious hypha (dikaryotic)
- Conjugating sporidia (H-structure)
- Systematic infection of wheat plant
- Mycelium invading wheat head
- Intercellular hyphae become teliospores
- Haploid sporidia
- Teliospore cross-section (dikaryotic)
- Basidium
- Teliospore
- Karyogamy followed by meiosis
- Bunted kernels
- ‘Bunt balls’
- Infected head
Management of Hill Bunt of Wheat

• **Disease control:**
The disease is externally seed borne and systemic infection is there - as such spray of fungicides is not effective in controlling the disease.

• **Seed treatment** Systemic fungicides like carboxin, vitavax and benlate @ 2.0 g/Kg seed and Tilt (propioconazole) 25 EC @ 0.1% are used for seed treatment. A combination of vitavax with thiram is very effective for disease control.

• **Use of resistant varieties:**
The most successful method is the use of resistant varieties.

• **Crop rotation:**
Crop rotation at suitable intervals is also effective in disease control.
Karnal Bunt of Wheat

**DISEASE:** Karnal Bunt or Partial bunt

**PATHOGEN:** *Tilletia indica* (Syn. *Neovossia indica*)

**HOSTS:** Wheat
History of Karnal bunt

Karnal bunt is so named because it was discovered in 1931 on wheat grown near Karnal, India by Mitra. Since then, it has been found in all major wheat-growing states of India, as well as in Pakistan, Iraq, Mexico, and Afghanistan.
Karnal bunt is difficult to identify in the field. Developing wheat kernels are randomly infected and usually only partially converted to the fungus and hence, called partial bunt.
The disease is not usually noticed until the grain is threshed and partially smutted kernels are exposed. Generally a few florets per spike are affected.
While diseased seeds usually retain a partial seed coat, the embryo and part of the endosperm have been converted to masses of small black spores, which emit a fishy odor (trimethylamine).
Symptoms and Signs

Teliospores are dark brown to black in colour.
Ellipsoidal to spherical in shape.
Characteristic apiculus and episore is reticulate with curved spines.

Infected kernels and teliospores
Predisposing Factors

- Rains Splashes
- Moderate rains
- Moderate temperatures (16-24 degrees Celsius)
Life cycle of *Tilletia indica*
Disease cycle of Karnal Bunt

VULNERABLE
Allantoid spores on boot leaf

MOST VULNERABLE
Spores run down to boot sheath
Primary infection site
Rundown to boot sheath

VULNERABLE
Secondary (minor) infection site
Can escape

VULNERABLE
Dew or rain induced wash down

Move up to higher leaf
Multiply & disperse

Allantoid sporidia
Soil surface sporidia
Filiiform secondary sporidia
Whorl

Germinating primary sporidia on whorl (sickle shape)
Germinating teliospores
Teliospores (soil borne)

Left over sheaves and farmyard manure
Infected grains
Combining & threshing

Until ED stage schematic
Partial systemic spread
Harvest
Management of Karnal bunt of Wheat

• **Disease control:**
The disease is basically seed and soil borne.

• **Use of resistant varieties:**
The most successful method is the use of resistant varieties.

• **Crop rotation:**
Crop rotation at suitable intervals is also effective in disease control.

• **Use disease free Seed**

• **Spray of Tilt** (propioconazole) 25EC @ 0.1% at heading (anthesis) stage.
Seed treatment

(a) Hot water treatment:
The seeds are first soaked in water for five hours at 20°C, the water is drained off and then they are treated with hot water at 49°C for about a minute and finally with hot water at 52°C for 11 minutes. Immediately after the hot water treatment, the seeds are cooled off by dipping in cold water and dried. The dormant mycelium inside the seed dies off by this treatment.

(b) Use of systemic fungicides:
Fungicides like carboxin, vitavax, and benlate @ 2.0 g/Kg seed and propioconazole @ 0.1% are used for seed treatment to reduce the pathogen infectivity. A combination of vitavax with thiram is very effective for disease control.
DISEASE: Flag smut
PATHOGEN: *Urocystis agropyri* (Syn. *U. tritici*)
HOSTS: Wheat
Symptoms and Signs

**Flag smut**: a smut fungus, causing a smut in cereals, affects leaves, leaf sheath, culm and stems, and is characterized by chains of sori (linear sori) within the plant tissue that later rupture releasing black masses of spores. These streaks have a greasy appearance. Diseased plants remain stunted and produce excessive numbers of tillers. There is a twisting of affected leaves which droop down like a flag and finally wither away.
The fungus forms small spore balls consisting of 1 to 4 teliospores surrounded by a layer of smaller sterile cells. The teliospores are red-brown, smooth and round. They germinate to produce a short hypha (promycelium) with 3 to 4 basidiospores (sporidia) near the tip.
Disease cycle of Flag smut

Flag smut

Urocystis agropyri

Sori develop between veins on leaves and glumes.

Fungus grows systemically within plant.

In autumn and spring sporidia germinate. Mycelium infects seedlings.

Teliospores germinate forming promycelium and sporidia.

Sori erupt releasing spore balls containing teliospores.

Overwinters as spore balls in soil and on seed.

Germinating teliospore.

Sporeball.
Management of **Flag smut** of Wheat

The disease is seed as well as soil borne.

- **Chemical**
  
  **Seed treatment:** Systemic fungicides like carboxin, vitavax and benlate @ 2.0 g/Kg seed and Tilt (propioconazole) 25EC @ 0.1%

- **Cultural.** Shallow planting is better than deep planting to reduce infection, probably because the seedling is in a susceptible stage for a shorter period of time. A 1- or 2-year break in wheat cropping may be beneficial.

- **Resistance.** Use resistant cultivars or at least avoid very susceptible cultivars
Glume blotch of Wheat

DISEASE: Glume blotch of Wheat
PATHOGEN: Septoria nodorum

HOSTS: Wheat, barley and oats
Symptoms and Signs

**Glume blotch**: Infected glumes develop gray-brown blotches. The septoria leaf and glume blotch pathogen survives within infested straw, seed and on volunteer wheat and serves as the source of inoculum with a new crop of wheat. Infections occur during periods of extended wetness.
Infected heads develop shriveled grain. seeds. Aged blotches develop dark pin-point structures called **pycnidia**. Pycnidia are the source of infectious spores. Pycnidia on discolored glumes are the best sign of this disease.
Pycnidium

Pycnidiospores inside pycnidium
Disease cycle of Glume blotch
Management of Glume blotch of Wheat

Management Strategy

• Grow moderately resistant varieties.
• Use disease-free seed (e.g. certified seed). Use seed grown in the drier areas of the province.
• Avoid excessive use of nitrogen
• crop rotation with non-cereal crops.
• General sanitation: Turn under the stubble and crop residue to reduce disease incidence and control volunteer wheat seedlings.
• Use wide row spacing and adequate but not excessive nitrogen levels. These practices lower canopy density and
DISEASE: Blast of Paddy

PATHOGEN: *Pyricularia grisea* ( *Magnaporthe grisea* )

HOSTS: Paddy
Symptoms and Signs

**Blast of Paddy:** Blast symptoms can occur on leaves, leaf collars, nodes and panicles. **Leaf blast,** the lesions are elliptical or spindle shaped with brown borders and gray centers. **Collar blast** or **node blast** occurs when the pathogen infects the collar that can ultimately kill the entire leaf blade. **Neck blast** occurs when the pathogen infects the neck of the **panicle.** The infected neck is girdled by a grayish brown lesion and the panicle falls over if the infection is severe. If neck blast occurs before the milk stage, the entire panicle may die prematurely, leaving it white and completely unfilled. The fungus produces two **toxins-μ-picolinic acid and piricularin**
Microscopically pyriform (pear-shaped) conidia are usually three-celled or 2 septate borne on conidiophores.
Factors favorable for blast:

- Upland conditions
- Extended dew periods
- High relative humidity
- Intermittent rain showers or drizzle
- High nitrogen fertilizer
Life cycle of Blast of Paddy

- The spores are released by dew or rain and are carried in the air to other plants.
- Then the fungus produces more spores.
- Airborne spores called conidia land on rice leaves.
- The fungus grows and produces leaf spots after 4-5 days.
- The spores germinate and the fungus penetrates the leaf surface or enters the leaf through the stomata.
Management of Blast of Paddy

- Use of resistant cultivars.
- Efficient use of nitrogen fertilizers.
- Soil amendment with silica.
- Spray Beam (tricyclazole) – melanin biosynthesis inhibitor fungicide for effective control
Brown spot of Paddy

DISEASE: Brown spot of Paddy

PATHOGEN: Bipolaris oryzae (Breda de Haan) Shoemaker
(Syn. Helminthosporium oryzae)

HOSTS: Paddy
**Historical Events**

*Bipolaris oryzae* (Breda de Haan) Shoemaker  
(anamorph) *Drechslera oryzae* (Breda de Haan) Subramanian & P. C. Jain  
(synonym) *Helminthosporium oryzae* Breda de Haan  
(synonym) *Cochliobolus miyabeanus* (Ito & Kuribayashi) *Drechsler ex Dastur* (teleomorph)

is a *fungus* that causes *brown spot* disease in *rice*. This disease was the causal agent of the *Bengal famine of 1943*.  
It was used by the *USA* as a *biological weapon* in Japan during *World War II*. 
Symptoms and Signs

Brown spot of Paddy: The symptoms of the disease appear on the coleoptile, the leaves, leaf sheath and also the glumes. Small circular to oval reddish-brown spots with gray center surrounded by a dark to reddish-brown margin.
The fungus may also infect the glumes, causing dark brown to black oval spots, and may also infect the grain, causing a black discoloration.
Conidia are generally curved, boat, or club-shaped, with 6 to 14 transverse septa or cross walls
At the point of attachment to a conidiophore with a minute, slightly protruding hilum (dot).
Brown Spot of Rice
Life cycle of Brown spot of Paddy

- Grain becomes infected when the disease develops in the panicles.
- The disease is transmitted by infected seeds.
- Disease spores germinate and enter the seedling roots or coleoptile.
- The spores germinate and infect the plant’s leaves or panicles.
- As the rice grows, spores are formed on leaf spots. These spores are then blown to the leaves and panicles of other plants.
Management of Brown spot of Paddy

- Use disease free seed
- Use of resistant varieties.
- Avoid excesses of N-application
  Use of silicon fertilizers (e.g., calcium silicate slag)
- Since the fungus is seed transmitted, a hot water seed treatment (53-54°C) for 10-12 minutes. And controls primary infection at the seedling stage.
- Seed treatment with captan, thiram or carbendazim.
- Seed treatment with tricyclazole followed by spraying of mancozeb + tricyclazole at tillering and late booting stages gave good control of the disease.
Sheath blight of rice

DISEASE: Sheath blight of rice

PATHOGEN: 
*Rhizoctonia solani* Kuhn

HOSTS: Paddy
Initial lesions are small, ellipsoidal or ovoid, greenish-gray and water-soaked and usually develop near the water line in lower leaves.
Older lesions are elliptical or ovoid with a grayish white center and light brown to dark brown margin. Lesions may coalesce forming bigger lesions with irregular outline and may cause the death of the whole leaf.
Severely infected plants produced **poorly filled or empty grains**, especially those on the lower portion of the panicles. **Brown sclerotia** of the fungi are observed on the infected area of the plant.
Sclerotia on the soil or floating on the water are assumed to be the sole source of inoculum in temperate regions.
Epidemiology

- Microclimatic conditions Warm (28-32°C) – humid (RH-95%) with low sunlight favor the disease.
- Disease development - in the early heading and grain filling growth stage
- Soil borne sclerotia, and to a lesser degree mycelium in plant debris, are means of pathogen survival between crops and are the primary inoculum.
- Sclerotia floating in the paddy field and infection occur near the water line.
Life cycle of Sheath blight of rice

- The disease spreads sideways and moves up the plants.
- The fungus grows on the plant.
- Sclerotia develop on lesions and drop to the soil.
- The fungus survives in sclerotia in the soil. The sclerotia float to the water surface during land preparation.
- The sclerotia germinate and the fungus penetrates the plant.
Management of Sheath blight of rice

Host resistance:
• No resistant cultivars.

Cultural practices:
• General sanitation-destruction of crop debris.
• Flooding of rice fields.
• Proper spacing.
• Efficient use of N-fertilizers.

Chemical control:
• Spray of Tilt (Propioconazole), a combination of fungicides (Azoxystrobin 18.2% + Difenoconazole 11.4% SC) was found highly effective against rice sheath blight.
DISEASE: False smut or green smut of Paddy

PATHOGEN: *Ustilaginoidea virens* (Cooke) Takah (anamorph), *Claviceps oryzae*-sativae Hashioka (teleomorph)

HOSTS: Paddy
Symptoms and Signs

- Individual rice grain transformed into a mass of yellow fruiting bodies.
- Growth of velvety spores that enclose floral parts.
- Only few grains in a panicle are usually infected and the rest are normal.
The causal organism is a fungus. Microscopically the chlamydospores or the conidia of the fungus are spherical to elliptical. They are pale and almost smooth when young, olivaceous and warty when mature.
Epidemiology

- presence of rain and high humidity
- presence of soils with high nitrogen content
- presence of wind for dissemination of the spores from plant to plant
- presence of overwintering fungus as sclerotia and chlamydospores
Life cycle of False smut or green smut of Paddy

- Eventually the grain or floral parts are replaced by a smut ball.
- Small infection bodies called chlamydospores are produced on the surface of the smut balls.
- The spores either infect the developing spikelets at the flowering stage or the mature grain later in the season.
- The spores are blown in the air and infect the panicles of other plants.
Management of False smut or green smut of Paddy

Management Strategy

• Removal and proper disposal of infected plant debris.
• Use of disease-free seeds that are selected from healthy crop.
• Seed treatment with carbendazim 2.0g/kg of seeds.
• Spraying of copper oxychloride at 2.5 g/litre or Propiconazole at 1.0 ml/litre at boot leaf and milky stages will be more useful to prevent the fungal infection.
DISEASE: **Bacterial blight of rice**

PATHOGEN: *Xanthomonas oryzae pv. oryzae* (Ishiyama) Swings et al.

HOSTS: **Rice**
Symptoms and Signs

- ‘Kresek’ occurs in early stage (Seedling wilt-Plants withers and dies up).
- In later stage blightening starts from the tip of leaves to the base.
- Straw turned yellow to white.
- Partially filled grains.
- Appearance of bacterial ooze (a milky or opaque dew drop) on young lesions.
The gram negative bacteria causing the disease are rod-shaped and devoid of capsules.
Life cycle of Bacterial blight of rice

After the initial leaf lesions appear, bacteria from ooze droplets on the leaf surface are spread throughout an area by wind and rain, particularly by typhoons or irrigation water.

Rice plants can become infected with bacterial blight from many sources: diseased stubble, diseased seeds, paddy water, and diseased straw.

The bacteria multiply inside the plant and enter the veins of the leaf. Bacteria that enter the roots plug the water-conducting tissue and cause the plant to wilt.

The bacteria enter through the water pores of the hyathodes or wounds of the leaf or root and multiply inside the plant.

High temperature and humidity during crop growth increase the incidence of bacterial blight.
Life cycle

The bacteria enter the leaf through injured tissues or leaf stomata. The bacteria multiply and remain in localized tissues just beneath the surface. They do not spread throughout the plant like the bacteria causing bacterial blight.

High temperatures and high humidity favor disease development.

The bacteria are spread by wind, rain, irrigation water, and diseased seeds.

Bacterial ooze forms small, round, yellow beads on new leaf lesions.
Management of Bacterial blight of rice

Management Strategy

• The use of resistant varieties.
• Seed treatment with bleaching powder (100µg/ml) and zinc sulfate (2%)
• Field sanitation such as removing weed hosts, rice straws, ratoons and volunteer seedlings.
• Use balanced fertilizer doses, especially nitrogen.
• Proper plant spacing.
• Spray Blitox (Copper oxychloride) 50 W P alternate with Streptocycline with the appearance of disease
DISEASE:

• Local Name: Tungro virus rog
• PATHOGEN: Rice Tungro Spherical Virus (RTSV)

HOSTS: Rice
Symptoms and Signs

- Yellow discoloration begins from leaf tip and extends down to the blade or the lower leaf portion
- Infected leaves may also show mottled or striped appearance - stunting
- Reduced tillering.
- Delayed flowering, which may delay maturity.
- Most panicles sterile or partially filled grains and covered with dark brown blotches.
In field the symptoms of tungro are confused with the symptoms of nitrogen or zinc deficiency. In case of zinc and nitrogen deficiency—uniform yellow discoloration of plants is there. But virus infected symptoms usually appear in isolated patches.
Tungro in the field
RTSV and RTBV
Predisposing Factors

• Presence of the virus sources.
• Presence of the vector.
• Age and susceptibility of host plants.
• Synchronization of the three above factors.
• All growth stages of the rice plant specifically the vegetative stage
Tungro disease of rice
Spherical virus

- RNA

transmission

Bacilliform virus

- DNA

symptoms

RTSV  +  RTBV

transmission and symptoms
Management of Tungro disease of rice

Management Strategy

• The use of resistant varieties- IR 36, IR 50, ADT 37, Ponmani, Co 45, Co 48, Surekha, Vikramarya, Bharani, IR 36 and white ponni.

• Eradication of tungro hosts- destroy stubbles.

• Elimination of vectors.
  - light traps
  - spraying Monocrotophos @ 1.6 to 2.2ml/litre of water

• Crop rotation.
Disease: Banded Leaf and Sheath blight of Maize
Pathogen: *Rhizoctonia solani* Kühn f. *sp.sasakii* Exner
Host: Maize (*Zea mays*)
Characteristic symptoms include **concentric bands and rings** on infected **leaves** and **sheaths** that are **discolored, brown, tan or grey** in color. The disease develops on leaves and sheaths and can spread to the ears causing **ear rot**.
Dull white to brown sclerotial formation is there on affected plant parts.
Predisposing Factors

• Warm - humid conditions
• Water logged conditions.
• High crop densities impact disease severity.
• Hybrids are much prone to the disease
Life cycle

**Rhizoctonia stunt**

*Rhizoctonia solani*

- Root infection weakens seedlings causing patches of poor growth
- Affected plants often remain stunted
- In autumn and spring sclerotia germinate producing infectious hyphae
- Overwinters in soil and on host debris as sclerotia and mycelium
Rhizoctonia solani survives in the soil and on infected crop debris as sclerotia or mycelium. Sclerotia are known to survive for several years in the soil. The fungi spread by water (flooding), irrigation, movement of contaminated soil, and plant debris. At the onset of the growing season, in response to favorable humidity and temperatures (15 to 35°C), fungal growth is attracted to freshly planted host crops by chemical stimulants released by growing plant cells. The fungi infect plants, leading to characteristic symptoms on the stem, sheaths, leaves and ears. The fungi overwinter as sclerotia or in infected crop debris.
Management Strategy

Host resistance
- Lack of resistant commercial varieties.
- Available tolerant germplasm should be cultivated.

Biological control
- *Trichoderma* bioformulations—through seed dressing and soil application

Cultural control
- Stripping of 2-3 lower leaves.
- Composting of hardwood.
- Fields should be well drained prior to planting.
- Seeds should be planted on raised beds
Northern maize leaf blight or Turcicum leaf blight of Maize

Disease: Northern leaf blight or Turcicum leaf blight of Maize

Pathogen:
Anamorph (asexual phase)
• *Exserohilum turcicum* or (syn. *Helminthosporium turcicum*)

Teliomorph (sexual phase)
• *Setosphaeria turcica*

Host: Maize
Lesions are **elliptical and tan** in color, developing distinct dark areas as the fungus sporulates. Lesions typically first appear on **lower leaves and spread upwards**. Under severe infection, lesions may coalesce, blighting the entire leaf.
Conidia are 3 - 8 septate, spindle-shaped and have a protruding hilum
Predisposing Factors

- Less warm and wet conditions
- Monoculture
- Reduced tillage
Disease cycle

Northern Leaf Blight Disease Cycle

Exserohilum turcicum (Helminthosporium turcicum)

- Secondary spread of conidia from leaf lesions
- Infection and symptom development
- Infected plant
- Conidia spread by wind, rain to leaves
- Fungus overwinters as mycelia and conidia in infected leaves, husks and other plant parts
Disease management

• The use of resistant varieties.
• General sanitation—Destruction of crop debris.
• Rotation with non-host species.
• Spray Zineb/Maneb @ 0.25% at 10 days intervals
Southern Leaf Blight of Maize

Disease: Southern Leaf Blight of Maize

Pathogen:
Anamorph (asexual phase)
• *Bipolaris maydis* [Syn. *Helminthosporium maydis*]

• Teliomorph (sexual phase) *Cochliobolus heterostrophus*

Host: Maize
Symptoms

There are 3 physiological races of *Bipolaris maydis*: Race T, Race O and Race C. Race O causes long, tan, lesions that have brown borders on leaves. Race T and race C are known to be specifically virulent to corn with cytoplasm male-sterile T and cytoplasm male-sterile C, respectively.
Lesions on the leaves caused by Race 0 of the fungus are elongated tan coloured between the veins up to one inch long.

Lesions produced by Race T (which was very prevalent in the early 1970s) are tan spindle-shaped or elliptical, with yellow-green or chlorotic halos. Later, the Race T lesions often have dark, reddish-brown borders and may occur on all parts of the plants.
Predisposing Factors

• Warm, temperatures (70 - 90° F) and wet conditions
• Monoculture
• Reduced tillage
Disease Cycle

- Wind and rain spread spores
- Infection and lesion development
- Spores produced in lesions
- Fungus overwinters in debris
- Spores produced in spring
- Wind and rain move spores to plant
- Infected plants
- Infection and lesion development
Disease management

- Use of resistant hybrids
- Crop rotation with non-host species
- General sanitation-Destruction of crop debris.
- Spray Zineb/Maneb @ 0.25% at 10 days intervals
Brown spot Of Maize

**Disease:** Brown spot of Maize

**Pathogen:** *Physoderma maydis*

**Host:** Maize
Symptoms and sign

Lesions occur mainly on the leaf, but may also occur on leaf sheath, stalks, outer ear husks and tassels producing small round-to-oblong a chocolate brown to reddish brown spots which may merge together to form large brown blotches. Spots on the mid-ribs are circular and dark brown.
Physoderma maydis sporangia
Brown spot is favored by
• high temperatures and
• high humidity.
Disease Cycle

• Overwintering **fungal mycelium** and **sporangia** in infected maize debris or soil.

• **Sporangia** germinate to produce infective **zoospores** under conditions of moisture and light.
Disease management

• Use of tolerant varieties
• Crop rotation, as the fungus survives in infected crop residue
• Good cultural practices
  - Sanitation
  -- proper spacing
Head Smut of Maize

- **DISEASE:** Head Smut
- **PATHOGEN:** *Sorosporum reilianum* (Syn. *Sphacelothecaraeliana, Ustilago reiliana*)
- **HOSTS:** Maize (*Zea mays*), teosinte (*Zea mexicana*)
Characteristic symptoms of Head Smut of Maize-ears and tassels may be replaced with smut sori filled with teliospores.
Teliospores
The fungus survives as thick-walled black teliospores in the soil produced by the head smut fungus. Plants are systemically infected as seedlings and head smut develops systemically into the ear and tassel (invades the meristematic tissue).
Disease Cycle and Epidemiology

The saprophytic budding stage can be maintained indefinitely on artificial media.

Mating and infection filaments are in nuclear cycle arrest, and subsequent development requires an appropriate host.

Young leaves and kernels are most commonly infected.

Growth in planta is intracellular at first, but in the tumor, most hyphae is intercellular. Tumors initially contain primarily host cells, but eventually fungal cells predominate.

Meiosis occurs as teliospores germinate, restoring the haploid, saprophytic stage.
The disease is soil borne. But seed can also carry teliospores.

- **Chemical**
  - Seed treatment: Systemic fungicides like Carboxin, Vitavax and Benlate @ 2.0 g/Kg seed and Tilt (propioconazole) 25EC @ 0.1%

- **Cultural.**
  - Rogue out –removal of smutted heads.

- **Resistance.** Use resistant cultivars or at least avoid very susceptible cultivars.
Common smut of corn (Syn. boil smut, blister smut)

- **DISEASE:** Common smut (Syn. boil smut, blister smut)
- **PATHOGEN:** *Ustilago maydis* (Syn. *Ustilago zeae*)
- **HOSTS:** Maize (*Zea mays*), teosinte (*Zea mexicana*)
Grayish smut galls develop on the stalks, ears and tassels, while smaller galls often appear on the leaves. The galls initially have a white membrane cover that eventually breaks and releases dark-brown or black powdery spores (teliospores) which are wind borne. **Common smut** incidence increases where plants have been **wounded**.
Young actively growing parts of the plant are susceptible to infection. Smutted plant parts - tumor-like galls are found most frequently on ears, tassels, stalks, nodal shoots, and mid-ribs of leaves.
Teliospores
Disease development is favoured by
• rain showers,
• high humidity
• and warm temperatures
• in conjunction with physical plant injury.
During spring and summer, the black globose or subglobose spiny diploid (2N) teliospores, survive in the soil, germinate to form a 4-celled basidium (promycelium) that produces oval - haploid (N) sporidia or basidiospores, which are of two sexes, (+) and (-). The sporidia are blown about by air currents or are splashed by water to young developing tissues of corn plants. Infection occurs when the + and - sporidia germinate to form fine hyphae that penetrate into corn tissue through stomata, wounds, or directly through cell walls and stimulates an increase in size and number of cells in
Disease management

• Use resistant hybrids or varieties.
• Rogue out infected plant parts.
• Avoid injury of roots, stalks, and leaves during cultivation.
DISEASE: Bacterial Stalk rot of Maize

PATHOGEN: *Erwinia chrysanthemi* pv. *zeae* (Syn. *Erwinia carotovora* - bacterium)

HOSTS: Maize
Symptoms are similar to those of *Pythium stalk rot*. The basal internodes develop **soft rot** and give a **tan or brown - water soaked** appearance. The **pith** of infected plants will **decay** and be **mushy or soft**. A mild sweet **fermenting odour** accompanies such rotting. Leaves some time show signs of wilting or water loss and affected plants within a few days of infection **lodge or topple down**.
Stalk Rot of Maize
Predisposing Factors

Conditions Favoring Disease

• High Temperatures (88-95°F)
• High amounts of rainfall and flooding
• Overhead irrigation
• Insect injury
The bacterium is able to survive on plant debris or in the soil. High humidity and free water favour spread and penetration of the bacteria. *Erwinia chrysanthemi* is a soft rot pathogen degrading succulent fleshy plant organs infecting plants through natural openings or wounds caused by heavy rain, high winds, hail damage and insect feeding. The bacterial ooze from the diseased plant is carried and transmitted by wind, rain and insects. Disease development is dependent on high temperatures, generally 25-30°C.
Disease management

- Use of disease resistance varieties, i.e. Hybrids Ganga Safed-2, DHM 103.
- Sanitation - removal of infected crop residues.
- Avoid waterlogging and poor drainage.
- Avoid excessive irrigation.
- Avoid injury to plant parts.
- Three application of bleaching powder (10%) @ 16.5 kg/ha at the time of sowing, earthing up and tesseling stage.
DISEASE: Downy mildew of Maize
PATHOGEN: Downy mildew fungi
Peronosclerospora philippinensis, P. sorghii, P. sacchari, P. maydis and Sclerophthora graminicola S. rayssiae
HOSTS: Maize
• Downy mildew of Maize is characterized by local lesions and systemic infection.
• All *Peronosclerospora* spp. induce both local and systemic infection.
• Chlorosis, white stripes/streaks, stunting with downy fungal growth on both leaf surfaces are the characteristic symptoms.
• Severe infection also incites blotching.
• Systemic infection can induce malformation of tassels, ears and other plant parts.
• Wilting generally starts from the top leaves; Leaves become dull green, eventually loose colour and become dry.
Brown stripe downy mildew of maize – *Sclerophthora rayssiae var. zeae*

*Sclerophthora rayssiae var. zeae* causes leaf lesions only. Lesions are initially interveinal and appear as chlorotic, brownish, or reddish stripes on the leaves.
Philippine downy mildew of maize
Peronosclerospora philippinensis

Young plants and seeds may be infected systemically. Malformation of vegetative or floral tissues.
Downy mildew of maize
Epidemiology

• Cool - humid weather i.e. high relative humidity with a temperature of 20-25° C favours the disease development.

• Zinc deficiency predisposes plants to infection.

• Collateral and wild hosts near maize field harbour the downy mildew fungi.
The fungus perpetuates in the form of oospores in the seed or soil or plant debris or sporangia on collateral hosts. The Oospores present in soil serve as source of primary infection. Secondary infection is about by sporangia producing zoospores formed on the lower surface of infected leaves.
Disease management

• General sanitation - The eradication of collateral and wild hosts along with maize debris.
• Seed treatment with Metalaxyl at 4 g/kg
• Spray of Mancozeb 2.5 g/l or Ridomil MZ at 2g/l is recommended.
• Use of resistant varieties like DMR 1, DMR 5 and Ganga 11.