PLANT PATHOLOGY

Introduction

- The health of plants is essential to mankind as they play an important role in balancing the ecosystem as producers.
- Plant pathology is study of disease causing agents, spread of the disease, symptoms, physiological effects, prevention and control of plant diseases.
- Malfunctioning process of plant due to continuous irritation by the pathogen or agent is called as disease.
- External manifestation of improper and invisible functioning of plant is called as symptom. Symptoms occur either singly or in combinations or sequence.
- Diseases caused by other living organisms to plants are called as infectious diseases. These organisms are mycoplasmas, viruses, bacteria, fungi, nematodes, protozoans, and parasitic flowering plants.
- The non-infectious diseases cause disorders in plants and caused by abiotic agents such as air, water, soil, frost, pollutants, mineral nutrients and adverse environmental conditions.
- Late Blight of Potato caused by Phytophthora infestans was responsible for the Great Irish Famine in 1845.
- Brown leaf spot of rice caused by Helminthosporium oryzae was responsible for the famous Bengal famine in 1942-43.
- In systemic disease the whole plant body is affected by the pathogen.
- In localised disease only certain parts of the plant body are affected by the pathogen.
- Organism that incites disease in plant is called as pathogen.
- The ability of host plant to resist the pathogen to cause disease is called as resistance.
- The capacity of pathogen to incite disease is called as pathogenecity.
- The series of events from infection to appearance of disease symptoms is called as pathogenesis.
- Ainsworth classification is followed to understand the systemic position of the pathogen.

PADDY BLAST

Host : *Oryza sativa*
Pathogen : *Pyricularia oryzae*

Division	: Eumycota
Sub division	: Deuteromycotina
Class	: Hypomycetes
Order	: Moniliales
Family	: Moniliaceae

Introduction

- It was first recorded in **China** in 1637.
- In India it was first reported in **Tanjore** district of Tamil Nadu.

Symptoms

- The **first** symptoms of the disease are seen in the leaves when the crop is young. It is known as **leaf blast**.
- Leaves show the formation of **brown flecks** which later transform into **spindle shaped spots** with pointed ends called as **lesions**.
- The **central portion** of the lesion is **dull greyish** or **pale green** and **water soaked** in appearance. The rim is **dark brown** in colour and the centre is **grey** or almost **straw coloured**.
- Same type of lesions are seen on leaf sheaths, nodes and culms.
- When dry the leaf sheath becomes **black** and **breaks** apart. The plant has bluish patches on the stem.
- At the time of **panicle emergence**, the neck shows the formation of **necrotic lesions** causing **neck rot**. This stage is known as **Neck blast** or **Black neck**. It is the **most serious phase** of the disease. Infected part has **fluffy mycelial growth**.
- If the neck is infected before the emergence of inflorescence, the panicle stands erect. If it is infected after the emergence of inflorescence, the panicle hangs down. The panicle breaks and falls off.

Causal organism

- The fungus *Pyricularia oryzae* causes it.
- Its mycelium is branched filamentous, septate, **hyaline** or **olive** coloured and intercellular.

- It is present in the **lesions** of infected plant part.
- Its **conidiophores** emerge from the stomata or by rupturing the cuticle.
- **Conidiophores** are simple or rarely branched, **2 – 4 septate** with olive colour.
- Conidia are produced terminally on the conidiophores.
- Each conidium is **3-celled, pyriform, obclavate**, hyaline to **pale olive** in colour.
- It reproduces sexually in culture. Sexual stage is known as *Magnaporthe grisea*.
- It produces a toxin known as Piricularin.

Disease cycle

- The pathogen survives in the form of mycelium and conidia on diseased straw, seed and weed hosts.
- The sources of **primary infection** are **straw and debris**, **seed** and **collateral hosts**.
- The mycelium **surviving in the straw** infects the plant at **seedling stage**.
- The pathogen present in the **seed** infects the plant during **seed germination**.
- The hosts other than the usual host on which the pathogen can complete its life cycle are called as **Collateral hosts**. The conidia produced on these hosts serves as inoculum for the next season. Such inoculum can infect the rice plant at any stage. The collateral hosts are *Panicum repens*, *Digitaria marginata*, *Setaria intermedia*, *Brachiaria mutica* and *Leersia hexandra*.
- 90% or above relative humidity and **20 – 25^{o}C** temperature are the favourable conditions for the production and release of conidia.
- The conidia are dispersed through wind, germinate on **wet surface** and enter the host through **stomata**.
- Younger plants are more susceptible than older plants.
- In favourable conditions, the lesions appear within **4 to 5 days**.
- The conidia are produced and released within **few hours** of appearance of primary lesions and start infecting the new plant parts or new host plants.
- Conidial release between **midnight** and **sunrise** is continued for several days.
- These cause secondary infections.
- The fungus perennates in the form of **Chlamydospores** and **Sclerotia**.
- High **nitrogen fertilizers**, **prolonged wetness**, **90% or more relative humidity** and **night temperatures around 20^{o}C** favour the development of disease.

Control measures

- **Using disease resistant and tolerant varieties**: Paddy varieties must be changed periodically. Varieties with high silicon content in their leaves are resistant to this disease.
- **Early planting and low nitrogen fertilizer usage**.
- **Chemical Control:** Spraying **fungicides** such as Mancozeb, Cerasan and organophosphates and **antibiotics** and tricyclazole.
- **Field Sanitation:** The infected plants or their parts should be destroyed by burning.
- **Destroying the collateral hosts** minimises the incidence of this disease.
- **Seed treatment** with **Agrosan GN** eliminates the pathogen.

RED ROT OF SUGARCANE

Host : *Saccharum officinarum*
Pathogen : *Colletotrichum falcatum*

Division	: Eumycota
Sub division	: Deuteromycotina
Class	: Coelomycetes
Order	: Melanconiales
Family	: Melanconiaceae

- This disease caused severe loses in India during **1939 – 42** in **Bihar** and **Uttar Pradesh** and remained **endemic**.
- It causes heavy loses to both **cultivators** and **millers**.

Symptoms

- It is difficult to recognise the disease in the field during early stages of the crop.
- First symptoms are seen **after rainy season** when the **vegetative growth** of the plant is stopped and **sucrose** formation begins.

- **Discolouration** of the leaves is the **first symptom** in the field. Drooped leaves show the withered margins and tips. This discolouration from tip to the base is continued till all the leaves of the crown wilt.
- Elongated **dark lesions** appear at the mid rib regions. They later turn **blood red** with dark margins.
- The infected canes are shrivelled and **longitudinally wrinkled** with sunken rind.
- Infected canes are **light in weight** and easily **broken**.
- Longitudinally split open infected stems have **reddened pith** with **transverse white bands** showing the mycelium. In severe cases the reddened region is **dirty brown** in colour. Cavities have **white** or **greyish** mycelium.
- Minute velvety **dark dots** of the fungus are formed near the nodes of the diseased canes or in the shrunken areas.
- Juice of the infected canes emits **alcoholic smell** and does not set well upon boiling due to conversion of sucrose to **glucose** and **alcohol**.

Causal Organism

- It is *Colletotrichum falcatum*. Its mycelium is both **intracellular** and **intercellular**.
- Hyphae are thin, septate, **hyaline** and profusely branched.
- Asexual fruiting bodies known as **Acervuli** are minute, velvety and formed on the surface of the host part.
- Acervulus has a mat of vegetative hyphae of the mycelium formed below the epidermis of host plant known as **Stroma.** The fertile layer formed above it is known as **Hymenium**. Hymenium has long, rigid, septate, bristle like **Setae** with dark brown basal region and light coloured apex. Setae rupture the epidermis and the conidiophores of the hymenium are exposed.
- The **conidiophores** are hyaline, **linear** or **club-shaped** and present between the setae. They produce conidia at their tips.
- Each conidium is single celled, thin walled, uninucleate, colourless, **sickle shaped** (Falcate) and have granular protoplasm with a large **oil globule**.
- The fungus also produces thick walled, greenish black **Chlamydospores**.
- The sexual stage is known as *Glomerella tucumanensis*. It is responsible for the survival of the fungus on decaying leaves and formation of new pathological races.

Disease Cycle

- **Primary infection** source is either the **infected canes** used as sets or the pathogen present in the soil on the plant debris.
- **Secondary infection** takes place through wind, water and agricultural implements.
- Pathogen present in the soil enters into the host through cut ends of the cane **sets**, **leaf scars** and **root primordia**. Conidia germinate only in the presence of water.
- Acervuli appear during the **later stages** of development of disease. Conidia are released and spread only when the **acervuli** are wet. **Conidia** are short lived. These conidia produced on infected plants serve as **secondary infection** inoculum and cause infections to other healthy plants of the same field or other parts of the same plant.
- The fungus reproduces sexually by the formation of **perithecia**. Perithecia have **asci** in which ascospores are formed. Sometimes the ascospores act as primary source of inoculum.
- The favourable conditions for disease development are 1. **High atmospheric humidity**, 2. **Water-logged** conditions of the soil, 3. **Lack** of **cultural practices** that result in the growth of weeds, 4. Continuous cultivation of same variety in the field and 5. Presence of **susceptible varieties** in the surroundings.

Control Measures

- **Seed-sets** must be selected from disease free areas.
- **Deep tillage** to burry the plant debris of the crop.
- **Field sanitation** by removing and burning the diseased plants and their parts.
- **Crop rotation** by growing some other crop for **2-3** years to minimise the soil borne inoculum.
- **Seed treatment** by the dipping the seed-sets in fungicide such as **Bordeaux** mixture.
- Complete elimination of infection through roots by dipping the sets in **Benomyl** or **Aretan** for **18 minutes** at **52°C**.
- **Pest control** to avoid wounds to canes.
- Avoiding **Ratooning** in endemic conditions of the disease. (Development of new shoots from the cut sugarcane stems left over in the field after harvesting is known as Ratooning).
- Using **disease resistant** varieties.

GRAIN SMUT OF SORGHUM

Division : Eumycota
Sub-division : Basidiomycotina

Class : Teliomycetes
Order : Ustilaginales
Family : Ustilaginaceae

- It is also known as **Kernel smut, Covered smut** and **Short smut**.
- It causes upto **95%** decrease in the yield.

Symptoms

- Disease symptoms are not seen in the **vegetative stage** of the plant.
- Symptoms are seen at the time of **emergence of inflorescence** and grain formation.
- Either a **few or most** of the grains are infected depending on the susceptibility of the variety.
- The grains are converted to **ash or black** coloured spore sacs called **sori**. These infected grains are **larger** than healthy grains. The spore sac is surrounded by the glumes at the base.
- Usually stamens are not developed or converted into **smut sori**. Stigma is not transformed into smut sorus. Spores are **not formed** in other parts of the host.
- Sorus is **oval** or **conical** in shape and consists of central hard **columella** made of host tissue with **fibrovascular** bundles. Columella may be **branched** and exposed after the liberation of **smut spores**. The wall of the sorus is made of **fungal hyphae** forming **pseudoparenchyma**. The wall is broken at the time of threshing the ear and the smut spores are liberated.
- The infected plants have **few and shorter** internodes.

Causal organism

- It is *Sphacelotheca sorghi*. Its mycelium is branched filamentous, septate and grows **intercellular** and **intracellular** manner in the host tissue.
- Cells of the mycelium are **dikaryotic** and consist of one **+stain** and one **–strain** nucleus.
- Though the mycelium is distributed throughout the plant body, it forms spores only in the **ovary** or **anther**, which become as smut sori.
- The spores are **dark brown** coloured or **black** coloured and **binucleate**. These spores are known as Chlamydospores or Teleutospores.
- Each smut spore wall is thick and consists of outer thick **exospore** and inner thin **endospore**. The spores either germinate immediately when water is available or remain dormant for many years in **dry** conditions.

Disease Cycle

- It is externally seed borne disease. The smut spores are adhered to the grains during **threshing** and remain dormant till the grains germinate.
- During germination, the two nuclei of the **teleutospore** unite and form **diploid** nucleus. This diploid nucleus undergoes meiosis and forms four haploid nuclei.
- During this, the **exospore** ruptures and the inner endospore comes out in the form of a tube like structure which is four nucleated and **3-septate**. This is known as **promycelium**.
- Promycelium produces **uninucleate**, **spindle** shaped structures known as **sporidia** or **basidiospores**.
- The **sporidia** or **basidiospores** germinate and form monokaryotic mycelium that invades the germinating seed. Some times the **promycelium** directly infects the seedling through **mesocotyl**.
- The monokaryotic + and – stain mycelia unite and form dikaryotic cells. These cells divide and form dikaryotic mycelium that completely **invades** the plant body. The tips of the mycelium are present in the **apical meristem** of the plant and infect the flower to produce the **smut sori**.
- Symptoms start appearing only during seed setting stage.

Control measures

- **Seed treatment**: It effectively controls the disease because it is externally **seed-borne**. Seeds are soaked in **0.5% Formalin** solution for **2 hours** and dried immediately. Seeds can be soaked in **0.5 – 3% copper sulphate** solution for **10 – 15 minutes**, dried and sown. **2-4 gm of Sulphur, Organic mercurials, thiram or captan** found to be effective seed treatment.
- **Crop Rotation**: Crops on which the pathogen cannot survive must be grown for a few years.
- **Hot water treatment**: The seeds are soaked in water for **4 – 5** hours. Later the temperature of the water is raised to **55 – 58°C** for about **10** minutes, which kills the pathogen. Afterwards the grains are dried in hot sun for **5 – 6** hours.
- **Disease resistant** varieties must be used.

CITRUS CANKER

- This disease reduces the **fruit quality** and causes **premature fruit drop**.

- It was introduced into Florida, **USA** from **Japan**. This disease is eradicated from **South Africa, Australia** and **New Zealand**.
- Strict **quarantine** laws are maintained to import citrus plants and fruits.
- In our country it was reported from **Andhra Pradesh**, **Uttar Pradesh**, **Karnataka, Tamil Nadu, Punjab** and **Rajasthan**.

Symptoms

- Disease symptoms are seen on all **aerial parts** of the plant and roots.
- The lesions appear on the **lower surface of the leaves** as **small, slightly raised, round, water soaked greyish white spots** (cankers). These later spread to the upper surface. Greyish white lesions **enlarge**, **rupture** and become **brown** with **crater** like depression in the centre. The lesions become corky and the margins of these brown lesions are surrounded by **yellow halo**.
- Similar symptoms are seen on the fruits without halo and prominent crater like depression. It does not affect the **pulp** or **juice**.
- Lesions of fruit and leaves unite and form **bigger lesions** with up to 1cm diameter.
- In severe conditions, the plant becomes stunted; twigs and branches become weak and show early defoliation.
- In old branches, the necrotic lesions are irregular, **rough** and **more prominent**.

Causal Organism

- It is a bacterium known as *Xanthomonas axonopodis citri* (X. **campestrix** pv. **citri**).
- It is short, **rod – shaped**, **gram –negative**, **aerobic** and **monotrichous**.
- It measures 1.5 – 3.0 x 0.5 – 1.5 μ.

Disease Cycle

- This bacterium does not survive in the soil or on the fallen plant parts.
- Lesions of infected old leaves, twigs and fruits are the **source of infection**.
- In rainy and warm conditions, the bacteria come out and spread the infection.
- It enters the fresh host through **stomata** or wounds.
- It multiplies in the intercellular spaces, dissolves the middle lamellum and establishes in the cortex and mesophyll of host tissue.
- Later cankerous outgrowths develop in which the **bacteria** multiply. These bacteria are oozed out with sticky exudations that attract the insects.
- Wind, water, air and insects disseminate the disease. The rainwater and wind borne rains splash the bacteria from lesions.
- **20–35^{o}C** temperature is essential for the infection.

Control measures

- **Exclusion of pathogen:** Entry of pathogen must be prevented by strictly following quarantine laws.
- **Eradication of Pathogen:** The infected plants with adjacent trees and pruned infected parts must be burnt before monsoon.
- **Chemical control:** One of the chemicals such as **Copper oxychloride, Phytomycine, Streptomycin** sulphate and **Streptocycline** must be sprayed once in a fortnight. These chemicals must be sprayed on fruits when they are about marble size to prevent fruit infection and another time after **30 days**.
- **Neem cake** solution is also effective to control this disease.
- **Cultivation of disease resistant** varieties.