1 & 2	**Rice: economic importance, symptoms, cause, disease cycle and integrated management- blast, brown spot, sheath rot , stem rot, sheath blight, false smut, bacterial leaf blight, bacterial leaf streak and tungro**
3 & 4	**Wheat- economic importance, symptoms, cause, disease cycle and integrated management- black stem rust, brown rust and yellow rust, loose smut and *Karnal* bunt, leaf blight, soil borne mosaic and tundu disease / yellow slime disease**
5	**Pearl millet- economic importance, symptoms, cause, disease cycle and integrated management- downy mildew, rust, sugary disease and smut,** **Finger millet- blast, smut and mosaic**
6	**Sorghum- economic importance, symptoms, cause, disease cycle and integrated management- anthracnose, rust, sugary disease, grain molds, leaf blight, grain smut, charcoal rot, downy mildew and *Striga***
7 & 8	**Maize- economic importance, symptoms, cause, disease cycle and integrated management- turcicum leaf blight, post flowering stalk rot and *Cephalosporium* wilt, charcoal rot, banded leaf and sheath blight and downy mildew, Yellows disease**

Lecture 1&2 Rice diseases – blast, brown spot, Sheath rot, Stem rot, narrow brown leaf spot.

sheath blight, False smut, Bacterial leaf blight, Bacterial leaf streak, Rice Tungro Disease,

DISEASES OF RICE *(ORYZA SATIVA)*

Blast

Pyricularia oryzae (Syn: P. grisea) (Sexual stage: Magnaporthe grisea)

The disease was first recorded in China in 1637. In Japan, it is believed to have occurred as early as in 1704. In Italy the disease called "**brusone**" was reported in 1828 and in USA in 1876. The disease was first recorded from Tanjore district of Tamil Nadu in 1918.

Economic importance: The pathogen cause yield loss ranging from 30-61 per cent depending upon the stage of infection. In severe cases, losses amounting to 70-80 per cent of grain yield are reported.

Symptoms

The fungus attacks the crop at all stages from seedlings in nursery to heading in main field. The typical symptoms appear on leaves, leaf sheath, rachis, nodes and even the glumes are also attacked.

- **Leaf blast:** On the leaves, the lesions start as small water soaked bluish green specks, soon enlarge and form characteristic **spindle shaped or diamond shaped**

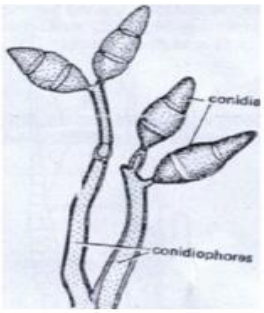
spots with grey centre and dark brown margin. The spots join together as the disease progresses and large areas of the leaves dry up and wither. Similar spots are also formed on the sheath. Severely infected nursery and field show a **burnt** appearance.

- **Node blast:** In infected nodes, irregular black areas that encircle the nodes can be noticed. The affected nodes may break up and all the plant parts above the infected nodes may die **(Node blast).**
- **Neck blast:** At the flower emergence, the fungus attacks the peduncle which is engirdled, and the lesion turns to brownish-black. This stage of infection is commonly referred to as rotten neck/neck rot/neck blast/panicle blast. **In early neck infection, grain filling does not occur and the panicle remains erect like a dead heart caused by a stem borer.** In the late infection, partial grain filling occurs. Small brown to black spots also may be observed on glumes of the heavily infected panicles.

LEAF BLAST

NODE BLAST

Partial grain filling in dead heart like panicle



NECK BLAST

3 CELLED PYRIFORM CONIDIA

Etiology

The causal organism was first detected by **Cavara** in 1891 from Italy. **Mycelium** of the fungus, is hyaline to olivaceous, septate and highly branched. Conidia are produced in clusters on long septate, olivaceous slender conidiophores. Conidia are **pyriform** to obclavate or somewhat top shaped, attached at the broader base by a hilum. Conidia are

hyaline to pale olive green, usually **3 celled**. The perfect state of the fungus is *M. grisea.* It produces perithecia. The ascospores are hyaline, fusiform, 4 celled and slightly curved. The pathogen produces few toxins namely, **α-picolinic acid, Pyricularin and pyriculol.**

Disease cycle

P.I:Mycelium and **conidia** in the infected straw and seeds are important sources of primary inoculum. The seed borne inoculum fails to initiate the disease in the plains due to high soil temperature in June. In both tropical and temperate regions, the fungus overwinters in straw piles or grain. In tropics, one method of survival is through infection of collateral hosts such as *Panicum repens, Digitaria marginata, Brachiaria mutica, Leersia hexandra, Dinebra retroflexa, Echinochloa crusgalli, Setaria intermedia* and *Stenotaphrum secondatum.* The most probable source of perennation and initiation of the disease appear to be the grass hosts and early sown paddy crop.

S.I: The disease cycle is short and most damage is caused by secondary infections. Air can carry the conidia for long distances. The **conidia** from these sources are carried by air currents to cause secondary spread. Most conidia are released at night in the presence of dew or rain.

Favourable Conditions

Application of excessive doses of nitrogenous fertilizers, intermittent drizzles, cloudy weather, high relative humidity (93-99 per cent), low night temperature (between 15-20 ^{0}C or less than 26 ^{0}C), more number of rainy days, longer duration of dew, cloudy weather, slow wind movement and availability of collateral hosts.

Forecasting

Forecasting blast of rice can be made on the basis of minimum night temperature range of 20-26 ^{0}C in association with a high relative humidity range of 90 per cent and above lasting for a period of a week or more during any of the three susceptible phases of crop growth, viz., seedling stage, post transplanting tillering stage and neck emergence stage. In Japan, the first leaf blast model was developed and named as **BLAST**. Later based on different field experiments various models were developed namely, PYRICULARIA, PYRIVIEW, BLASTAM, and P BLAST. A model to forecast the disease called **"Epi- Bla"** has been evolved in India.

Management

- Use of seeds from a disease free crop
- Growing resistant varieties like Simhapuri, Tikkana, Sriranga, Phalguna, Swarnadhan, Swarnamukhi, MTU 7414, MTU 9992, MTU 1005, Swathi, IR 64, IR 36, Sravani, Jaya, Vijaya, Ratna, RP 4-14, IET 1444, IR20, TKM 6, MTU-3 & 5 and NLR 9672 & 9674 in different tracts of Andhra Pradesh.
- **Pusa Basmati 1847, Pusa Basmati 1885 and Pusa Basmati 1886 were developed by IARI in 2021with inbuilt resistance to bacterial blight and blast disease**
- Removal and destruction of the weed hosts in the field bunds and channels.
- Split application of nitrogen and judicious application of nitrogenous fertilizers
- Treating the seeds with Tricyclazole at 2 g/kg.
- Seed treatment with biocontrol agent *Trichoderma asperellum*@ 10g/kg or *Pseudomonas fluorescens* @ 10g/kg of seed. Avoid close spacing of seedlings in the main field.
- Spray the main field with **Tricyclazole** @0.06% or **kasugamycin or biomycin**@0.2%
- Spraying the crop in the main field twice with trifloxystrobin+tebuconazole@350ml/ once

  for every 10-day interval at 15 DAT until the 50% heading stage.

Brown Spot or Sesame leaf spot or Helminthosporiose *Helminthosporium oryzae* (Syn: *Drechslera oryzae*) (Sexual stage: *Cochliobolus miyabeanus)*

In India, this disease is the principal cause of **Bengal famine of 1942-43**. The first report of the disease in India was made by Sundararaman from Madras in 1919, and now is reported from all of the rice growing states. Under highly favourable conditions, the disease causes a reduction in yield ranging upto 90 per cent.

Symptoms

The fungus attacks the crop from seedling in nursery to milk stage in main field. Symptoms appear as lesions (spots) on the coleoptile, leaf blade, leaf sheath, and glumes, being most prominent on the leaf blade and glumes. The disease appears first as minute brown dots, later becoming cylindrical or **oval to circular**. The several spots coalesce and the leaf dries up. The seedlings die and affected nurseries can often be recognized from a distance by their brownish scorched appearance. Dark brown or black spots also appear on glumes which contain large number of conidiophores and conidia of the fungus. It causes failure of seed germination, seedling mortality and reduces the grain quality and weight. The disease is associated with a physiological disorder known as **akiochi in Japan**. Abnormal soil conditions (Deficiency of potassium) predispose the plants to heavy infection.

Fusiform 6-14 septate golden brown conidia

Etiology

H. oryzae produces greyish-brown to dark brown septate mycelium. Conidiophores may arise singly or in small groups. They are straight, sometime **geniculate**, pale to brown in colour. Conidia are usually fusiform,curved with a bulge in the centre and tapering towards the ends occasionally almost straight, pale olive green to golden brown colour and are 6-14 septate. The perfect stage of the fungus is *C. miyabeanus.* It produces

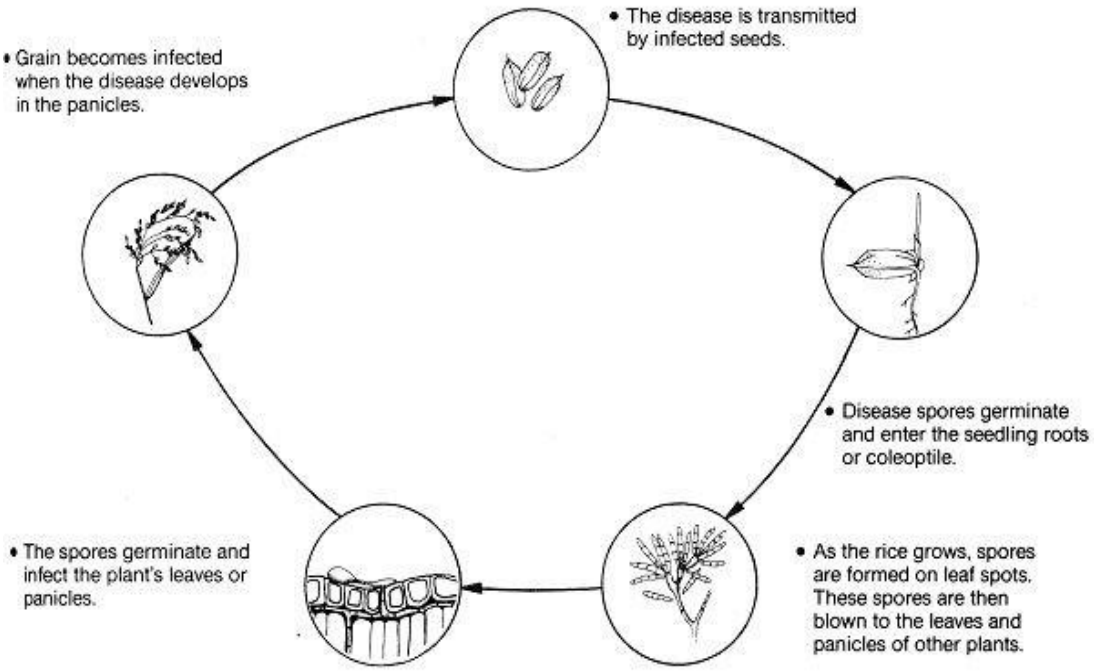
perithecia with asci containing 6-15 septate, filamentous or long cylinderical, hyaline to pale olive green ascospores. It produces C25 terpenoid phytotoxins called **ophiobolin A**, (or Cochliobolin A), **ophiobolin B** (or cochliobolin B) and **ophiobolin I**. Ophiobolin A is most toxic. These result in the breakdown of the protein fragment of cell wall resulting in partial disruption of integrity of cell.

Disease cycle

P.I: The fungus overwinters mainly in the infected plant parts. It is not soil borne. The infected seeds are the most common source of primary infection. Diseased seeds(externally seed borne) may give rise to the seedling blight, the first phase of the disease. The young seedlings show infection symptoms soon after germination. Pale yellowish- brown spots appear on the coleoptiles, spreading to cover the other tissues of the seedling. The fungus reproduces on the spots and is disseminated by air currents. The fungus also survives on collateral hosts like *Digitaria sanguinalis, Leersia hexandra, Echinochloa colonum, Pennisetum typhoides, Setaria italic*a and *Cynodon dactylon*.

S.S.I: Wind borne conidia

The symptoms of potassium deficiency are somewhat similar to that of brown spot making it often difficult to ascribe the symptoms to fungus attack or nutrient deficiency.



Favourable Conditions

Temperature of 25-30^{0}C with relative humidity above 80 per cent are highly favourable. Excess of nitrogen aggravates the disease incidence.

Management

- Use of disease free seeds.
- Field sanitation-removal of collateral hosts and infected debris in the field.
- Crop rotation,
- Adjustment of planting time
- Proper fertilization
- Use of slow release nitrogenous fertilizers is advisable.
- Good water management
- Use of soil amendments
- Grow disease tolerant varieties *viz.,* Bala, BAM 10, IR-20, Jaya, Ratna, Tellahamsa and Kakatiya.
- Spraying **aureofungin@0.005%**
- Spraying the crop in the main field twice with trifloxystrobin+tebuconazole@350ml/ once

  for every 10-day interval at 15 DAT until the 50% heading stage.

Sheath rot

Sarocladium oryzae

(Syn: *Acrocylindrium oryzae)*

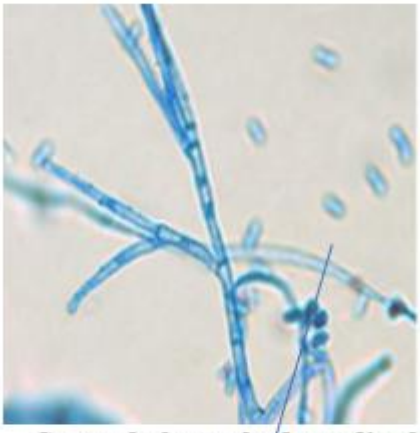
Economic importance

Sheath rot was first described in Taiwan in 1922. It is reported in all countries in South Asia. In A.P, sheath rot was found to be severe in Godavari delta, Nellore and Chittoor. 10 to 25% tillers may occasionally be infected.

Symptoms

Sheath rot occurs usually at the **booting** stage of the crop. Initial symptoms are noticed only on the upper most leaf sheath enclosing young panicles. The flag leaf sheath show oblong or irregular greyish brown spots. They enlarge and develop grey centre and brown margins covering major portions of the leaf sheath. The young panicles may remain within the sheath or emerge partially. The affected sheath and panicles rot and abundant whitish powdery fungal growth is formed inside the leaf sheath. The grain discolours and shrivels.

Oblong or irregular greyish brown spots on leaf sheath



Sparsely branched condiophore with cylindrical conidia

Etiology

The fungus produces whitish, sparsely branched and septate mycelium. Conidiophore is slightly thicker than the vegetative hyphae. Conidia are **hyaline**, smooth, **single celled** and cylindrical in shape.*Sarocladium oryzae* produces **phytotoxins helvolic acid and cerulenin**

Favourable Conditions

Closer planting, high doses of nitrogen, high humidity and temperature around 25-30^{O}C. Injuries made by leaf folder, brown plant hopper and mites increase infection.

Disease cycle

P.I: Externally seedborne

S.SI: Mainly through **wind borne conidia**

Management

- Applying recommended doses of fertilizers.
- Adopting optimum spacing.
- Spraying twice with Carbendazim 12% + Mancozeb 63% @0.2% or tebuconazole+ trifloxystrobin @0.1% at boot leaf stage and 15 days later.
- Soil application of gypsum in 2 equal splits (500 kg/ha) reduce the sheath rot incidence.

Stem rot

C.O.Sclerotium oryzae

*Sclerotium hydrophilum(*Nellore and few places)

(Sexual stage: *Magnaporthe salvinii)*

Economic importance

Stem rot was reported in Japan in 1910 and in India in 1913. Early reports indicated heavy losses from stem rot. In India 18 to 56% loss was reported. IRRI studies show

that the stem rot fungus is a wound parasite. Due to the damage caused by injuries the disease incidence initiates.

Symptoms

Small black lesions are formed on the outer leaf sheath near the water line and they enlarge and reach the inner leaf sheath also. The affected tissues rot and abundant sclerotia are seen in the rotting tissues. The culm collapses and plants lodge. If the diseased tiller is opened, profuse mycelial growth and large number of sclerotia can be seen. The sclerotia may be seen in the stubbles after harvest.

Black lesions on leaf sheath **Sclerotia in rotting tissue**

Etiology

White to greyish hyphae produces smooth, spherical black and shiny **sclerotia**, visible to naked eyes as black masses.

Disease cycle

P.I: In the field, sclerotia are mostly distributed in the upper 5 to 10 cm of the soil. These sclerotia float on the water during ploughing, puddling, weeding and other operations. Propagules in contact with the leaf sheath produce appressoria and may start infection. Infection takes place readily in the presence of a wound. After harvest the fungus continues to grow on stubbles producing large quantities of sclerotia.

S.SI: Irrigation water carries the sclerotia to other fields.

Favourable Conditions

Infestation of leaf hoppers and stem borer and high doses of nitrogenous fertilizers aggravates the disease.

Management

- Use of recommended doses of fertilizer.
- Deep ploughing in summer and burning of stubbles and infected straw
- Use of resistant or non-lodging varieties (Basumati 3, Basumati 370, Mushkan 7, Mushkan 41 and Bara 62 were found resistant to stem rot in Punjab)

- Draining off the irrigation water and allow the soil to dry
- Avoiding flow of irrigation water from infected fields to healthy fields.
- Spraying Captan 70% + Hexaconazole 5%@750 g/ha is effective

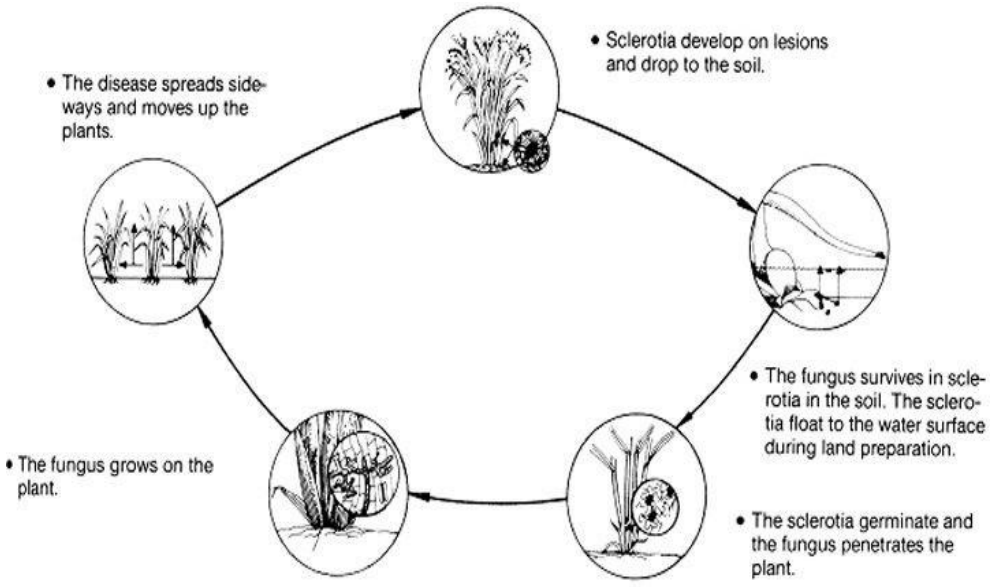
Sheath blight

Rhizoctonia solani

(Sexual stage : *Thanetophorus cucumeris)*

Symptoms

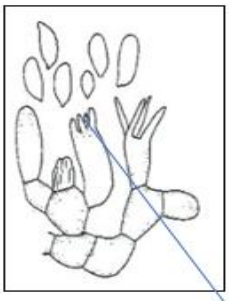
The fungus affects the crop from **tillering** to **heading** stage. Initial symptoms are noticed on leaf sheaths near water level. On the leaf sheath oval or elliptical or irregular greenish grey spots are formed. As the spots enlarge, the centre becomes greyish white with an irregular blackish brown or purple brown border. Lesions on the upper parts of plants extend rapidly coalesing with each other to cover entire tillers from the water line to the flag leaf. The presence of several large lesions on a leaf sheath usually causes death of the whole leaf, and in severe cases all the leaves of a plant may be blighted in this way. The infection extends to the inner sheaths resulting in death of the entire plant. Older plants will be highly susceptible. Five to six weeks old leaf sheaths are highly susceptible. Plants heavily infected in the early heading and grain filling growth stages



produce poorly filled grain, especially in the lower part of the panicle.

Oval or elliptical or irregular greenish grey spots on leaf sheath

Hypha arising at right angles



Basidia and basidiospores of *Thanetophorus cucumeris*

Pathogen

The fungus produces usually long cells of septate mycelium which are hyaline when young, yellowish brown when old. It produces large number of globose **sclerotia**, which are initially white, later turn to brown or purplish brown.

Disease cycle

P.I: The pathogen can survive as sclerotia or mycelium in dry soil for about 20 months but for 5-8 months in moist soil. It infects more than 188 crop species in 32 families.

S.SI: Sclerotia spread through irrigation water.

Favourable Conditions

High relative humidity (96-97 per cent), high temperature (30-32 ^{0}C), closer planting and heavy doses of nitrogenous fertilizers.

Management

- Avoid excess doses of fertilizers.
- Adopt optimum spacing.
- Eliminate weed hosts.
- Apply organic amendments.
- Avoid flow of irrigation water from infected fields to healthy fields.
- Deep ploughing in summer and burning of stubbles.
- Grow disease tolerant varieties like **Shiva** (WGL 3943)
- Spray Propiconazole@0.1% or Hexaconazole@0.2% or **Validamycin@0.2%**
- Seed treatment with *Pseudomonas fluorescens* @ of 10g/kg of seed followed by seedling dip @ of 2.5 kg of product/ha dissolved in 100 litres and dipping for 30 minutes.
- Soil application of *P.fluorescens* @ of 2.5 kg/ha after 30 days of transplanting (This product should be mixed with 50 kg of FYM/Sand and then applied.
- Spray Propiconazole@0.1% or Hexaconazole@0.2% or Validamycin@0.2% from 45 days after transplanting at 10 days interval for 3 times depending upon the intensity of disease. Trifloxystrobin 25% + Tebuconazole 50% (Nativo 75 WG) @ 0.4 g/l performed better in reducing the sheath blight

False smut

C.O:Ustilaginoidea virens

(P.S: *Villosclava virens*)

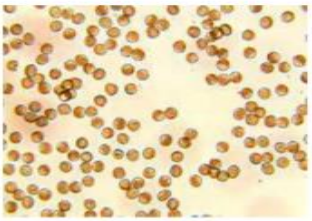
Economic importance

Most countries in Asia, Latin America and Africa have reported the presence of the disease. There was severe epidemic in Burma in 1935. Its presence was believed to indicate a good crop year. This belief is still common in South-east Asia.

Symptoms

The fungus transforms individual grains into yellow or greenish spore balls of velvety appearance which are small at first and 1 cm or longer at later stages. At early stages the spore balls are covered by a membrane which bursts with further growth. Due to the development of the fructification of the pathogen, the ovaries are transformed into large velvety green masses. Usually only a few spikelets in a panicle are affected.

Individual grains are transformed into yellow or greenish spore balls of velvety appearance



Chlamydospores (spherical to elliptical, waxy and olivaceous) on spore balls

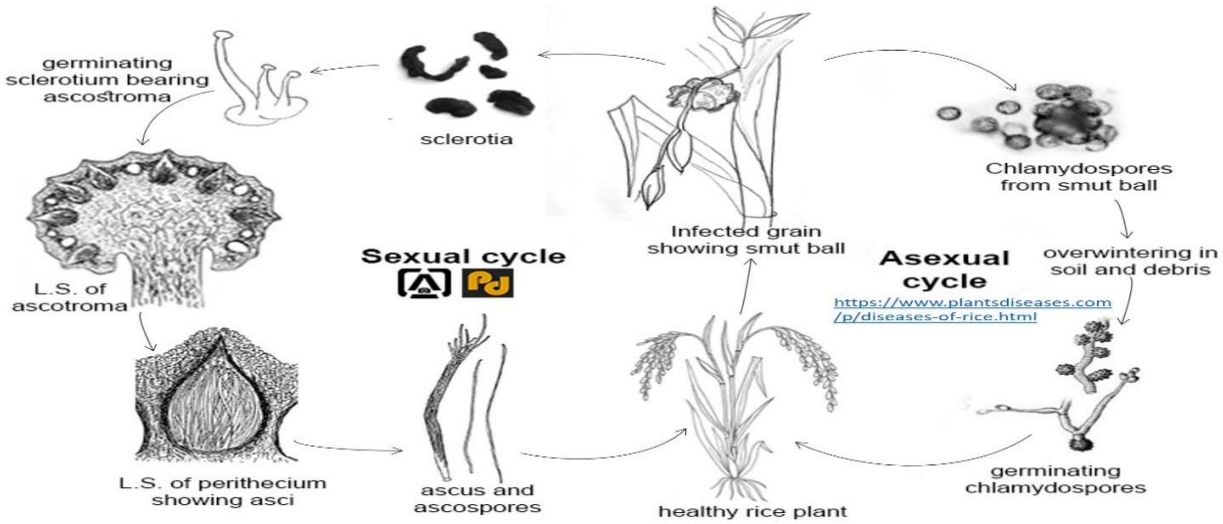
Etiology

Chlamydospores are formed on the spore balls, they are spherical to elliptical, waxy and olivaceous.*Villosclava virens* is an ascomycete produced from ascostroma produced from germinating sclerotia of *Ustilaginoidea virens.* The *U. virens* could produces large amounts of mycotoxins, the **ustilotoxins**

Disease cycle

P.I:In temperate regions, the fungus survives the winter through sclerotia as well as through chlamydospores. Ascospores produced on the over wintered **sclerotia** apparently start primary infection.

S.S.I: Chlamydospores are important in secondary infection which is a major part of the disease cycle. Infection usually occurs at the booting stage of rice plants. **Chlamydospores** are air borne, but do not free them from spore ball easily because of the presence of sticky material.



Favourable conditions

Rainfall and cloudy weather during the flowering and maturity periods are favourable.

Management

- Spray copper oxychloride@0.3% or mancozeb63%+carbendazim12%@0.2% at panicle emergence stage

Bacterial leaf blight

Xanthomonas oryzae pv. oryzae

Economic importance

The disease was first observed in Japan (1884). In Indonesia, **Kresek** disease was reported to kill young seedlings completely in 1950. In India, BLB was first reported in 1959. A severe outbreak of the disease occurred in Bihar and Uttar Pradesh in 1963. In the tropics the disease is usually referred as bacterial blight as it often kills entire young seedlings

Yield losses in severely diseased fields range from 20-30% and occasionally 50%. In India, millions of hectares are infected every year. Yield losses have been as high as 60% in some states and Godavari district of Andhra Pradesh which are endemic to this disease. **Taichung Native 1** is highly susceptible.

Symptoms

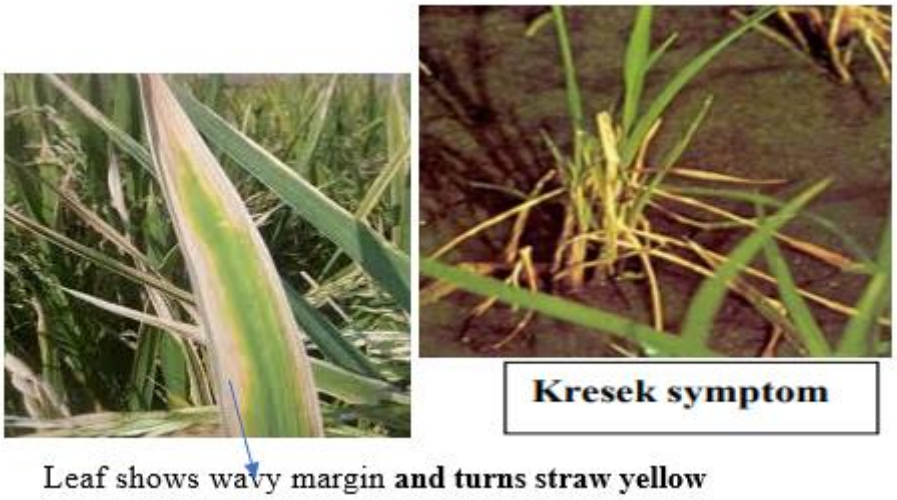
The bacterium induces either wilting of plants or leaf blight. Wilt syndrome known as **Kresek** is seen in seedlings within 3-4 weeks after transplanting of the crop. Kresek results either in the death of whole plant or wilting of only a few leaves. The bacterium enters through the hydathodes and cut wounds in the leaf tips, becomes systemic and cause death of entire seedling.

The disease is usually noticed at the time of heading but in severe cases occur earlier also. In grown up plants water soaked, translucent lesions appear usually near the leaf margin. The lesions enlarge both in length and width with a **wavy margin** and turn straw yellow within a few days, covering the entire leaf. As the disease progresses, the lesions cover the entire leaf blade which may turn white or straw coloured. Lesions may also be seen on leaf sheaths in susceptible varieties. Milky or opaque dew drops

containing bacterial masses are formed on young lesions in the early morning. They dry up on the surface leaving a white encrustation. The affected grains have discoloured spots surrounded by water soak areas. If the cut end of leaf is dipped in water, bacterial ooze makes the water turbid.

Etiology

The bacterium is strict aerobe, gram negative, non spore forming, rod shaped with **monotrichous** polar flagellum of at one end. The bacterial cells are capsulated and are joined to form an aggregate mass. Colonies are circular, convex with entire margins, whitish yellow to straw yellow and opaque. The bacterium has many strains that differ in ability to infect rice plants. Strains in tropical countries are usually more virulent than those in temperate areas like Japan.



Bacterial ooze

Disease cycle

P.I:The bacterium enters the plant through water pores (**hydathodes**) along the edges of the leaf and through injuries in roots or leaves. It does not enter through stomata. BLB is primarily a vascular or **systemic** disease. Bacterial cells move along the vascular tissues causing wilting. Rain storms and typhoons help in the spread of the disease. Irrigation water also carries the organism from field to field. The primary source of infection is through bacterium overwintering in seed (**husk and endosperm**). Bacteria may survive in soil, plant stubbles and debris. The pathogen also survives on collateral hosts like *Leersia hexandra, Leersia oryzoides, Zizania latifolia, Cyprus rotundus, Cyprus deformis, Phalaris arundinacea, Cyanodon dactylon*, etc.

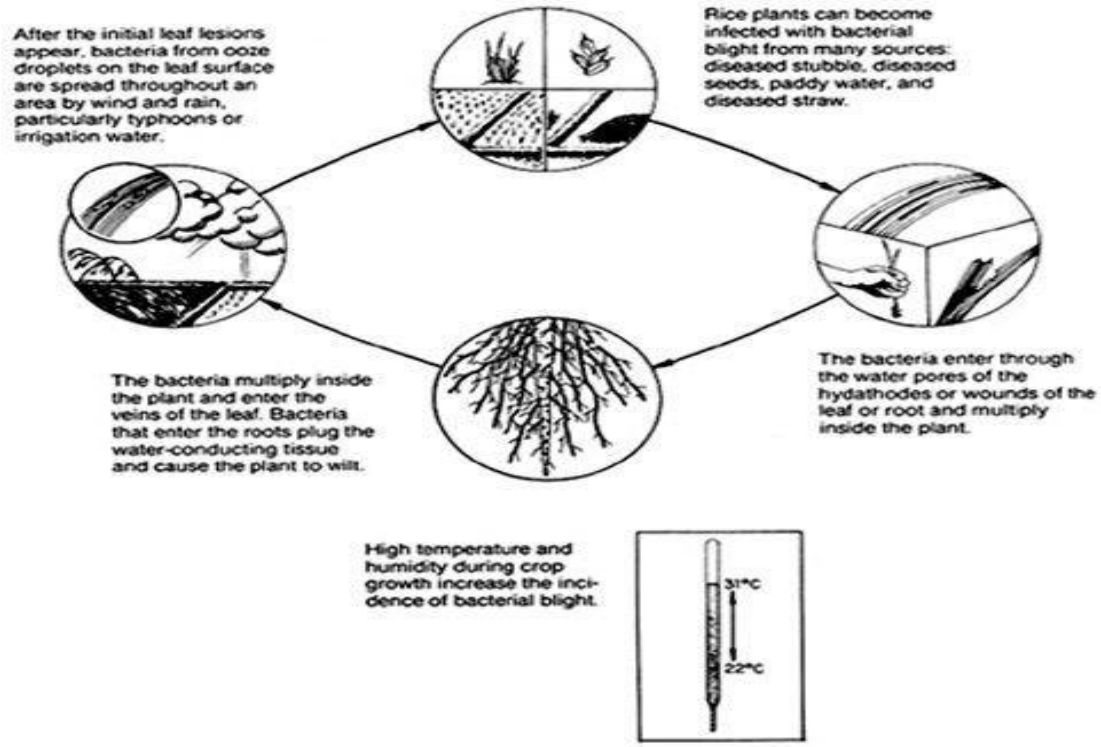
S.S.I: Bacteria spread through irrigation water, rain storms and typhoons

Favourable Conditions

Clipping of tip of the seedling at the time of transplanting, heavy rain, heavy dew, flooding, deep irrigation water, severe wind, temperature of 25-30^{0}C and application of excessive nitrogen, especially late top dressing.

Management

- Grow resistant cultivars like MTU 9992, Swarna, Ajaya, IR 20, IR 42, IR 50, IR 54, TKM 6, Mashuri, IET 4141, IET 1444, IET 2508, Chinsura Boro, etc.
- Resistant donors: Tetep, Tadukan, Zenith, etc.
- Affected stubbles are to be destroyed by burning or through ploughing
- Judicious use of nitrogenous fertilizers
- Avoid clipping of tip of seedling at the time of transplanting.
- Avoid flooded conditions or drying of the field (not at the time of flowering)
- Avoid flow of irrigation water from infected to healthy field



- Remove and destroy weed hosts.
- Soaking seeds for 8 hrs in Agrimycin (0.025%) followed by hot water treatment for 10 minutes at 52-54 ^{0}C eradicates the bacterium in the seed
- Spray **poshamycin (100 ppm) along with copper oxychloride (0.3%)**

Bacterial leaf streak

Xanthomonas oryzae p.v. oryzicola

Economic importance

Bacterial leaf streak was first found in Phillippines in 1918. The disease is common in tropical Asia, but is not present in Japan or other parts of the world. In India, it is reported by Srivastava from U.P, MP, AP, Maharashtra, Karnataka, Orissa, Haryana and West Bengal. **IR 8, Jaya** and **Padma** are highly susceptible to BLS.

Symptoms

Fine translucent streaks appear between the veins of the leaf are the first symptoms. The lesions enlarge lengthwise and advance over larger veins laterally and turn brown. On very susceptible varieties a yellow halo appears around the lesions. On the surface of the lesions, **bacteria ooze** out and form small yellow band-like exudates under humid conditions. In severe cases the leaves may dry up.

Fine translucent streaks appear between the veins of the leaf

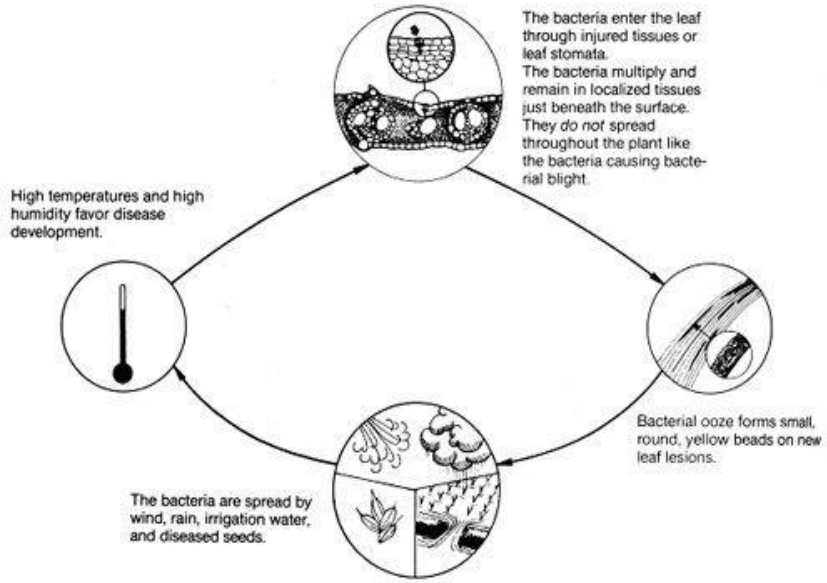
Etiology

The organism is short rod, about 1.2µ x 0.3 to 0.5µ, and gram negative. The strains of the bacteria differ in pathogenicity, the virulent strains causing longer streaks.

Disease cycle

P.I:The pathogen can survive in infected **seed** but not in crop debris. The bacteria enter the leaves through stomata and wounds. It mainly infects the parenchymatic cells but does not enter the vascular systems. BLS is not a systemic disease. When the leaves are wet, exudate from infected leaf spread to other portions of the leaf and to other plants.

S.S.I:Bacteria spread through irrigation.Rain storms and typhoons favour the spread of the disease.



Favourable conditions

High relative humidity (83-93%) or dew during morning hours for 2 to 3 hours

Management

- Grow resistant varieties
- IR 20, Krishna and Jagannath are tolerant to BLS
- Affected stubbles are to be destroyed by burning or through ploughing
- Judicious use of nitrogenous fertilizers
- Avoid clipping of tip of seedling at the time of transplanting.
- Avoid flooded conditions or drying of the field (not at the time of flowering)
- Avoid flow of irrigation water from infected to healthy field
- Spray Kasugamycin+copper oxychloride@0.25%

Tungro disease

Economic importance

Penyakitmerah which has been known in Malaysia since 1938 was identified as Tungro in 1965. The **mentak** disease of Indonesia is also identified as Tungro. Tungro is commonly found in Bangladesh and India. In India, it is seen in states of West Bengal, Kerala and other parts of India. Tungro is one of the most widely distributed and most destructive diseases in tropical Asia. The loss was estimated during 1940 as 30% or 1.4 million hectares annually. In Thailand a severe epidemic occurred in 1966 affecting more than 3 lakh hectares. An outbreak of Tungro in 1971 affected hudred and thousands of hectares in Phillippines.

Symptoms

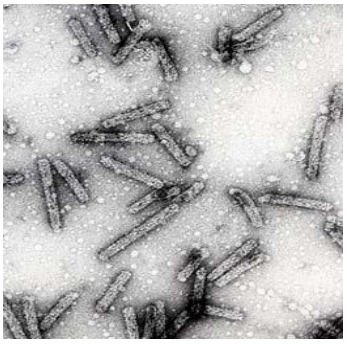
Infection occurs both in the nursery and in the main field. Plants are markedly **stunted**. Stunting is more severe on susceptible varieties and slight on more resistant varieties. Leaves show **yellow to orange discoloration** and interveinal chlorosis. Yellow discoloration is commonly seen in “Japonica” varieties, while “Indica” varieties show orange discoloration. Yellowing starts from the tip of the leaf and may extend to the lower part of the leaf blade. Young leaves are often mottled with pale green to whitish interveinal stripes and the old leaves may have rusty streaks of various sizes. The plants may be killed if infected early. Tillering is reduced with poor root system. The infected plants have few spikelets and panicles are small with discoloured grains.

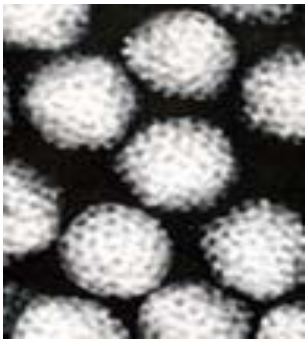
Tungro infected plants can be chemically identified by **Iodine Test.** Ten cm long leaf tip is cut in the early morning before 6 A.M. and dipped in a solution containing 2g Iodine and 6 g Potassium Iodide in 100 ml of water for 30 minutes. Tungro infected leaves show dark blue streaks.

Etiology

It is a composite disease caused by two morphologically unrelated viruses: rice tungro bacilliform virus (RTBV) and rice tungro spherical virus (RTSV). **RTBV** has a bacilliform capsid or bullet-shaped particles (130 x 30 nm) made up of a single piece of coat protein of MW 36 K and a single molecule of circular **ds DNA** of 8.3 KbP. **RTSV** has a isometric capsid, 30 nm in diameter comprising two to three polypeptide pieces and a single piece of polyadenylated **ss RNA** of about 10 KbP.

Yellow to orange discolouration of leaves





Yellow or orange RTBV RTSV discolouration

Two types of virus particles are associated with the disease. **Bacilliform** particles cause majority of the **symptoms** of the disease. **Spherical** particles help in the **transmission** of bacilliform virus by the green leaf-hoppers. If the bacilliform virus particles are alone present in the rice plant they will not be transmitted by the leafhopper vector.

Disease cycle

P.I:The virus causes severe damage only in area where the host plants and the insect vector multiply the year round. In the areas where the rice is not grown continuously, collateral hosts, especially wild rice are probable sources of inoculum. Stubbles of infected plants from the previous season also serve as a source of inoculum. Grassy weeds such as *Eleusine indica, Echinochloa colonum, Echinochloa crusgalli* may be infected occasionally.

S.S.I: Viral particles dispersed through **leafhoppers** viz, *Nephotettix virescens, N. nigropictus, N. parvus, N.malayanus* and *Recilia dorsalis* transmit the virus in a **non-persistent** manner.

Management

- Summer deep ploughing and burning of stubbles.
- Destroy weed hosts of the virus and vectors.
- Grow disease tolerant cultivars like MTU 9992, 1002, 1003, 1005, Suraksha, Vikramarya, Bharani, IR 36, IET 2508, RP 4-14, IET 1444, IR50 and Co45.
- Maintain 2.5 cm of water in the nursery and broadcast anyone of the following in 20 cents. Carbofuran 3 G 3.5 kg. Phorate 10 G 1.0 kg.
- Spray phosphamidan @1ml/lt or Ethophenphos@1.5ml/lt or Neem oil @3 per cent in the main field 15 and 30 days after transplanting to control leaf hoppers.

Lecture 3&4 Wheat- economic importance, symptoms, cause, disease cycle and integrated management- black stem rust, brown rust and yellow rust, loose smut and *Karnal* bunt, leaf blight, soil borne mosaic and tundu disease / yellow slime disease

Wheat (*Triticum* sp.) **Black or stem rust** *Puccinia graminis* f.sp.*tritici*

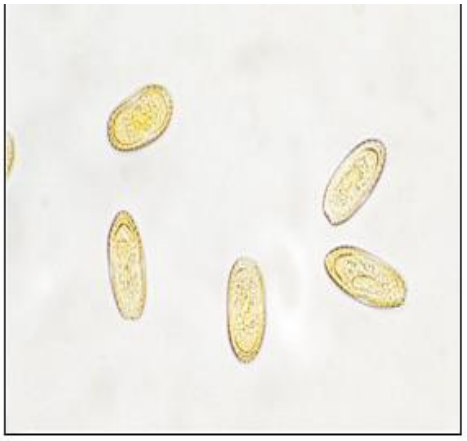
Economic importance

The most important and destructive disease throughout the world where ever wheat is grown. The rust epidemics of 1946-47 in M.P, Maharashtra, Rajasthan and U.P destroyed over two million tonnes of grain. In 1956-57 rust was severe in W.B, Bihar and Eastern parts of U.P causing heavy damage and rendered the grain in some tracts unfit to harvest. In India though black stem rust is prevalent in all parts of the country it normally appears in epidemic form only in central, southern and eastern parts of the country where high temperatures prevailed during crop season. In Northern India, the disease usually appears during March when the crop is reaching maturity causing only a limited loss to the grain yield, whereas, in Southern parts, it appears during Nov to Dec causing severe losses. **Barley** is also susceptible to this rust.

Symptoms

The first symptom of rust infection is **flecking** of leaves, leaf sheaths, culms and floral structures. These flecks soon develop as oblong, **reddish brown uredo-pustules**, frequently merging into one another, finally bursting to expose a mass of brown uredospores. When large number of uredosori burst and release their spores, the entire leaf blade and other affected parts will have a brownish appearance even from a distance. Later in the season, **teleutosori** are produced. They are conspicuous, linear or oblong, dark brown to black, and often merging with one another, to cause linear patches of black lesions, which account for the name black rust. On maturity the teleutosori burst open, exposing masses of dark brown teleutospores. In the transitional stage, there is a mosaic of brown and black masses of spores on the affected tissues, which dry up prematurely. Moreover, in the case of severe infections the diseased plants are stunted and produce small spikes and shrivelled grains, or no grain at all.

Blackish teliopustules on stem



Reddish brown uredospores of black stem rust

Dark brown teleutospores of black stem rust

Pathogen

Black stem rust is **heteroecious** full cycle rust. It requires more than one host species to complete its life cycle. The uredial and telial stages occur on wheat, barley and some grasses and the pycnial and aecial stages on the species of ***Berberis*** (Barbery) and ***Mahonia***, the alternate hosts. The uredospores are brown, oval shaped, thick walled and marked with thin short spines and borne singly on stalks. The teleutospores are dark or chestnut brown, two celled, germinating by producing thin walled, hyaline four celled promycelium (basidium). The fungus is highly specialized and has number of physiological races (over 250). Races 11, 15c, 34-A and 122 are most predominant appearing in virulent form in wheat growing tracts of India.

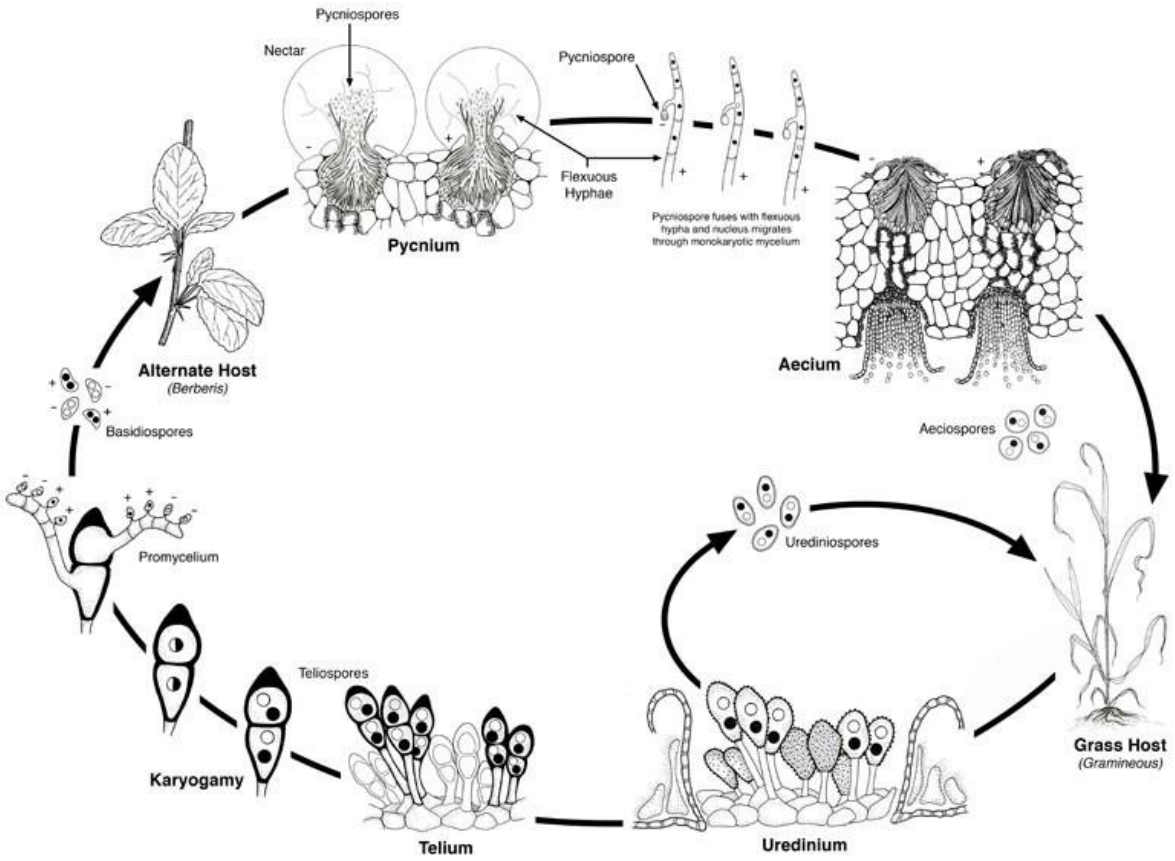
Disease cycle

PSI: Teliospores in plant debris .Primary infection is mainly through **barberry**, i.e., *Berberis vulgaris*. These barbery plants play a role in USA, Europe and Australia, where as in India they are not known to play any role in the perpetuation of the fungus. The source of inoculum for black rust comes from south, i.e., **Nilgiri** and **Pulney** hills. In plains of North India during summer months the uredospores cannot survive because of the high temperatures. The possibilities of the fungus surviving on ratoon tillers or **self**

sown wheat plants, late and **off season wheat** crops and certain grasses growing in cool areas particularly in the foot hills of Himalayas in the North, the Nilgiris and Pulney hills in the South appear to be great.

The grasses, viz., ***Briza minor, Bromus patula, Brachipodium sylvaticum*** and ***Avena fatua***, harbor the fungus in the off-season. It is believed that the fungus over summers on the wheat plants and grasses in the hilly areas and spreads to the plains in the main wheat crop season. In the central Nepal, the wheat crop sown in August and harvested in December, January becomes infected by *P. graminis tritici* from October. This may be a source of inoculum for the main crop sown in the plains, which becomes infected from February each year.

. SSI: Wind borne uredospores



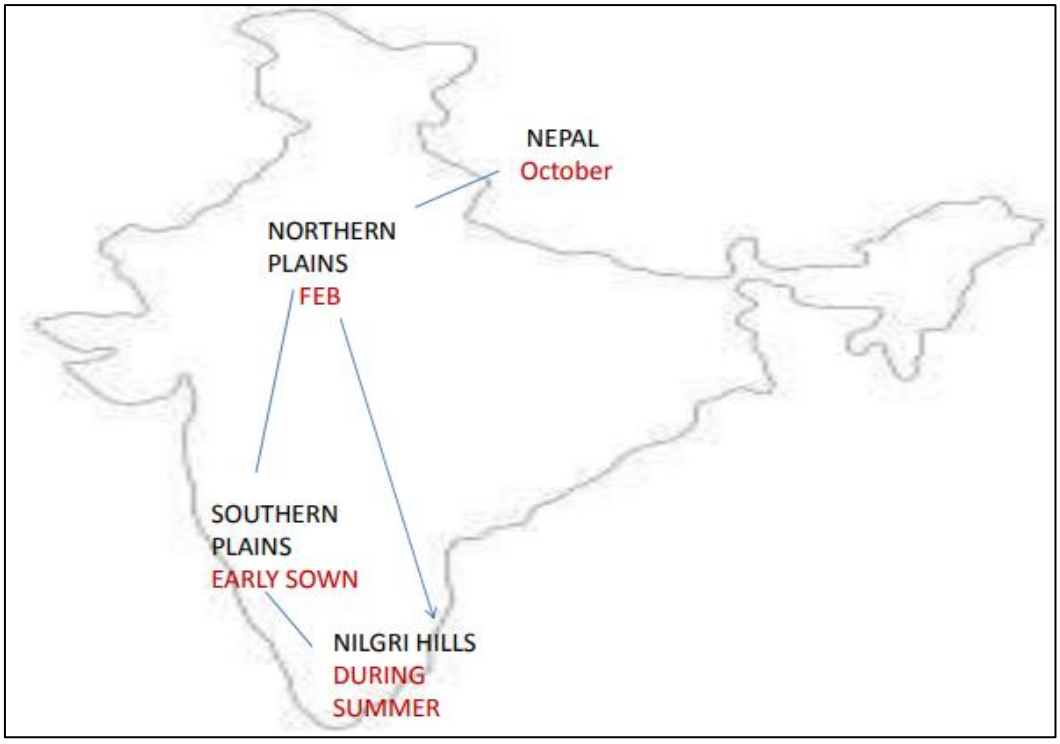
***Puccinia* pathway:**It is path followed by *Puccinia graminnis* f.sp.*tritici* uredospores during crop season and off season in India.

Dr.K.C.Mehta & his associates through aeroscopic studies on *Puccinia* pathway brought into light the mystery behind the spread of uredospores in the subcontinent.

- In central Nepal wheat is sown during August & harvested in January. It gets infected by *P.graminis tritici* during October.
- The uredospores from central Nepal acts as the inoculum for the infection of main crop in the Northern plains.

- Uredospores travel to south via Terai region

- The wheat crops in northern plains are infected during February each year.
- The uredospores travel to summer wheat crops of Nilgri & Palani hills & also spreads to early sown crops of Mysore & other neiboughring places.
- Fungi multiplies on summer hill crops & spreads to early sown crops in southern plains.
- The fungi multiplies & spreads Northward during October- November. The Gangetic plains are severely affected during February – March.



Nagarajan and Singh(1975) framed certain synoptic upper air rules called the "Indian stem stem rust rules".Combinations of weather conditions which satisfy these rules result in transportation and deposition of inoculum from the Nilgiri hills area.They found that the path of airborne inoculum could be correlated with the visible cloud movements detected by Satellite television

Management

- Eradication of selfsown wheat plants and weed hosts
- Adjust time of sowing

Grow resistant varieties like Kalyanasona, Sonalika, Choti Lerma, Lerma Rojo, Safed lerma, NP 700 & 800.

- Avoid late sowing
- Balanced application of nitrogenous fertilizers
- Seed dressing with Plantavax@0.1% followed by two sprays with the same chemical.
- spray twice or thrice with Plantavax@0.1%, at 15 days interval or Azoxystrobin@0.05%

Leaf, brown or orange rust

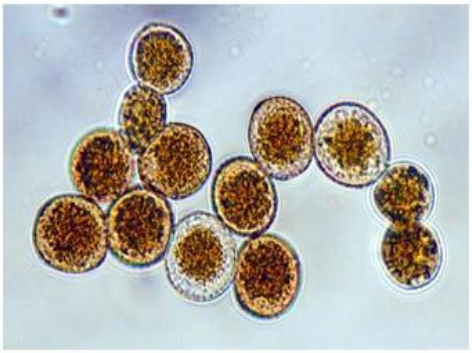
Puccinia recondita

Economic importance

In India it is the most common rust in the northern and eastern parts. In Punjab, Bihar and UP it causes more damage than stem rust. In South India, it is found in the crops grown both in the hills and in the plains. Ten per cent yield losses are reported.

Symptoms

The first symptom of the disease is the appearance of minute, round, **orange sori**, irregularly distributed on the leaves, **rarely on the leaf sheath and stem**. The sori turn brown with maturity. As the disease advances, the telial stage may be found in the same pustule. The telia are small, oval to linear, black and covered by the epidermis. The telia are also found on the leaf sheath. Severe rusting of leaves causes reduction in yield.



Brownish uredospore of leaf rust

Teliospores of leaf rust with round apex

Pathogen

The fungus, *Puccinia recondita*, is heteroecious. The uredial and telial stages appear on wheat and some other grasses and aecial and pycnial stages on species of ***Thalictrum***. In India, the role of *Thalictrum javanicum* and *T. flavum* as alternate hosts has not been precisely determined. In Russia, ***Isopyrum fumaroides*** is known to act as a natural alternate host. The uredospores are brown, spherical and minutely echinulate with 7-10 germ pores. Telia are rare, but when formed are found mostly on the lower surface of the leaf and do not rupture. Teleutospores are smooth, oblong, thick walled and brown with a rounded and a prominent thickened apex.

Disease cycle

PSI: Teliospores in plant debris. Alternate host, species of *Thalictrum*, helps the fungus to oversummer in other countries. The role of *Thalictrum* is not clear in India. In early

January, the rust gets well established in the foot hills of Himalayas and also in the plains of Tamil Nadu and Karnataka in the South. The first buildup of inoculum takes place in the plains of Karnataka and moves northwards to Maharashtra and Madhya Pradesh. The inoculum from the foot hills of Bihar and UP moves to the northern plains.Therefore the brown rust appears slightly later in the Western hills of North India. The rust population of the north and the south moves in opposite directions, finally merging into each other, and causes serious disease in the wheat growing states.

SSI: Wind borne uredospores

Management

- Grow resistant varieties like Sonalika, NP 700 & 800, Lerma Rojo and Safed Lerma.
- Spray Carbendazim12%+ Mancozeb 63%@0.25%
- Seed dressing with Plantavax@0.1% followed by two sprays with the same chemical

Yellow or stripe rust

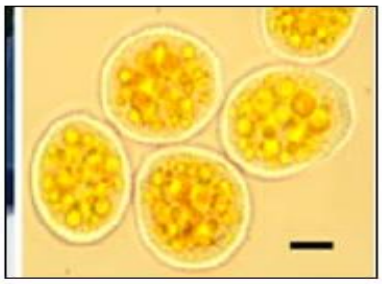
Puccinia striiformis

Economic importance

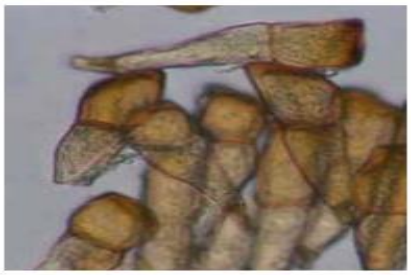
It is confined to the cooler parts particularly the foot hills of Himalayas, Punjab, Himachal Pradesh, Haryana, U.P, and parts of Rajasthan and Bihar. It is totally absent from South India except in Nilgiris and Pulney hills. It appears every year, but the damage is seen only in occasional years. **Sonara-64** is susceptible to yellow rust.

Symptoms

The uredosori appear as **bright yellow pustules** chiefly on the leaves. But in severe infections they may be seen on leaf sheaths also. The sori are elongated and are arranged in **linear rows** between the veins of the leaf and hence it is referred as stripe rust. The sori are mostly sub-epidermal and are remained covered by the epidermal layer and break only at the time of crop maturity. The teleutosori appear late in the season and are also arranged in linear rows. They are compact, elongated, and black which remain sub- epidermal. They do not break through epidermis for a long time remaining as black crust.



Bright yellowish uredosori in linear rows on leaf



Compact /flattened teliospores

Pathogen

Uredospores are yellow, spherical to oval with a spiny wall. The teleutospores are dark brown, two celled, thick walled and **flattened at the top**. The teleutosori are filled with numerous unicellular, brown lengthy paraphyses.

Disease cycle

PSI: Fungus overwinter on volunteer wheat plants.

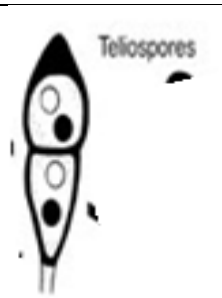
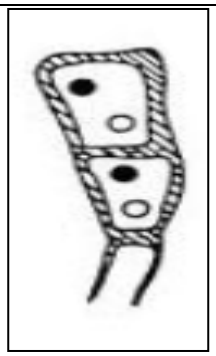
The fungus overwinters in its uredial stage in England and other countries. Its persistence in India is not known. It may overwinter on volunteer wheat plants at an altitude of about 1500 to 1800 meters in the Himalayas. The uredospores germinate after a period of dormancy and form a source of inoculum for early sown wheat crop. In U.P early sown crop is severely infected by the fungus than the late sown crop. Some weeds like ***Agropyron semicostatum, Bromus catharaticus, Bromus japonicus*** and ***Hordeum murinum*** also serve as primary source of inoculum. Secondary infection is by wind borne uredospores. There are about 40 races in the world including 13, 14, 19, 20, 24 and 31 A which are wide spread in India.

SSI: Wind borne uredospores

Management

- Grow resistant varieties like Lerma Rojo, Safed Lerma, Sonalika and Choti Lerma
- Spray plantavax@0.1%
- Removal and destruction of weed hosts

S.No	Character	***Puccinia graminis***	***Puccinia recondita***	***Puccinia striiformis***
1	Disease	Black stem rust of wheat	Brown rust of wheat	Yellow rust of wheat
2.	.Infected part	mainly stem, then leaf sheath, leaf blade and ear.	. Mainly leaf, rarely leaf sheath and stem.	mainly leaf, rarely leaf sheath and stem.
3.	Uredosori	large elongated, dark brown to rusted in colour. Available mainly on stem, elongated and black in colour.	small round to oval, bright orange in colour.	small round to oval, lemon- yellow in colour
4.	Uredospore	Oval, brown, thick walled with 4-round equatorial germ pores. Available mainly on stem, elongated and black in colour.	oval or round, bright orange with 7-10 scat-tered germ pores.	oval to spherical, yellow, with 6-16 scattered germ pores.
5.	Teleutosorus	mainly on stem, elongated and black in colour. Burst on maturity.	develops rarely; if pre-sent, they are confined to the lower surface of the leaf and black in colour. Do not burst.	confined to lower sur-face of the leaf, elongated and black. Do not burst.
6.	Teleutospore	2-celled, chestnut brown to black in colour, thick walled with very thick round or pointed apex.	2-celled, dull black in colour, thick walled with flattened and less thick at the apex.	2-celled, dark-brown in colour, thick-walled with flattened and less thick at the apex.

7.	Alternate host	*Berberis vulgaris, Mahonia sp.* and Mahoberberis sp.	*Thalictrum falvum, Isopyrum fumarioides,*	*Muehlenbeckia hugeli.*
8.	Collateral hosts	*Bromus japonicus*	*Bromus japonicus*	*Aegilops sp*.
9.	Teliospore diagram			

Loose smut

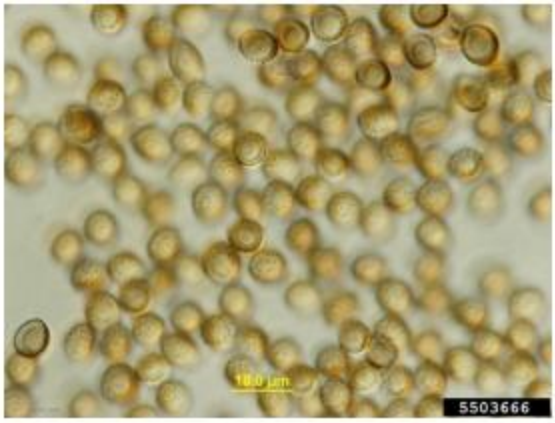
Ustilago tritici

Economic importance

Loose smut is one of the major diseases on wheat. There was loose smut epidemic during 1970-75 in Punjab, Haryana and Western U.P. In Sonalika, the incidence was 5 to 6%. Incidence is more in North than in southern parts of India. Country wide loss is about 2- 3% in yield.

Symptoms

The symptoms are evident only at the time of emergence of the panicle from boot leaf. All the spikelets in a panicle transform into a mass of **black powdery spores**. The infected panicle emerges two days earlier than healthy and the spores are covered with the silvery membrane. This thin membrane gets ruptured exposing the mass of black spores. The spores are easily blown by wind leaving the bare rachis.

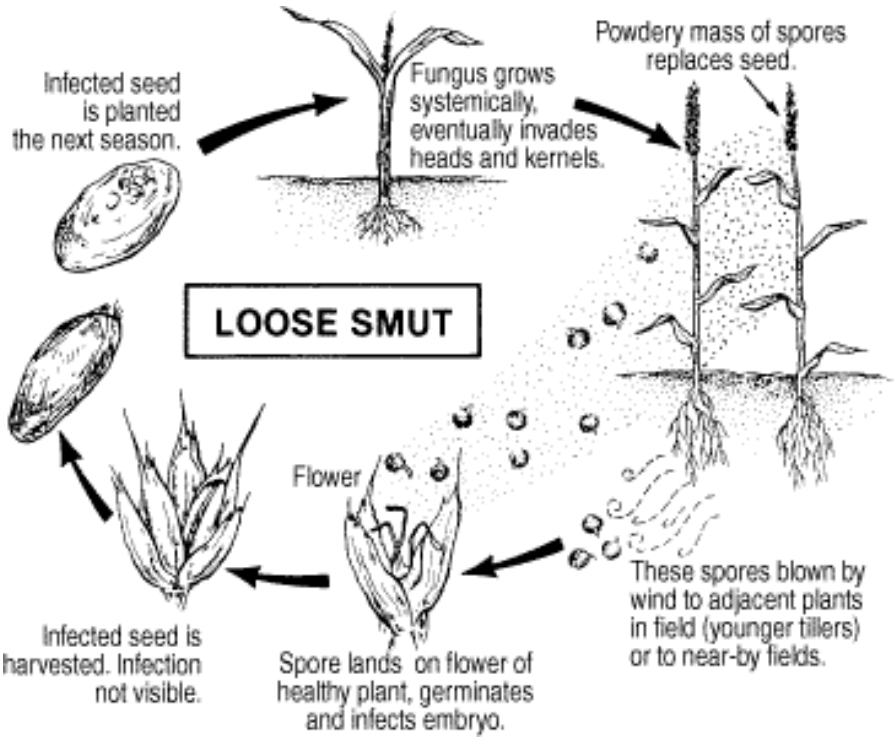


Pale, olive brown, spherical to oval smut spores

Spikelets transformed into a mass of black powdery spores covered with the silvery membrane

Pathogen

Chlamydospores of the fungus are pale, olive brown, spherical to oval in shape. These smut spores germinate and produce promycelium or sporidium. The promycelial cells fuse and give rise to germ tubes that enter the ovary through the stigma and become established in the embryo remaining dormant until seed germination.



Disease cycle

P.S.I:Dormant mycelium in **internally seed borne** and is systemic. The fungus is carried over in the seed as dormant mycelium. When the planted seed germinates the mycelium becomes active. It grows along with the plant and when the panicle is produced the mycelium reaches the ovaries and transforms the ovaries into a mass of black smut spores. Secondary spread occurs through wind borne smut spores. The sporidia infect the healthy flowers. The mycelium enters the ovary and remains in the seed as dormant mycelium.

SSI: Wind borne chlamydospores and sporidia

Management

- Grow resistant varieties kalyanasona, PV 18, WG 307 and HD 450.
- ***Hot water treatment*** (**Jensen**, 1908): Soak the seed in cold water for 4 hours and then immerse the seed in hot water at a temperature of 132 ^{0}F or 52^{0}C for about 10 minutes. Dry the seed in shade before sowing.
- ***Solar seed treatment*** (**Luthra and Sattar**, 1934): Soak the seed in water for 4 hours (8 AM to 12 Noon) and expose the seed to the hot sun for 4 to 5 hours (from 12 Noon to 5 PM) on cement or rocky surface. This can be practiced in the areas where the summer temperatures are high (42-44^{0}C)

- ***Anaerobic seed treatment*** (USA): Soak the seeds for 2-4 hours in water between 60- 70^0F and keep the moist seeds in air tight containers for 65-70 hours and there after dry the seed.
- Seed treatment with systemic chemicals like vitavax@0.2%

Karnal bunt or partial bunt

Neovossia indica (formerly Tilletia indica)

Economic importance

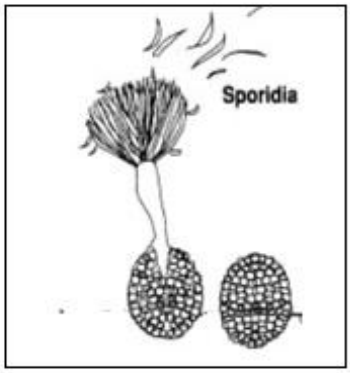
The disease was first reported in India from Karnal (Haryana) by **Mitra** in 1931. The disease was less severe till 1970's, however it assumed greater importance in early seventies with the adoption of high yielding, semi dwarf nutrient responsive varieties. The disease appeared in epidemic form in different parts of India in 1976, 1979, 1981-83 and 1986.

Symptoms

The infection is usually confined to a **few grains** in the spike with irregular arrangement. In some cases the infection may spread to only a part of the grains. In severe cases, the grain is reduced to black shiny sac of teliospores. As the grains mature the outer glumes spread and the inner glumes expand, exposing the bunted grains. The bunt balls are first enclosed by the pericarp but when it bursts the masses of bunt spores are exposed. The bunt affected plants emits a foul smell which is mainly due to the presence of **Trimethyl amine**.

Blackened areas surrounding the partially smutted grains

Bunted grains with partial seed coat, the embryo and part of the endosperm converted to masses of small black spores



Teliospore with needle shaped sporidia

Allantoid(sickle shaped) sporidia

Pathogen

Teliospores are smooth walled measuring 22-49µ in diameter and require a long resting period. Teliospores germinate and produce a large number (60-120) of **needle shaped** primary sporidia on a short stout basidium. Later, sickle shaped (**allantoid**) secondary sporidia are produced which help in the dispersal of karnal bunt.

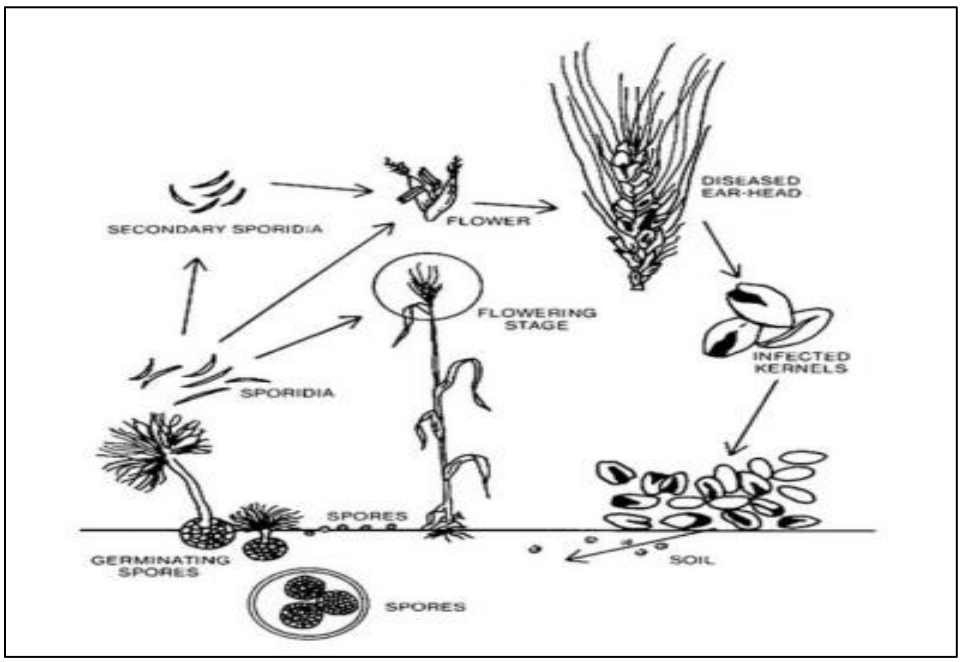
Disease cycle

PSI: Teliospores in soil and plant debris

SSI: Wind borne primary and secondary sporidia

The teliospores in soil germinate producing primary sporidia. The sporidia become air borne and deposit on leaves of host plants. Under high humid conditions they produce a secondary crop of secondary sporidia (allantoids). If boot emergence stage coincides with drizzle, the secondary sporidia get washed down to sheath. The sporidia germinate on glumes to enter epidermal cells to penetrate ovary. The sporidial germ tubes penetrate stomata in rachis, glumes, lemma and palea. The disease progresses

systemically to other florets within an infected spikelet. The infection mostly starts from the embryonal end and spreads **along the grain suture**. The hyphae grow through the base of the glumes into sub-ovarian tissue and enter pericarp through funiculus. Hyphae of the pathogen proliferate, remain restricted to pericarp and produce teliospores terminally. In severe cases grain is reduced to black shiny sack of teliospores. The embryo and endosperm are not colonized. The pericarp ruptures during threshing and teliospores deposit in soil and adhere to the surface of the seed.



Favourable conditions

Moderate temperatures (19-23^{0}C), high humidity (>70%) and cloudiness or rainfall during anthesis favours disease development in susceptible host varieties.

Management

- Grow tolerant varieties, viz., WL 1562, HD 2281, etc. Use resistant sources like wild species of *Aegilops* and *Triticum*, HD 2329, HD 29 and HD 20 for breeding programme.
- Follow strict quarantine measures
- Use disease free seed for sowing
- Judicious application of nitrogenous fertilizers
- Adjust date of sowing
- Intercropping with Gram or Lentil
- Seed treatment with copper carbonate or propiconazole@0.1%

- Spray with carboxin@0.2% or propiconazole @0.1%

Tundu disease or yellow slime disease

Anguina tritici (Nematode) + Clavibacter tritici(Rathayibacter tritici)

Economic importance

The disease was first reported by **Hutchinson** (1917) from Punjab in India.

Symptoms

The tundu disease is characterized by the twisting of the stem, distortion of the ear head and rotting of the spikelets with a **profuse oozing of yellow liquid** from the affected tissues. The ooze contains masses of bacterial cells.

The nematode alone causes winkling, twisting and various other distortion of the leaves, stem and produce small round galls on the leaves. The infected plants are shorter and thicker than healthy plants. In the distorted earheads dark galls are found in place of kernels.

When the bacterium is associated with the nematode, the disease symptoms are intensified at the flowering stage and yellow ear rot sets in due to combined action of the nematode and bacterium. The earhead becomes chaffy and the kernels are replaced by dark

nematode galls which also contain the bacterium. The infected plants produce more tillers than the healthy ones. Another interesting feature is the **early emergence of ears** in the nematode infected plants which is about 30 to 40 days earlier than the healthy ones.

Twisting,distortion and rotting of spikeletss in tundu infected panicles

Pathogen

Rathayibacter tritici or Clavibacter tritici is rod shaped, Gram positive and is motile by single polar flagellum.The female is about 3.8 mm long and spirally coiled. Although females exhibit some movement, they are effectively immobile. The stylet is relatively short at 8 to 10 µm long. Males are not as robust as the females, curved rather than coiled and only about 2.4 mm long.

Disease cycle

PSI: Seed contaminated with nematode galls

The disease starts from the seeds contaminated with the nematode galls. When such contaminated seeds are sown in the field, they absorb moisture from the soil and the larvae (juveniles) escape from the galls and climb upon the young wheat plants. At the time of flowering, the nematodes enter the floral parts and form galls in the ovaries. When once the nematode is inside the tissues of the ovary, the bacterium becomes active and causes rotting. The yellow ooze coming out of the rotting earhead provides the inoculum for the secondary spread of the disease which is favoured by wind and rain. The nematode probably functions as a vector transporting the bacterium to otherwise inaccessible meristematic regions of the host. The nematodes secrete some substances in the presence of the host bacterium which can remain viable for atleast 5 years in the galls of *A. tritici*. The nematode galls are reported to remain in the soil for 20 years or more and the bacterium can also survive for the same period inside the nematode gall.

SSI: Bacterial ooze disseminated through rain splashes and nematode juveniles

Management

- Seed cleaning and crop rotation together can achieve complete control to the point of localised extinction. Modern harvesting, seed cleaning and testing practices are more than adequate to ensure that seed is free of the nematode. The most effective control is by mechanical seed cleaning. Galls may be also removed by submersion of the seed in 20% brine solution (galls float to the surface), followed by thorough washing in water. Hot water treatment at 54°C for 10 min is also reported to be effective in killing the nematodes.
- Modern harvesting, seed cleaning and testing practices are more than adequate to ensure seed free of the nematode. For small seed lots, where this technology is not available, brine (10%) flotation is more efficient than hand sieving.
- In addition to clean seed, carryover from one crop to the next in the same or adjacent fields needs to be avoided. Despite having a remarkable longevity when stored dry, only a small proportion of juveniles will survive to a second season in the field, hence rotation with a non-host crop and control of volunteer wheat will provide effective field control.

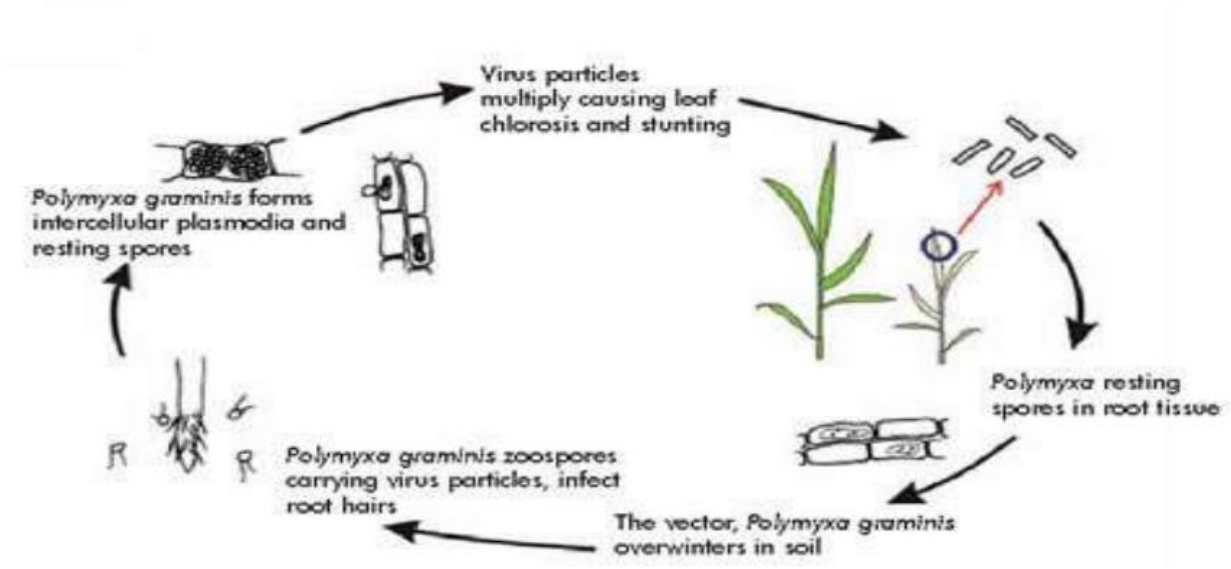
➢ Wheat, barley or oat should not be sown in the infested soil.

Wheat soil borne mosaic virus

Economic significance

Early in the history of SBWM research, host genotypes susceptible to rosette stunting (Figure 15) were common, and the resulting yield loss was potentially greater than 50%. As a result of the exclusion of cultivars exhibiting rosette symptoms, the devastating rosette phenotype is seldom seen today. However, many contemporary cultivars still exhibit the mosaic phenotype and may experience significant yield losses. In many grain producing regions, SBWM results in significant annual yield losses, but, since SBWM symptoms are short-lived and mimic nutritional deficiencies, its economic significance is often underestimated. SBWMV infection causes a reduction in kernel weight, tiller number, and test weight, cumulatively leading to lower grain yields.

Symptoms: ϖYellow green mosaic on young leaves, stunting, severe rosetting and excessive tillering. ϖThis virus is transmitted by soil borne fungi.



Pathogen: Wheat yellow mosaic virus (WYMV)

- Conditions: Long periods of fluctuating temperatures in early spring.
- Transmission: Soilborne fungal vector (Polymyxa graminis) and sap transmission

ϖPolymyxa graminis,is the reported vector of wheat soil borne mosaic virus is an obligate root parasite of wheat.

DISEASE: **Soil-borne wheat mosaic**

PATHOGEN: **Soil-borne wheat mosaic virus**

HOSTS: **Wheat, triticale, rye, barley, and other grasses**

Close-up of SBWM nursery

First described in 1919, *Soil-borne wheat mosaic virus* (SBWMV) can cause severe stunting and mosaic in susceptible wheat, barley and rye cultivars. The disease was often misdiagnosed as a nutritional problem, but this has actually contributed to the fortuitous visual selection by breeding programs of resistant genotypes. Currently, major gene resistance is incorporated into the most popular cultivars grown where SBWM already occurs. Nevertheless, in recent years SBWMV distribution has spread to various wheat growing regions of the U.S. and abroad. This has resulted in a resurgence of research interest in the virus and control of the disease.

Symptoms and Signs

Soil-borne wheat mosaic (SBWM) affects autumn-sown small grains. The first visual evidence of *Soil-borne wheat mosaic virus* (SBWMV) infection in a growing season usually occurs in the spring, after the crop begins to green up. However, in some years SBWM symptoms are expressed in the late autumn or early winter, especially in warmer climates. Irregular, chlorotic patches in the field suggest soil-borne viral infection, although similar symptoms may be caused by a number of other viruses or other biotic or

abiotic factors. In dry environments, patches of symptomatic SBWMV-infected plants usually occur in low-lying, wet regions of the field (Figure 2) that are conducive for infection by the swimming zoospores of its protozoan viral vector, *Polymyxa graminis.* In wetter or more humid climates, these patches may occur anywhere in the field (Figure 3).

Upon closer inspection, SBWMV-infected plants are often stunted (Figure 4) and the leaves may have a general chlorotic mosaic or irregular mottling and streaking (Figure 5). A few strains of the virus cause rosetting in highly susceptible wheat cultivars; leaves and tillers remain short, growth is bunchy or compact, and tillering is excessive (Figure 6). Perhaps the most diagnostic characteristic of SBWMV infection is that symptoms are not expressed on leaves that emerge after the average temperature rises above 20 °C (68 °F). Thus, SBWM chlorotic patches tend to disappear in late spring. SBWM is most often confused with a disease caused by another soil-borne virus, *Wheat spindle streak mosaic virus* (WSSMV), which can also induce stunting and chlorosis and is also temperature sensitive. A good diagnostic feature to distinguish between these two viruses is that WSSMV induces chlorotic streaks that are elongated and spindle-shaped and often have a dark green island in their center (Figure 7).

Figure 4

Figure 5

Figure 6

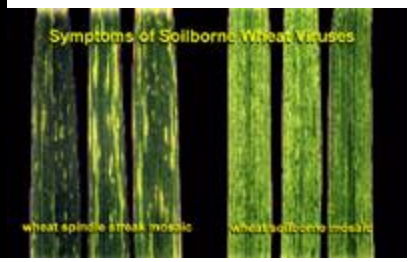


Figure 7

However, accurate diagnosis of SBWM is complicated by the fact that symptom expression varies by host genotype and, as previously mentioned, similar symptoms may be caused by a range of biotic and abiotic factors. Therefore, additional diagnostic tests are often conducted to verify the presence of SBWMV. The most commonly used tests are detection of viral coat protein by enzyme-linked immunosorbent assay (ELISA) or detection of a portion of the RNA genome by reverse-transcription polymerase chain reaction (RT-PCR).

Pathogen Biology

SBWMV is the type member of the *Furovirus* genus. Members of the genus are characterized by rigid rod-shaped particles (Figure 8) and positive sense (directly serving as mRNA) RNA genomes consisting of two molecules that are packaged into separate particles (Figure). The two particles differ in length, and the shorter particle (138-160 nm) is 10 to 20 times more prevalent in infected tissue than the longer particle (281-300 nm), but both particles are required for infection.

Figure

The longer particle contains RNA 1, which is approximately 7100 nucleotides long and encodes three proteins. Two of these, measuring 150 kDa and 209 kDa, are associated with virus replication (Figure 9). The 37 kDa protein is a cell-to-cell movement protein. The 150 kDa and 209 kDa proteins are translated directly from the message sense viral RNA, whereas the 37 kDa protein is expressed via a subgenomic mRNA.

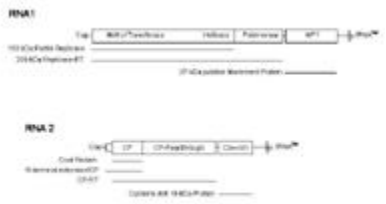


Figure 9

The shorter particle contains RNA 2 (approximately 3600 nucleotides), which also encodes three different proteins (Figure 9). The first is the 19 kDa coat or capsid protein (CP). Sometimes, the coat protein UGA termination codon is suppressed allowing translation of an 84 kDa CP-readthrough protein, which is thought to be required for virus transmission by its vector, *Polymyxa graminis* (Figure 10). The third protein is a 19 kDa cysteine-rich protein that is expressed via a subgenomic mRNA and may function as a suppressor of post transcriptional gene silencing (Figure 9). Post transcriptional gene silencing is a mechanism that can provide host resistance to a virus.

Figure 10

Disease Cycle and Epidemiology

Polymyxa graminis (Figure 10), the vector of SBWMV, belongs to the endoparasitic slime molds (Plasmodiophoromycota). This organism produces resting spores that harbor viral RNA and movement protein. These resting spores can remain dormant in soil for up to 30 years, waiting to infect the next graminaceous host germinating in an environment conducive for infection, at which time the virus-containing zoospores germinate. Soil water is critical for the swimming zoospore to reach a host root. The threshold matric potential is between –20 and –40 kPa. This means that soil pores between 7.4 and 14.7 µm must be filled with water in order for significant transmission to occur.

On reaching the host root, a *P. graminis* zoospore encysts on the surface of a cortical root cell, and develops a spear-like "stachel," which when mature will shoot through the adjoining zoospore and host walls . Along with the stachel, the zoospore contents and presumably the contaminating SBWMV particles are emptied into the host cortical cell. Thus begin the life cycles of the vector and of the virus. How the virus is attached to or carried by the zoospore, and exactly how the virus is transferred from the zoospore to the plant root are not fully understood, although SBWMV RNA and movement protein, but not capsid protein, have been detected within *P. graminis* sporosori.

Optimal temperatures for *P. graminis* infection vary among virus isolates and often reflect temperatures where the isolate is commonly found. Thus, isolates from India are active at warmer temperatures (27-30 °C/80-86 °F) than isolates from Belgium, Canada, Japan and France (15-18 °C/ 59-64 °F). In New York state, the optimal temperature for transmission of SBWMV is 15 °C (59 °F), with significant transmission occurring in the

growth chamber in less than 24 hours, given conducive soil moisture. Importantly, no significant transmission occurs at 6.5 °C (44 °F), suggesting that SBWMV is either transmitted in the autumn or in the spring in temperate climates, but not during the winter months. Given the period of time required for SBWMV systemic movement and symptom expression and the brevity of spring temperatures conducive to SBWM symptom expression, spring infections by *P. graminis* are not likely to contribute to viral symptom expression.

After the stachel-mediated infection, one of two types of plasmodia (multinucleate non-motile masses of protoplasm) of *P. graminis* may form in a single host cortical root cell (Figure 13). The plasmodia differentiate to give rise either to secondary zoospores or resting spores.

Infection of plant roots by *P. graminis* is the first step in the poorly defined SBWMV infection process. Viral replication within the initially infected cell and movement to adjacent host cortical cells does not appear to be inhibited in wheat genotypes resistant to systemic foliar SBWMV infection. There is some evidence that SBWMV moves systemically via xylem. Since viruses move within the living cell contents of plant tissues, this suggests that SBWMV must be inoculated into young tissues before the living protoxylem cells have differentiated to become mature, dead xylem cells. Protoxylem infection is most likely to occur at root tips and where lateral roots initiate, which coincides with the region of the rhizosphere that is most likely to attract *P. graminis* zoospores.

Disease Management

Chemical control

The only chemicals that have been reported to provide reproducible control against *P. graminis* in the field are soil fumigants, which are not economically feasible in small grains systems.

Crop rotation

Continuous wheat amplifies *P. graminis* inoculum, and SBWM-susceptible cultivars increase the proportion of viruliferous zoospores. However, because of the long-term

survival of the virus in *P. graminis* resting spores, crop rotation is not an effective management strategy for SBWM.

Sanitation

Any equipment or mechanism that transports soil has the potential to transport SBWMV-infested inoculum. In growers' fields in New York State, where SBWMV was recently introduced, we observe SBWMV-infected patches most often associated with areas where machinery first entered the field. This observation stresses the importance of sanitation (e.g., cleaning machinery) to avoid the introduction of new soil-borne viruses (Figure 16).

Lecture No. 5 Bajra diseases – Downy mildew/green ear, rust, ergot, smut.

Downy mildew or Green ear

Sclerospora graminicola

Economic importance

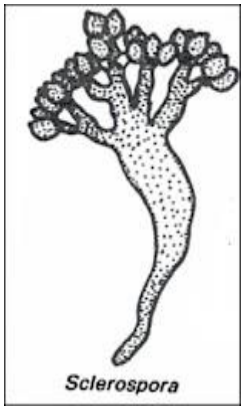
It occurs in many parts of Africa, as well as in India, where it was first reported by Butler in 1907. Disease is severe in **ill drained** and **low lying** areas. Losses due to the disease may be as high as 30-45 per cent in the high yielding varieties. The disease occurred in epidemic form in 1970 and 1983 devastating the popular hybrids, viz., **HB 3** and **BJ 104**.

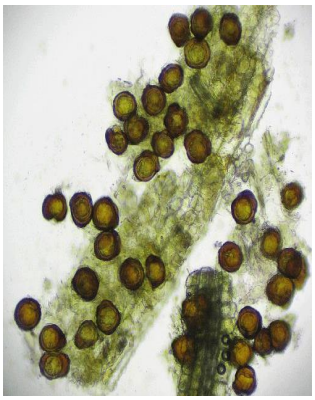
Symptoms

Infection is mainly systemic and symptoms appear on the leaves and the earhead. The first symptoms can appear in seedlings at three to four leaf stage. The affected leaves show patches of light green to light yellow colour on the upper surface of leaves and the corresponding lower surface bears **white downy growth** of the fungus. The downy growth seen on infected leaves consists of sporangiophores and sporangia. The **yellow discolouration** often turns to streaks along veins. The infected plants tiller excessively and are dwarfed. As the disease advances, the streaks turn brown and the

leaves shred at the tips only. But shredding is not as prominent as in Jowar.

In affected plants, ears fail to form or if formed, they are completely or partially malformed into twisted green leafy structures; hence the name **green ear** disease. The infection converts the various floral parts, including glumes, palea, stamens and pistil into green linear leafy structures of variable length. As the disease advances, the green leafy structures become brown and dry bearing masses of oospores.





Ears malformed into green leafy structures **Sporangiophore**

Oospores

Pathogen

The mycelium is systemic, non-septate and intercellular in the parenchymatous tissues. Short, stout, hyaline sporangiophores arise through stomata and branch irregularly to produce sterigmata bearing the sporangia. Sporangia are hyaline, thin walled and elliptical, and bear prominent papilla. Oospores are round in shape, surrounded by a smooth, thick and yellowish brown wall.

Disease cycle

P.I:The oospores remain viable in soil for five years or longer giving rise to the primary infection on the host seedling. Oospores attached to the seed also cause primary and systemic infection of seedlings. Secondary spread is through sporangia, which are active during rainy season, disseminated by air and water.

S.S.I: Secondary infection may not develop into systemic infection, but leads to local infection. The pathogen readily infects **teosinte** (*Euchlaena mexicana*) and ***Setaria italica***.

Favourable Conditions

Formation of sporangiophores and sporangia is favoured by very high humidity (90 per cent), presence of water on the leaves and low temperature of 15-25^{O}C.

Management

- Selection of seed from healthy crop
- Collect diseased plants, especially before oospores are formed, and burn them
- Summer deep ploughing
- Rogue out infected plants.
- Prolonged crop rotation
- Grow resistant varities like **WCC 75**, PHB 10, ICMH 451 ICTP 8203, Mallikarjuna, HB-1, HB 5 and PHB 14
- Grow tolerant varieties like MBH 118, CM 46, Balaji composite, Nagarjuna composite, Visakha composite, New vijaya composite, RBS 2, etc.
- Treat the seeds with Metalaxyl (Apron 35SD)@6g/kg
- Spray Mancozeb@0.25% or Metalaxyl (Ridomil MZ)@0.2% starting from 30 days after sowing in the field.

Rust

Puccinia penniseti

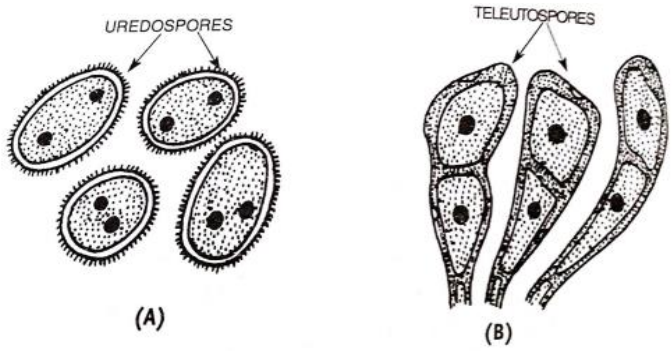
Economic importance

Rust occurs in all Bajra growing areas.

Symptoms

Symptoms first appear mostly on lower leaves as minute, round raised reddish brown pustules. Uredosori occur in groups on both surfaces of leaf and leaf sheath. The

pustules may also be formed on stem and peduncles. Dark brown to black teliospores are produced late in the season in the uredosori or teleutosori. In severe infections, whole leaf may wither completely presenting a scorched appearance to the field.



26.10. **(A)** *Uredospores, and* **(B)** teleutospores of *Puccinia penniseti.*

Reddish brown pustules on leaf

Pathogen

The rust is **heteroecious**. The fungus has a long life cycle producing uredial and telial stages on bajra and aecial and pycnial stages on several species of ***Solanum***, including **brinjal**. *(Solanum melongena)*. Uredospores are oval, elliptic or pyriform with four germpores, sparsely echinulated and pedicellate. Teliospores are dark brown in colour, 2 celled, cylindrical to club shaped, apex flattered, broad at top and tapering towards base.

Favourable Conditions

Closer spacing, presence of abundant brinjal plants and other species of *Solanum,* viz., *S.torvum, S. xanthocarpum* and *S. pubescens, S. panduriforme.* The uredial stage also

occurs on the species of *Pennisetum*, including *P. leonis, P. purpureum, P. orientale, P. spicatum* and *P. polystachyon*.

Disease cycle

P.I:Primary infection is from the alternate host, brinjal, in nature.

S.S.I:Secondary spread is through wind borne uredospores. The uredial stages also occur on several species of *Pennisetum.*

Management

- Removal and destruction of alternate hosts
- Spray thrice at 15 days interval with Wettable Sulphur@0.3% starting from 21 days after planting
- Grow resistant varieties like RT 814-3, PT 826/4, PT 829/5, etc.
- Spraying hexaconazole @ 0.1%is most effective

Ergot or Sugary disease

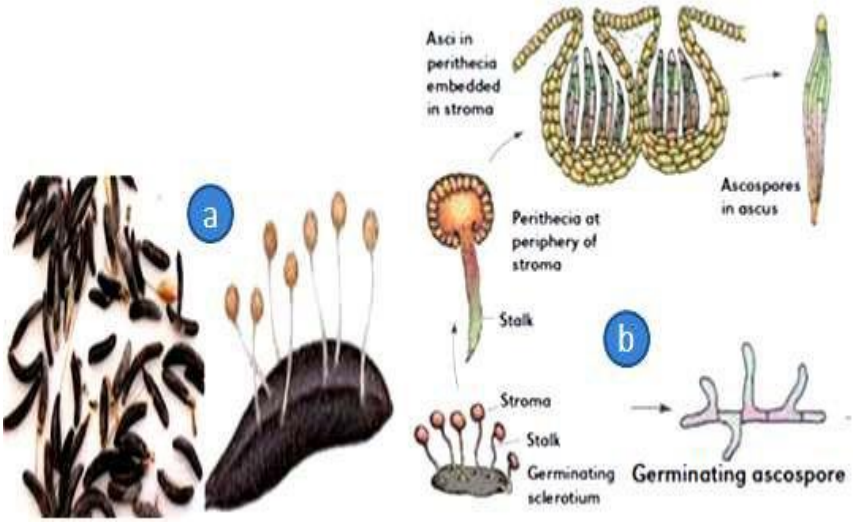
Claviceps fusiformis or C. microcephala

Economic importance

During 1967-78, the disease broke out in epidemic proportions on newly introduced hybrid Bajra varieties. On HB-1 and HB-2 hybrids, the disease occurred in epidemic form and caused 25% losses in grain yield in Bagalkot, Belgaum and Bijapur areas of Karnataka. In severe infections, 41 to 70% yield losses are also reported.

Symptoms

The symptom is seen by exudation of small droplets of light pinkish or brownish sticky fluid (honey dew) from the infected spikelets. Under severe infection many such spikelets exude plenty of honey dew which trickles along the earhead onto the upper leaves making them sticky. This attracts several insects. In the later stages, the infected ovary turns into small dark brown sclerotial bodies larger than the seed and with a pointed apex which protrude from the florets in place of grain.



Honey dew secretions from panicle

Pathogen

The fungus attacks the ovary and grows profusely producing masses of hyphae which form sclerotial bodies. The pathogen produces septate mycelium which produces conidiophores which are closely arranged. Conidia are hyaline and one celled. The **sclerotia** are small and dark grey but white inside. Sclerotia are 3-8 mm long and 0.3-15 mm broad.

Disease cycle

P.I:Sclerotia are viable in soil for 6-8 months. The primary infection takes place by germinating sclerotia present in the soilThe role of collateral hosts like *Cenchrus ciliaris* and *C. setigerus* in perpetuation of fungus is significant. The fungus also infects other

species of *Pennisetum.*

S.SI:Secondary spread is by insects or air-borne conidia (*Sphacelia sp.)*and ascospores.

Favourable conditions

Flowers are susceptible to the infection only after stigma emergence and before pollination and fertilization. Overcast sky, drizzling rain with a temperature of 20-30^{0}C during flowering period, favour the disease development.

Management

- Adjust the sowing date so that the crop does not flower during September when high rainfall and high relative humidity favour the disease spread.
- Immerse the seeds in 10 per cent common salt solution and remove the floating sclerotia.
- Eradication of collateral hosts
- Grow resistant varieties like PHB 10, 14; Co 2, 3 and Bajra 24.
- Spray with carbendazim 12%+ Mancozeb63%@0.2% at boot leaf and flowering stage

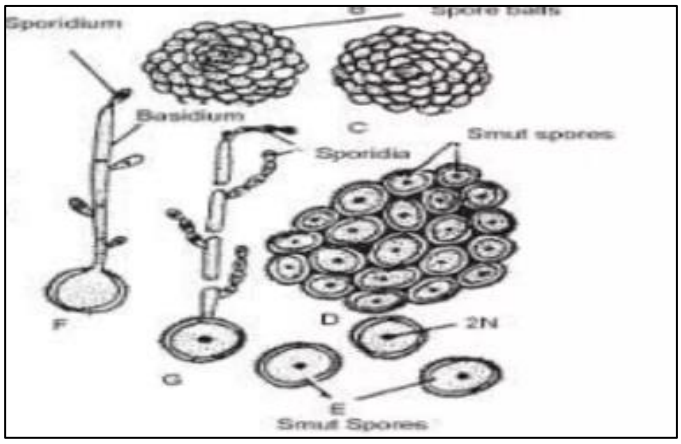
Bajra smut

(*Tolyposporium penicillariae*) *(Syn:Moesziomyces bullatus)*

Symptoms

Symptoms of the disease become apparent at the time of grain setting. The pathogen infects few florets and transforms them into large oval shaped sacs (sori) containing black powder (smut spores). Initially the sori are larger and greener than normal healthy grains and when the sori mature they become dark brown and are easily broken and release millions of black smut spore balls.

Smut sori on panicle



SPORE BALLS

Pathogen

The fungus infects developing flowers and the mycelium aggravates in the ovary and rounds off into chlamydospores. Meanwhile, a wall partly of host and partly of fungus tissues forms into a sorus. The fungus is mostly confined to the sorus. The sori contain spores which are usually in balls and are not easy to separate. No columella is present. Each spore is angular to round and light brown coloured with a rough wall. the spores germinate to produce four celled promycelium on which the sporidia are formed.

Disease cycle

P.I:It survives as spore balls in the **seed** and **soil** and serves as primary source of inoculum. The air borne spores germinate to produce the sporidia that enter the spikelets and infect the ovary.

S.SI: The secondary spread is through wind borne chlamydospores.

Favourable Conditions

Spikelets are mostly susceptible before stigma and anthers come out. High humidity and successive cropping with bajra is conducive for disease occurrence.

Management

- Removal and destruction of affected earheads
- Use Trichoderma virens (6g/kg seeds) and raw cow milk or raw goat milk (about 18h 50% dilution with water) as seed treatment.

- Grow resistant varieties like DC 7, MPP 7131 and MPP 7108.
- Spray Carboxin @0.2%

Ragi/Fingermillet diseases- blast, smut, mosaic and leaf spot

Blast or Gray leaf spot

Pyricularia grisea

Economic importance

It is the most important disease on ragi. It causes heavy damage to the crop under favourable environmental conditions. In **Chittoor** district of A.P, it is more or less endemic. Yield loss may range from 50 to 90%.

Symptoms

Infection may occur at all stages of plant growth. Young seedlings may be blasted or blighted in the nursery bed as well as developing young plants in the main field. There are three stages in disease development.

Leaf blast: It is more severe in tillering phase. The disease is characterized by spindle shaped spots on the leaves with gray centres surrounded by reddish brown margins.

Node blast: Infection on stem causes blackening of the nodal region and the nodes break at the point of infection. All the parts above the infected node die.

Neck blast: At flowering stage, the neck just below the earhead is affected and turns sooty black in colour and usually breaks at this point. In early neck infections, the entire earhead becomes chaffy and there is no rain set at all. If grain setting occurs, they are shrivelled and reduced in size.

Spindle shaped spots on the leaves with

Blackened node(node blast)

gray centres surrounded by reddish brown

margins.

Chaffy ear head

Pathogen

Young hyphae are hyaline and septate and turns to brown when become old. Numerous conidiophores and conidia are formed in the middle portion of the lesions. Conidiophores are slender, thin walled, emerging singly or in groups, unbranched, and pale brown in colour. Conidia are thin walled, sub-pyriform, hyaline 1-2 septate, mostly 3 celled with a prominent hilum.

Pyriform 3 celled conidia

Disease cycle

P.I:The fungus is seed-borne and the primary infection takes place through the seed-borne conidia and also through diseased plants, stubbles and weeds.

S.S.I:The secondary spread is through air-borne conidia.

Favourable Conditions

Application of high doses of nitrogenous fertilizers, low night (20^0C) and day (30^0C) temperatures with high relative humidity (92-95%) and rain or continuous drizzles favour the disease development. Presence of collateral hosts like bajra, wheat, barley and oats.

Management

- Destruction of collateral hosts and infected plant debris

- Treat the seeds with mancozeb+ Carbendazim at 2 g/kg.
- Grow resistant varieties like **Ratnagiri, Padmavati, Gowtami and Godavari**
- Spray with Iprobenphos (IBP)@0.1% first spray immediately after symptom appearance and second spray at flowering stage.

Smut

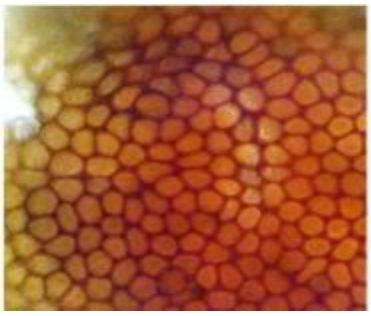
Melanopsichium eleusinis

Economic importance

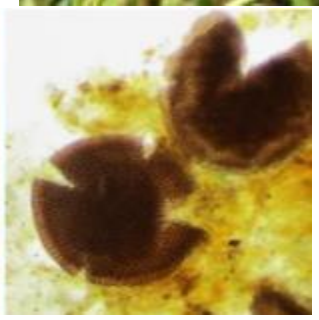
The disease is of minor importance being found only in certain places of Karnataka and Maharashtra.

Symptoms

Disease appears mostly during kharif at grain setting stage. Only few scattered grains in a head are attacked and transformed into globose galls of 5-15 mm diameter, greenish at first and turning black at maturity. The sorus ruptures releasing black mass of spore



Spore ball



Crushed layer of sporeball

Pathogen

The fungus is mostly confined to the spikelets, being present in the form of hyphae with thickened cells or **chlamydospores**. The spores are globose with a rough, spiny or pitted

spore wall. They measure 7-11µ in diameter and readily germinate in water producing **sporidia** on septate promycelium.

Disease cycle

P.I:The disease is mainly air borne, infecting only few spikelets in the panicle. The spores are released from the sac while on the panicle or they may reach the soil subsequent to harvest.

S.S.I:During the following season the spores germinate to produce masses of sporidia which become air borne and infect spikelets.

Management

- Crop rotation
- Rouging and destruction of affected earheads reduces smut incidence.
- Grow resistant varieties

Mosaic

Sugarcane mosaic virus

Economic importance

During 1965-67 this disease occurred in epidemic form in **Chittoor** and some parts of Karnataka. The disease is severe in Karnataka and Andhra Pradesh in summer crop. 100% loss in grain yield was reported.

Symptoms

All stages of crop growth are susceptible to disease and prominent symptoms are noticed from 4-6 weeks after planting. Leaves become chlorotic, mottled, plants stunted and inflorescence may become **sterile**. Ears, if formed are chaffy. In advanced stages plants wither prematurely.

Chlorotic,mottled plants

Disease cycle

P.I:Besides ragi, the virus also infects Setaria, maize, sugarcane and sorghum

S.S.I: is transmitted by **aphids**. The principal vectors are *Rophalosiphum maydis, Aphis gossypii* and *Myzus persicae*.

Management

- Application of phosphatic fertilizers
- Rogue out infected plants and destroy
- Spray dimethoate@2ml/lt to control the vector

Lecture 6.DISEASES OF SORGHUM *(SORGHUM BICOLOR)*

Anthracnose or red leaf spot

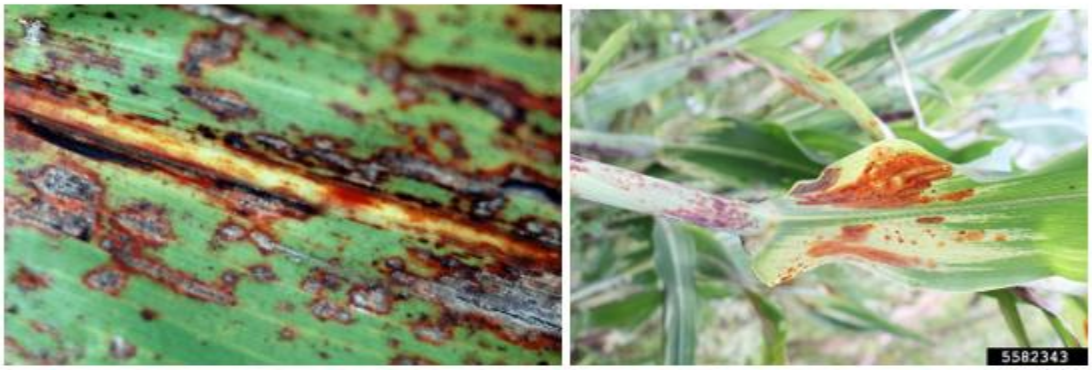
*Colletotrichum graminicola (*Syn:*Colletotrichum sublineola* -
Teliomorph stage : *Glomerella graminicola*)

Economic importance

This is wide spread and prevalent in all sorghum growing areas. In India anthracnose is severe in Andhra Pradesh, Madhya Pradesh, Rajasthan, Tamil Nadu and Delhi.

Symptoms

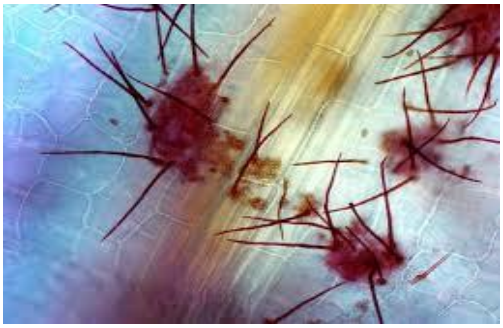
The fungus causes both leaf spot (anthracnose) and stalk rot (red rot) in sorghum. The disease appears as small red coloured spots on both surfaces of the leaf. The centre of the spot is white in colour encircled by red, purple or brown margin. Numerous small black dots are seen on the white surface of the lesions which are the fruiting bodies (acervuli) of the fungus. Many lesions coalesce and kill large leaf portions. In midrib region, elongate elliptical, red or purple regions with black acervuli are formed. Stalk and inflorescence infection can be characterized externally by the development of circular cankers. Infected stem when split open shows discoloration, which may be continuous over a large area or more generally discontinuous giving the stem a marbled appearance. The stem lesion also shows acervuli.

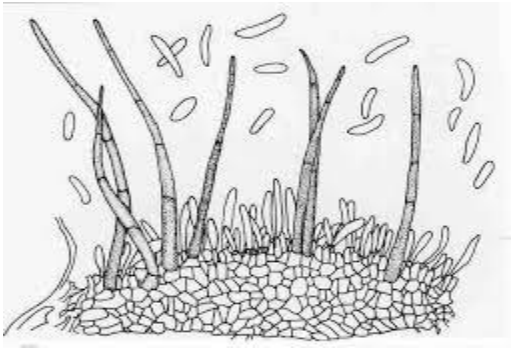


Small red coloured spots on both surfaces of the leaf with whitish centre with red, purple or brown margin along with black dot like acervuli on leaf and leaf sheath

Etiology

The mycelium of the fungus is localized in the spot. Arvuli with long dark setae arise through epidermis. The conidiophores are short, single celled and colourless. Conidia are short, hyaline, **single celled**, vacuolate and **falcate** in shape.





Section showing black acervuli

Falcate conidia

Disease cycle

Fungus has wide host range and survives on **Johnson grass, Sudan grass, maize, barley** and **wheat**. Also survives in seed and infected plant debris. Primary infection is from the conidia produced on the infected plant debris and infected seed. Disease spread within the season is through air borne conidia, which are produced on first infected plants.

Favourable Conditions

Continuous rain, temperature of 28-30^{0}C and high humidity aggravates the disease.

Management

- Destruction of infected plant debris and collateral hosts
- Crop rotation with non-host crops
- Grow resistant varieties like SPV 162, CSV 17, Texas Milo and Tift sudan etc.
- Seed treatment with Carbendazim 12% + Mancozeb 63% wp) @ 0.3% + two foliar spray of neem oil @ 0.5%
- Spray the crop with Pyraclostrobin+fluxapyroxad@0.29 l/ha

Rust

Puccinia purpurea

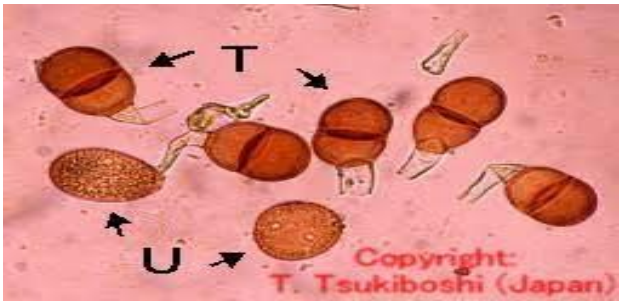
Economic importance: Occurs in warmer regions. In India it is recorded in all states. Damage caused by rust depends on the time of infection and varieties affected. If infection occurs early the premature drying of leaves results in reduction of yields. In India rust is prevalent in all seasons. Both irrigated and rainfed crops are damaged.

Symptoms

The fungus affects the crop at all stages of growth. The intensity of rust infection is generally severe after flag leaf stage of the crop growth. The first symptoms are small **reddish brown flecks** on the lower surface of the leaf (purple, tan or red depending upon the cultivar). Pustules (uredosori) appear on both surfaces of leaf as purplish spots which rupture to release reddish powdery masses of **uredospores**. The pustules are elliptical and lie between and parallel with the leaf veins. Pustules are surrounded by a reddish or yellow halo. In highly susceptible cultivars, the pustules occur so densely that almost the entire leaf is destroyed. **Teliopores** develop later sometimes in the old uredosori or in teliosori, which are darker and longer than the uredosori. The pustules may also occur on the leaf sheaths and on the stalks of inflorescence.

Reddish brown flecks on the lower surface of the leaf infected by rust

Pustules on stalk



Telio & Uredospore

Etiology

The uredospores are pedicellate, elliptical or oval, thin walled, echinulate and brown in colour with 4 to 5 germ pores. Club shaped paraphyses are also found in uredosorus. The teliospores are reddish or brown in colour and two celled, rounded at the apex with one germ pore in each cell. The teliospores germinate and produce promycelium and basidiospores. Basidiospores infect ***Oxalis corniculata*** (alternate host) where **pycnial** and **aecial** stages arise after infection.

Disease cycle

The fungus is **long cycled rust** with *Oxalis corniculata* as the alternate host with aecial and pycnial stages. Presence of alternate host helps in perpetuation of the fungus. The uredospores survive for a short time in soil and infected debris. Air borne uredospores help in the secondary spread of the disease.

Favourable Conditions

Low temperature of 10 to 12^{0}C favours teliospore germination and a spell of rainy weather favours the onset of the disease.

Management

- Grow resistant varieties like CSH 5, SPV 13, 81, 126, PSH 1, CSV 17, etc.
- Remove and destroy the alternate host *Oxalis corniculata.*
- Spray the crop with propiconazole @0.1%
- Dusting of sulphur@25 kg/ha

Ergot or Sugary disease

Claviceps sorghi or Sphacelia sorghi

Economic importance

Mc Rae first described this disease from Tamil Nadu. In South India, disease is prevalent during **October** to **January** when cold weather prevails at crop maturity. **CK 60 A**, male sterile line is highly susceptible

Symptoms

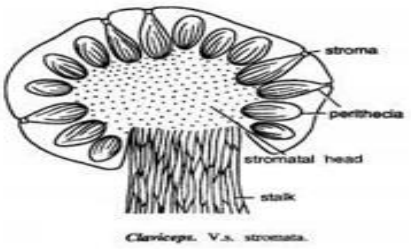
The disease is confined to individual spikelets. The first symptom is the secretion of honey dew (creamy sticky liquid) from infected florets. The honey dew secretion attracts large number of insects and ants which help in spreading the disease. Often the honey dew is colonized by *Cerebella sorghivulgaris* which gives the head a blackened appearance. Under favourable conditions, grain is replaced by long (1-2cm), straight or curved, cream to light brown, hard sclerotia. At the base of the affected plants white spots can be seen on the soil surface, denoting the drops of honey dew which had fallen on the soil.

Secretion of honey dew (creamy sticky liquid) from infected florets

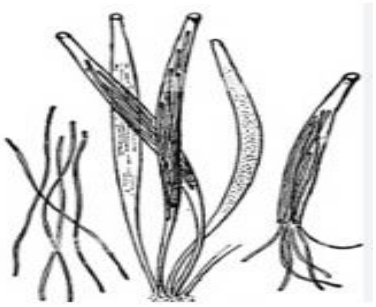
Black, convoluted mass in panicle due to the saprophyte Cerebella species associated with Sphacelia sorghi

Etiology

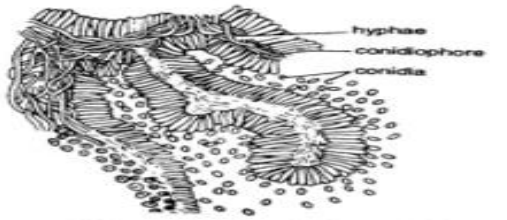
The fungus produces septate mycelium. The honey dew is a concentrated suspension of conidia, which are single celled, hyaline, elliptic or oblong in shape and slightly constricted in the middle. The sclerotial bodies produced by the fungus are 10-12mm long and 2mm thick, hard and tough



Claviceps. V.s. stromata.



Asci with filiform ascospores



Claviceps. Mycelium showing conidiophores.

Sphacelia sorghi conidia

Disease cycle

Primary source of infection is through the germination of sclerotia which produce ascospores, which infect the ovaries. The secondary spread takes place through air and insect borne conidia, which settle in the spikelets. Rain splashes also help in spreading the disease.

Favourable Conditions

A period of high rainfall and high humidity during flowering season. Cool night temperature (20-25 ^{0}C) and cloudy weather during anthesis encourages disease spread rapidly causing severe losses in hybrid seed production. Male sterile lines are highly susceptible.

Management

- Adjust the date of sowing so that the crop does not flower during the periods of high rainfall and high humidity.
- Grow resistant varieties like SPV 191, CSH 5, SPH 1 and CS3541.
- Deep summer ploughing
- Soaking seeds with 2% saline solution will aid to remove ergot infested seeds, as ergot infested seeds will float in the salt solution.
- Seed treatment with fungicides such as propiconazole@0.1 %

- Spray tebuconazole@0.1% at emergence of earhead (5-10 per cent flowering stage) followed by a spray at 50 per cent flowering and repeat the spray after a week, if necessary.
- Control of ergot with fungicides such as Propiconazole or Tebuconazole has proved to be cost effective in seed production plots.

Head mould/Grain mould/Head blight

More than thirty two genera of fungi were found to occur on the grains of sorghum. Some of them are pathogens, while many others are only saprophytes.

Symptoms

If rains occur during the flowering and grain filling stages, severe grain moulding can occur. Infected grains are covered with pink or black mold and such grains disintegrate during threshing process. *Fusarium semitectum* and *F.moniliforme* develop a fluffy white or pinkish colouration. *C. lunata* colours the grain black.

Discolured and moldy grains in panicle

Fungi from many genera have been isolated from the infected sorghum grains and the most frequently occurring genera are *Fusarium, Curvularia, Alternaria, Aspergillus, Cheatomium, Rhizopus, Helminthosporium* and *Phoma.* Moldy grains contain toxic mycotoxins and are unfit for human consumption and cattle feed.

Disease cycle

The fungi mainly spread through air-borne conidia. The fungi survive as parasites as well as saprophytes in the infected plant debris.

Favourable Conditions

Wet weather following the flowering favours grain mould development and the longer the wet period the greater the mould development. Compact ear heads are highly susceptible.

Management

- Adjust the sowing time.
- Grow resistant varieties like GMRP 4, GMRP 9, GMRP 13 and tolerant varieties like CSV 15.
- Seed disinfestation with propiconazole@0.1% will prevent seedling infection.
- Spray carbendazim 12%+Mancozeb 63%@0.25%, in case of intermittent rainfall during earhead emergence, a week later and during milky stage.

Grain smut/Kernel smut / Covered smut / Short smut

Sphacelotheca sorghi or Sporisorium sorghi

Economic importance

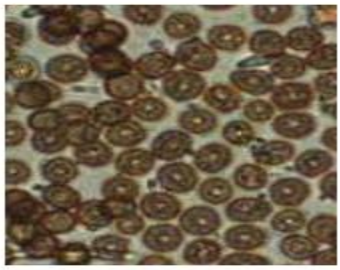
It is considered as the most destructive disease among all the smuts of sorghum. The extent of damage is even upto 25% of the grain yield. It is widely prevalent in Maharashtra, UP, AP, Tamil Nadu, Gujarat and Karnataka when the seed is not disinfected with fungicides. However, recently the losses due to this disease are reduced since the seeds are being treated by fungicides before sowing. Most varieties of the cultivated species of *Sorghum vulgare* are susceptible, along with ***S. halapense*** and ***S. sudanens***.

Symptoms

The disease becomes apparent only at the time of grain formation in the ear. The **individual grains** are replaced by **smut sori** which can be localized at a particular part of the head or occur over the entire inflorescence. The sori are dirty white to gray in colour, oval or cylindrical and are covered with a tough white cream to light brown skin (**peridium**) which often persists unbroken upto threshing. The glumes are unaltered and may be found adhering to the sides of the sorus. Sometimes the stamens may develop normally protruding out of the sorus. The size, colour and degree of breakage of the sori vary considerably with race of the fungus and the sorghum cultivar. Ratoon crops exhibit higher incidence of disease.

Infected panicle with smut sori

Promycelium



Smut spores

Etiology

The fungus is systemic. The mycelium occupies the growing point of the seedling and continues to grow along the plant without producing any external symptoms until the earhead is put forth. The mycelium aggregates in the immature ovary and the chlamydospores are formed by the rounding off of the mycelium. The sorus wall is formed mean while mainly by the outer layer of the mycelium, and partly by the host tissue. The fungus is present in the form of sorus, which has a tough wall and a long,

hard, central tissue called **columellum**. The columella is bulbous at the base and narrowed towards the tip. A dense mass of black to dark brown, smooth, thick walled spores, which are mostly single and measure 5-9µ in diameter, fill the space between the columellum and sorus wall. They germinate immediately if moisture is available, usually by producing a four celled promycelium which buds off sporidia.

Disease cycle

The disease is **externally seed borne** and **systemic**. The spores germinate with the seed and infect the seed by penetrating through the radicle or mesocotyl to establish systemic infection that develops along the meristematic tissues. At the time of flowering, the fungal hyphae get converted into spores, replacing the ovary with the sori. If the diseased ears are harvested with the healthy ones and threshed together, the healthy grains become contaminated with the smut spores released from the bursting of the sori. The spores remain dormant on the seed until next season.

Management

- Use disease free seeds.
- Grow resistant varieties like T 29/1, PJ 7K, PJ 23K, Nandyal and Bilichigan.
- Treat the seed with fine sulphur powder @0.5% or carboxin@0.1% or metalaxyl@0.25%
- Follow crop rotation.
- Collect the smutted ear heads in cloth bags and dip in boiling water.

Charcoal rot or **hollow stem** or **stalk rot blight**

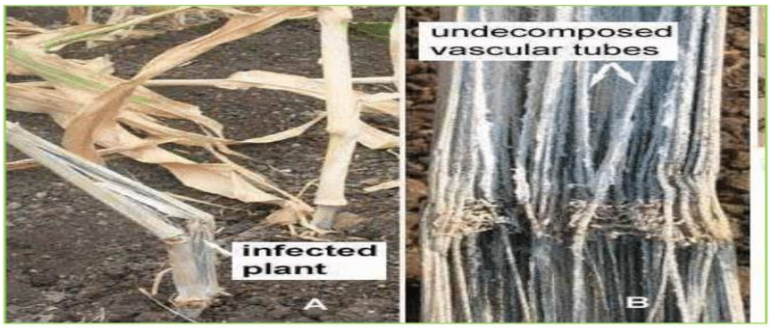
Macrophomina phaseolina

Economic importance

It is a major problem in the warmer regions. It causes seedling blight and stalk rot of plants. It is severe in Kurnool and Khammam districts of A.P.

Symptoms

This disease is characterized by sudden wilting and death of the diseased plant resulting in lodging. If the infected stalk is split open, the pith is found to be disintegrated with longitudinal **shredding of the tissue** into fibers. Small black sclerotial bodies are seen in the infected tissues. The stalk is weak, hollow inside and break easily. The stem, breaks near the ground level. Premature ripening takes place and the heads are poorly developed.



Blackening of roots **Shredding of tissue**

Disease cycle

Pathogen survives in soil, plant debris and many cultivated and wild plants. Secondary spread is through sclerotial bodies.

Favourable conditions

Disease is favoured by soil temperature of 35^0C and moisture stress conditions preceding crop maturity and application of more nitrogenous fertilizers.

Management

- Thin plant population should be maintained in problematic areas (60,000 plants/ha)
- The infected plants along with trash should be collected and burnt immediately
- Avoid moisture stress at flowering
- Treat the seeds with *Trichoderma asperellum @ 4 g/kg of seed.*
- Grow resistant varieties like E-36-1, CSV 5, CSH 7-R, SPV 126 and SPV 193.

Downy Mildew or **Leaf shredding**

Peronosclerospora sorghi

Disease is severe in delta regions of Andhra Pradesh where cool humid conditions prevail.

Symptoms

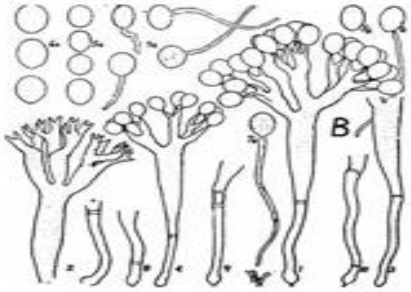
The first few leaves that show symptoms are only partially infected with green or yellow colouration of the infected portion. Abundant **downy whitish growth** is produced on the lower surface of the leaves. The downy growth spreads over a major portion of the upper surface. As the plant grows, **white streaks** appear on both the surfaces of the leaves. The tissues then tear along the streaks causing shredding of the leaves which is the most characteristic symptom and hence the name **leaf shredding**. The tissue then turns brown in colour. Numerous oospores are found in the shredded leaves. The affected parts are stunted and sterile. In the standing crop healthy plants are infected

due to secondary infection by sporangia.**Crazy top symptom(distorted ,twisted leaves) in sorghum is due to *Sclerophthora macrospora***

White streaks appear on both the surfaces of the leaves

Crazy top symptoms in sorghum

Leaf shredding and brownish tissue



Peronosclerospora with stout sporangiophore with dichotomous branching with non-papillate sporangia

Pathogen biology

P. sorghi is systemic in young host plant in the form of intercellular, non-septate mycelium. It is an obligate parasite. Sporangiophores emerge through the stomata in single or in clusters which are stout and dichotomously branched with pointed sterigmata. On each sterigmata a single hyaline, globose thin walled and non-papillate sporangium is formed which germinates directly by a germ tube without any zoospores. Oospores are typically produced abundantly in parallel bands between fibro vascular strands of the shredded leaf tissue which are three walled, more or less round, thick walled and golden yellow in colour.

Disease cycle

The primary infection is by means of **oospores** present in the soil which germinate and initiate the systemic infection. The oospores persist in the soil for several years. Presence of mycelium of the fungus in the seeds of systemically infected plants is also a source of infection. Secondary spread is by means of **air-borne sporangia**.

Favourable Conditions

Maximum sporulation takes place at 100 per cent relative humidity. Optimum temperature for sporulation is 21-23^{0}C. Light drizzling accompanied by cool weather is highly favourable.

Management

- Destroyal of affected plants by burning before oospore formation, reduce the inoculum potential
- Crop rotation with other crops like pulses and oilseeds.
- Grow tolerant varieties like CSH 2, CSV 5, SPV 101, 165 and 190.
- Seed treatment with Metalaxyl (Apron 35 SD) @4g/kg seed
- Spray Metalaxyl (Ridomyl MZ) @0.2%

Phanerogamic parasite (*Striga or Witch Weed)*

Striga asiatica and Striga densiflora

Symptoms

The root exudates of sorghum stimulate the seeds of the parasite to germinate. The parasite then slowly attach to the root of the host by haustoria and grow below the soil surface and produce underground stems and roots for about 1-2 months. The parasite grows faster and appears at the base of the plant. Severe infestation causes yellowing and wilting of the host leaves. The infected plants are stunted in growth and may die prior to seed setting, if the infection occurs early

Etiology

It is a partial root parasite and occurs mainly in the rainfed sorghum. It is a small plant with bright green leaves, grows upto a height of 15-30 cm. It always occurs in clusters of 10-20/host plant. *S. asiatica* produces red to pink flowers while. *S. densiflora* produces white flowers. Each fruit contains minute seeds in abundance which survives in the soil for several years.

Disease cycle

In the absence of host, the seeds remain viable in soil for many years. The seeds can germinate only in contact with roots of host. Seeds can germinate even one foot below soil. Soil temperature of 35^0C and soil moisture of 30 per cent is favourable for Striga infection.

Management

- Hand weeding of the parasites before flowering
- Crop rotation with cowpea, groundnut and sunflower
- Mixing of **ethrel** with soil triggers germination of Striga in the absence of host. After germination, Striga can be removed and destroyed.
- Spray **Fernoxone** (sodium salt of 2, 4-D) or Agroxone (MCPA) at 450g/500 liters of water
- 1% Tetrachloro dimethyl phenoxy acetate can be used for instant killing of Striga, if water is in scarce.

Lecture 7&8 Maize- economic importance, symptoms, cause, disease cycle and integrated management- turcicum leaf blight, post flowering stalk rot and *Cephalosporium* wilt, charcoal rot, banded leaf and sheath blight and downy mildew, Yellows disease

Turcicum Leaf blight

Setosphaeria turcica/Exserohilum turcicum

Northern corn blight(Helminthosporium turcicum)

Helminthosporium maydis-Southern corn blight

Economic importance

The pathogen is spread worldwide. In the USA it occurs mainly in the northern Corn Belt. Therefore this pattern of damage is known as “Northern corn leaf blight” .In Andhra Pradesh, it is severe in the districts of Warangal, Karimnagar, Medak, Nizamabad, Adilabad and Ranga Reddy.

Symptoms

Disease is characterized by long **elliptical** grayish green or tan lesions on the leaves measuring 2.5 to 25 cm in length and upto 4 cm in width. The fungus affects the maize

plant at young stage. Small yellowish round to oval spots are seen on the leaves. The spots gradually increase in area into bigger elliptical spots and are straw to grayish brown colour in the centre with dark brown margins. The spots coalesce to form bigger spots and gives blighted appearance. The surface is covered with olive green velvety masses of conidia and conidiophores. Under high humidity the whole leaf area becomes necrotic and plant appears as dead. Lesions may be extended to husk. Southern corn leaf blight lesions typically remain in the lower canopy and are smaller than lesions of NCLB, but can resemble lesions resulting from a resistant (hybrid) reaction to NCLB.

Figure 10. Southern corn leaf blight

Southern corn leaf blight is caused by the fungus Helminthosporium maydis/*Bipolaris maydis*. There are two races of the pathogen. Race O normally attacks only leaves. Lesions are tan, somewhat rectangular in shape, and have reddish-brown margins. Race T attacks leaves, husks, stalks, leaf sheaths, shanks, ears, and cobs. Race T lesions are rectangular to elliptical (¼ to ½ inch wide by ¼ to ¾ inch long) with a reddish-brown border that may be surrounded by a chlorotic zone. Stalk and leaf infections initially appear as purple spots that develop tan-gray centers. Ear infections result in a gray-black lesion on

the husk that extends into the kernels, appearing as a black felt-like mold. Seedlings from infected kernels are often blighted.

Resistant hybrids and inbreds are available. Foliar fungicides labeled for southern corn leaf blight are available.

Southern corn leaf blight lesions.
Image: C. Grau

Ear mold from southern corn leaf blight.

Large elliptical spots with straw to grayish brown colour in the centre with dark brown margins(Northern corn blight)

Spots on cob

Distinctly curved, fusiform, and pale to golden brown conidia

Pathogen

Conidiophores are in group, **geniculate**, mid dark brown, pale near the apex and smooth. Conidia are distinctly curved, fusiform, and pale to golden brown with 5-11 pseudosepta.

Disease cycle

P.I:Fungus survives in plant debris, seed and collateral hosts. The fungus is **externally seed borne**. It also infects Sudan grass, Johnson grass, sorghum, wheat, barley, oats, sugarcane and spores of the fungus are also found to associate with seeds of green gram, black gram, cowpea, and **Teosinte**. S.S.I:Secondary spread is through wind borne conidia.

Favourable Conditions

Optimum temperature for the germination of conidia is 18 to 27^{O}C provided with free water on the leaf. Infection takes place early in the wet season.

Management

- Crop rotation
- Grow resistant hybrids like DHM-1
- Treat the seeds with *Trichoderma harzianum@20g/kg seed*
- Spray zineb 68%+hexaconazole 4%@1250g/ha

Post flowering stalk rot

***Cephalosporium* wilt (Black bundle disease and late wilt)**

Cephalosporium acremonium(Acremonium strictum/

Cephalosporium maydis/Harpophora maydis

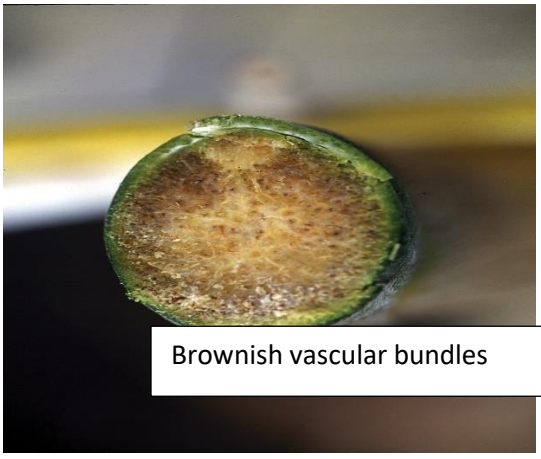
Symptoms

Infection caused by *C. acremonium* becomes apparent when maize has reached the dough stage. One of the first symptoms is the purpling of leaves and stalks. The most characteristic symptom is the **restricted blackening of vascular bundles** in the stalk with shredding of the intermodal pith region. Blackening of the vascular bundles extends through several internodes. Barren plants, excessive tillering and multiple ears are the other diagnostic symptoms.

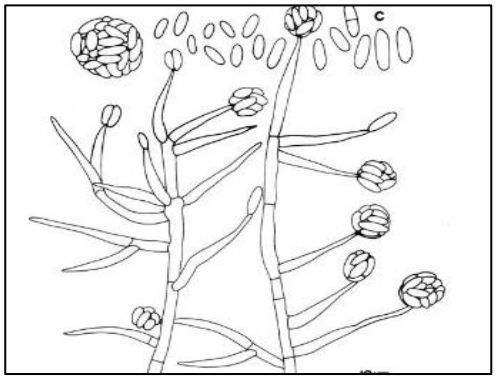
Symptoms caused by *C. maydis* appear only after flowering stage and plants start wilting basipetally giving a dull green appearance of the leaves which later dry up. The lower internode turns discoloured, become reddish brown, shrunken and soft, and subsequently becomes dry and hollow. When diseased stalks are split open, reddish brown vascular bundles are seen.

Barren plants, excessive tillering and multiple ears

Plants with excessive tillering and multiple ears



Brownish vascular bundles



Shredding of the intermodal pith region

Pathogen

Conidiophores swollen or slender; conidia are one celled, hyaline and are produced successively at the tip and usually embedded in a slimy drop. It produces antibiotics cephalosporins.

Disease cycle

P.I:Both the pathogens survive in soil, plant debris and seed

S.S.I:wind borne conidia

Favourable Conditions

High temperature and low soil moisture (drought) favour the disease.

Management

- Crop sanitation
- Crop rotation
- Avoid water stress at flowering
- Seed treatment with azoxystrobin@0.0025g/kg
- Grow resistant varieties like DHM-103, DHM 105, Hi-Starch and Ganga Safed-2.

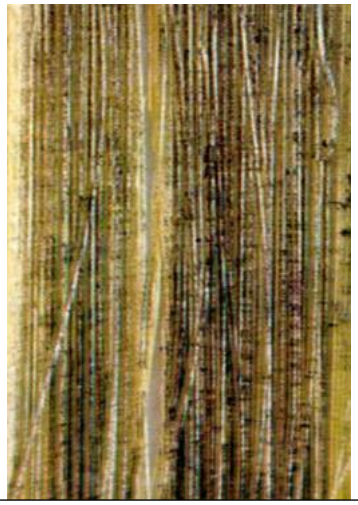
Charcoal rot

Macrophomina phaseolina(Sclerotial stage: Rhizoctonia bataticola)

Economic importance:Prevalent particularly in *Rabi*, when temperature during post-flowering periods become comparatively high (35-45^{0}C). Yield losses upto 10-50% are common.

Symptoms

Charcoal rot commonly attacks plants approaching maturity. The fungus produces brown, water soak lesions on the roots that later turn black. As the plant matures, the infection extends into the lower portions of the stem where gray streaks develop on the stem surface of lower internodes leading to premature ripening, shredding and breaking of the crown. Split open stalks have numerous which is a characteristic symptom of the disease. Sclerotia may also be found on the roots.



Black sclerotia on vascular strands, giving the interior of the stalks charred appearance

Pathogen

The fungus produces large number of sclerotia which are round and black in colour. Sometimes, it produces pycnidia on the stems or stalks.

Disease cycle

P.I:The fungus has a wide host range, attacking sorghum, bajra, ragi and pulses. It survives for more than 16 years in the infected plant debris. The primary source of infection

is through soil-borne sclerotia. S.S.I:Sclerotia spread through irrigation water.

Favourable Conditions

Development of charcoal rot is favoured by dry weather with high temperature (37^0C) at the time of silking. Imbalanced fertilizer application and high plant density influence disease prevalence and severity.

Management

- Long crop rotation with crops that are not natural host of the fungus.
- Field sanitation
- Irrigate the crops at the time of earhead emergence to maturity.
- Treat the seeds with *Trichoderma harzianum @20* g/kg.
- Grow disease resistant varieties, viz., DHM 103, DHM 105 and Ganga Safed 2.

Banded leaf and sheath blight

Rhizoctonia solani

(Perfect stage: *Thanetophorus sasakii*)

Symptoms

Large, discoloured areas alternating with irregular dark bands are typical symptoms of the disease. Severe infection leads to blotching of the leaf sheath as well as leaves. The symptoms under favourable conditions extend upto silk, glumes and kernels. Disease generally appears at pre-flowering stage. Symptoms also appear on stalk and the internodes break at the point of infection. Symptoms of this disease which develop on leaves and sheaths are characteristic **concentric spots that cover large areas of infected leaves and husks**. The main damage in the humid tropics is a brownish rotting of ears, which show conspicuous light brown cottony mold with small, round, black sclerotia. The developing ear is completely damaged and dries up prematurely with cracking of the husk leaves.

Large, discoloured areas alternating with irregular dark bands on leaf sheath,cobs

Survival

P.I: The fungus survives on weeds and in the plant debris.

S.S.I:Sclerotia spread through irrigation water

Management

- Clean cultivation
- Destruction of crop debris
- Removal of lower leaves nearer to soil
- Treat the seeds with *Trichoderma harzianum @20* g/kg
- Spray metalaxyl @0.1% or propiconazole@0.1%

Downy mildew

Sorghum DM - Peronosclerospora sorghi / P. philippinensis

Crazy top DM – Sclerophthora macrospora

Brown stripe DM – Sclerophthora rayssiae var zeae

Sugarcane DM – Peronosclerospora sacchari

Rajasthan DM-Peronosclerospora heteropogoni

Symptoms

The most characteristic symptom is the development of **chlorotic streaks** appears on the leaves and the plants exhibit a stunted and bushy appearance due to the shortening of the internodes. White downy growth can be seen not only on the lower surface of leaf but also on the chlorotic streaks. **Affected leaves often tear linearly causing leaf shredding**. The downy growth also occurs on bracts of green unopened male flowers in the tassel. The important symptom of the disease is the partial or complete malformation of the tassel into a mass of narrow, twisted leafy structures. **Proliferation of axillary buds on the stalk of tassel as well as the cobs is very common (Crazy top)**

Crazy top downy mildew

Crazy top is caused by infection of *Sclerophthora macrospora*, an oomycete pathogen. Crazy top-affected plants are distorted and/or stunted. The leaves below the tassel may proliferate, resulting in a very bushy appearance of the top of the plant, which lends this disease its name. Internodes may be short or long; there may be a proliferation of ear shoots, leaves that are narrow and strap-like, excessive tillering, or a complete lack of ear and tassel formation.Crazy top is more common in fields that are flooded early in the growing season. Proper soil drainage will reduce the risk of infection.

Tassel proliferation, resulting in a very bushy appearance of the top of the plant

Brown stripe downy mildew(*Sclerophthora rayssiae* var. *zeae*)

Lesions on the leaves develop as narrow chlorotic stripes, variable in length, 3-7 mm

wide. These stripes extend in parallel fashion and have well-defined margins because of their being vein limited. Stripes in advanced stages become necrotic and present a brown burnt appearance to the leaves. A fine downy growth can be seen on the stripes.Brown stripe downy mildew is not a systemic disease, and the pathogen will only be present in infected leaves.

Narrow chloratic/brown stripes that run parallel to veins

Sorghum downy mildew

Systemic infection in maize seedlings is characterized by chlorosis which normally appears two weeks after sowing. The leaves of infected plants tend to be narrower and more erect than those of healthy plants. Plants infected early usually die approximately four weeks after infection . In late infected plants, the chlorosis may be more noticeable on the lower half of the leaf which is often called **half-leaf symptom**. This chlorosis gradually covers the entire leaf surface at later stage. Under warm humid conditions, a white downy growth is produced on the lower leaf surface some times on both surfaces also. This growth is a combination of conidia and conidiophores. In maize, **leaf shredding is rare but it is common in sorghum** . At flowering stage of growth, infected plants produced a bushy top, referred to a crazy top in place of the tassel. Systemically infected maize plants generally do not form cob. In some cases when cobs formed, these are small and poorly filled.

Underside of maize leaf showing downy growth

Bushy top, referred to a crazy top in place of the tassel

when cobs formed small and poorly filled

Sugarcane downy mildew

Spots on leaves lead to internal infections causing **yellow to white stripes**, often along the length of leaves with downy growth and spores. Plants may be stunted, with deformed ears and tassels.

Spread is by wind-blown spores.

Photo 2. Spore masses of maize downy mildew, *Peronosclerospora sacchari*, on the white stripes (centre).

Yellowish to white stripes on leaf

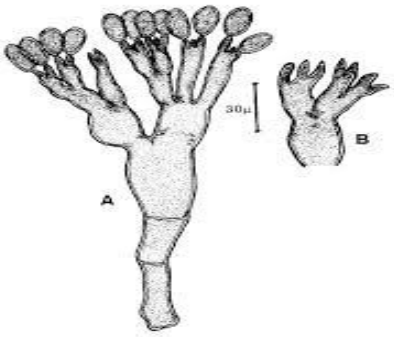
At first, small, round, yellow spots on the leaves leading to pale yellow to white stripes as the oomycete grows in the leaves . Several stripes may be present on each leaf, often extending the entire length. A white downy fungal growth develops on both sides of the leaves, and on the husks. The downy growth develops at night, especially when dew is present. The most identifiable symptoms are creamy white leaf stripes that turn red with age and stunting of infected stools. After warm humid nights, down may be seen on the

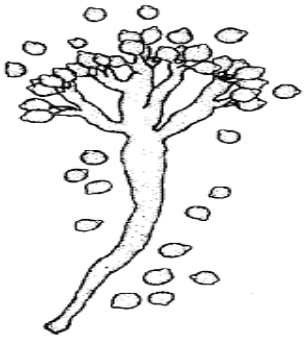
underside of leaves showing stripe symptoms. Less common symptoms include elongated, thin stalk growth (jump ups); brown lesions on external stalk surfaces and leaf shredding, tassels may be deformed, and ears may be aborted.

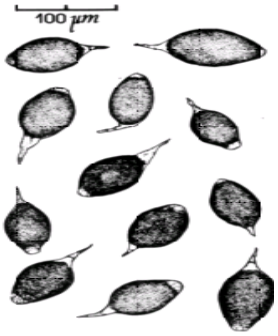
Philippine Downy mildew on maize: It is characterised by chlorotic stripes of leaves at early stage.Often older leaves become abnormally erect and dried.

Early symptoms of Philippine downy mildew as chlorotic stripes on leaves

Abnormal erect and dried leaves







Scleropthora macrospora **Sporangia of *Scleropthora***

Peronosclerospora sacchari

Pathogen

The fungus grows as white downy growth on both surface of the leaves, consist of sporangiophores and sporangia. Sporangiophores are quite short and stout, branch profusely into series of pointed sterigmata which bear hyaline, oblong or ovoid sporangia (conidia). Sporangia germinate directly and infect the plants. In advanced stages, oospores are formed which are spherical, thick walled and deep brown.

Favourable Conditions

High relative humidity (90 per cent), water logging condition, light drizzles with a temperature of 20-25^{0}C favours the disease development. Young plants are highly susceptible.**Zinc d**eficiency will predispose downy mildew.

Mode of Spread and Survival

P.I:The fungus survives in soil, plant debris and graminaceous collateral hosts (***Sorghum bicolor,***

Sorghum halapense, etc). In Punjab, ***Digitaria sanguinalis*** serve as primary source of infection. S.S.I:Secondary spread is through wind-borne sporangia

P.philippinensis and *P.sacchari* are morphologically identical but differ from each other in that the former does not produce oospores The **oospores** survive in the soil as well as in the infected plant debris. *P. philippinensis* survives on sorghum, kansas grass. Seed-borne presence of *P.phillipinensis* was mentioned in DM of maize while mycelium is present in all parts of the seeds,70% embryos contained oospores and 80% in P.sorghi.

- *P. maydis , P. sacchari , S. rayssiae zeae* : Internally seed borne

Management

- Destruction of plant debris
- Removal and destruction of collateral hosts
- Grow resistant hybrids like DHM-1, DHM-103, DMR-5 and Ganaga II.
- Seed treatment with Metalaxyl (Apron 35SD) at 4g/kg
- Deep summer ploughing
- Crop rotation with pulses
- Spray the crop, 3-4 times, with Metalaxyl MZ (Ridomil MZ)@0.2% starting from 20th day after sowing.

Yellows disease

A group of plant viruses and individual viruses have emerged as major threats to crop production worldwide. Emergent plant viruses are often mediated via an insect vector and with increasing global trade. Also, the emergence of a virus in a new geographical area may be initiated by the introduction of infected plant materials (propagative

materials or seeds). The novel virus, named Maize yellow mosaic virus (MaYMV) consists potentially in a new constraint to maize production worldwide. MaYMV epidemics are multi-component systems resulting from interactions among the viruses, vectors and host plants. MaYMV was transmitted by corn leaf aphid, Rhopalosiphum maidis, but not mechanically. In addition to maize, MaYMV was harbored by alternative hosts such as sugarcane, itch grass sugarcane, Panicum miliaceum, and Sorghum bicolor. Knowledge of the ways in which a virus maintains itself in the absence of a living host and spreads in the field is essential for the development of effective management measures. More research is needed to assess MaYMV in weeds and wild hosts, whether transmitted through seeds and the relative abilities of different aphid species to transmit MaYMV within and between different grass species to know the virus ecology and infection pathway to maize, to aid the development of an effective integrated disease management tactic.

Symptoms

The virus was identified from the symptomatic field-grown maize plants showing yellowing, whitish to yellow stripes, and mosaic on maize

The novel virus, called Maize yellow mosaic virus (MaYMV, classified in genus Polerovirus of the family Luteoviridae), was first reported to infect maize (Zea mays L.) in China in 2016 (Chen et al., 2016). All members of the Luteoviridae

MaYMV Alternative hosts In-plant virus disease management, it is important to understand the role of alternate hosts in the emergence and development of disease epidemics (Regassa et al., 2021). Since the emergence of MaYMV, this virus was found to infect diverse plants in the family Poaceae, including maize (Gonçalves et al., 2017), itch grass (Rottboellia cochinchinensis) and sugarcane (Saccharum spp.) in Nigeria (Yahaya et al., 2017), Panicum miliaceum, and Sorghum bicolorin South Korea (Lim et al., 2017). MaYMV infecting sugarcane also reported in China (Sun et al., 2019). In addition to maize, the detection and identification of MaYMV in perennial sugarcane, itch grass, Panicum miliaceum, and Sorghum bicolor, indicate their potential roles as reservoir hosts of the virus.

Management of MaYMV

Generally, plant virus disease management measures are aimed either at decreasing the virus source, or at preventing virus spread within the crop.

MaYMV is newly emerged in the world; and hence, much is not known about its epidemiology except vector (aphids) and few alternative hosts identified Plant virus diseases including MaYMV are intrinsically difficult to manage directly by use of chemical pesticides; however, integrated management methods which include cultural practices such as removal of infection sources, field sanitation, removal of alternative hosts, use of healthy seed (virus free seeds);

chemical pesticides to control insect vectors indirectly through seed treatment and foliar spray are the most possible management measures of plant viral diseases . The removal of symptomatic plants, known as roguing, is a phtyosanitary control measure that is widely used to remove sources of virus infection from within crops

11	**Lentil- economic importance, symptoms, cause, disease cycle and integrated management-Rust and wilt.** **Pigeonpea- economic importance, symptoms, cause, disease cycle and integrated management- *Phytophthora* blight, wilt and sterility mosaic**
12	**Soybean and Cowpea- economic importance, symptoms, cause, disease cycle and integrated management- rust, soybean mosaic, bacterial leaf spot and Cowpea mosaic**
13	**Rapeseed and Mustard- economic importance, symptoms, cause, disease cycle and integrated management- white rust, downy mildew, powdery mildew and *Alternaria* leaf spot**
14	**Sesame economic importance, symptoms, cause, disease cycle and integrated management- *Alternaria* leaf spot, powdery mildew, phyllody, *Macrophomina* stem rot and bacterial leaf spot**
15	**Linseed- economic importance, symptoms, cause, disease cycle and integrated management-Rust, Powdery mildew, Alternaria blight and wilt.**
16	**Sunflower- economic importance, symptoms, cause, disease cycle and integrated management- *Alternaria* leaf blight, rust, powdery mildew, head rot, sclerotial wilt, downy mildew, mosaic and sunflower necrosis virus.**

Lecture No.9 Gram- economic importance, symptoms, cause, disease cycle and integrated management- wilt, rust, *Ascochyta* blight, *Macrophomina* stem and root rot

BENGAL GRAM *(Cicer arietinum)*

Wilt

Fusarium oxysporum f.sp. ciceri

Symptoms

The disease occurs at two stages of crop growth, seedling stage and flowering stage or adult stage. The field symptoms of wilt are death of seedlings or adult plants in patches. Seedlings collapse and lie flat on the ground retaining their dull green colour. When split open or cut transversely, brown to black discolouration of the internal tissues can be seen. Grown up plants show typical symptoms of wilting, i.e., drooping of petioles, rachis and leaflets. All the leaves turn yellow and then light brown. **Vascular**

discolouration is observed on longitudinal splitting of stem. Sometimes only a few branches are affected, resulting in partial wilt.

Pathogen

The fungus produces hyaline to light brown, septate and profusely branched hyphae. **Microconidia** are oval to cylindrical, hyaline, single celled, normally arise on short conidiophores. **Macroconidia** which borne on branched conidiophores, are thin walled, 3 to 5 septate, fusoid and pointed at both ends. **Chlamydospores** are rough walled or smooth, terminal or intercalary, may be formed singly or in pairs in chains.

Disease cycle

The fungus may be **seed-borne** and survives in infected plant debris in **soil**.

P.S.I:The primary infection is through chlamydospores in soil, which remain viable upto next crop season. The weed hosts also serve as a source of inoculum.

S.S.I:The secondary spread is through irrigation water, cultural operations and implements.

Favourable Conditions

High soil temperature (Above 25^0C), high soil moisture, monocropping and presence of weed hosts like *Cyperus rotundus, Tribulus terrestris* and *Convolvulus arvensis.*

Management

- Treat the seeds with Carbendazim or Thiram at 2 g/kg or treat the seeds with *Trichoderma viride* at 4 g/kg or *Pseudonomas fluorescens* @ 10g/kg of seed.
- Apply heavy doses of organic manure or green manure.
- Follow 6-year crop rotation with non-host crops.
- Grow resistant cultures like **Kranthi** (ICCC 37), **Swetha** (ICCV-2), ICCV 10,

Avrodhi, G 24, C 214, BG 244, Pusa 212 and JG 315.

Rust

Uromyces ciceris-arietini

Symptoms

The infection appears as small oval, brown, powdery lesions on both the surface, especially on lower surface or leaf. The lesions, which are uredosori, cover the entire leaf surface. Sometimes a ring of small pustules can be seen around larger pustules which occur on both leaf surfaces. Late in the season dark teliosori appear on the leaves. The rust pustules may appear on petioles, stems and pods. It is heteroecious rust, but the pycnial and aecial stages are unknown.

Pathogen

The uredospores are spherical, brownish yellow in colour, loosely echinulated with 4-8 germ pores. Teliospores are round to oval, brown, single celled with thickened apex and the walls are rough, brown and warty.

Disease cycle

P.S.I:The fungus survives as uredospores in the legume weed ***Trigonella polycerata*** during summer months and serve as primary source of infection.

S.S.I:The spread is through wind- borne uredospores.

Management

- Destroy weed host.
- Dust Sulphur at 20 kg/h

Ascochyta blight

Ascochyta rabiei

(Perfect stage: Mycosphaerella pinodes)

Symptoms

All above ground parts of the plant are attacked. The disease is usually seen around flowering and podding time as patches of blighted plants in the field. On leaves, small water-soaked necrotic spots appear that enlarge rapidly under favourable conditions leading to blighting of leaves. Pycnidia are observed on the blighted parts. In hot dry weather, the infection remains in the form of discrete lesions on the leaves, stems, pods and seeds. On leaflets, the lesions are round or elongated, with grey centres surrounded by brownish margin. Similar spots may appear on the stem and pods. The spots on the stem and pods have **pycnidia** arranged in **concentric circles** as minute block dots. The stem and petioles usually break at the point of infection due to girdling. If the main stem is girdled at the collar region, the whole plant dies.

Pathogen

The fungus produces hyaline to brown and septate mycelium. Pycnidia are spherical to sub-globose with a prominent ostiole. Conidia are borne on short conidiophores inside the pycnidia. They are hyaline, oval to oblong, straight or slightly curved and single celled, occasionally bicelled. The perfect or perithecial stage is also seen on infected host tissues, usually after the plant is dead. The perithecia are globose, dark coloured and contain asci which are typically 8 spored. The ascospores are hyaline, thin walled and two celled.

Favourable Conditions

Night temperatures of 10^0C and day temperature of 20^0C, rains accompanied by cloudy weather and excessive canopy favour the disease spread.

Disease cycle

The fungus survives in the infected plant debris as pycnidia. The pathogen is also **externally** and **internally** seed-borne.

P.S.I:The primary spread is from seed-borne pycnidia and plant debris in the soil.

S.S.I:The secondary spreads is mainly through air-borne conidia. Rain splash also helps in the spread of the disease.

Management

- Grow resistant/tolerant varieties like **Gaurav**, C 235, G 543, GG 588, GG 688, BG 261 and GNJ 214.
- Remove and destroy the infected plant debris in the field.
- Follow crop rotation with cereals.
- Deep sowing of seeds, i.e., 15cm or deeper.
- Intercropping with wheat, barley and mustard.
- Treat the seeds with mancozeb+ Carbendazim 2 g /kg.
- Exposure of seed at 40-50^{0}C reduced the survival of *A. rabiei* by about 40-70 per cent.

Stem and Root rot or dry root rot

Rhizoctonia bataticola

(Pycnidial stage: *Macrophomina phaseolina*)

(Sexual stage: *Thanatephorus cucumeris)*

Symptoms

The disease generally appears around flowering and podding time in the form of scattered dried plants. The seedlings can also get infected. The first symptom of the disease is yellowing of the leaves. The affected leaves, petioles and leaflets droop within a day or two. The leaves and stems of the affected plants turn straw coloured and plants wilt within a week. The lower portion of the tap root usually remains in the soil when plants are uprooted. The tap root is dark showing signs of rotting and is devoid of most of the lateral and finer roots. Dark minute sclerotial bodies can be seen on the roots exposed or inside the wood.

Pathogen

The hyphae of the fungus are dark brown, filamentous and septate with constrictions in hyphal branches at the junction with main hypha. The sclerotia are brown and irregular in shape. The fungus has its sexual stage, *T. cucumeris,* which produces 2-4 basidiospores in terminal clusters on a celled hypha.

Disease cycle

P.S.I:The pathogen survives in the soil in infected host debris as sclerotia for several years.

S.S.I:The secondary spread is through farm implements, irrigation water and rain splash.

Favourable conditions

Maximum ambient temperatures above 30^0C, minimum above 20^0C, and moisture stress favour disease development.

Management

- Treat the seeds with seed pelleting with *Trichoderma viride* at 4 g/kg or *Pseudomonas fluorescens* @ 10g/kg of seed.
- Apply farmyard manure at 10 t/ha.
- Grow tolerant genotypes like ICCV 10

LECTURE 10 Urdbean and Mungbean- economic importance, symptoms, cause, disease cycle and integrated management- powdery mildew, rust, *Cercospora* leaf spot, *Corynespora* leaf spot, angular black spot, root rot, bacterial leaf spot, yellow mosaic virus, crinkle virus disease and *Cuscuta*

BLACK GRAM *(Vigna mungo)*and G**REEN GRAM** *(Vigna radiata)*

Powdery mildew(C.O:*Erysiphe polygoni)*

Economic importance

Powdery mildew is one of the wide spread diseases of several legumes including peas, black gram and green gram.

Symptoms

Small, irregular powdery spots appear on the upper surface of the leaves. These spots gradually increase in size and become circular covering the lower surface also. When the infection is severe, both surfaces of the leaf are completely covered by whitish powdery growth. In severe infections, foliage becomes yellow causing premature defoliation. The disease becomes severe during flowering and pod development stage. The white powdery spots completely cover the petioles, stem and even the pods. The plant assumes greyish white appearance. Often pods are malformed and small with few ill-filled seeds. The disease causes forced maturity of infected plants which results in heavy yield losses.

Pathogen

The fungus is ectophytic, spreading on the surface of the leaf, sending haustoria into the epidermal cells. Conidiophores arise vertically from the leaf surface, bearing conidia in short chains. Conidia are hyaline, thin walled, elliptical or barrel shaped or cylindrical and single celled. Later in the season, **cleistothecia** appear as minute, black, globose structures with myceloid appendages. Each cleistothecium contains 4-8 asci and each ascus contains 3-8 ascospores which are elliptical, hyaline and single celled.

Disease cycle

The fungus is an obligate parasite and survives as **cleistothecia** in the infected plant debris. P.S.I:Primary infection is usually from ascospores from perennating cleistothecia.

S.S.I: The secondary spread is carried out by the **air-borne conidia**. Rain splash also helps in the spread of the disease.

Favourable Conditions

Warm humid weather favours disease development. The disease is severe generally during late *kharif* and *rabi* seasons.

Management

- Remove and destroy infected plant debris.
- Spray twice with Carbendazim or Thiophanate methyl or Tridemorph @0.1%, one immediately after disease appearance and the second after 15 days.
- Grow tolerant black gram cultivar like **Krishnayya** and green gram cultivars like JGUM 1, TARM 1, Pusa 9072, WGG 48 and WGG 62.

Rust *Uromyces phaseoli typica* (Syn: *U.appendiculatus*)

Symptoms

The fungus infects both black gram and green gram. The disease is mostly seen on leaves, rarely on petioles, stem and pods. The fungus produces small, round, reddish brown uredosori mostly on lower surface. They may appear in groups and several sori coalesce to cover a large area of the lamina. In the late season, teliosori appear on the leaves which are linear and dark brown in colour. Intense pustule formation causes drying and shedding of leaves.

Pathogen

It is **autoecious**, long cycle rust and all the spore stages occur on the same host. The uredospores are unicellular, globose or ellipsoid, yellowish brown with echinulations. The teliospores are globose or elliptical, **unicellular**, pedicellate, chestnut brown in colour with warty **papillae at the top**. Yellow coloured pycnia appear on the upper surface of leaves. Orange coloured cupulate aecia develop later on the lower surface of leaves. The aeciospores are unicellular and elliptical.

Disease cycle

The pathogen survives in the soil as teliospores and as uredospores in crop debris.

P.S.I:Primary infection is by the basidiospores developed from teliospores.

S.S.I: Secondary spread is by wind-borne uredospores. The fungus also survives on other legume hosts.

Favourable Conditions

Cloudy humid weather, temperature of 21-26^{0}C and nights with heavy dews favour the disease.

Management

- Remove and destroy the infected plant debris.
- Spray Wettable sulfur@0.3%, immediately on the set of disease and repeat after 15 days.
- Grow tolerant black gram cultivar like **LBG 648**.

Cercospora leaf spot

Cercospora canescens

Economic importance

This is an important disease of black gram and green gram and it usually occurs in a severe form, causing heavy losses in yield particularly when humidity is high.

Symptoms

Small, circular spots develop on the leaves with grey centre and reddish brown margin. The several spots coalesce to form brown irregular lesions. Under favourable environmental conditions, severe leaf spotting and defoliation occurs at the time of flowering and pod formation. The brown lesions may be seen on petioles, branches and pods in severe cases. Powdery growth of the fungus may be seen on the centre of the spots.

Pathogen

The fungus produces clusters of dark brown septate conidiophores. The conidia are linear, hyaline, thin walled and 5-6 septate.

Disease cycle

P.S.I:The fungus survives on diseased plant debris in soil and on **seeds**.

S.S.I:The secondary spread is by air-borne conidia.

Favourable Conditions

Humid weather and dense plant population favour disease development.

Management

- Remove and burn infected plant debris.
- Spray Mancozeb@0.25% or Carbendazim@0.1%
- Grow tolerant black gram varieties like UG 135, TPU 4, TPU 5, TPU 11, TPU 12, AKU 4 and SP 21.

Corynespora leaf spot

Corynespora cassicola

Economic importance

The disease attacks a wide range of crops including many legumes such as black gram, green gram and cowpea.

Symptoms

Symptoms develop on leaves when the crop reaches flowering stage. Lesions begin as **dark reddish brown circular spots** usually on the upper surface of the leaf. They expand with marked, **narrow concentric banding** to become larger spots. The **concentric rings** are made up of dead tissue. In advanced stages, the spots coalesce to form patches. **Shot holing** and severe defoliation is a marked symptom in advanced stages of infection. Yields decrease drastically.

Disease cycle

P.S.I:The fungus is seed borne and can survive on host debris for two years.

S.S.I:The secondary spread is through air borne conidia.

Management

- Remove and burn infected plant debris.
- Spray Mancozeb@0.25% or Carbendazim@0.1%
- Grow tolerant black gram varieties like LBG 167.

Angular black spot

Protomycopsis phaseoli or P. patelii (Syn: Erratomyces patelii)

Economic importance

The disease is restricted to green gram cultivated in Krishna, Khammam and Northern Telangana districts of Andhra Pradesh.

Symptoms

Symptoms appear from 3 weeks after sowing. Small light yellow spots appear on older leaves, enlarge gradually turning into angular black spots. When infection is severe,

several such spots coalesce resulting in drooping, drying and defoliation. Yields are greatly reduced due to poor pod set and reduction in seed size.

Pathogen

In India and in the American tropics angular black spot disease on leaves of Vigna spp. is caused by *Protomycopsis patelii*. The fungus is related to smut fungi of the genus Tilletia because it produces relatively large, opque teliospores which have a partition layer in their wall and which germinate with holobasidia carrying needle-shaped basidiospores. In contrast to species of Tilletia and related genera, the teliospores are scattered in intercellular spaces in the mesophyll without rupturing it and develop mostly intercalaryly. Additionally taking into account the distinct host family, the agent of angular black spot disease of beans cannot be classified in any known genus. The new genus ***Erratomyces*** is proposed.

Disease cycle:

P.S.I:Protomycopsis produces finely punctuate (rough) chlamydospores which are formed terminally on the mycelium and helps in survival of the fungus.

S.S.I:Rainsplash will dessminate bacteria

Management

- Grow tolerant green gram cultivars like LGG 407, LGG 450, LGG 421, WGG 295 and Pusa 105.
- Remove and destroy infected plant debris

Dry root rot

Rhizoctonia bataticola

(Pycnidial stage : *Macrophomina phaseolina)*

Symptoms

The disease symptom starts initially with yellowing and drooping of the leaves. The leaves later fall off and the plant dies with in week. Dark brown lesions are seen on the stem at ground level and bark shows shredding symptom. The affected plants can be easily pulled out leaving dried, rotten root portions in the ground. The rotten tissues of stem and root contain a large number of black minute sclerotia.

Pathogen

The fungus produces dark brown, septate mycelium with constrictions at hyphal branches. Minute, dark, round sclerotia are produced in abundance. The fungus also produces dark brown, globose ostiolated pycnidia on the host tissues. They pycnidiospores (conidia) are thin walled, hyaline, single celled and elliptical.

Disease cycle

The fungus survives in the infected debris and also as facultative parasite in soil.

P.S.I:The primary spread is through seed-borne and soil-borne sclerotia.

S.SI: The secondary spreads is through pycnidiospores which are air-borne.

Favourable Conditions

Day temperature of 30^0C and above and prolonged dry season followed by irrigation.

Management

- Treat the seeds with Carbendazim or Thiram at 4 g/kg or pellet the seeds with *Trichoderma viride* at 4 g/kg or *Pseudonomas fluorescens* @ 10g/kg of seed.
- Apply farm yard manure or green leaf manure *(Gliricidia maculate)* at 10 t/ha or neemcake at 250 kg/ha.

Bacterial leaf spot

Xanthomonas phaseoli

Symptoms

The disease usually attacks green gram and black gram in kharif season. The disease is characterized by many brown, dry, raised spots on the leaf surface. The spots first appear as superficial eruptions and gradually invade the tissues giving **corky or rough appearance**. When the disease is severe spots coalesce and leaves turn yellow and fall off prematurely. The lower surface of the leaf appears red in colour due to the formation of raised spots. The stem and pods also get infected.

Disease cycle

P.S.I:The bacterium is seed borne and grows through perennial vines.

S.S.I: Rain splashes play an important role in the development and spread of the

disease.

Management

- Grow tolerant green gram varieties like LGG 407, LGG 444, JAL 781, NDM 88-14 and ML 537.
- Soak the seed in 500 ppm streptocycline solution for 30 minutes before sowing.
- Spray twice with paushamycin or plantomycin 100 mg in combination with 3 g of COC per liter at an interval of 12 days.

Yellow mosaic

Mungbean yellow mosaic virus

Economic importance

The disease is prevalent in black gram and green gram in Andhra Pradesh, T.N., U.P., M.P., Bihar, Punjab, Haryana, Himachal Pradesh, Rajasthan and Orissa.

Symptoms

Initially small yellow patches or spots appear on young leaves. The next trifoliate leaves emerging from the growing apex show irregular yellow and green patches alternating with each other. The yellow discoloration slowly increases and newly formed leaves may completely turn yellow. Infected leaves also show necrotic symptoms. The infected plants normally mature late and bear a very few flowers and pods. The pods are small and distorted. The early infection causes death of the plant before seed set.

Disease cycle

P.SI:The virus survives in the weed hosts and other legume crops.

S.S.I: The disease spreads through white fly, *Bemisia tabaci.*

Favourable Conditions

Summer sown crops are highly susceptible. The presence of weed hosts viz., *Croton sparsiflorus, Acalypha indica, Eclipta alba* and *Cosmos pinnatus* and legume hosts.

Management

- Rogue out the diseased plants upto 40 days after sowing.
- Remove the weed hosts peiodically.
- Increase the seed rate (25 kg/ha).
- Grow resistant black gram varieties like Teja, LBG 752, Pant-30 and Pant-90.
- Grow resistant green gram varieties like LGG 407 and ML 267.
- Cultivate the crop during rabi season.
- Follow mixed cropping by growing two rows of maize (60 x 30 cm) or sorghum (45 x 15 cm) for every 15 rows of black gram or green gram.
- Grow seven rows of sorghum as border crop
- Treat seeds with Imidacloprid 70 WS @ 5ml/kg to control vector.
- Give one foliar spray of systemic insecticide (Dimethoate @ 750 ml/ha) on 30 days after sowing.

Leaf crinkle

Leaf crinkle virus

Symptoms

The symptom appears initially in young leaves. The enlargement of 4th or 5th leaf is seen four or five weeks after sowing. Later crinkling and curling of the tips of leaflets are seen. The petioles as well as internodes are shortened. The infected plant gives a stunted and bushy appearance. Flowering is delayed by 8-10 days, inflorescence, if formed, is malformed and turns with small size flower buds and fails to open. The age of the plant is prolonged with dark green leaves till harvest. Pod setting is curtailed which decreases the yield drastically.

Disease cycle

P.S.I:The virus is **seed-borne** and primary infection occurs through infected seeds.

S.S.I:White fly, *Bemisia tabaci*, helps in the secondary spread. The virus is also transmissible through aphids and **Epilachna** beetles.

Favourable Conditions

The presence of weed hosts like *Aristolochia bracteata* and *Digera arvensis.* Closs planting. Kharif season crop is highly susceptible. Continuous cropping of other legumes which also harbour the virus.

Management

- Use increased seed rate (25 kg/ha).
- Hot water treatment of the seed at 55^0C for 30 minutes.
- Rogue out the diseased plants at weekly interval upto 45 days after sowing.
- Cultivate seed crop during rabi season.
- Remove weed hosts periodically.
- Spray Monocrotophos or Methyl demeton on 30 and 40 days after sowing at 500 ml/ha.

Cuscuta

Symptoms

- In field, it is noticed as small masses of branched thread- like, leafless stems which twine around the stem or leaves of host (Complete stem parasite)
- Leaves of parasite are represented by minute functionless scales
- When stem comes in contact with the host, haustoria penetrate the host cortex reaching fibro-vascular bundles
- Infected plants appears sick as the parasitic vine increases in size
- Plants die under severe infection

Survival and spread

- P.S.I:Perpetuates through seed which fall onto the ground
- S,.S.I:Dispersed through birds and grazing animals

Management

- Crop seed should be free from dodder seeds
- Do not allow grazing animals to move in dodder infested field
- Badly infested crop should be burnt before the parasite produces flowers and seeds
- Five year crop rotation with non-host crop
- Spot treatment with Glyphosate, Pentachloro phenol or 2,4-D
- Spray herbicide, pursuit (200ml/acre), in problematic areas when the crop is at 20 days followed by urea spray (1%) within 5-7 days after herbicide treatment.

Lecture 11 .- economic importance, symptoms, cause, disease cycle and integrated management-Rust and wilt.
Pigeonpea- economic importance, symptoms, cause, disease cycle and integrated management- *Phytophthora* blight, wilt and sterility mosaic

Lentil rust

Uromyces viciae-fabae

• It is regarded as the most important foliar disease of lentil.

• Complete crop failures can occur due to this disease.

• Rust disease is a potential threat to lentil cultivation and causes substantial yield losses ranging from 60- 69 per cent.

• In 1978, severe outbreak of lentil rust was recorded in the Narmada Valley of Madhya Pradesh. • In tarai region of Uttarakhand state and its surrounding areas, rust has been a major constraint affecting yield adversely.

• Rust pustules can be seen on leaf blade, petiole & stem.

• Rust starts with the formation of yellowish-white pycnidia and aecial cups on the lower surface of leaflets and on pods, singly or in small groups in a circular form.

• Later, brown uredial pustules emerge on either surface of leaflets, stem and pods.

• Pustules are oval to circular and up to 1 mm in diameter. They may coalesce to form larger pustules. • In severe infections leaves are shed and plants dry prematurely.

• The affected plant dries without forming any seeds in pods or with small shriveled seeds.

Epidemiology • The disease generally starts from low-lying patches in the paddock and radiates towards the border. • Rust is an autoecious fungus, completing its life cycle on lentil. High humidity, cloudy or drizzly weather with temperatures 20 to 22°C favour disease development. • The disease generally occurs during the flowering /early podding stage.

Management

• Lentil varieties Pant L-639, Pant L-406, Pant L-6, pant L-7 and Pant L-8 are resistant.

• Use of foliar fungicides as Hexaferb give best control.

• Fungicides as Bayleton (0.05% a.i) and Calixin (0.2% a.i.) are found effective against the pathogen. • Foliar spray of benomyl, carboxin, metalaxyl, oxycarboxin

Orange-brown rust pustules on leaf

Fusarium wilt

Fusarium oxysporum f.sp. lentis

• It is the most important biological constraints to productivity of lentil worldwide.

• The pathogen causes serious disease and is widespread in India.

• It is a soil borne, root pathogen colonizing the xylem vessels and blocking them completely to cause wilting.

Symptoms • The disease appears in the field in patches at both seedling and adult stages.

• It is characterized by sudden drooping, followed by drying of leaves and seedling death.

• The roots appear healthy, with reduced proliferation and nodulation and usually no internal discoloration of the vascular system.

• Adult wilt symptoms appear from flowering to late pod-filling stage and are characterized by sudden drooping of top leaflets of the affected plant.

• dull green foliage followed by wilting of the whole plant or individual branches.

• Seeds from plants affected in mid-pod-fill to late pod-fill are often shrivelled.

Epidemiology • The fungus is soil borne, which can survive in the soil and plant debris in the absence of its host for a period of 3-4 years. • The disease is favoured by low soil temperature, 30%soil water holding capacity and increasing plant maturity. • Yield losses depend on the stage at which the plant wilts it can be 100% when wilt occurs at pre pod stage, about67% when it occurs at the pre harvest stage.

Management • Using resistant varieties, a number of which are now available as Pant L-4, Pant L-6, Pant L-8 and Noori. • Seed treatment with benornyl (0.3%) or thiram + benomyl (1:1, 0.3%) reduces wilt incidence and increases grain yield. • Soil amendment with organic matter enhances antagonism with other soil microorganisms. • Ploughing of the field during summer. • Following crop rotation with cereal crops which are not affected by wilt pathogen. • Using antagonistic microorganisms like Bacillus subtilis, Trichoderma harzianum, T. viride @ 4 g/kg seed etc.

Fusarial wilt of lentil

Vascular discoloration

RED GRAM *(Cajanus cajan)*

Economic importance

Phytophthora blight / Stem blight *Phytophthora drechsleri f. sp. cajani*

A devastating disease that kills young (1 to 7 week old) plants, leaving large gaps in plant stands. Yield losses are usually higher in short duration pigeonpeas than in medium and long duration types.

Symptoms

Phytophthora blight resembles damping off in that it causes seedlings to die suddenly. Infected plants have water soaked lesions on their leaves and brown to black, slightly sunken lesions on their stems and petioles. Infected leaves loose turgidity, and become desiccated. Lesions girdle the affected main stems or branches which break at this point and foliage above the lesion dries up. When conditions favour the pathogen, it is common for many plants to die. Pigeonpea plants that are infected by blight, but not killed often produce large galls on their stems especially at the edges of the lesions. The pathogen infects the foliage and stems but not the root system.


Pathogen

Fungus produces hyaline, coenocytic mycelium. The sporangiophores are hypha-like with a swelling on the tip bearing hyaline, ovate or pyriform, non-papillate sporangia. Each sporangium produces 8-20 zoospores. Oospores are globose, light brown, smooth and thick walled.

Favourable Conditions

Cloudy weather and drizzling rain with temperatures around 25^0C favour infection that requires continuous leaf wetness for 8 hours to occur. Warm and humid weather following infection results in rapid disease development and plant death. Soils with

poor drainage, low lying areas, heavy rain during the months of July-September favours the disease. Pigeonpeas are usually not infected after they are 60 days old.

Disease cycle

The fungus survives in the soil and plant debris in the form of oospores, and dormant mycelium.

P.S.I:Primary infection is from oospores

S.S.I: secondary spread by zoospores from sporangia. Rain splash and irrigation water help for the movement of zoospores. *Cajanus scarabaeoides* var. *scarabaeoides*, a wild relative of pigeonpea is also a host of the blight pathogen.

Management

- Avoid sowing redgram in fields with low-lying patches that are prone to water logging.
- Adjust the sowing time so that crop growth should not coincide with heavy rainfall.
- Grow resistant varieties like BDN 1, ICPL 150, ICPL 288, ICPL 304, KPBR 80-1-4.
- Seed treatment with 4g *Trichoderma viride* formulation + 6g metalaxyl (Apron 35SD) per kg of seed.
- Spray Metalaxyl (Ridomyl MZ) at 0.2%.

Wilt

Fusarium oxysporum f. sp. udum

Economic importance

The annual losses due to wilt have been estimated at US $ 71 million in India. It is prevalent in A.P., Maharashtra, M.P., U.P and Bihar. In A.P., it is prevalent in Telangana districts and Kurnool.

Symptoms

The diseases may appear from early stages of plant growth (4-6 week old plant) upto flowering and podding. Patches of dead plants in the field when the crop is flowering or podding are the first indications of wilt. The most characteristic symptom is a **purple band** extending upwards from the base of the main stem. Vascular tissues exhibit brown discolouration in the region of purple band. Partial wilting of the plant is a definite

indication of *Fusarium* wilt and distinguishes from Phytophthora blight that kills the whole plant. Partial wilt is associated with lateral root infection, while total wilt is due to tap root infection. Foliar symptoms include loss of turgidity, interveinal clearing and chlorosis.

Pathogen

The fungus produces hyaline, septate mycelium. Microconidia are hyaline, small, elliptical or curved, single celled or two celled. Macroconidia are also hyaline, thin walled, linear, curved or fusoid, pointed at both ends with 3-4 septa. The fungus also produces thick walled, spherical or oval, terminal or intercalary chlamydospores singly or in chains of 2 to 3.

Favourable Conditions

Long and medium duration types suffer more wilt than short duration types. Mono-cropping and ratooning pre-disposes the plant to wilt. Disease incidence is more severe in Vertisols than in Alfisols. Early sowing, good weed management and good crop growth encourage wilt development. Soil temperatures of 17 to 25^{0}C favour the pathogen development.

Mode of Spread and Survival

The disease is **seed** and **soil** borne. The fungus survives in the infected stubbles in the field for about 3 years.

P.S.I:The primary spread is by soil-borne chlamydospores and also by seed contaminant. Chlamydospores remain viable in soil for 8-20 years.

S.S.I:The secondary spread in the field is through irrigation water and implements.

Management

Cultural:

- Follow long crop rotation with tobacco, sorghum or castor.
- Avoid successive cultivation of redgram in the same field.
- Adopt mixed cropping of sorghum in the field.
- Soil solarization in summer to reduce the inoculum of pathogen.
- Collect and destroy the diseased stubbles.

HPR:

- Grow resistant / tolerant varieties like Asha (ICPL 87119), Maruti (ICP 8863), Lakshmi (ICPL 85063), Durga (ICPL 84031), PRG 100, PRG 158, Muktha, Prabhat and Sharada.

Chemical:

- Seed treatment with Thiram @0.3% or Carbendazim @0.2%

Bio-control:

- Treat the seeds with *Trichoderma viride* at 4 g/kg.
- Multiply 2 Kg *T. viride* formulation in 50 kg of Farm Yard Manure and apply to soil.

Sterility Mosaic

Sterility mosaic virus

Economic importance

A serious problem in India and Nepal where it is estimated to cause annual pigeonpea grain losses worth US $ 282 million.

Symptoms

The disease attack can be seen in all stages of crop growth. In the field, the diseased plants appear as **bushy, pale green plants without flowers or pods**. Leaves of these plants are small and show typical light and dark green mosaic pattern. Symptoms initially appear as vein-clearing on young leaves. In severe cases, leaves become smaller and **cluster** near tip because of shortened internodes and stimulation of auxillary buds. The plants are generally stunted and do not produce pod. Plants infected at early stages (upto 45 days) of crop growth show near complete sterility and yield loss upto 95 per cent. As plants become older (after 45 days), their susceptibility

to the disease decreases and such plants show partial sterility. If pods develop, the seeds may be small, shrivelled and immature. Some pigeonpea varieties, e.g., ICP 2376 exhibit ring spot leaf symptoms, these indicate localized sites of infection of the pathogen, and such plants produce normal flowers and pods.

Disease cycle

.P.S.I: The self-sown redgram plants, perennial types of redgram (*Cajanus scarabaeoides* var. *scarabaeoides*) and the rationed growth of harvested plants serve as sources of infection.

S.S.I:The disease is transmitted by an Eriophyid mite ***Aceria cajani***

Favourable conditions

Disease incidence is high when pigeonpeas are inter- or mixed cropped with sorghum or millets. Shade and humidity encourage mite multiplication, especially in hot summer weather.

Management

- Rogue out infected plants in early stages of disease development
- Grow tolerant genotypes like ICPL 87119 (Asha), ICPL 227, Jagruti and Bahar
- Spray Dicofol 3ml or Sulphur 3g in one liter of water to control mite vector in early stages of disease development

Bacterial leaf spot and stem canker

Xanthomonas campestris pv. cajani

Symptoms

Leaf infection can occur at all stages of plant growth, stem infection usually occurs in

younger plants. In India the disease usually appears in the rainy season during July and August. It can be seen on lower leaves of plants that are about one month old as **small necrotic spots** surrounded by bright **yellow halos**. Later, rough, raised, **cankerous** lesions appear on the stem. Leaf spots do not usually cause defoliation. Cankers can cause stems to break, but the broken part usually attaches to the plant. Stems often break at the point where the primary leaves are attached. Often, the affected plants do not break, and the stem cankers increase in size until they are 15-25 cm long. In cases of severe infection the affected branches dry.


Pathogen

The bacterium is strict aerobe, gram negative, non spore forming, rod shaped with **monotrichous** polar flagellum of at one end. The bacterial cells are disseminated through rain splash.

Favourable conditions

Warm (25-30^{0}C) and humid weather favour the disease development. Disease incidence is generally higher in low-lying waterlogged areas of the field than in well drained areas.

Disease cycle:

P.S.I:Bacteria in infected plants

S.S.I:Bacterial cells spread through rain splash

Management

- Remove the infected plant debris and destroy.
- Spray antibiotics like Streptocycline@100ppm, 2-3 times at 10 days interval.

Lecture 12 Soybean and Cowpea- economic importance, symptoms, cause, disease cycle and integrated management- rust, soybean mosaic, bacterial leaf spot and Cowpea mosaic

SOYBEAN *(Glycine max)*

Rust (Phakopsora pachyrhizi)

Economic importance

This disease is the most destructive disease of soybean. It causes yield losses of 10-65% in Uttar Pradesh, 355 in Northeastern hilly region and 30-100% in Madhya Pradesh.

Symptoms

Symptoms appear on all above ground parts of the plant. Large number of light brown pustules appears on lower surface of the leaves in the initial stages, later turns to reddish brown to tan colour. Tan lesions consist of small uredia surrounded by slightly discolored necrotic areas on leaf surfaces. Early stages show an ostiole, or small hole, where uredospores emerge. As uredia become larger, they release masses of tan colored uredospores that appear as light brown or white raised areas. Uredial pustules become more numerous with advancing infection and often will coalesce forming larger pustules that break open releasing masses of uredospores. Lesions are generally restricted by veins giving angular appearance. In Kharif crop, the disease appears in the first week of September coinciding with flowering or pod formation. The leaves gradually become yellow and premature defoliation occurs resulting in yield loss.

Disease cycle

P.S.I:The pathogen survives as teleutospores in crop debris.

S.S.I:Secondary spread is through wind borne uredospores.

Favourable Conditions

The disease is favoured by temperature of 18 to 23^{0}C and R.H of 80%.

Management

- Early maturing cultivars escape rust infection.
- Spray twice with Saprol (Triforine)@0.05%, Delan (Dithianon)@0.2% or Mancozeb@0.1% at weekly interval, beginning at the first appearance of the disease are effective in controlling the disease.
- Grow resistant varieties like PK 73-84, PK-310, IC 89495, IC 89498, etc.

Soybean mosaic

Soybean mosaic virus

Symptoms

Infected plants can be recognized by their stunted growth, distorted and puckered leaves. The leaves are dwarfed, crinkled and narrow with their margins turned downwards. In severe cases, dark green blister like puckering along the veins takes place. Pod setting is drastically reduced. Infected plants produce distorted pods and fewer seeds. Seed discolouration can be seen under severe infection. The infected plants remain green even at the end of the growing season.

Disease cycle

P.S.I:The virus is seed borne

S.S.I:It is transmitted by aphids.

Management

- Use virus free seed from healthy crop.
- Rogue out infected plants and burn them.
- Spray dimethoate@2ml/lt to control the vector

Bacterial pustule

Xanthomonas axonopodis pv. glycines

Symptoms

Symptoms are evident as tiny, light green spots with elevated centers that later on turn into raised lightly coloured pustules, typically without exudates. Infection is more frequent on the lower ones. Spots may vary from minute specks to large, irregular, mottled brown areas. Severely affected portions of leaves are torn away by wind imparting a ragged appearance to plants, and results in premature defoliation. Raised red brown spots on pods may also develop. The disease reduces the yield as well as the oil content in seeds.

Disease cycle

P.S.I:The bacterium survives in crop residue and seed

S.s.I;Bacteria spread through rainsplash

Favourable conditions

The disease appears in a severe form when warm temperatures and frequent showers prevail during growing season.

Management

- Remove and burn infected plant debris.
- Crop rotation with grain crop is recommended
- Two sprays at 45and 55 DAS with a mixture of Copper oxychloride@0.2%+Streptocycline@250ppm effectively control the disease.

COWPEA *(Vigna unguiculata)*

Cowpea mosaic

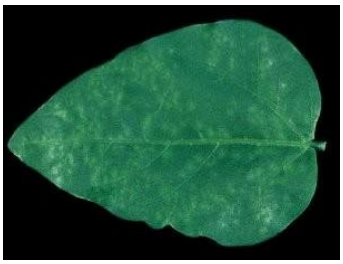
Cowpea yellow mosaic virus (Syn: Cowpea mosaic virus, yellow strain)

Economic importance

Yield reductions up to 95% have been reported. Also found in soybean (*Glycine max*), and pigeon pea (*Cajanus cajan*) which serves as a reservoir of the virus.

Symptoms

Chlorotic spots with diffuse borders (diam. 1-3 mm) are produced in inoculated primary leaves. Trifoliate leaves develop a bright yellow or light green mosaic. The severity increases in younger leaves with moderate distortion and reduction in size. The affected leaves are leathery. The infected plants produce a few pods which are small and distorted. Chlorotic spots are also produced on pods. Plants do not show necrosis.



Pathogen

Cowpea mosaic virus (CPMV) is a plant virus of the comovirus group. It is An RNA-containing virus with isometric particles about 28 nm in diameter. Its genome consists of

2 molecules of positive sense RNA (RNA-1 and RNA-2) which are separately encapsidated.

Disease cycle

PS.I:infected plants

S.S.I:Transmitted by various beetles with biting mouthparts. The transmission is characterised by short acquisition and inoculation access periods and an apparent lack of a latent period. Beetle vectors may remain viruliferous for 1-2 to more than 8 days depending on the species. Transmission efficiency and retention of infectivity are correlated with the amount of vector feeding. The virus is transmitted by chrysomelid beetles viz., *Ootheca mutabilis, Cerotoma variegata* and *C. ruficornis.*

Management

- Remove the infected plants as soon as symptoms appear.
- Grow resistant varieties
- Rogue out and destroy the weed hosts

Lectrue 13 Rapeseed and Mustard- economic importance, symptoms, cause, disease cycle and integrated management- white rust, downy mildew, powdery mildew and *Alternaria* leaf spot

Lect 13 Rapeseed and Mustard- economic importance, symptoms, cause, disease cycle and integrated management- white rust, downy mildew, powdery mildew and *Alternaria* leaf spot

Economic Importance

White rust *Albugo candida or A. cruciferarum*

The disease makes its first appearance in the beginning of January shortly after the attack of Alternaria leaf blight on the under surface of lower leaves.

Symptoms

Both local and systemic infection is observed. In case of local infection isolated white/ creamy yellow raised pustules appear on under surface of leaves which later coalesce to form patches. Systemic infection causes hypertrophy and hyperplasia resulting in malformation and distortion of floral parts. Entire inflorescence is replaced by swollen sterile structure (**Stag head**). Maximum damage occurs when systemic infection of the stem is noticed.



Pathogen

The fungus is an obligate parasite. The mycelium of the fungus is non-septate, intercellular which produces knob-like haustoria. Numerous short sporangiophores arise from the mycelium on which sporangia are produced in a basipetal succession. In systemic infection, Oogonia and antheridia join by means of a fertilization tube,

resulting in oospore. The oospores germinate to form zoospores in a vesicle. The zoospores are elliptical to kidney shaped and are biflagellate.

Disease cycle

The fungus survives through oospores formed in affected host tissues. The secondary spread is through zoospores disseminated by rain or irrigation water.

Management

- Collect and destroy infected plant debris
- Rotation with non-cruciferous crops
- Early sowing of the crop (in first week of October)
- Seed dressing with Metalaxyl (Apron 35SD)@6g/kg seed followed by a single spray with Metalaxyl (Ridomyl MZ)@0.2%
- Grow resistant varieties like RC 781, PYSR 8 and PR 10 (or) tolerant varieties like

  Kranthi and **Krishna**

Downy mildew

Peronospora parasitica

Symptoms

Symptoms appear on all aerial parts but usually on leaves and inflorescence. Greyish white irregular necrotic patches develop on the lower surface of the leaves. The most conspicuous and pronounced symptom is the infection of inflorescence causing hypertrophy of the peduncle or inflorescence (**Stag head**). The affected inflorescence does not produce any siliqua or seed.

Pathogen: The fungus is an obligate parasite. The mycelium of the fungus is non-septate, intercellular which produces haustoria. Sporangiophores are dichotomously branched with sterigmata which are pointed with acute angles usually of equal length. Oval and non-papillate sporangia are produced over the pointed sterigmata. Sporangia always germinate by germ tube and behave as conidia.

Disease cycle

The fungus survives through oospores formed in affected host tissues and on weed hosts. The secondary spread is through wind borne sporangia.

Management

- Collect and destroy infected plant debris
- Rotation with non-cruciferous crops
- Early sowing of the crop (in first week of October)
- Seed dressing with Metalaxyl (Apron 35SD)@6g/kg seed followed by a single spray with Metalaxyl (Ridomyl MZ)@0.2%
- Grow resistant varieties like RC 781, PYSR 8 and PR 10

Powdery mildew

Erysiphe cruciferarum

Symptoms

Symptoms appear as dirty white circular patches on both sides of lower leaves. Under favourable environmental conditions entire leaves, stems and siliquae are affected. The affected siliquae produce small and shrivelled seeds.

Pathogen

The fungus is ectophytic, spreading on the surface of the leaf, sending haustoria into the epidermal cells. Conidiophores arise vertically from the leaf surface, bearing

conidia in short chains. Conidia are of *Oidium* type. Later in the season, **cleistothecia** appear as minute, black, globose structures with myceloid appendages. Each cleistothecium contains 4-8 asci and each ascus contains 3-8 ascospores which are elliptical, hyaline and single celled.

Disease cycle

P.S.I:The fungus over-summers through cleistothecia as ascospores or as mycelium on volunteer host plants.

S.S.I:The disease spreads through wind borne conidia.

Favourable conditions

The disease is favoured by dry weather and becomes severe under irrigated conditions.

Management

- Collect and destroy infected plant debris
- Spray the crop with wettable sulphur@0.2%

Alternaria leaf spot

Alternaria brassicae , *A. brassicola* and *A.raphani*

Economic importance

The disease caused by *A. brassicae* is more damaging and occurs in all rapeseed-mustard growing areas.

Symptoms

Symptoms of the disease start with formation of spots on leaves, stem and siliquae. The spots produced by *A. brassicae* are usually gray compared to black sooty velvety spots produced by *A. brassicola*.

Table 1: Comparison of fungal structures among the three species of *Alternaria* encountered on rapeseed-mustard

Fungal structures	*A. brassicae*	*A. brassicicola*	*A. raphani*
Mycelium	Septate, brownish grey	Septate, olive grey to greyish black	Cottony whitish to greenish grey or dark olive
Conidiophore	Dark, septate, arise in fascicles, 14-74µ x 4-8µ	Olivaceous, septate, branched, 35-45µ x 5-8µ	Septate, olive-brown, single or branched, 29-160µ x 4-8µ
Conidia	Brownish black, obclavate, muriform, produced singly or in chains or 2-3	Dark cylindrical to oblong, muriform, produced in chains of 8-10 spores	Olive-brown to dark, obclavate, muriform, more-or-less pin-pointed at each end, appear singly or in chains of up to 6 spores
Spore body (µ)	96-114 x 17-24	45-55 x 11-16	45-58 x 13-21
Spore beak length (µ)	45-65	none	1-25
Spore			
Transverse septation	10-11	5-8	6-9
Longitudinal septation	0-6	0-4	3-6
Rate of growth and sporulation on media	Rudimentary slow growth	Black sooty colony with distinct zonations, fast growing with abundant sporulation	Cottony mycelial colony with less abundant sporulation
Infection	Penetrates leaf only through stomata	Penetrates leaf directly or through stomata	Direct penetration

Pathogen

The fungus produces dark brown, short, septate, irregularly bent conidiophores with a single conidium at the apex. The conidia are obclavate, light to dark brown in colour with both transverse and longitudinal septa, with a prominent beak.

Disease cycle

P.S.I:The pathogen survives in the infected crop debris as dormant mycelium.

S.S.I:The secondary spread is mainly by air-borne conidia.

Management

- Removal and destruction of infected plant debris
- Use disease free or treated seed carbendazim+Mancozeb @2.5g/kg seed
- Spray with carbendazim+mancozeb (@0.25%) or Iprodione (0.2%) at 10 days interval.

LECTURE 14 Sesame economic importance, symptoms, cause, disease cycle and integrated management- *Alternaria* leaf spot, powdery mildew, phyllody, *Macrophomina* stem rot and bacterial leaf spot

DISEASES OF GINGELLY *(SESAMUM INDICUM)*

Alternaria leaf spot

Alternaria sesami

Symptoms

Initially small, circular, reddish brown spots (1-8mm) appear on leaves which enlarge later and cover large area with concentric rings. The lower surface of the spots are greyish brown in colour. In severe blighting defoliation occurs. Dark brown lesions can also be seen on petioles, stem and capsules. Infection of capsules results in premature splitting with shriveled seeds.

Reddish brown spots with concentric rings

Pathogen

The mycelium of the fungus is dull brown and septate and produce large number of pale grey-yellow conidiophores which are straight or curved. The conidia are light olive coloured with transverse and longitudinal septa. There are around 3-5 septate and conidia are borne in chain over short conidiophore.

Disease cycle

The fungus is seed-borne and also soil-borne as it remains dormant in the infected plant debris.

Favourable Conditions

Low temperature (20-25^{0}C), high relative humidity, excessive rainfall and cloudy weather favour the disease.

Management

- Hot water treatment at 53^{0}C for 30 minutes gives good control of the disease.
- Treat the seeds with mancozeb63%+Carbendazim12%@0.2%

.Spray twice with Mancozeb 63%+carbendazim12%@0.2%

Powdery mildew

Leveillula taurica or Erysiphe cichoracearum

(Conidial stage: *Oidiopsis taurica* or *Oidium acanthosperma)*

Symptoms

Initially greyish-white powdery growth appears on the upper surface of leaves. When several spots coalesce, the entire leaf surface may be covered with powdery coating. In severe cases, the infection may be seen on the flowers and young capsules, leading

to premature shedding. The severely affected leaves may be twisted and malformed. In the advanced stages of infection, the mycelial growth changes to dark or black because of development of cleistothecia.

Leaves with greyish-white powdery growth

Pathogen

The fungus produces hyaline, septate mycelium which is ectophytic and sends haustoria into the host epidermis. Conidiophores arise from the primary mycelium and are short and non septate bearing conidia in long chains. The conidia are ellipsoid or barrel-shaped,

single celled and hyaline. The cleistothecia are dark, globose with the hyaline or pale brown myceloid appendages. The asci are ovate and each ascus produces 2-3 ascospores, which are thin walled, elliptical and pale brown in colour.

Disease cycle

The fungus is an obligate parasite and disease perenneates through cleistothecia in the infected plant debris in soil. The ascospores from the cleistothecia cause primary infection. The secondary spread is through wind-borne conidia.

Favourable Conditions

Dry humid weather and low relative humidity favours the disease.

Management

- Remove the infected plant debris and destroy.
- Spray Wettable sulphur@0.2% or dust Sulphur at 25 kg/ha and repeat after 15 days.
- Grow resistant varieties like **Rajeshwari**, SI-1926, KRR-2, etc.

Phyllody

Phytoplasma

Economic importance

It is a serious disease capable of causing heavy losses. One per cent increase in disease incidence reduces yield by 8 kg/ha. Its incidence in India ranges upto 20%.

Symptoms

The disease manifests itself mostly during flowering stage, where the floral parts are transformed into green leafy structures, which grow profusely. The plants bear cluster of leaves and malformed flowers at the tip. The flower is rendered sterile.

Floral parts are transformed into green leafy structure

The vein clearing can be seen in different floral parts. Stamens also become leaf like to certain extent. Anthers become green and do not dehisce. Ovary is transformed into an elongated out growth resembling a shoot. The plant is stunted with reduced internodes and abnormal branching gives a bushy appearance. The infected plants generally do not bear capsules, but if capsules are formed they do not yield quality seeds.

Disease cycle

The pathogen has a wide host range and survives on hosts like *Brassica campestris* var. *toria, B. rapa, Cicer arietinum, Crotalaria* sp., *Trifolium* sp., *Arachis hypogea* and some weed hosts. The disease is transmitted by jassid, ***Orosius albicinctus*** in a persistant manner. Optimum acquisition period of vector is 3-4 days and inoculation feeding period is 30 minutes. The incubation period of the pathogen in leaf hoppers may be 15-63 days and 13-61 days in sesamum. Nymphs are incapable of transmitting the phytoplasma. Vector population is more during summer and less during winter

months.

Favourable conditions

Dry weather, moderate temperature (25^0C), low humidity (65%), minimum rainfall (0.6mm) and dry season during February-March are congenial for the disease.

Management

- Remove all the reservoir and weed hosts.
- Delay sowing in the endemic areas to reduce the vector population and thereby the disease.
- Avoid growing sesamum near cotton, groundnut and grain legumes.
- Rogue out the infected plants periodically.
- *Sesamum mulayanum* is the resistant source to the pathogen.
- Spray 2-3 times with Dimethoate@0.2% at flowering stage reduces the vector population.
- Spray 500ppm tetracycline at flowering.

Root rot or stem rot or charcoal rot

Macrophomina phaseolina

(Sclerotial stage: *Rhizoctonia bataticola)*

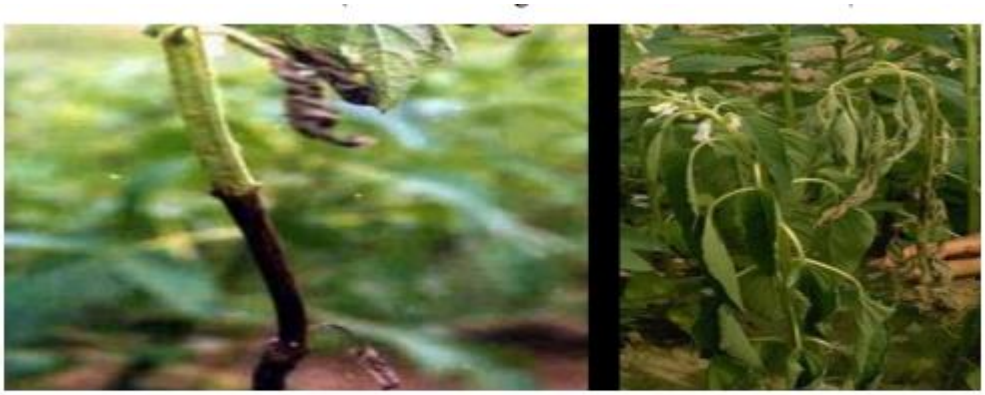
Economic importance

It is very destructive disease in all sesame growing areas in India. High incidence of the disease was reported in the states of Rajasthan, Maharashtra and Tamil Nadu during 1993- 94.

Symptoms

The disease symptom starts as yellowing of lower leaves, followed by drooping and defoliation. The stem portion near the ground level shows dark brown lesions and bark at the collar region shows shredding. The sudden death of plants is seen in patches. In the grown-up plants, the stem portion near the soil level shows large number of black pycnidia. The stem portion can be easily pulled out leaving the rotten root portion in the soil. The infection when spreads to pods, they open prematurely and immature seeds become shrivelled and black in colour. Minute pycnidia are also seen on the

infected capsules and seeds. The rotten root as well as stem tissues contains a large number of minute black sclerotia. The sclerotia may also present on the infected pods and seeds.



Stem portion near the ground level shows dark brown lesions and bark at the collar region shows shredding

Sudden death of plants is seen in patches

Pathogen

The fungus produces dark brown, septate mycelium showing constrictions at the hyphal junctions. The sclerotia are minute, dark black and 110-130µm in diameter. The pycnidia are dark brown with a prominent ostiole. The conidia are hyaline, elliptical and single celled.

Disease cycle

The fungus remains dormant as sclerotia in soil as well as in infected plant debris in soil. The infected plant debris also carries pycnidia. The fungus primarily spreads through infected seeds which carry sclerotia and pycnidia. The fungus also spreads through soil- borne sclerotia. The secondary spread is through the conidia transmitted by wind and rain water.

Favourable Conditions

Day temperature of 30^0C and above and prolonged drought followed by copious irrigation.

Management

- Treat the seeds with *Trichoderma viride* at 4g/kg or *Pseudomonas fluorescens* 10 g/kg

- Treat the seeds with tebuconazole@0.1%
- Apply farm yard manure or green leaf manure at 10t/ha or neem cake 250 kg/ha.
- Intercropping sesame with moth bean at 1:1 ratio is effective in managing the disease.
- Soil solarization with transparent polythene mulch of 50µ for 6 weeks during hot summer after ploughing and irrigation

Bacterial leaf spot

Pseudomonas sasami ***or*** *Ralstonia syringae pv. sesami*

Symptoms

Symptoms appear on all above ground parts of the plant. The disease appears as water- soaked yellow specks on the upper surface of the leaves. They enlarge and become angular as restricted by veins and veinlets. The colour of spot may be dark brown to purple with shiny oozes of bacterial masses. Under high rainfall or high humid conditions spots coalesce and ultimately defoliation occurs.

Leaves with light brown angular spots that turn blackish and purplish symptoms

Pathogen

The bacterium is gram negative and rod shaped. It is an aerobic bacterium with one or more polar flagella.

Disease cycle

The bacterium remains viable in the infected plant tissues.

P.S.I:It is internally seed-borne and

S.S.I: secondary spread through rain splash.

Management

- Keep the field free of infected plant debris.

- Seed treatment with hot water at 52^0C for 10 minutes.
- Steep the seed in Agrimycin 100 (250 ppm) for 30 minutes.

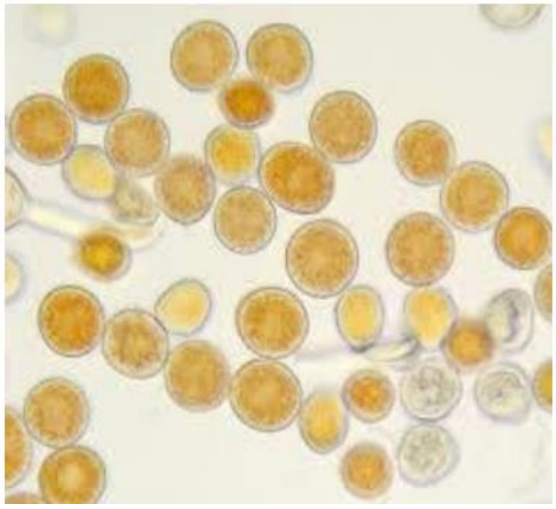
<u>Lceture 15</u> Linseed- economic importance, symptoms, cause, disease cycle and integrated management-Rust, Powdery mildew, Alternaria blight and wilt.

Rust - Melampsora lini

Symptoms:

• Bright orange and powdery pustules develop on leaves, stems and bolls but mostly on the underside of the leaves . • As the season progresses, the orange pustules turn black and produce overwintering teliospores . • Early infections may completely defoliate flax plants and reduce the seed yield Uredospore Teliospore Favourable conditions: • High humidity during cool nights and warmer day temperatures Management • Destruction of plant debris from the diseased field. • Seed treatment with Oxycarboxin. • Spray of fungicides like oxathin derivatives, Dithane M-45, Cuman L.

Uredospore



Teliospore

Fusarium Wilt - Fusarium oxysporum f. sp. Lini

Symptoms : • Yellowing and wilting of leaves, followed by browning and death of the plant. • The roots of dead plants appear ash-grey. • The tops of wilted plants

often turn downward, forming a "shepherd's crook". • Warm weather favours the disease

Mode of spread : • Seed-borne and soil-borne fungus • The fungus persists in the soil, while the mycelia and spores survive for many years in debris of flax and other organic tissue. • Wind-blown and run-off soil may spread the fungus from one field to another. shepherd's crook". Macroconidia Microconidia

Management : Crop rotation of at least 3 years. Seed treatment with Thiram or carbendazim@ 2.5g/kg seed. Soil drenching with Carbendaazim @ 0.1 % or

COC@ 0.25%

Powdery mildew – Oidium lini

Symptoms : • White powdery mass of mycelia that starts as small spots and rapidly spreads to cover the entire leaf surface . • Heavily infected leaves dry up, wither and die. • Early infections may defoliate the flax plant and reduce the yield and quality of seed Pathogen • Oidium type • Short conidiophore on which barell shaped conidia borne in chains. • Sexual fruiting body –Chasmothecium Management • Wettable sulphur @3% • Karathane 0.2%

Alternaria blight

Alternaria tenuis ,Alternaria linicola, A. solanai , A. linicola

Symptomatology

Alternaria blight Symptoms first appears on lower leaves of linseed as black point that gradually increased in size to become circular to oval or irregular in shape . Blighting of the leaves from leaf margins are also common. During severe infections, spots coalesce and covers the large area of the leaves . The affected leaves ultimately get dried up and curled. Target board like spots are not found in Alternaria leaf spot of linseed. Symptoms on floral parts first appears near the calyx on pedicel as minute dark brown spots. They enlarge, and spread all over passing into pedicel. The infected pods become distorted, blighted and discolored . Sometime partially filled capsules can be seen in disease affected plants. Symptoms on stem appears as light brown linear spot with darker margin of varying size which later become more linear and darker in colour.

The mycelium of A. linicola on Potato dextrose medium are septate, brown to brownish grey in colour. The conidiophores are dark, septate, arise in fascicles, measuring 18-56×2-5µm. Conidia are brownish black, obclavate, borne singly or sparingly in chains of 2-4, muriform with long beak and the overall conidial size ranges between 90-145 × 12-19 µm with 3-7 transverse and 1-6 longitudinal septa. Alternaria lini The mycelium of A. lini are septate, olive grey to grayish black in colour. The conidiophores are olivaceous, septate, branched measuring 26-80 µm in length and 3-7 µm in width. Conidia are dark, cylindrical to oblong, muriform

without beak measuring 42-60 µm in length and 3-7 µm in width with 2-7 transverse and 1-4 vertical septa . Toxins of A. linicola Alternaria linicola produces non-host specific phytotoxins tenuazonic acid, alternariol monomethyl ether, tentoxin and two destruxin-type compounds which closely resembled destruxin A and destruxin B.

seedlings which emerged was negatively correlated with the incidence of A. linicola on the seed.

The first symptoms appeared on cotyledons and leafs 4 and 6 days after inoculation, respectively. 8 hours of leaf wetness were found sufficient to initiate the disease at 25 ºC but not at 15 ºC, when 10-h period of leaf wetness were required. Percentage leaf area with symptoms was lower at 15 ºC than that at 25 ºC irrespective of the leaf wetness periods tested. Interruption of a continuous leaf wetness period by a 12-h dry period, occurring at any time between 1 and 18-h after inoculation, decreased the percentage cotyledons with symptoms and the disease severity, with the greatest reduction (60 and 100%, respectively) being observed when the dry period began 6-h after inoculation. A. linicola conidia were able to exploit successive 12-h periods of leaf wetness cumulatively to infect linseed plant. Disease incidence and severity were positively correlated with dark period following inoculation, but they were negatively related to the length of initial light period. Of these germplasm lines, one hundred forty lines were identified with significantly lower disease intensity of Alternaria blight on leaves than checks varieties.

Management:

Among the hybrids Padmini x Ayogi and PKVNL-260 X EC9825 were found resistant to Alternaria infestation.

Amongst the treatments, Mancozeb + Thiophana methyl was the most effective in controlling the disease. Ridomil MZ @ 0.25% and Blitox-50 @0.30% against Alternaria blight. carbendazim 12% + mancozeb 63%WP (0.125%), mancozeb 75WP (0.25%), propiconazole 25 EC (0.10%), hexaconazole 5 EC (0.10%), difenconazole 25 EC (0.05%) and iprobenphos 48 EC (0.10%) reported that all the

treatment were significantly effective against the disease. Propiconazole (tilt) 25 EC 0.10%, hexaconazole (contaf) 5 EC 0.10%, difenconazole (score) 25 EC 0.05% and iprobenphos (kitazin) 48 EC 0.10% have been noted to manage the disease economically. Maximum inhibition was recorded with Azadirachta indica (67.7%)

Alternaria blight of linseed with the integration Trichoderma viride, fungicides (mancozeb, thiophenole methyle) and plant extract (Neem leaf extract, garlic bulb extract), and reported that seed treatment with T. viride (4g/kg seed) followed 2 sprays of mancozeb (0.25%) was better in reducing the disease severity and increasing the seed yield.

Lecture 16 Sunflower- economic importance, symptoms, cause, disease cycle and integrated management- *Alternaria* leaf blight, rust, powdery mildew, head rot, sclerotial wilt, downy mildew, mosaic and sunflower necrosis virus.

DISEASES OF SUNFLOWER *(HELIANTHUS ANNUUS)*

Leaf blight

Alternaria helianthin,Alterniaster helianthi

Economic importance

It is the most destructive disease and is widely distributed wherever sunflower is grown. It occurs on all the varieties in the winter season and it spreads rapidly during the rainy season. This disease has been reported to reduce the seed yield by 27 to 80% and oil yield by 17 to 35%. The disease also affects the quality of seeds which adversely affects seed germination and vigour of seedlings.

Symptoms

The fungus produces brown spots on the leaves, but the spots can also be seen on the stem, sepals and petals. The lesions on the leaves are dark brown to black, circular to oval spots, ranging from 0.2 to 0.5mm in diameter. The spots are often surrounded by a chlorotic zone with necrotic center. The spots later enlarge in size with concentric rings and become irregular in shape. Under high atmospheric humidity, several spots coalesce to show bigger irregular lesions leading to drying and defoliation. The disease sometimes cause rotting of flower heads and affects the quality of seeds by reducing

the germination percentage.

Small, circular brown spots with yellow concentric rings

Dark brown lesions on stem

Conidia are light olive coloured with transverse and longitudinal septa

Pathogen

The fungus produces cylindrical conidiophores, which are pale grey-yellow coloured, straight or curved, geniculate, simple or branched, septate and bear single conidium. Conidia are cylindrical to long ellipsoid, straight or slightly curved, pale grey-yellow to pale brown, 1 to 2 septate with longitudinal septa.

Disease cycle

P.S.I:The fungus survives on seed, host debris and weed hosts.

S.S.I:The secondary spread is mainly through windblown conidia.

Favourable Conditions

Rainy weather, cool winter climate and late sown crops are highly susceptible.

Management

- Grow tolerant variety like BSH-1.
- Remove and destroy infected plant debris.
- Rogue out weeds at periodical intervals.
- Sow the crop early in the season (June sowing).
- Spacing of 60x30cm or 45x30cm reduces disease build up.

Treat the seeds with Carbendazim12%+mancozeb 63% at 2 g/kg.

Spray twice or thrice with Mancozeb63% +carbendazim12%@0.2% at 10 days interval starting from first appearance of the disease or 35 DAS.

Rust

Puccinia helianthi

Economic importance

Rust is the most common, wide spread and most severe diseases of sunflower. The disease is more common in temperate and sub-tropical region and is severe in winter months and causes a considerable yield reduction wherever it appears in early stages of crop growth. Under severe rust infection, the yield losses in susceptible hybrids may be 10-30%.

Symptoms

Small, reddish brown pustules (uredia) covered with rusty dust appear on the lower surface of bottom leaves. Infection later spreads to other leaves and even to the green parts of the head. In severe infection, when numerous pustules appear on leaves, they become yellow and dry. The black coloured telia are also seen among uredia on the lower surface.

Small, reddish brown pustules (uredia) covered with rusty dust appear on the lower surface of bottom leaves

Pathogen

The disease is **autoecious** rust. The pycnial and aecial stages occur on volunteer crops grown during off-season. The uredospores are round or elliptical, dark cinnamon-brown in colour and minutely echinulate. Teliospores are elliptical or oblong, two celled, smooth walled and chestnut brown in colour with a long, colourless pedicel.

Disease cycle

P.S.I:The pathogen survives in the volunteer sunflower plants and in infected plant

debris in the soil as teliospores.

S.S.I: The disease spreads by wind-borne uredospores.

Favourable conditions

Weather parameters like temperatures of 25.5 to 30.5^{0}C with RH of 86-92% favours rust disease severity. The incidence of rust increases with age, the maximum being on 75 days old plants.

Management

- Remove and burn the infected plant debris in the field.
- Remove the volunteer sunflower plants.
- Crop rotation for 3 years
- Grow tolerant variety like BSH-1.
- Spray carbendazim12%+Mancozeb 63%@0.2%, 2-3 times at 10 days interval.

Powdery mildew

Erysiphe cichoracearum

Economic importance

The disease is more common under dry conditions towards the end of winter months.

Symptoms

White to grey powdery growth appears on upper surface of older but still green foliage. Occasionally powdery growth is also seen on stem and bracts. As the plant matures black

pin head sized cleistothecia are visible in white mildew areas. The affected leaves curl, chlorotic, dry and defoliate.

White to grey powdery growth appears on upper surface on leaf

Pathogen

The fungus produces hyaline, septate mycelium which is ectophytic and sends haustoria into the host epidermis. Conidiophores arise from the primary mycelium and are short and non septate bearing conidia in long chains. The conidia are ellipsoid or barrel-shaped, single celled and hyaline. The cleistothecia are dark, globose with the hyaline or pale brown myceloid appendages. The asci are ovate and each ascus produces 2-3 ascospores, which are thin walled, elliptical and pale brown in colour.

Favourable Conditions

Dry humid weather and low relative humidity favours the disease.

Disease cycle

The fungus is an obligate parasite and disease perenneates through cleistothecia in the infected plant debris in soil.

P.S.I:The ascospores from the cleistothecia cause primary infection. S.S.I:The secondary spread is through wind-borne conidia.

Management

- Remove and destroy infected crop debris.
- Spray wettable sulphur@0.3%

Head rot

*Rhizopus sp. (*Mostly *R. arrhizus)*

Economic importance

Head rot generally affects the crop when there is intermittent rain or drizzling during heading stage. Almost total loss may result from this disease because of poor filling

and loss of seeds.

Symptoms

The affected heads show water soaked lesions on the lower surface, which later turn brown. The discoloration may extend to stalk from head. The affected portions of the head become soft and pulpy and insects are also seen associated with the putrified tissues. The larvae (*Helicoverpa armigera*) and insects which attack the head pave way for the entry of the fungus which attacks the inner part of the head and the developing seeds. The **seeds are converted into a black powdery mass**. The head finally withers and droops down with heavy fungal mycelial growth.

Withered head that droops down with heavy fungal mycelial growth

Pathogen

Fungus produces dark brown or black coloured, non-septate hyphae. It produces many aerial stolons and rhizoids. Sporangia are globose and black in colour with a central columella. The sporangiospores are aplanate, dark coloured and ovoid.

Disease cycle

P.S.I:The fungus survives as a saprophyte in host debris and other crop residues.

S.S.I:The disease is spread by windblown spores.

Favourable Conditions

Prolonged rainy weather at flowering and damage caused by insects and caterpillars.

Management

- Treat the seeds with mancozeb and carbendazim at 2g/kg.
- Control the caterpillars feeding on the heads.
- Spray fenthion 1ml plus wettable sulphur @2g per liter of water at the time of head

initiation.

- Spray the head with carbendazim12%+Mancozeb 63%at 1kg/ha during intermittent rainy season and repeat after 10 days, if the humid weather persists.

Sclerotial wilt/Collar rot

Sclerotium rolfsii

Economic importance

This disease was reported in India in 1973.

Symptoms

Initial symptoms of the disease appear 40 days after sowing. Infected plants can be spotted from a distance by their sickly appearance, later the entire plant withers and dies. White cottony mycelium and mustard seed sized sclerotial bodies are formed on the affected stem near soil level.

Witherd and dried plant

White cottony mycelium and mustard seed sized sclerotial bodies on stem

Pathogen

The pathogen produces dark brown to black sclerotia.

Disease cycle

P.S.I:The fungus survives as **sclerotia** in soil and plant debris.

S.S.I:The secondary spread of the disease occurs through sclerotia by implements and irrigation water.

Favourable Conditions

Alternate periods of high soil moisture and water stress conditions predispose the disease.

Management

- Collection and destruction of plant debris

- Seed treatment with captan or carboxin@0.3%
- Drench the base of the plant with cheshunt compound@0.3%
- Addition of soil amendments like oat straw and finely grounded castor and neem cakes reduces disease incidence.
- Use of antagonistic fungi such as *T. harzianum*.

Downy mildew

Plasmopara halstedii

Economic importance

This disease was reported from Latur and Beed districts of Marathwada region of Maharashtra with intensity ranging from 5 to 60%. Later the disease was reported from Karnataka and A.P.

Symptoms

Various kinds of symptoms are being produced by the pathogen like damping off, systemic infection, local lesions and basal rot or stem gall, etc. In systemic infections plants are severely stunted. Chlorosis starts through midribs causing ultimately abnormally thick, down ward curled leaves that show prominent yellow and green epiphyllous mottling. A hypophyllous downy growth of the fungus develops.

downy growth of the fungus

Hypophyllous Erect Head

Flower heads of affected plants remain sterile. Local foliar lesion symptoms are characterized by small angular greenish yellow spots on leaves. Development of basal gall symptoms occur independently of the infection that results in systemic infection. In infected plants **flower heads are erect**.

Disease cycle

P.S.I:Primary infection of the crop occurs through soil borne oospores.

S.S.I:Secondary spread of the disease is through wind borne sporangia and zoospores.

Management

- Regulatory measures to prevent races (other than race 1) of pathogen into India.
- Follow spacing of 60x30cm or 45x30cm
- Rogue out infected plants and destroy
- Cropping sequence of sunflower followed by groundnut reduces the disease.
- Seed treatment with Metalaxyl@0.6% (Apron 35SD) followed by foliar spray with Metalaxyl@0.2% (Ridomyl MZ) is effective.
- Hybrids like LSH-1, LSH-3, KBSH-1, Jwalamukhi, etc had high degree of resistance.

Mosaic

Virus

Symptoms

In infected plants, leaves show irregular yellow or light green patches alternating with normal green areas. Small, chlorotic circular spots develop on leaves which coalesce to form typical mosaic pattern. Cupping and malformation of leaves, poorly developed root system and reduction in pollen fertility are the other symptoms of the disease.

Virus

Irregular yellow or light green patches alternating with normal green areas

Disease cycle

P.S.I: The virus can survive in **amaranthus**.

S.S.I: The virus is transmitted through sap, seed and white flies, *Bamesia tabaci*.

Management

- Rouging of infected plants
- Spray Triazophos 1ml per litre of water.

Sunflower necrosis virus (SND)

Tobacco streak virus

The appearance of SND was observed for the first time during 1997 at Bangalore which later spread to other parts of Karnataka, TN, A.P and Maharashtra. The disease was observed on all stages of crop growth in Kharif as well as in Rabi and the incidence ranged from 5-70%.

Symptoms

Initially small, irregular, necrotic patches appear on leaf lamina more near to the midrib. As the necrosis advances it results into twisting of the leaf, later it extends through one side of the leaf lamina to the petiole and stem and finally terminates at shoot of the plant leading to paralytic symptom. Necrosis at bud formation stage makes the capitulum to bend and twist. The necrosis symptoms appear on bracts and capitulum also. The early infected plants become stunted, weak and die before flowering,. Necrosis affected flower heads fail to open and no seed filling takes place.

Initially small, irregular, necrotic patches appear on leaf lamina, stem,head

Disease cycle

Tobacco streak virus of Ilar group causes the disease. The virus can be transmitted through mechanical, sap inoculation from sunflower to other 22 hosts and vice versa. The virus is transmitted by thrips through infected pollen as carrier. Weed hosts particularly, *Parthenium, Ageratum, Commelina* and *Achyranthus* harbour the virus.

Management

- Removal of weeds plants from the field and adjoining areas of crop.
- Rouging of infected plants before flowering helps to destroy the virus source and spread of the disease.
- Avoid growing of chrysanthemum and marigold close to sunflower.
- Growing 5-7 rows of border crop all around sunflower with sorghum or Bajra
- Seed treatment with Imidachlorpid (Gaucho 70WS) @5g/kg followed by 2-3 sprays at 15 days interval starting from 25 days old seedlings to pre-seed setting stage with Imidachlorpid (Confidor 200SL)@0.05% control the insect vector.