

LECTURE 1

DISEASES OF RICE (*ORYZA SATIVA*)

Blast

Pyricularia oryzae (Syn: *P. grisea*)

(Sexual stage: *Magnaporthe grisea*)

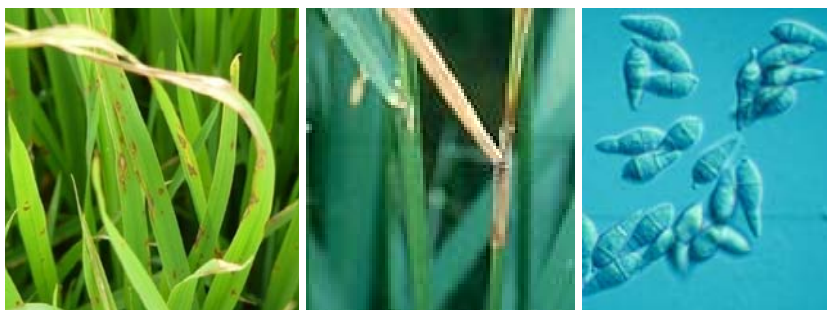
The disease was first recorded in China in 1637. In Japan, it is believed to have occurred as early as in 1704. In Italy the disease called “**brusone**” was reported in 1828 and in USA in 1876. The disease was first recorded from Tanjore district of Tamil Nadu in 1918.

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** 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

3 celled 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

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 secundatum*. The most probable source of perennation and initiation of the disease appear to be the grass hosts and early sown paddy crop. 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 °C or less than 26 °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 °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
- Grow 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.
- Remove and destroy the weed hosts in the field bunds and channels.
- Split application of nitrogen and judicious application of nitrogenous fertilizers
- Treat the seeds with Captan or Thiram or Carbendazim or Carboxin or Tricyclazole at 2 g/kg.
- Seed treatment with biocontrol agent *Trichoderma viride*@ 4g/kg or *Pseudomonas fluorescens* @ 10g/kg of seed. Avoid close spacing of seedlings in the main field.
- Spray the nursery with Carbendazim 25 g or Edifenphos 25 ml for 8 cent nursery.
- Spray the main field with Edifenphos@0.1% or Carbendazim@0.1% or **Tricyclazole** @0.06% or Thiophanate Methyl@0.1%.

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.



Oval to circular spots



Olive green to golden brown conidia

Etiology

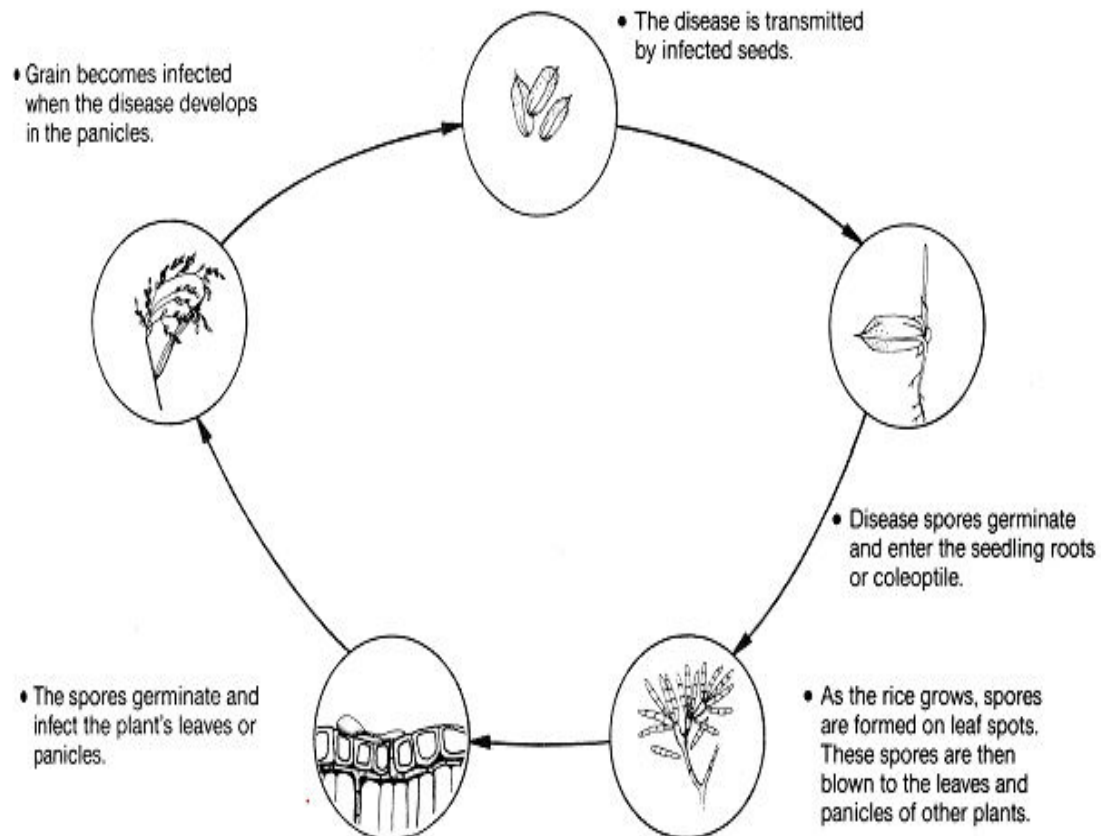
H. oryzae produces greyish-brown to dark brown septate mycelium. Conidiophores may arise singly or in small groups. They are straight, sometime **geniculate**, pale to brown in colour. Conidia are usually curved with a bulge in the centre and tapering towards the ends occasionally almost straight, pale 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 cylindrical, 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

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 italica* and *Cynodon dactylon*.

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 25-30⁰C with relative humidity above 80 per cent are highly favourable. Excess of nitrogen aggravates the disease incidence.

Management

- Use 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.
- Treat the seeds with Thiram or Captan at 4 g/kg and with Mancozeb @0.3%

- Spray the crop in the main field twice with [Mancozeb@0.2%](#), once after flowering and second spray at milky 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 Chittor. 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.



Infection on flag leaf sheath

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.

Favourable Conditions

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

Mode of Spread and Survival

Mainly through air-borne conidia and also seed-borne.

Management

- Apply recommended doses of fertilizers.
- Adopt optimum spacing.
- Spray twice with Carbendazim @0.1% or Benomyl@0.05% or Mancozeb@0.2% or Chlorothalonil@0.2% 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

Sclerotium oryzae

(Sexual stage: *Leptosphaeria 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

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. 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 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
- Avoid flow of irrigation water from infected fields to healthy fields.

Narrow brown leaf spot

Cercospora oryzae

(Sexual stage: *Sphaerulina oryzae*)

Economic importance

It is a minor disease, present in almost all rice growing countries of the world. In the year 1953-54, 40% loss from the disease was reported in Surinam. In Asia, narrow brown leaf spot is important on very susceptible varieties.

Symptoms

The fungus produces short, linear brown spots mostly on leaves and also on sheaths, pedicels and glumes. The spots appear in large numbers during later stages of crop growth. It may also occur as long and about 1mm narrow, short and dark on resistant varieties, but wide and light brown on susceptible ones.



Narrow elongated spots



Cylindrical 3-10 septate conidia

Etiology

Conidiophores are produced in small groups and are brown/dark at the base and pale at the apex with three or more septa. Conidia are hyaline or sub hyaline, cylindrical and 3-10 septate.

Disease cycle

Primary source of inoculum is by means of infected plant debris. Secondary spread of the disease is by means of air borne conidia produced on leaves.

Management

- Destruction of infected plant debris.
- Spray [Mancozeb@0.2%](#) or [Carbendazim@0.1%](#) twice at 15 days interval starting with disease appearance.

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 coalescing 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 are highly susceptible. Five to six week 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.

Pathogen

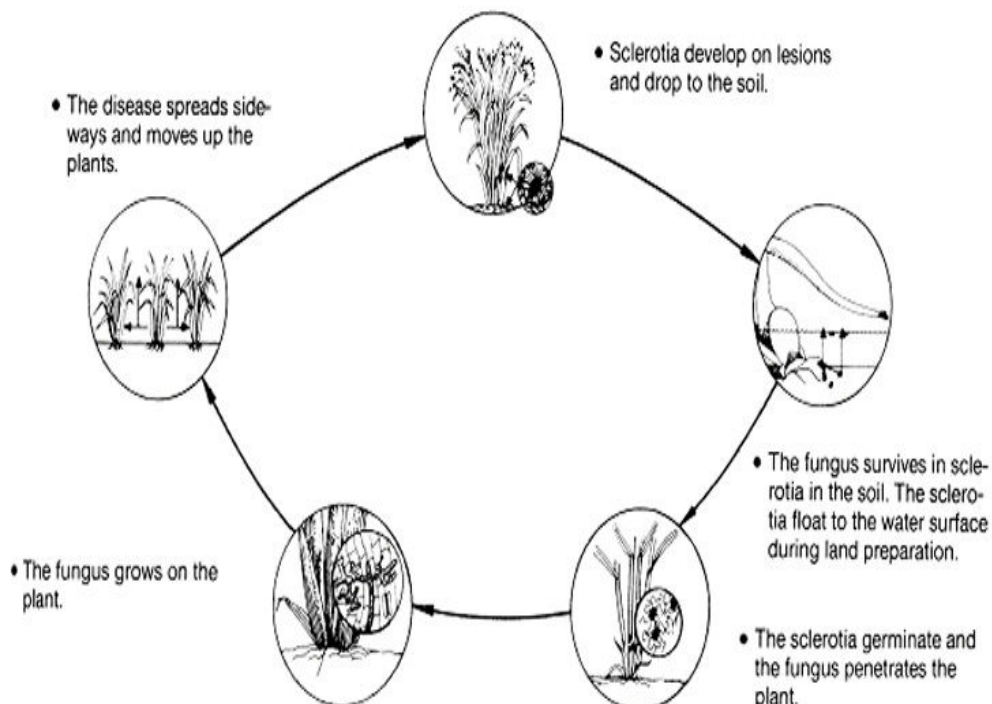
The fungus produces usually long cells of septate mycelium which are hyaline when young, yellowish brown when old. It produces large number of globose **sclerotia**, which are initially white, later turn to brown or purplish brown.

Disease cycle

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. Sclerotia spread through irrigation water.

Favourable Conditions

High relative humidity (96-97 per cent), high temperature (30-32 °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.
- Foliar spray at 0.2% concentration commencing from 45 days after transplanting at 10 days interval for 3 times depending upon the intensity of disease.

False smut

Ustilaginoidea virens

(P.S: *Claviceps oryzae - sativa*)

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.



Yellow or greenish spore balls

Etiology

Chlamydospores are formed on the spore balls, they are spherical to elliptical, waxy and olivaceous.

Disease cycle

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. 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 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 [carbendazim@0.1%](#) 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 soaked 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.

Disease cycle

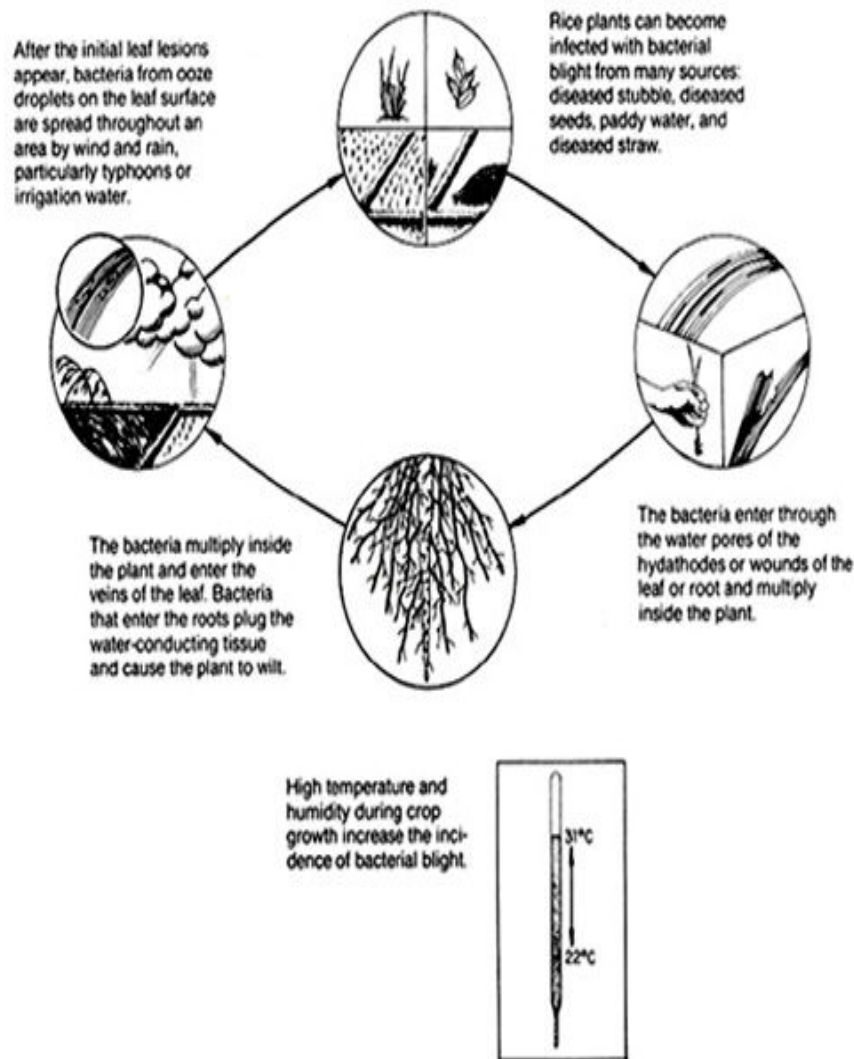
The bacterium enters the plant through water pores (**hydathodes**) along the edges of the leaf and through injuries in roots or leaves. It does not enter through stomata. BLB is primarily a vascular or **systemic** disease. Bacterial cells move along the vascular tissues causing wilting. Rain storms 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. The bacterial ooze serves as secondary inoculum and cause secondary infection.

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 25-30⁰C 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 °C eradicates the bacterium in the seed
- Spray Streptomycin (250 ppm) along with copper oxychloride (0.3%)

Bacterial leaf streak

Xanthomonas campestris p.v. orydicola

Economic importance

Bacterial leaf streak was first found in Phillipines 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.

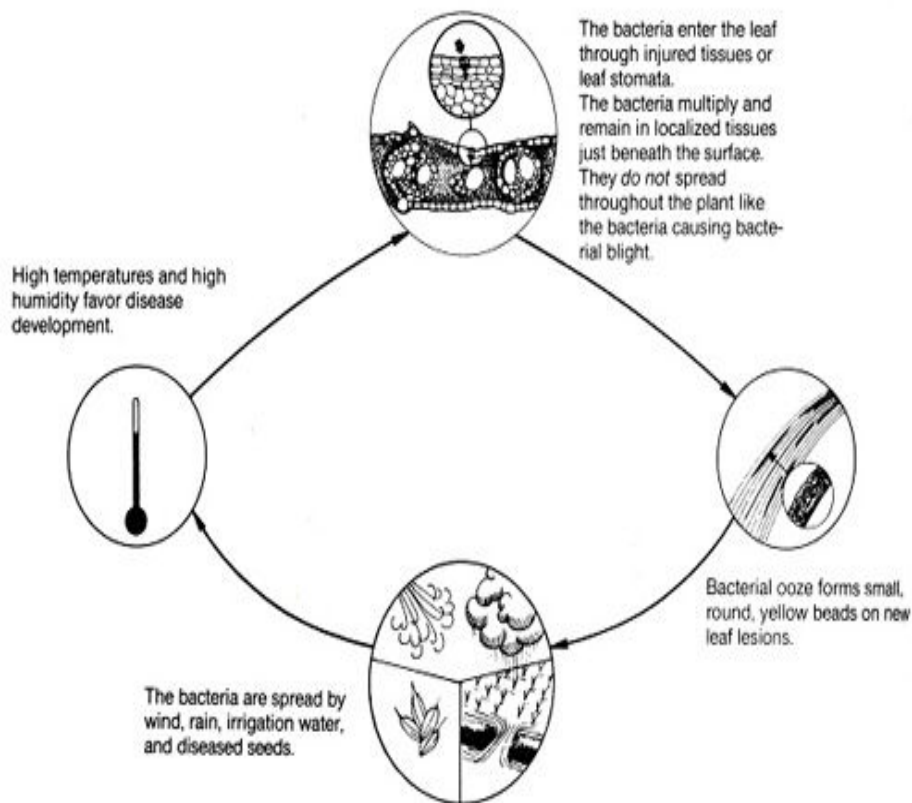


Etiology

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

Disease cycle

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. 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
- Soak the seed in Streptocycline (250 ppm) followed by hot water treatment at 52 °C for 30 minutes eradicates seedling infection.
- Spray Streptocycline (250 ppm) along with copper oxychloride (0.3%)

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 hundred and thousands of hectares in Phillipines.

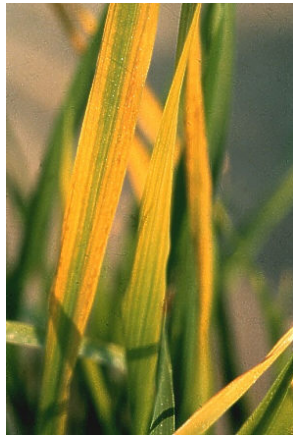
Symptoms

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.

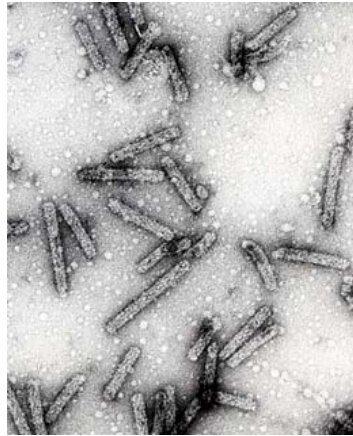
Tungro infected plants can be chemically identified by **Iodine Test**. Ten cm long leaf tip is cut in the early morning before 6 A.M. and dipped in a solution containing 2g Iodine and 6 g Potassium Iodide in 100 ml of water for 30 minutes. Tungro infected leaves show dark blue streaks.

Etiology

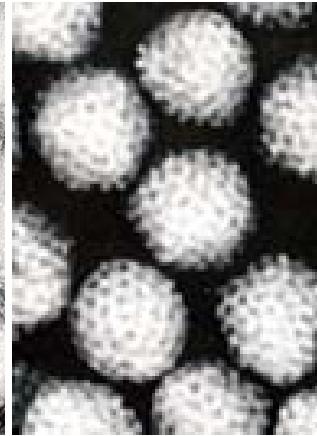
It is a composite disease caused by two morphologically unrelated viruses: rice tungro bacilliform virus (RTBV) and rice tungro spherical virus (RTSV). **RTBV** has a bacilliform capsid or bullet-shaped particles (130 x 30 nm) made up of a single piece of coat protein of MW 36 K and a single molecule 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 or orange
discolouration**



RTBV



RTSV

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

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. The **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.
- Control the vectors in the nursery by application of carbofuran granules @170 g/cent, 10 days after sowing and @10kg/ac in main field
- Spray Monochrotophos@2.2 ml/lt or 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 6 & 7

DISEASES OF SORGHUM (*SORGHUM BICOLOR*)

Anthracnose or red leaf spot

Colletotrichum 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.



Anthracnose

Stalk rot

Black acervuli

Falcate conidia

Etiology

The mycelium of the fungus is localized in the spot. Acervuli 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.

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°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.

- Treat the seeds with Captan or Thiram @3 g/kg.
- Spray the crop with Mancozeb @0.25% or carbendazim@0.1%

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

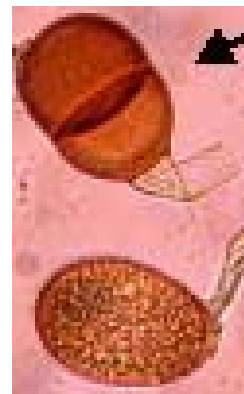
The fungus affects the crop at all stages of growth. The intensity of rust infection is generally severe after flag leaf stage of the crop growth. The first symptoms are small **reddish brown flecks** on the lower surface of the leaf (purple, tan or red depending upon the cultivar). Pustules (uredosori) 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. **Teliospores** 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.



Rust on leaf



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°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 Mancozeb @0.25%
- 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.



Honey dew

Ergots

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

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 °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 Captan or Thiram@4g/kg seed
- Spray **Ziram** (or) Zineb (or) Captan (or) Mancozeb @0.2% 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.



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 disinfection with [Thiram@0.3%](#) will prevent seedling infection.
- Spray Mancozeb (0.25%) or captan (0.2%) or captan 2g + Aureofungin 200ppm per liter, in case of intermittent rainfall during earhead emergence, a week later and during milky stage.

Leaf blight or leaf stripe

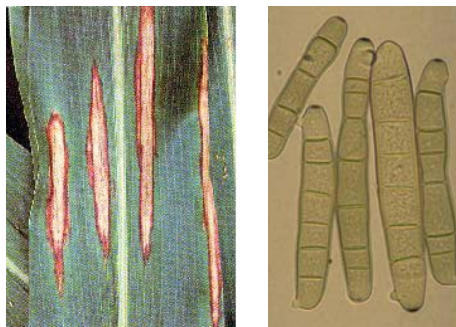
Exserohilum turcicum or *Trichometasphaeria turcica*
(Syn : *Helminthosporium turcicum* or *Drechslera turcicum*)

Economic importance

In India, the disease is more prevalent in MP, AP and Karnataka causing heavy losses. If leaf blight is established on susceptible cultivars before earhead emergence, grain yield losses may be upto 50%.

Symptoms

The leaf blight pathogen also causes seed rot and seedling blight of sorghum. The disease appears in the form of small narrow elongated spindle shaped spots in the initial stage. But in due course, they extend along the length of the leaf becoming bigger.



Elliptical lesion

Conidia

On older plants, the typical symptoms are **long elliptical necrotic lesions**, straw coloured in the centre with dark margins. The straw coloured centre becomes darker with the sporulation of the fungus. The lesions can be several centimeters long and wide. Many lesions may develop and coalesce on the leaves, destroying large areas of leaf tissue, giving the crop a distinctly burnt appearance leading to premature drying of leaves.

Pathogen

The mycelium is brown and intercellular and is localized in the infected lesion. Conidiophores emerge through stomata and are long, olivaceous, septate and geniculate. Conidia are olivaceous brown, 3-8 septate and thick walled.

Disease cycle

The fungus is found to persist in seed, soil and infected plant debris. Seed borne conidia are responsible for seedling infection. The secondary spread of the disease is through wind-borne conidia and seed.

Favourable Conditions

Cool moist weather, high humidity (90 per cent) and high rainfall.

Management

- Use disease free seeds.
- Rotation with non susceptible crops
- Collect and destroy infected plant debris
- Treat the seeds with Captan or Thiram at 4 g/kg.
- Spray the crop with [Mancozeb@0.25%](#) at the age of 40 days and the spraying have to be repeated twice at 15 days interval
- Tift-Sudan is resistant to this disease

Grain smut/Kernel smut / Covered smut / Short smut

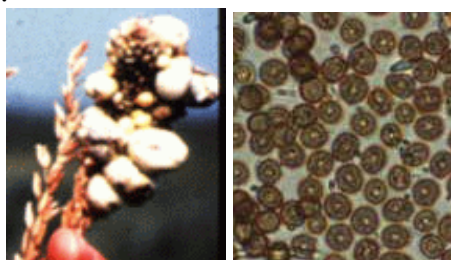
Sphacelotheca sorghi or *Sporisorium sorghi*

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.



Smut sori

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 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 5-9 μ 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 Captan or Thiram @0.3%.
- 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.

Disease cycle

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



Blackening of roots Shredding of tissue

Favourable conditions

Disease is favoured by soil temperature of 35⁰C 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
- 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.



White streaks on leaves Downy white growth Leaf shredding

Pathogen biology

P. sorghi is systemic in young host plant in the form of intercellular, non-septate mycelium. It is an obligate parasite. Sporangioophores 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 21-23⁰C. 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% or Mancozeb @0.25%

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°C 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 or Praquat@1kg/ha.
- 1% Tetrachloro dimethyl phenoxy acetate can be used for instant killing of *Striga*, if water is in scarce.

LECTURE 7& 8

DISEASES OF MAIZE (*ZEA MAYS*)

Turcicum Leaf blight

Helminthosporium turcicum

(Syn : *H. maydis*)

Economic importance

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.



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

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**. Secondary spread is through wind borne conidia.

Favourable Conditions

Optimum temperature for the germination of conidia is 18 to 27°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 Captan or Thiram at 4 g/kg
- Spray [Mancozeb@0.25%](#)

Post flowering stalk rot
***Cephalosporium* wilt (Black bundle disease and late wilt)**
Cephalosporium acremonium/ Cephalosporium 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.

Pathogen

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

Disease cycle

Both the pathogens survive in soil, plant debris and seed

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 Thiram or captan@3g/kg seed
- 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 (35-45⁰C). Yield losses upto 10-50% are common.

Symptoms

Charcoal rot commonly attacks plants approaching maturity. The fungus produces brown, water soaked 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 black sclerotia on vascular strands, giving the interior of the stalks a charred appearance which is a characteristic symptom of the disease. Sclerotia may also be found on the roots.

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

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. Sclerotia spread through irrigation water.

Favourable Conditions

Development of charcoal rot is favoured by dry weather with high temperature (37°C) 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 Carbendazim or Captan at 2 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



Survival

The fungus survives on weeds and in the plant debris.

Management

- Clean cultivation
- Destruction of crop debris
- Spray carbendazim 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*

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**).

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 20-25⁰C favours the disease development. Young plants are highly susceptible.

Mode of Spread and Survival

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. Secondary spread is through air-borne conidia. The **oospores** survive in the soil as well as in the infected plant debris.

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.

LECTURE 9

DISEASES OF BAJRA OR PEARL MILLET (*Pennisetum typhoides*)

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

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

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. 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°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 varieties 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 or Thiram or Captan@4g/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.

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 germ pores, 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

Primary infection is from the alternate host, brinjal, in nature. 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% or [Mancozeb@0.2%](#) starting from 21 days after planting
- Grow resistant varieties like RT 814-3, PT 826/4, PT 829/5, etc.

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.

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

Sclerotia are viable in soil for 6-8 months. The primary infection takes place by germinating sclerotia present in the soil. Secondary spread is by insects or air-borne conidia and ascospores. The role of collateral hosts like *Cenchrus ciliaris* and *C. setigerus* in perpetuation of fungus is significant. The fungus also infects other species of *Pennisetum*.

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°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 [Ziram@0.2%](#) or [Carbendazim@0.1%](#) or [Mancozeb@0.2%](#) at boot leaf and flowering stage

Smut

Tolyposporium penicillariae

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.

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

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. 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
- Seed treatment with Thiram or Captan@3g/kg seed
- Grow resistant varieties like DC 7, MPP 7131 and MPP 7108.
- Spray Carboxin or Zineb@0.2%

LECTURE 10

RAGI / FINGER MILLET (*Eleusine coracana*)

Blast

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 grain set at all. If grain setting occurs, they are shrivelled and reduced in size.



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.

Disease cycle

The fungus is seed-borne and the primary infection takes place through the seed-borne conidia and also through diseased plants, stubbles and weeds. The secondary spread is through air-borne conidia.

Favourable Conditions

Application of high doses of nitrogenous fertilizers, low night (20⁰C) and day (30⁰C) 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 Captan or Thiram@3g/kg or Carbendazim at 2 g/kg.
- Grow resistant varieties like Ratnagiri, Padmavati, Gowtami and Godavari
- Spray with [Carbendazim@0.2%](#) or Iprobenphos (IBP)@0.1% or Edifenphos@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 spores.



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

Disease cycle

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. 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.

Disease cycle

Besides ragi, the virus also infects Setaria, maize, sugarcane and sorghum and 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 [monochrotophos@1.5ml/lt](#) or dimethoate@2ml/lt to control the vector

LECTURE 11, 12 & 13

Wheat (*Triticum* sp.)

Black or stem rust

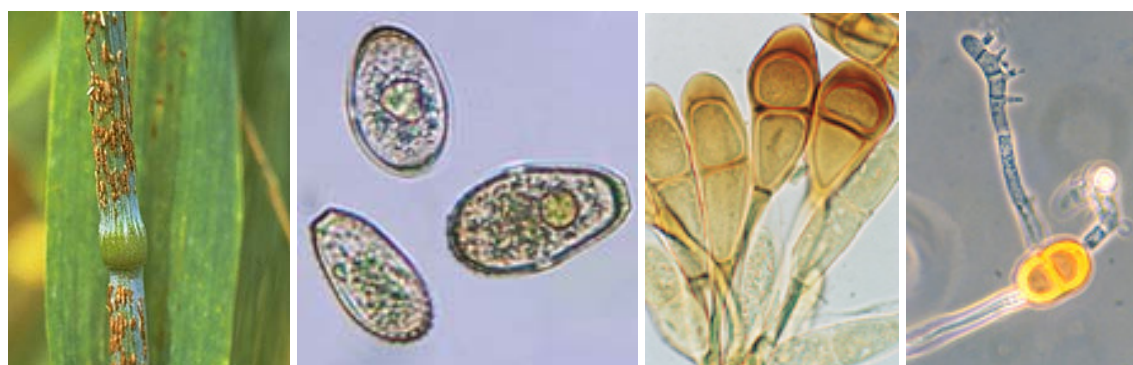
Puccinia graminis tritici

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.



Stem rust

Uredospores

Teliospores

Promycelium

Pathogen

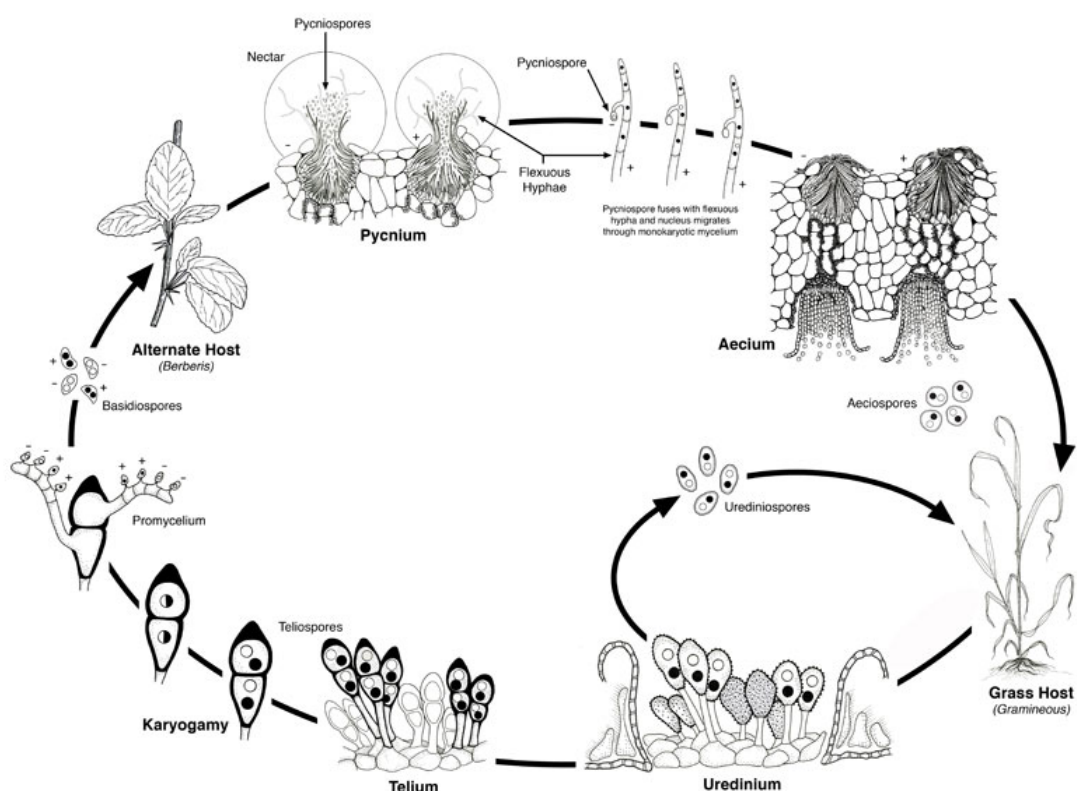
Black stem rust is **heteroecious** full cycle rust. It requires more than one host species to complete its life cycle. The uredial and telial stages occur on wheat, barley and some grasses and the pycnial and aecial stages on the species of *Berberis* (Barbery) and *Mahonia*, the alternate hosts. The 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

Primary infection is mainly through **barberry**, i.e., *Berberis vulgaris*. These barberry 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.



Management

- Eradication of self sown 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 [Zineb@0.25%](#) or [Mancozeb@0.25%](#) or [Plantavax@0.1%](#) , at 15 days interval.

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.



Brown rust

Teliospores

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

Alternate host, species of *Thalictrum*, helps the fungus to overwinter 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 build up 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.

Management

- Grow resistant varieties like Sonalika, NP 700 & 800, Lerma Rojo and Safed Lerma.
- RH-124, an Indofil product is very specific to brown rust (or) spray dithiocarbamates like [zineb@0.25%](#) or [Mancozeb@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.



Yellow rust



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

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 catharticus*, *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.

Management

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

Loose smut

Ustilago nuda 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.

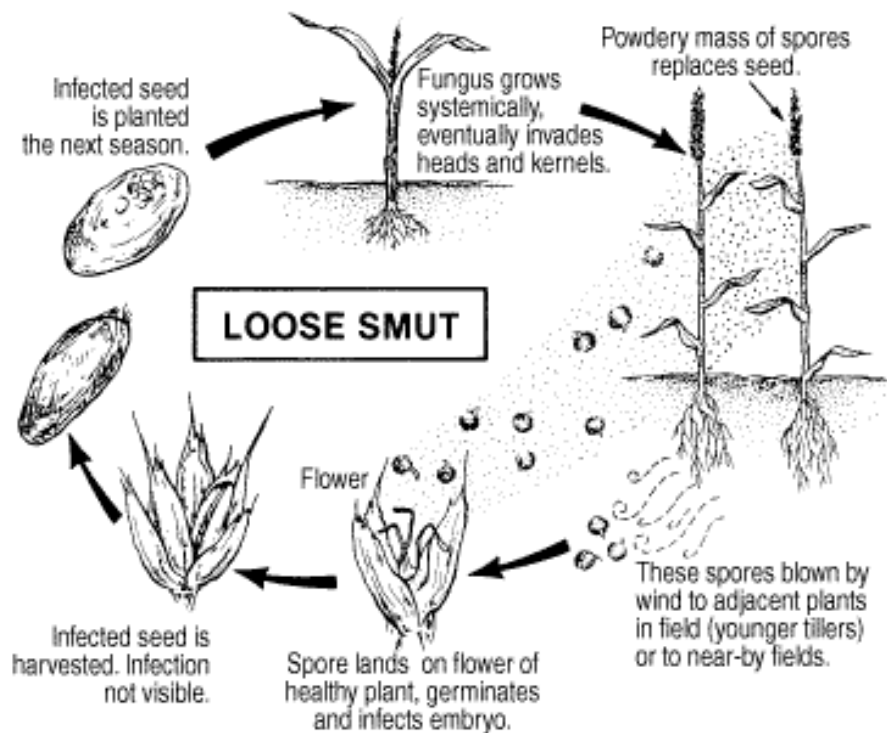
Symptoms

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.



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

It is **internally** and **externally 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.

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 °F or 52°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°C)
- **Anaerobic seed treatment (USA)**: Soak the seeds for 2-4 hours in water between 60-70°F 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%](#) or [Benlate@0.2%](#)

Karnal bunt

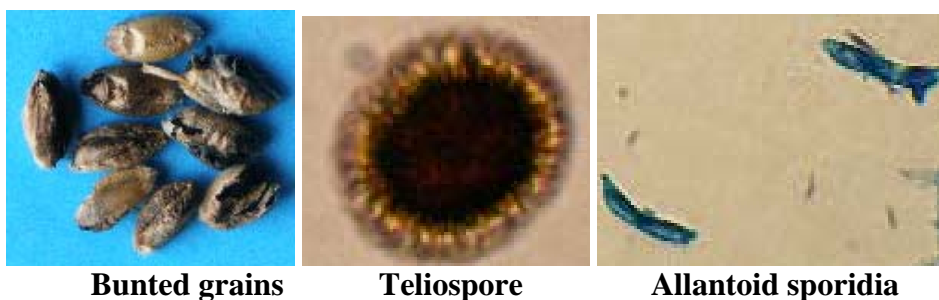
Neovossia indica (formerly *Tilletia indica*)

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 sack 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**.



Pathogen

Teliospores are smooth walled measuring 22-49 μ 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

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 (19-23⁰C), 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 Thiram@3g/kg seed
- Spray with carbendazim@0.1% or carboxin@0.2% or [Mancozeb@0.25%](#) or bitertanol

Leaf blight

Alternaria triticina

Economic importance

Reported by **Prasad** and **Prabhu** in 1962 from India. It is prevalent in parts of Maharashtra, Bihar, West Bengal and UP. Seedlings are not prone to infection.

Symptoms

Reddish brown oval spots appear on young seedlings with bright yellow margin. In severe cases, several spots coalesce to cause drying of leaves. The young leaves are not usually infected. Heavily infected fields display a burnt appearance even from a distance. In some varieties reduction in grain yield is as high as 90% if the infection takes place at or before the boot leaf stage.

Pathogen

Fungus produces light brown coloured multicellular conidia, with 1-10 transverse septa and 1-5 longitudinal septa, singly or in chains (2-4).

Disease cycle

Pathogen over summers in plant debris and soil. Primary spread is by **externally** and **internally** seed-borne conidia. Secondary infection is mainly through wind-borne conidia.

Favourable Conditions

Temperature of 25 °C and high relative humidity favours the disease.

Management

- Soak the seeds in water for 4 hrs followed by 10 min. dip in hot water at 52°C.
- Grow resistant varieties like Co.25, Sonalika, Arnautka, E6160 and K7340.
- Spray the crop with [Mancozeb@0.25%](#) or [Zineb@0.25%](#)

Tundu disease or yellow slime disease

Anguina tritici (Nematode) + *Corynebacterium tritici* or *Clavibacter 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 winking, 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.

Pathogen

Corynebacterium is rod shaped, Gram positive and is motile by single polar flagellum.

Disease cycle

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.

Management

- Sow gall free seeds. Separate the galls from the seed by floating in brine at 160 g of sodium chloride in liter of water.
- Wheat, barley or oat should not be sown in the infested soil.
- Spray the crop with streptocycline, 1g in 10 liters of water.

LECTURE 14

DISEASES OF COTTON (*GOSSYPIUM SPP.*)

Bacterial blight or Angular leaf spot or Black arm

Xanthomonas campestris pv. *malvacearum*

Economic importance

This disease was first observed in Tamil Nadu in 1918. It is an important disease in Maharashtra, Karnataka, A.P., Tamil Nadu and Madhya Pradesh.

Symptoms

The bacterium attacks all stages from seed to harvest. Usually five common phases of symptoms are noticed.

- i) Seedling blight:** Small, water-soaked, circular or irregular lesions develop on the cotyledons. Later, the infection spreads to stem through petiole and cause withering and death of seedlings.
- ii) Angular leaf spot:** Small, dark green, water soaked areas develop on lower surface of leaves, enlarge gradually and become angular when restricted by veins and veinlets and spots are visible on both the surface of leaves. As the lesions become older, they turn to reddish brown colour and infection spreads to veins and veinlets.
- iii) Vein blight or vein necrosis or black vein:** The infection of veins causes blackening of the veins and veinlets, gives a typical 'blighting' appearance. On the lower surface of the leaf, bacterial oozes are formed as crusts or scales. The affected leaves become crinkled and twisted inward and show withering. The infection also spreads from veins to petiole and cause blighting leading to defoliation.
- iv) Black arm:** On the stem and fruiting branches, dark brown to black lesions are formed, which may girdle the stem and branches to cause premature drooping off of the leaves, cracking of stem and gummosis, resulting in breaking of the stem which hang typically as dry black twig to give a characteristic "black arm" symptom.
- v) Square rot / Boll rot:** On the bolls, water soaked lesions appear and turn into dark black and sunken irregular spots. The infection slowly spreads to entire boll and shedding occurs. The infections on mature bolls lead to premature bursting of bolls. The bacterium spreads inside the boll and lint gets stained yellow because of bacterial ooze and loses its appearance and market value. The pathogen also infects the seed and causes reduction in size and viability of the seeds.



Angular leaf spot



Veinal necrosis



Boll rot

Pathogen

The bacterium is a short rod with a single polar flagellum. It is gram negative, non-spore forming and measures 1.0-1.2 X 0.7-0.9 µm. The bacterium is aerobic, capsule forming and produces yellow colonies in culture medium.

Disease cycle

The bacterium survives on infected dried plant debris in soil for several years. The bacterium is also seed-borne and remains in the form of slimy mass on the fuzz of seed coat. It multiplies soon after the seed is sown and infects the seedling through the micropyle. Volunteer plants that arise from the bolls falling off prematurely also provide a source of primary infection. The bacterium also attacks other hosts like *Thurbaria thespesioides*, *Eriodendron anfructuosum* and *Jatropha curcas*. The primary infection starts mainly from the seed-borne bacterium. The secondary spread of the **bacteria** may be through wind, wind blown rain splash, irrigation water, insects and other implements. The bacterium enters through **natural openings** or insect caused **wounds**.

Favourable Conditions

Optimum soil temperature of 28⁰C, high atmospheric temperature of 30-40⁰C, relative humidity of 85 per cent, early sowing, delayed thinning, poor tillage, late irrigation and potassium deficiency in soil. Rain followed by bright sunshine during the months of October and November are highly favourable.

Management

- Remove and destroy the infected plant debris.
- Rogue out the volunteer cotton plants and weed hosts.
- Follow crop rotation with non-host crops.
- Early thinning, good tillage, early irrigation, early earthing up and addition of potash to the soil reduces disease incidence.
- Grow resistant varieties like HG-9, BJA 592, G-27, Sujatha, 1412 and CRH 71. Suvin is tolerant.
- *Gossypium herbaceum* and *G. arboreum* are almost immune. *G. barbadense*, *G. hirsutum*, *G. herbaceum var typicum* and *G. herbaceum var acerifolium* have considerable resistance.
- Delint the cotton seeds with **concentrated sulphuric acid** at 125ml/kg of seed.
- Treat the delinted seeds with Carboxin at 2 g/kg seed or soak the seeds in 1000 ppm Streptomycin sulphate overnight or treat the seed with hot water at 52-56⁰C for 10-15 minutes.
- Spray with Streptomycin sulphate (Agrimycin 100), 500 ppm along with Copper oxychloride at 0.3%.

Fusarium wilt

Fusarium oxysporum f.sp. *vasinfectum*

Symptoms

The disease affects the crop at all stages. The earliest symptoms appear on the seedlings in the **cotyledons** which turn yellow and then brown. The base of petiole shows brown ring, followed by wilting and drying of the seedlings. In young and grown up plants, the first symptom is **yellowing of edges of leaves** and area around the veins, *i.e.* discolouration starts from the margin and spreads towards the midrib. The leaves lose their turgidity, gradually turn brown, droop and finally drop off. Symptoms start from the older leaves at the base, followed by younger ones towards the top, finally involving the branches and the

whole plant. The defoliation or wilting may be complete leaving the stem alone standing in the field. Sometimes partial wilting occurs; where in only one portion of the plant is affected, the other remaining free. The taproot is usually stunted with less abundant laterals. **Browning or blackening of vascular tissues** is the other important symptom, black streaks or stripes may be seen extending upwards to the branches and downwards to lateral roots. In severe cases, discolouration may extend throughout the plant starting from roots extending to stem, leaves and even bolls. In transverse section, discoloured ring is seen in the woody tissues of stem. The plants affected later in the season are stunted with **fewer bolls** which are very small and open prematurely.



Marginal chlorosis & necrosis



Browning of vascular bundles

Pathogen

The fungus produces three types of spores. **Macroconidia** are 1 to 5 septate, hyaline, thin walled, falcate with tapering ends. The **microconidia** are hyaline, thin walled, spherical or elliptical, single or two celled. **Chlamydospores** are dark coloured and thick walled. The fungus also produces a vivotoxin, **Fusaric acid** which is partially responsible for wilting of the plants.

Disease cycle

The fungus can survive in soil as saprophyte for many years and **chlamydospores** act as resting spores. The pathogen is both externally and internally seed-borne. The primary infection is mainly from dormant hyphae and chlamydospores in the soil. The secondary spread is through **conidia** and **chlamydospores** which are disseminated by irrigation water.

Favourable Conditions

Soil temperature of **20-30°C**, hot and dry periods followed by rains, heavy black soils with an alkaline reaction, increased doses of nitrogen and phosphatic fertilizers, soil amendment with manganese and wounds caused by nematode (*Meloidogyne incognita*) and grubs of Ashweevil (*Myloccerus pustulatus*).

Management

- Treat the acid-delinted seeds with Carboxin or Chlorothalonil at 4 g/kg or Carbendazim@2g/kg seed
- Remove and burn the infected plant debris in the soil after deep summer ploughing.
- Apply increased doses of potash with a balanced dose of nitrogenous and phosphatic fertilizers.
- Multiply *Trichoderma viride* (2kg) in 50 kg of Farm yard manure for 15 days and then apply to the soil.

- Apply heavy doses of farm yard manure or other organic manures at 10 t/ha. Follow mixed cropping with non-host plants to lower the soil temperature below 20°C by providing shade.
- Soil amendment with zinc.
- Grow disease resistant varieties of *G. hirsutum* and *G. barbadense* like Varalakshmi, Vijaya, Pratap, Jayadhar, Jarila, Jyothi, G 22 and Verum.

Verticillium wilt *Verticillium dahliae*

Economic importance

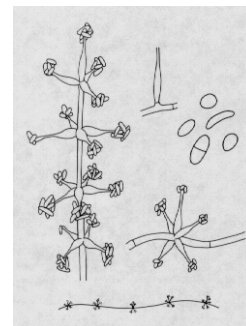
The disease is a major disease in cotton in **USA** and **USSR** and was first reported in India during 1968 on *hirsutum* cottons in Coimbatore, Tamil Nadu. The disease usually appears in November and December when the crop is in **squares** and **bolls**, about three months after sowing.

Symptoms

The symptoms are seen when the crop is in squares and bolls. Plants infected at early stages are severely stunted. The first symptoms can be seen as distinct **mottling** of leaves with pale yellowish irregular areas at the margins and between the principal veins. The yellowish areas become pale, more whitish and extensively necrotic. The **necrosis** of the leaves spreads from lower to upper leaves and there is heavy defoliation. The affected leaves fall off leaving the branches barren. Infected stem and roots, when split open, show a **pinkish to pinkish brown discolouration** of the woody tissue which may be continuous or interrupted. Pinkish streaks alternating with healthy tissue (**Tiger stripe**) are seen on removing the bark of the roots, stem and petiole. The affected plants may bear a few smaller bolls with immature lint.



Pinkish discolourations



Conidiophore & conidia

Pathogen

The fungus produces hyaline, septate mycelium and two types of spores. The conidia are single celled, hyaline, spherical to oval, borne singly on verticillate conidiophores. The micro sclerotia are globose to oblong, measuring 48-120 X 26-45um.

Disease cycle

The fungus also infects the other hosts like **brinjal**, **chilli**, **tobacco** and **bhendi**. The fungus can survive in the infected plant debris and in soils as **microsclerotia** upto 14 years. The seeds also carry the microsclerotia and conidia in the fuzz. The primary spread is through the micro sclerotia or conidia in the soil. The secondary spread is through the

contact of diseased roots to healthy ones and through dissemination of infected plant parts through irrigation water and other implements.

Favourable Conditions

Low temperature of **15-20°C**, low lying and ill-drained soils, heavy soils with alkaline reaction and heavy doses of nitrogenous fertilizers favours the disease.

Management

- Treat the delinted seeds with Carboxin@4g/kg or Carbendazim at 2 g/kg.
- Remove and destroy the infected plant debris after deep ploughing in summer months.
- Apply heavy doses of farm yard manure or compost at 10t/ha.
- Follow crop rotation by growing **paddy** or **Lucerne** or **chrysanthemum** for 2-3 years.
- Spot drench with 0.05 per cent Benomyl or Carbendazim.
- Grow disease resistant varieties like Sujatha, Suvin and CBS 156.

Root rot

Rhizoctonia bataticola

(Pycnidial stage: *Macrophomina phaseolina*)

Economic importance

This disease is severe in many parts of India, especially in Punjab and Gujarat.

Symptoms

The fungus causes three types of symptoms, *viz.*, seedling disease, sore-shin and root rot. Germinating seedlings of one to two weeks old are attacked by the fungus at the **hypocotyl** and cause black lesions, girdling of stem and death of the seedling, causing large gaps in the field. In sore-shin stage (4 to 6 weeks old plants), **dark reddish-brown cankers** are formed on the stems near the soil surface which later turns dark brown or black and plant breaks at the collar region leading to drying of the leaves and subsequently the entire plant. Typical **root rot** symptom appears normally at the time of **maturity** of the plants. The most prominent symptom is sudden and complete wilting of plants in patches in **concentric circles**. Initially, all the leaves droop suddenly and die within a day or two. The affected plants when pulled reveal the rotting of entire root system except tap root and few laterals. The bark of the affected plant shreds and even extends above ground level. In badly affected plants the woody portions may become **black** and **brittle**. A large number of dark brown **sclerotia** are seen on the wood or on the shredded bark.



Pathogen

The fungal hyphae are septate and fairly thick and produce black, irregular **sclerotia** which measure 100 µm in diameter.

Disease cycle

The disease is mainly soil-borne and the pathogen can survive in the soil as **sclerotia** for several years. The spread is through sclerotia which are disseminated by **irrigation water**, implements, heavy winds and other cultural operations.

Favourable Conditions

Dry weather following heavy rains, high soil temperature (**35-39°C**), low soil moisture (**15-20 per cent**), cultivation of favourable hosts like vegetables, oil seeds and legumes preceding cotton and wounds caused by ash-weevil grubs and nematodes.

Management

- Treat the seeds with *Trichoderma viride* @ 4g/kg or *Pseudomonas fluorescens* @ 10g/kg of seed.
- Treat the seeds with Carboxin or Thiram at 4 g or Carbendazim at 2g/kg.
- Spot drench with 0.1% Carbendazim or 0.05% Benomyl.
- Apply farm yard manure at 100 t/ha or neem cake at 2.5t/ha.
- Adjust the sowing time, early sowing (First Week of April) or late sowing (Last week of June) so that crop escapes the high soil temperature conditions.
- Adopt intercropping with **sorghum** or **moth bean** (*Phaseolus aconitifolius*) to lower the soil temperature.
- Grow resistant varieties like KH 33/1146, 15/KW-2 (MB)

Grey or Areolate mildew

Ramularia areola

(Sexual stage: *Mycosphaerella areola*)

Economic importance

The disease is common in many parts of India and is severe in low-lying areas with high humidity.

Symptoms

The disease usually appears on the under surface of the lower leaves when the crop is nearing maturity. **Irregular to angular pale translucent lesions** which measure 1-10 mm (usually 3-4 mm) develop on the lower surface, usually bound by veinlets. On the upper surface, the lesions appear as light green or yellow green specks. Whitish grey or frosty powdery growth, consisting of conidiophores of the fungus, appears on the lower surface. When several spots coalesce, the entire leaf surface is covered by **white to grey powdery growth**. The infection spreads to upper leaves and entire plant may be affected. The affected leaves dry up from margin, turn yellowish brown and fall off prematurely.

Pathogen

The fungus produces endophytic, septate mycelium. Conidiophores are short, hyaline and branched at the base. **Conidia** are borne singly or in chains at the tips of conidiophores. The conidia are hyaline, irregularly oblong with pointed ends, sometimes rounded to flattened ends, unicellular or 1-3 septate. The perfect stage of the fungus produces **perithecia** containing many asci. The ascospores are hyaline and usually two celled.

Disease cycle

The fungus survives during the summer in the infected crop residues. The perennial cotton plants and self-sown cotton plants also harbor the pathogen during summer months. The

primary infection is through conidia from infected plant debris and secondary spread is through wind, rain splash, irrigation water and implements.

Favourable Conditions

Wet humid conditions during winter cotton season, intermittent rains during North-East monsoon season, low temperature (20-30⁰C) during October-January, close planting, excessive application of nitrogenous fertilizers, very early sowing or very late sowing of cotton and growing highly susceptible varieties/hybrids like Suvin, DCH 32, MCU 5, and MCU 9.

Management

- Remove and burn the infected crop residues.
- Rogue out the self-sown cotton plants during summer months.
- Avoid excessive application of nitrogenous fertilizers/manures.
- Adopt the correct spacing based on soil conditions and varieties.
- Spray the crop with Carbendazim@0.1% or BM@1% or Wettable sulphur at 1.25-2.0 kg/ha, repeat after a week.
- Grow the resistant varieties like Sujatha and Varalakshmi.

Anthracnose

Colletotrichum capsici

Symptoms

The fungus infects the seedlings and produces small reddish circular spots on the cotyledons and primary leaves. The lesions develop on the collar region, stem may be girdled, causing seedling to wilt and die. In mature plants, the fungus attacks the stem, leading to stem splitting and shredding of bark. The most common symptom is boll spotting. Small water soaked, circular, reddish brown depressed spots appear on the bolls. The lint is stained to yellow or brown, becomes a solid brittle mass of fiber. The infected bolls cease to grow and burst and dry up prematurely.



Pathogen

The pathogen forms large number of **acervuli** on the infected parts. The conidiophores are slightly curved, short, and club shaped. The conidia are hyaline and **falcate**, borne single on the conidiophores. Numerous black coloured and thick walled **setae** are also produced in acervulus.

Disease cycle

The pathogen survives as dormant mycelium in the seed or as conidia on the surface of seed for about a year. The pathogen also perpetuate on the rotten bolls and other plant

debris in the soil. The secondary spread is by air-borne conidia. The pathogen also survives in the weed hosts, viz., *Aristolachia bractiata* and *Hibiscus diversifolius*.

Favourable Conditions

Prolonged rainfall at the time of boll formation and close planting predispose the disease.

Management

- Treat the delinted seeds with Carbendazim or Carboxin@2g/kg or Thiram or Captan at 4g/kg.
- Remove and burn the infected plant debris and bolls in the soil.
- Rogue out the reservoir weed hosts.
- Spray the crop at boll formation stage with Mancozeb@0.25% or Copper oxychloride@0.3% or Ziram@0.25% or Carbendazim@0.1%.

Leaf spots – *Alternaria*, *Cercospora*, *Helminthosporium* and *Myrothecium*

***Alternaria* leaf spot** *Alternaria macrospora*

Symptoms

The disease may occur in all stages but more severe when plants are 45-60 days old. Small brown, round spots surrounded by a purple margin appear on leaves. On older leaves the necrotic center of the spots may be marked by a pattern of concentric zonation. Several spots coalesce together to form blighted areas. Under humid weather conditions the spots appear as sooty black leading to premature defoliation. Sometimes stem lesions are also seen. In severe cases, the leaf stalk and bolls become infected with spherical or elliptical purple spots.



Disease cycle

The pathogen survives in the infected crop debris as dormant mycelium. The secondary spread is mainly by air-borne conidia.

Pathogen

The fungus produces dark brown, short, 1-8 septate, irregularly bent conidiophores with a single conidium at the apex. The conidia are obclavate, light to dark brown in colour with 3-9 transverse septa and 4 longitudinal septa, with a prominent beak.

Favourable Conditions

High humidity, intermittent rains and moderate temperature of 25-28⁰C favours the disease incidence.

Management

- Remove and destroy the infected plant residues.
- Deep summer ploughing

- Avoid seeds from infected crop
- Spray Mancozeb@0.25% or Copper oxychloride@0.3% at the initiation of the disease. Four to five sprays may be given at 15 days interval.

***Cercospora* Leaf spot**

Cercospora gossypina

Symptoms

Usually the symptoms appear on lower leaves. At first, small water soaked lesions appear on upper surface of leaf. The spots enlarge and develop into circular or irregular spots with grayish white centre surrounded by brown margin. Many such spots coalesce to form big irregular patches. The centre of the spot may fall off leading to shot hole formation. The leaves may drop off.

Disease cycle

The pathogen survives in the infected plant debris as conidia. The secondary spread is mainly by air-borne conidia.

Management

- Remove and destroy the infected plant residues.
- Spray Mancozeb@0.25% or Copper oxychloride@0.3% or zineb@0.2% or vitavaz@0.1% at the initiation of the disease.

***Helminthosporium* Leaf spot**

Helminthosporium gossypii

Symptoms

The spots are grayish white in colour with deep purple margin. Usually circular spots may coalesce leading to the drying of leaves.

Disease cycle

The pathogen survives in the dead leaves as conidia. The secondary spread is mainly by air-borne conidia.

Management

- Remove and destroy the infected plant residues.
- Spray Mancozeb@0.25% or Copper oxychloride@0.3% or BM@1% at the initiation of the disease.
- Grow resistant varieties like SRT-1, AC 738, PS 10 and JR 78.

RUST

Phakopsora gossypii (Tropical rust), *Puccinia cacabata* (South western rust),

Puccinia schedonnardi (Cotton rust-USA)

Economic importance

The disease usually occurs after September, *i.e.*, at the end of the season. It is difficult to assess the loss due to bacterial blight.

Symptoms

Most common symptom is the appearance of bright yellow orange spots usually on under surface of the lower leaves. These pustules are surrounded by purple borders. Spots

become brown with age. Spots may appear on any of the above ground parts including bracts and bolls. Severe infections may cause defoliation and reduction in the size of the bolls. On stems and petioles these pustules are usually elongated and are not much raised.



Pathogen

Phakopsora uredia are yellowish brown, varying from 0.5 to 3.0 mm in diameter and are surrounded by purplish borders. Uredia first appear as oval, corky pustules and then become round.

The **pycnial** and **aecial** stages of *Puccinia* occur on **cotton**, whereas uredial and telial stages occur on grasses, most of which are species of *Bouteloua* (*Gramma grass*).

Disease cycle

Pycnial pustules occur mostly on upper leaf surfaces of cotton and are bright yellow to orange in colour. Aecia of similar colour occur on lower leaves. The spores from cotton infect gramma grass (*Bouteloua*) producing elongate brownish spots (Uredial stage). The black telial stage appears on gramma grass later. The spores produced from telial stage during summer rains infect cotton to complete the cycle.

Favourable Conditions

High humidity and moderate temperatures are conducive for the disease.

Management

- Remove and destroy the infected gramma grass.
- Spray Mancozeb@0.25% prior to first spore showers from gramma grass.

LECTURE 16 & 17

DISEASES OF SUGARCANE (*SACCHARUM OFFICINARUM*)

Red rot

Colletotrichum falcatum

(Sexual stage: *Physalospora tucumanensis* or *Glomerella tucumanensis*)

Economic importance

Red rot has been reported from all cane producing regions but it is usually a major problem in **sub-tropical** countries such as the Southern United States, India and Southern Queensland.

Symptoms

The first external symptoms appear as **discolouration** of the young leaves. The margins and tips of the leaves wither and the leaves droop. The discolouration and withering continues from the tip to the leaf base until the whole crown withers away in four to eight days. In a single stool, most of the stalks may wither almost simultaneously. Typical symptoms of red rot are observed in the internodes of a stalk by splitting it longitudinally. These include the **reddening of the internal tissues**, especially the vascular bundles, which are usually elongated at right angles to the long axis of the stalk. The presence of **cross-wise white patches** interrupting the reddened tissues are the important diagnostic character of the disease. Split open stems emit a characteristic **acidic-sour odour**. As the disease advances the entire stem rots and the stalk becomes hollow and covered with white mycelial growth. Later the rind shrinks longitudinally with minute black, velvety fruiting bodies protruding out of it.

The pathogen also produces tiny reddish lesions on the upper surface of leaves with dark dots in the centre. Minute **red spots** also appear on the centre of the mid-rib and develop in both directions forming small or long lesions. The lesions are initially blood red with dark margins and later on with straw coloured centres. Often the infected leaves may break at the lesions and hang down, with large number of minute black dots.



Pathogen

The fungus produces thin, hyaline, septate, profusely branched hyphae containing oil droplets. The fungus produces black, minute velvety **acervuli** with long, rigid bristle-like, septate pointed setae on the surface of rind, leaf midrib and sometimes in the pith region. Conidiophores are closely packed inside the acervulus, which are short, hyaline and single celled. The conidia are single celled, hyaline, **falcate**, granular and guttulate. Five pathotypes of the fungus have been reported. In general, light coloured physiological races sporulate readily and are more virulent than the dark coloured strains that sporulate sparsely. The perfect stage of the fungus produces large number of globose and dark brown to black perithecia with a papillate ostiole. Asci are clavate, unitunicate and eight-

spored. Large number of hyaline, septate, filiform paraphyses is also present among asci. Ascospores are ellipsoid or fusoid, hyaline, straight or slightly curved and unicellular.

Disease cycle

The fungus is sett-borne. The fungus also persists in the soil on the diseased clumps and stubbles as **chlamydospores** and **dormant mycelium**. The primary infection is mainly from **infected setts**. Secondary spread in the field may be through irrigation water and cultivation tools. The rain splash, air currents and dew drops also help in the spread of conidia from the diseased to healthy plants in the field. The fungus also survives on collateral hosts like *Sorghum vulgare*, *S. halepense* and *Saccharum spontaneum*.

Favourable Conditions

Mono-culturing of sugarcane, successive ratoon cropping, water logged conditions and injuries caused by insects.

Management

- Removal and destruction of infected plant debris, stubbles and trash.
- Deep tillage to incorporate the left over debris.
- Adopt crop rotation by including rice and green manure crops.
- Select the setts from the disease free fields or disease free area.
- Avoid ratooning of the diseased crop.
- Avoid flow of irrigation water from diseased to healthy plants.
- Soak the setts in 0.1% Carbendazim solution for 20 minutes before planting.
- Hot water treatment of setts at 52⁰C for 30 min or 50⁰C for 2 hours followed by steeping in 0.1% carbendazim solution.
- Setts can be treated with aerated steam at 52⁰C for 4 to 5 hours and by moist hot air at 54⁰C for 2 hours.
- Grow resistant varieties like CO 6907, CO 7219, CO 8013, CO 8021, CO 7706, CO A 7602, CO A 89082, CO A 89085, 87 A 397, CO T 8201, etc.

Whip Smut

Ustilago scitaminea

Economic importance

It is considered as an important disease and is common throughout the world. In India, it is widely prevalent in almost all the sugarcane growing tracts.

Symptoms

The affected plants are stunted and the central shoot is converted into a **long whip-like, dusty black** structure. The length of the whip varies from few inches to several feet. In early stages, this structure is covered by a thin, white papery membrane. The whip may be straight or slightly curved. On maturity it ruptures and millions of tiny black smut spores (**teliospores**) are liberated and disseminated by wind. All the shoots arising from the diseased clump produce whip like structures. The smutted clumps also produce mummified arrows in which lower portion consists of normal inflorescence with typical flowers and the upper portion of the rachis is converted into a typical smutted whip. Occasionally smut sori may develop on the leaves and stem. The ratoon crops are severely affected.



Pathogen

The fungal hyphae are primarily intercellular and produce tiny black **teliospores**. The thin membrane which covers the smut whip represents the host epidermis. The smut spores are light brown in colour, spherical and echinulate. Smut spores germinate to produce 3-4 celled, hyaline promycelium and produce 3-4 sporidia which are hyaline and oval shaped with pointed ends.

Disease cycle

Teliospores may survive in the soil for long periods, upto 10 years. The spores and sporidia are also present in the infected plant debris in the soil. The smut spores and dormant mycelium also present in or on the infected setts. The primary spread of the disease is through **diseased seed-pieces** (setts). In addition, **sporidia** and spores present in the soil also spread through rain and irrigation water and cause soil-borne infection. The secondary spread in the field is mainly through the smut spores developed in the whips, aided by air currents. The fungus also survives on **collateral hosts** like *Saccharum spontaneum*, *S. robustum*, *Sorghum vulgare*, *Imperata arundinacea* and *Cyperus dilatatus*.

Favourable Conditions

Mono-culturing of sugarcane, continuous ratooning and dry weather during tillering stage favours the disease.

Management

- Plant healthy setts taken from disease free area.
- Remove and destroy the smutted clump (Collect the whips in a thick cloth bag/polythene bag and immerse in boiling water for 1 hr to kill the spores).
- Discourage ratooning of the diseased crops having more than 10 per cent infection.
- Follow crop rotation with green manure crops or dry fallowing.
- Grow redgram as a companion crop between 2 rows of sugarcane.
- Treat the setts in hot water at 50⁰C for 2 hours.
- Grow resistant varieties like **Co 6806** and **Co 62175**

Wilt

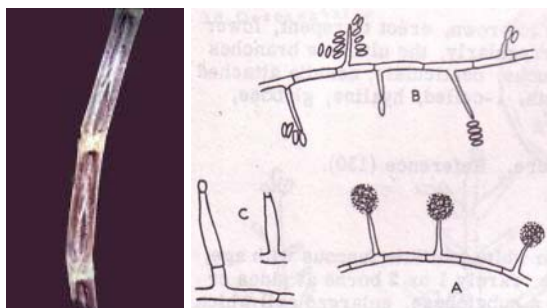
Cephalosporium sacchari

Economic importance

This is an important disease of sugarcane and is common in **Srikakulam**, **Vishakapatnam** and **Nizamabad** districts of our state. The disease occurs singly or in combination with red rot. The disease is more in wilt sick soils and in alkaline soils. Moisture stress aggravates the disease.

Symptoms

The first symptom of the disease is visible in the canes of **4-5 months age**. The canes may wither in groups. The affected plants are **stunted** with yellowing and withering of crown leaves. The midribs of all leaves in a crown generally turn yellow, while the leaf lamina may remain green. The leaves dry up and stem develop **hollowness** in the core or pith. The pith shows **reddish discolouration** with longitudinal red streaks passing from one internode to another. In severe cases, spindle shaped cavities tapering towards the nodes develop in each internode. The canes emit a **disagreeable odour**, with lot of white mycelial threads of the fungus covering the cavity. Weight gets reduced due to hollow canes.



Pathogen

The fungal mycelium is hyaline, septate and thin walled. The conidiophores are simple, slender or swollen on which hyaline, single celled, hyaline, oval to elliptical **microconidia** collecting in a slime drop. Macroconidia are not produced.

Disease cycle

The fungus is primarily sett borne and also survives in the soil as saprophyte for 2-3 years. The disease is primarily transmitted through infected seed setts. The secondary spread is aided by wind, rain and irrigation water.

Favourable Conditions

High day temperature (30-35°C), low humidity (50-60 per cent), low soil moisture, alkaline soils and excess doses of nitrogenous fertilizers.

Management

- Select the seed material from the disease-free plots.
- Avoid the practice of ratooning in diseased fields.
- Burn the trash and stubbles in the field.
- Grow **coriander** or **mustard** as a companion crop in the early stages of crop.
- Avoid alkaline soils for growing the crop
- Treat the setts in hot water at 50°C for 2 hours followed by dipping in 0.05% Carbendazim for 15 minutes.
- Dip the setts in 40ppm **Boron** or **Manganese** for 10 minutes
- Grow resistant varieties like CO 617 and BP 17.

Ring spot

Leptosphaeria sacchari

Symptoms

Disease symptoms first appear on the foliage as **dark green oval or spherical spots** developing straw colour from August onwards. When grown in size, central portion of the spots die and turns straw coloured surrounded by thin reddish brown band. Under severe conditions, leaves collapse and dry prematurely. In the central straw coloured portion

many pin head sized fruiting bodies (**perithecia**) develop in concentric rings. Juice quality is affected.



Pathogen

The fungus produces globose **perithecia**, each crowned with a short cylindrical beak with clavate asci carrying 8 ascospores which are three septate and pale yellow in colour.

Disease cycle

The disease is primarily transmitted through **infected seed setts** and plant debris. The secondary spread is through ascospores aided by wind, rain and irrigation water.

Favourable Conditions

Cool weather and heavy application of nitrogenous fertilizers usually favours the disease.

Management

- Select the seed material from the disease-free plots.
- Judicious use of nitrogenous fertilizers
- Burn the trash and stubbles in the field.
- Spray thrice with Copper oxy chloride@0.4% or carbendazim@0.1% or Mancozeb@0.3% at 7 days interval starting from disease initiation.

Grassy shoot

Phytoplasma

Economic importance

In India the grassy shoot has become an important disease in TN, AP, Karnataka, Bihar and UP, next to red rot and smut. It is more severe in ratoon crops and reduces juice quality, plant height and cane yield drastically.

Symptoms

The disease symptoms are usually seen two months after planting. The disease is characterized by the production of **numerous lanky tillers** from the base of the affected shoots. Leaves become pale yellow to completely **chlorotic**, thin and narrow. The plants appear **bushy** and 'grass like' due to reduction in the length of internodes, premature and continuous tillering. The affected clumps are stunted and in many instances exhibit **premature proliferation of axillary buds**. In a diseased clump one or two thin, weak and small canes are produced. In plant crop, young leaves of diseased plants are white (**Albino**) and the buds on such canes are usually white, **papery** and abnormally elongated.



Pathogen

Phytoplasma is found in the sieve cells of infected plants. Two types of bodies are noticed, spherical bodies of 300-400 nm diameter and filamentous bodies of 30-53 nm diameter.

Disease cycle

The pathogen is transmitted through planting material and within the crop by **aphids**, viz., *Aphis maidis*, *Rhopalosiphum maidis*, *Longiunguis sacchari*, *Melanaphis sacchari* and *M. indosacchari*. In addition, leaf hopper, *Proutista moesta* also involves in the transmission. **Sorghum** serves as a natural collateral host.

Management

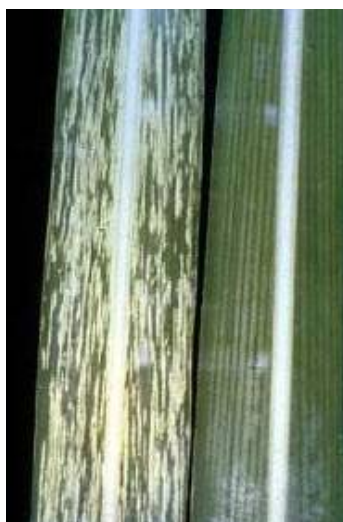
- Plant disease free setts.
- Remove and burn the infected clumps periodically.
- Avoid ratooning in problem areas
- Hot Water Treatment (HWT) of setts at 52⁰C for 30min or Aerated Steam Therapy (AST) at 50⁰C for 1 hr followed by steeping in fungicidal solution of carbendazim@0.05% for 15 minutes.
- Control vector by spraying Malathion or Dimethoate@2ml/lt

Mosaic

Sugarcane Mosaic Virus

Symptoms

The disease appears more prominently on the basal portion of the younger foliage as chlorotic or yellowish stripes alternating with normal green portion of the leaf imparting the mosaic pattern. As infection becomes severe, chlorotic area considerably increases over the normal green area and yellow stripes appear on the leaf sheath and stalks. Elongated necrotic lesions are produced on the stalks and stem splitting occurs. The necrotic lesions also develop on the internodes and the entire plant becomes stunted and chlorotic.



Pathogen

Sugarcane mosaic virus is rod shaped, measuring 650-770 X 12-15 μ m. It belongs to Potato Virus Y group. In India atleast six strains, viz., A, B, C, D, E, and F have been identified. Strain **B** is the most common which produces a mild mottle of the leaf.

Disease cycle

The virus is mainly transmitted through infected setts. The virus remains viable on several other hosts like *Zea mays*, *Sorghum vulgare*, *Pennisetum americanum*, *Eleusine indica*, *Setaria lutescens*, *Echinochloa crusgalli*, *Stenooaphorum secundatum*, *Digitaria didactyla* etc., which serve as potential sources of virus inoculum. The disease mainly spreads through sap and **aphids**, viz., *Rhopalosiphum maidis*, *Hysteroneura setariae*, *Toxoptera gramineum*, *Melanaphis sacchari* and *M. indoscchari*.

Management

- Rogue out the diseased clumps periodically.
- Select setts from the healthy fields as the virus is sett-borne.
- Treat the setts in hot water as follows: 52⁰C for 20 minutes on the first day, 57.3⁰C for 20 minutes on the second day and 57⁰C for 20 minutes on the third day or Aerated Steam Therapy (AST) at 56⁰C for 3 hrs.
- Vector control with malathion or dimethoate@2 ml/lt
- Use *Saccharum spontaneum* and *S. berberi* for breeding programme.

Ratoon stunting

Clavibacter xyli pv. *xyli* (*Xylem limited fastidious bacteria*)

Economic importance

The disease is present throughout the sugarcane growing areas of our country. **CO 419** variety was worst affected in parts of Karnataka where it was withdrawn from cultivation. The disease appears in both plant and ratoon crop, but more pronounced in ratoon crop.

Symptoms

Diseased clumps usually display **stunted** growth, reduced tillering, thin stalks with shortened internodes and yellowish foliage (**mild chlorosis**). When mature canes are split open, vascular bundles appear discoloured. In young canes, **pink colour** is seen in the form of minute pin head like areas near the nodes. The disease reduces the length, girth and the number of canes per clump.



Pathogen

The pathogen is known to present in the xylem cells of infected plants. They are small, thin, rod shaped or coryneform.

Disease cycle

The disease spreads through use of **diseased setts**. The disease also spreads through cane harvesting implements contaminated with the juice of the diseased canes. **Maize, sorghum, Sudan grass** and *Cynodon* are some of the collateral hosts of the pathogen.

Management

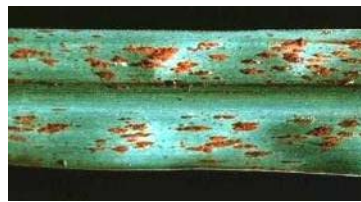
- Select the setts from disease free field.
- Remove and burn the clumps showing the disease.
- Sterilization of cutting knives with lysol or any other antiseptic solution.
- Hot air treatment of setts at 54⁰C for 8 hours or hot water treatment at 50⁰C for 2 hrs or aerated steam treatment at 50⁰C for 1 hour.

Rust

P. melanocephala, P. kuehnii (Syn: *Puccinia erianthi*)

Symptoms

Minute, elongated, **yellow spots (uredia)** appear on lower surface of young leaves. Later the pustules appear on upper surface also. The pustules turn brown on maturity. Late in the season, **dark brown to black telia** appear on the lower surface of leaves. In severe cases, the uredia also appear on the leaf sheath and the entire foliage looks brownish from a distance. The disease affects cane yield and reduces juice quality.



Pathogen

The mycelium is hyaline, branched and septate. *P.kuehnii* produces ovoid or pear shaped, single celled uredospores with apical thickening and golden yellow in colour. Teliospores are produced in abundance, which are pale to brick colour, two celled, smooth walled and slightly constricted at septum. Occurrence of pycnial and aecial stages and the role of alternate host are unknown.

Disease cycle

The fungus survives on collateral hosts like *Erianthus fulvus* and *Saccharum spontaneum*. The uredospores also survive in the infected stubbles in the soil. The disease is mainly spread through air-borne uredospores.

Favourable Conditions

Temperature of 30⁰C, relative humidity between 70 and 90 per cent during winter months, high wind velocity and continuous cloudiness favours disease development.

Management

- Remove the collateral hosts.
- Spray Tridemorph@0.1% or Mancozeb@0.3%, twice or thrice.

LECTURE 18 & 19

DISEASES OF TOBACCO (*NICOTIANA TABACUM*)

Black shank

Phytophthora parasitica var. *nicotianae*

Economic importance

It is a common disease of tobacco in many parts of India, particularly in the areas with heavy rainfall.

Symptoms

The pathogen may affect the crop at any stage of its growth. Even though all parts are affected, the disease infects chiefly the **roots** and **base of the stem**.

In the transplanted crop, the disease appears as **minute black spot on the stem**, spreads along the stem to produce irregular black patches and often **girdling** occurs. The upward movement leads to development of **necrotic patches on the stems**. The infected tissues shrink, leaving a **depression** and in advanced condition the stem shrivels and plant wilts. When the affected stem is split open, the pith region is found to be dried up in **disc-like plates** showing black discoloration. On the leaves large water soaked spots appear during humid weather, which enlarge to blight the leaves.

Seedlings in the nursery show **black discoloration of the stem** near the soil level and blackening of roots, leading to wet rot in humid condition and seedling blight in dry weather with withering and drying of tips. The pathogen also spreads to the leaves and causes blighting and drying of the bottom leaves.



Pathogen

The fungus produces hyaline and non-septate mycelium. The **sporangia**, which are hyaline, thin walled, ovate or pyriform with papillae, develop on the sporangiophores in a sympodial fashion. Sporangia germinate to release **zoospores** which are usually kidney shaped. The fungus also produces globose and thick walled **chlamydospores**. **Oospores** are thick walled, globose, smooth and light yellow coloured.

Disease cycle

The fungus lives as a saprophyte on organic wastes and infected crop residues in soil. The fungus also presents in the soil as dormant mycelium, oospores and chlamydospores for more than 2 years. The primary infection is by means of **oospores** and **chlamydospores** in the soil. Secondary spread is by **sporangia** or **zoospores** disseminated by wind or water. The pathogen in the soil spreads through irrigation water, transport of soil, farm implements and animals.

Favourable Conditions

Frequent rainfall, high soil moisture and high population of root knot nematodes, *Meloidogyne incognita*, favours the disease.

Management

- Collect and burn plant residues and debris.
- Select disease free seedlings for transplanting.
- Remove and destroy the affected plants in the field.
- Spot application of Bordeaux mixture@0.2% or copper oxy [chloride@0.2%](#) or [metalaxyl@0.2%](#) in planting points offer good protection.
- Burn the seed beds with paddy husk or groundnut shell at 15-20 cm thick layer. Provide adequate drainage in the nursery.
- Leaf blight and black shank phases of the disease can be effectively managed by two sprays of [metalaxyl@0.2%](#) or 3-4 sprays of copper oxy chloride@0.2%.

Damping off

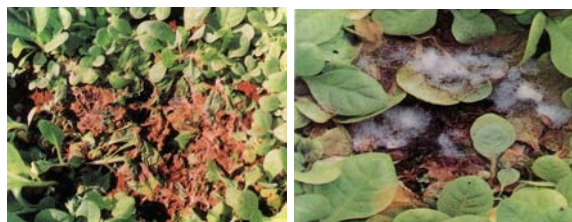
Pythium aphanidermatum

Economic importance

It is a common disease occurring in tobacco nurseries. The disease causes extensive damage in tobacco tracts of Andhra Pradesh, Maharashtra and Gujarat.

Symptoms

The fungus may attack the seedling at any stage in the nursery. **Sprouting seedlings** are infected and wither before emergence from the soil (**Pre-emergence damping off**). Water soaked minute lesions appear on the stems near the soil surface, soon girdling the stem, spreading up and down in the stems and within one or two days stem may rot leading to **toppling** over of the seedlings (**Post-emergence damping off**). The young seedlings in the nursery are killed in **patches** and infection spreads quickly. Under favourable conditions, the entire seedlings in the nursery are killed within 3 to 4 days. A thick **weft of mycelium** may be seen on the surface of the soil.



Pathogen

The fungus produces thick, hyaline, thin walled, non-septate mycelium. It produces irregularly **lobed sporangia** which germinate to produce **vesicle** containing **zoospores**. The zoospores are kidney shaped and biflagellate. **Oospores** are spherical and light to deep yellow or yellowish brown coloured.

Disease cycle

The fungus survives in the soil as oospores and chlamydospores. The primary infection is from the soil-borne **oospores** and secondary spread through **sporangia** and **zoospores** transmitted by wind and irrigation water.

Favourable Conditions

Overcrowding of seedlings, ill drained nursery beds, heavy shade in nursery, high atmospheric humidity (90-100 per cent), high soil moisture, low temperature (below 24⁰C) and low soil temperature of about 20⁰C.

Management

- Raised seed beds of 15-45 cm height should be formed.
- Avoid overcrowding of seedlings by using optimum seed rate of **3-3.5 kg/ha** (1 to 1.5g/2.5m²)
- Provide adequate drainage facility and avoid excess watering of the seedlings.
- Burn the seed beds with paddy husk or dry twigs before sowing.
- Drench the seed bed with 0.4% per cent Bordeaux mixture or 0.2 per cent Copper oxy chloride, two days before sowing.
- Spray the nursery beds twice with 0.4% Bordeaux mixture or 0.2 Copper oxychloride or Metalaxyl or Mancozeb at 20 and 30 days after germination.
- Soil incorporation of *Trichoderma viride* or *T. harzianum* in seed beds one week before seed sowing and thereafter BM should be sprayed at 0.4 per cent.

Frog eye spot

Cercospora nicotianae

Economic importance

This is an important disease in nurseries raised in light soils as well as black soils. Normally the disease appears in nurseries **beyond 6 weeks age** and its severity increases with the age of the nursery under favourable weather conditions. Intermittent rains and warm weather favour the development and spread of the disease.

Symptoms

The disease appears mostly on mature lower leaves as **small ashy grey spots with brown border**. The typical spots has a **white centre**, surrounded in succession by grey and brown portions, surrounded by a **dark brown to black margin**, resembling the eyes of a frog. Under favourable conditions, several spots coalesce to form large necrotic areas, causing the leaf to dry up from the margin and **wither prematurely**. Both yield and quality are reduced greatly. The disease may occur in the seedlings also, leading to withering of leaves and death of the seedlings.



Pathogen

The mycelium is intercellular which aggregates beneath the epidermis and produce clusters of conidiophores which emerge through stomata. The conidiophores are septate, dark brown at the base and lighter towards the top bearing 2-3 conidia. The conidia are hyaline, slender, slightly curved; thin walled and 2-12 septate.

Disease cycle

The primary infection is from **plant debris** in the soil. The secondary spread is through wind-borne **conidia**.

Favourable Conditions

Temperature of 20-30⁰C, high humidity (80-90 per cent), **close spacing**, frequent irrigation and excess application of nitrogenous fertilizers favours the quick spread of the disease.

Management

- Remove and burn plant debris in the soil.
- Avoid excess nitrogenous fertilization.
- Adopt optimum spacing.
- Regulate irrigation frequency.
- Spray the crop with 0.2 per cent Bordeaux mixture (20g copper sulphate + 20g lime in 10 liters of water) or Thiophanate Methyl or carbendazim or benomyl@0.1% or zineb@0.2%. Spray 2-3 times of systemic fungicides or 4-6 times with non-systemic fungicides at weekly interval.

Brown spot

Alternaria alternata

Symptoms

Brown spots with concentric circles are formed on leaves. Many spots may coalesce resulting in leaf blight.



Pathogen

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

Disease cycle

The pathogen survives in the infected crop debris as dormant mycelium. The secondary spread is mainly by air-borne conidia.

Management

- Remove and burn plant debris in the soil.
- Spray once or twice with fungicides like 0.4% Bordeaux mixture or zineb@0.2% or Copper oxy chloride@0.2% or Mancozeb@0.25%

Mosaic

Tobacco Mosaic Virus (TMV) or Nicotiana virus I

Economic importance

Mosaic is the most important and very common virus disease on tobacco in India appearing in every tobacco growing tract of the country.

Symptoms

The disease appears as light discoloration along the veins of the youngest leaves. Soon the leaves develop a characteristic light and dark green pattern, the dark green areas are usually associated with the veins. The dark green areas later develop into irregular **blisters** due to more rapid growth. The plants that become infected early in the season are usually very much stunted with small, chlorotic, mottled and curled leaves. In severe infections, the leaves are narrowed, puckered, thin and **malformed** beyond recognition. Later, **dark brown necrotic spots** develop under hot weather and this symptom is called “**Mosaic burn**” or “**Mosaic scorching**”.



Pathogen

Tobacco mosaic is caused by *Nicotiana virus I* (*Marmor tabaci* var. *vulgare*). It is a rod shaped particle measuring 300 X 150-180µm with a central hollow tube of about 4µm diameter. It is made up of centrally placed Ribonucleic acid molecules (**RNA**) covered with a protein coat. It is capable of remaining infective when stored dry for over 50 years. The thermal inactivation point (TIP) of the virus is 90⁰C for 10 minutes.

Disease cycle

The virus has a wide host range, affecting nearly 50 plant species belonging to nine different families. Virus produces different types of symptoms on several species of *Nicotiana*, tomato, brinjal, chilli, *Datura stramonium*, *Solanum nigrum* and *Petunia*. The virus is sap-transmissible and enters the host through wounds. The virus is not seed-transmitted in tobacco but tomato seeds transmit the virus. In the field, the virus is transmitted by contact. The farm workers engaged in topping and clipping operations transmit it through their dresses, chewing tobacco and snuff to the standing crop. The implements used in the field also transmit the virus.

Management

- Remove and destroy infected plants.
- Keep the field free of weeds which harbour the virus.
- Wash hands with soap and running water before or after handling the plants or after weeding.
- Prohibit smoking, chewing and snuffing during field operations.
- Spray the nursery and main field with leaf extracts of *Bougainvillea* or *Basella alba* at 1 litre of extract in 150 litres of water, two to three times at weekly intervals.
- Adopt crop rotation by growing non-host plants for two seasons.
- Spray tannic acid 1% at 30th, 40th and 50th day after planting

- Grow resistant or tolerant varieties like CTRI special (M.R), Jayasree (M.R), Virginia Tobacco 1158, **Prabhat**, Gautami, Blankat 1, Godavari special, TMV RR-2, 3, 4, 6.

Leaf curl

Tobacco leaf curl virus or Nicotiana Virus 10 (Ruga tabaci)

Economic importance

Wide spread in India and occurs in severe form in **Punjab**. Sporadic in some areas in South India, though at times found in severe form in few tracts.

Symptoms

Disease usually appears in the field **4-6 weeks after transplanting** and is characterized by **downward curling** of young leaves. Leaf margins turn downwards and come together at the bottom exposing the middle upper surface of the leaf blade. The thickened leaf blade usually exhibits **vein clearing** symptoms. As the disease advances the plant becomes dwarfed and most of the leaves curl. Inflorescence is greatly condensed and the veins of the calyx are thickened and turn green. **Enations** or leaf like outgrowths along the veins are also common.



Pathogen

The virus is spherical measuring 35µm in diameter. The virus is Nicotiana virus 10 or *Ruga tabaci*.

Disease cycle

The virus has wide host range infecting 63 crops species belonging to fourteen families. The virus also attacks tomato, chilli, papaya, sunhemp, *Zinnia*, *Petunia*, *Datura*, *Sida*, *Euphorbia*, *Ageratum*, *Solanum nigrum* and *Physalis peruvia*. The virus is not transmissible through sap or seed. It is graft-transmissible. The whitefly, *Bemisia tabaci* is the vector responsible for transmission in the field.

Management

- Remove and destroy the infected plants.
- Rogue out the reservoir weed hosts which harbour the virus and whiteflies.
- Avoid growing solanaceous crops like tomato near tobacco fields.
- Spray chloropyriphos@2.5ml/lt or monocrotophos or Methyl dematon@1.5 ml/lt to control the vectors.

Phanerogamic parasite
Orobanche or Broom rape
Orobanche cernua var. *desertorum*

Economic importance

It is one of the worst Phanerogamic parasites of crop plants known and occurs on tobacco in many areas in India. It is known by different names in different parts of India such as 'Malle' in A.P and 'tokra' in North India.

Symptoms

The affected tobacco plants are **stunted** and show withering and drooping of leaves. Plants show wilting symptoms during day time which usually recover at nights. These indicates underground parasitism of the tobacco roots by the parasite. The young shoot of the parasite emerges from the soil at the base of the plants **5-6 weeks after transplanting**. Normally, it appears as clusters of 50 shoots around the base of a single tobacco plant. The plants which are attacked very late exhibit no external symptoms but the quality and yield of leaves are reduced.



Parasite

It is a total root parasite. It is an annual, fleshy flowering plant with a short, stout stem, 10-15 inches long. The stem is pale yellow or brownish red in colour and covered by small, thin, brown scaly leaves and the base of the stem is thickened. White-coloured flowers appear in the leaf axils. The floral parts are well developed with a lobed calyx, tubular corolla, superior ovary with numerous ovules and a large four-lobed stigma. The fruits are capsules containing small, black, reticulate and ovoid seeds.

Disease cycle

The seeds of the parasite remain dormant in the soil for several years. Primary infection occurs from the seeds in the soil. The seeds spread from field to field by irrigation water, animals, human beings and implements. The dormant seeds in the soil are stimulated to germinate by the root exudates of tobacco. The germinated seeds of the parasite attach to the roots of tobacco by forming haustoria. Later, it grows rapidly to produce shoot and flowers. *Orobanche* also attacks the crops like brinjal, tomato, cauliflower, turnip and other cruciferous crops.

Management

- Rogue out the tender shoots of the parasite before flowering and seed set.
- Deep ploughing in the off-season helps in burying the seeds of the parasite deep into the soil

- Spray 0.1 per cent **Allyl alcohol** or 25 per cent Copper sulphate.
- Grow decoy or trap crops like chilli, mothbean, sorghum or cowpea to stimulate seed germination and kill the parasite.

LECTURE 20 & 21

DISEASES OF GROUND NUT (*Arachis hypogea*)

Tikka leaf spots

Early leaf spot: *Cercospora arachidicola* (Sexual Stage: *Mycosphaerella arachidis*)

Late leaf spot: *Phaeoisariopsis personata* (Sexual stage: *Mycosphaerella berkeleyi*)
(Syn: *Cercosporidium personatum*)

Economic importance

This is the most destructive disease of groundnut occurring where ever groundnut is cultivated. In India this disease occurs in all the groundnut areas causing severe damage, particularly when the plants are affected early in the season. The loss in pod production may be sometimes as high as 50%. The **late spot symptoms are common in our state** than the early leaf spots.

Symptoms

The tikka disease occurs as two distinct types of leaf spots, caused by two species of *Cercosporidium*.

Early leaf spot (*Cercospora arachidicola*): Symptoms usually appear within 35 DAS. The most conspicuous symptoms are observed on the leaflets. But symptoms may also appear on rachis, petioles, stipules and stalks etc, as elongated, elliptical spots with definite border. The disease usually appears early (before 35 DAS) than the *Cercosporidium personatum* and hence is known as early spot. The leaf spots are irregularly circular (1-10 mm in diameter), reddish brown or dark brown on the upper surface and are surrounded by a bright yellow halo. On the under surface, spots are light brown to tan coloured. Several spots coalesce and result in drying of the leaves.



Early LS

Lesions on stem

Late LS

Late leaf spot (*Cercosporidium personatum*): Leaf spots due to *C. personatum* appear (after 35 DAS) later than those due to *Cercospora arachidicola* and hence the symptoms are called late spots. The spots on leaves are circular with bright yellow halo around mature spots, usually **darker** than early leaf spots. On the under surface of the leaves the halo is not seen. The spots are deep black in colour with clusters of conidiophores bearing conidia, arranged in concentric manner. Severely diseased leaves dry up and results in heavy defoliation.

In both the cases generally lower leaves are first attacked but later on the disease spreads to other leaves also. Both the fungi produce lesions also on petiole, stem and pegs. Loss of photosynthetic tissue leads to reduction in yield and quality of nuts.

Pathogen

C. arachidicola (Sexual stage: *M. arachidis*)

The fungus is both intercellular and intracellular. The fungus produces abundant sporulation on the **upper** surface of the leaves. Conidiophores are olivaceous brown or yellowish brown in colour, short, 1 or 2 septate, unbranched and geniculate and arise in clusters. Conidia are sub hyaline or pale yellow, obclavate, often curved **3-12 septate**, 35-110 x 2.5 - 5.4 um in size with rounded to distinctly truncate base and sub-acute tip. The fungus in its perfect stage produces asci in **pseudothecia** which are globose or broadly ovate with papillate ostiole. Asci are cylindrical to clavate and contain 8 ascospores. Ascospores are hyaline, slightly curved and two celled, apical cell larger than the lower cell.

P. personata (*C. personatum*) (Sexual stage: *M. berkeleyii*)

The fungus produces both intercellular and intracellular mycelium. The conidiophores are long, continuous, 1-2 septate, geniculate, arise in clusters on **lower** surface of leaves and are olive brown in colour. The conidia are cylindrical or obclavate, short, measure 18-60 x 6-10um, hyaline to olive brown, usually straight or curved slightly with 1-9 septa, but mostly **3-4 septate**. The fungus in its perfect stage produces asci in pseudothecia which are globose or broadly ovate with papillate ostiole. Asci are cylindrical to ovate, contain 8 ascospores. Ascospores are 2 celled and constricted at septum and hyaline.

Disease cycle

The fungi survive for a long period in the infected plant debris as conidia, dormant mycelium and pseudothecia. The volunteer groundnut plants also harbour the pathogens. The fungi also survive on contaminated pods and seeds. The primary infection is by ascospores or conidia liberated from infected plant debris. The secondary spread is by wind blown conidia. Rain splash also helps in the spread of conidia.

Favourable Conditions

Prolonged high relative humidity for 3 days, low temperature (25-30°C) with dew on leaf surface, heavy doses of nitrogen and phosphorus fertilizers and deficiency of magnesium in soil favours the disease

Management

- Remove and destroy the infected plant debris.
- Eradicate the volunteer groundnut plants.
- Crop rotation with millets
- Treat the seeds with Captan or Thiram at 4g/kg or Carbendazim@0.2%
- Spray Carbendazim@0.1% or Mancozeb@0.2% or Chlorothalonil@0.2% and if necessary, repeat after 15 days.
- Grow resistant varieties like **Vemana** (early and late leaf spots), Naveen, Tirupathi-3 (early leaf spot only).

Rust

Puccinia arachidis

Economic importance

In India it was first observed in Punjab and recorded for the first time at Tirupati in 1971. Maximum loss in yield was upto 38% in case of early infection.

Symptoms

The disease attacks all aerial parts of the plant. The disease is usually found when the plants are about 6 weeks old. Small, minute pale yellow to light brown pustules (uredosori) appear on the lower surface of leaves. The pustules enlarge in size and reach a maximum size of 1mm diameter. The epidermis ruptures and exposes a powdery mass of uredospores. Corresponding to the sori, small, necrotic, brown spots appear on the upper surface of leaves. The rust pustules may be seen on petioles and stem. Late in the season, brown teliosori, as dark pustules, appear among the necrotic patches. In severe infection lower leaves dry and drop prematurely. The severe infection leads to production of small and shriveled seeds.



Pathogen

The fungus produces both uredial and telial stages. Uredial stages are produced in abundance on groundnut and production of telia is limited. Uredospores are pedicellate, unicellular, yellow, oval or round and echinulate. Teliospores are dark brown with two cells. Pycnial and aecial stages have not been recorded and there is no information available about the role of alternate host.

Disease cycle

The pathogen survives as uredospores on volunteer groundnut plants. The fungus also survives in infected plant debris in soil. The uredospores also spread as **contaminants of seeds** and pods. The spread is mainly through wind-borne inoculum of uredospores. Rain splash and implements also help in dissemination. The fungus also survives on the collateral hosts like *Arachis marginata* and *A. prostrata*.

Favourable Conditions

High relative humidity (above 85 per cent), heavy rainfall and low temperature (20-25⁰C) favours the disease.

Management

- Avoid mono-culturing of groundnut.
- Remove volunteer groundnut plants and collateral hosts.
- Spray Chlorothalonil or Tridemorph@0.2%.
- *Arachis glabarata* can be used in breeding programme.

Pepper leaf spot or leaf scorch

Leptosphaerulina crassiasca

Symptoms

Leaf spot: Minute, numerous, irregular to circular, dark brown to black spots appear on lower leaves. The size may vary from 0.5 to 1.0 mm. Such spots appear on both sides of the leaflet; but are more common on upper surfaces. The infected leaflets remain attached to the axis for about 30 days without notable change in the size of the spots.

Leaf scorch: The most common symptom frequently develops on the tips and occasionally on the margins of the leaflets. The wedge shaped lesions have a bright yellow zone along the periphery of their advancing margins. The drying tissue becomes dark brown and tends to break along the leaflet margins.

Disease cycle

P.S.I: Ascospores or Ascus

S.I: Wind borne ascospores

Management

- Remove and destroy the infected plant debris.
- Eradicate the volunteer groundnut plant
- Treat the seeds with Captan or Thiram at 4g/kg or Carbendazim@0.2%
- Spray Carbendazim@0.1% or Mancozeb@0.2% or Chlorothalonil@0.2% and if necessary, repeat after 15 days.

Stem rot

Sclerotium rolfsii

Symptoms

Mostly collar region of stem is affected. Sudden wilting of branches occurs. Leaflets become chlorotic to light green and then turn brown. Subsequently the adjacent branches become infected and wilt. Wilting is caused due to invasion of stems at or near the soil surface. **White mycelium** and brown to **dark brown sclerotia** are seen on the affected stems in advanced stages of infection. Pegs, roots, pods and kernels are also affected. Orange to brown coloured spots can be observed on pods. Under severe infection, a weft of white mycelium is seen on pods leading to rotting of pods. On the kernels, **bluish-gray** or ashy blue spots can be observed on the **testa**.



Pathogen

The pathogen produces dark brown to black sclerotia.

Disease cycle

The fungus is seed and soil borne. 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

- Avoid mono-culturing of groundnut.
- Deep summer ploughing to incorporate plant debris deep into the soil and to expose the dormant structures of fungi to direct sunlight.
- Grow **Bahia grass** as a trap crop for stem rot in groundnut
- Seed treatment with [Thiram@0.3%](#) followed by *Trichoderma viride*@4g/kg seed

- Multiply *T.viride* in farm yard manure for 15 days (2kg *T.viride* formulation + 50kg FYM) and apply to soil before sowing.

Bud necrosis or Peanut spotted wilt or groundnut ring mosaic

Tomato spotted wilt virus (TSWV-Tospovirus)

Economic importance

It occurred in severe form during the year 1974-75 in Amaravathi region. It appears generally a month after sowing and causes yield loss upto 50%.

Symptoms

First symptoms are visible 2-6 weeks after sowing as **ring spots** on leaves. The newly emerging leaves are small, rounded or pinched inwards and rugose with varying patterns of mottling and minute ring spots. Necrotic spots and irregularly shaped lesions develop on leaves and petioles. Stem also exhibits **necrotic streaks**. As the plant matures, it becomes generally stunted with short internodes and **short auxillary shoots**. Leaflets formed on these auxillary shoots show a wide range of symptoms including reduction in size, distortion of the lamina, mosaic mottling and general chlorosis. In advanced conditions, the **necrosis of bud** occurs. Drastic reduction in flowering is noticed and seeds produced are abnormally small and wrinkled with the dark black lesions on the testa.



Disease cycle

The virus perpetuates in the weed hosts viz., *Bidens pilosa*, *Erigon bonariensis*, *Tagetes minuta* and *Trifolium subterraneum*. The virus is transmitted by thrips, viz., *Scirtothrips dorsalis*, *Frankliniella schultzei*, and *Thrips palmi*.

Favourable conditions

- Early sown crop (first half of June) shows lower incidence of PBNB than late sown crop (late June)
- Higher incidence where plant population is less (<23 plants/m²) as against optimum population (33 plants/m²). Sub-optimal plant population leaves bare patches in the field which attract thrips.

Management

- Grow resistant varieties like Kadiri 3, Kadiri 4, Vemana, ICGS-11, etc.
- Maintain optimum plant population and adopt spacing of 15x15cm
- Intercropping with **Bajra**.
- Spray [monochrotophos@1.6ml/lt](#) or dimethoate@2ml/lt for vector control

Peanut Stem necrosis disease (PSND)

Tobacco streak virus (Ilarvirus)

Economic importance

The disease appeared in an epidemic form in **Anantapur** district of Andhra Pradesh during **Kharif 2000**. The losses were estimated to exceed Rs. 300 crores.

Symptoms

Symptoms first appear on young leaves as necrotic lesions and **veinal necrosis**. The necrosis later spreads to the petiole and stem. Necrotic lesions on the stem later spread upwards killing the bud. Majority of the plants infected within a month after sowing die due to necrosis. In some cultivars, pods harvested from the PSND infected plants show necrotic lesions. Some early infected plants are killed leaving gaps in the field. The surviving plants show stunting, small, clumped leaves with or without chlorosis. In some cases stunted plants with small leaves having distinct mosaic patterns are also seen.

Disease cycle

The virus perpetuates in the weed hosts, especially *Parthenium hysterophorous*. Of the crop species, **sunflower** and **marigold** also acts as a source of inoculum. Natural incidence of TSV was also detected in Mung bean, urd bean, safflower and sunhemp. The virus is transmitted by **thrips** viz., *Frankliniella schultzei*, *Scirtothrips dorsalis*, *Thrips palmi*, etc. while feeding on these hosts the thrips pick up **infected pollen grains** on their bodies. When these thrips attack groundnut plants, the pollen grains get dislodged from their bodies and during feeding both groundnut leaf tissues and pollen grains get damaged allowing the virus present in the pollen grains to infect groundnut plants.

Favourable Conditions

Higher incidence where plant population is less (<23 plants/m²) as against optimum population (33 plants/m²). Sub-optimal plant population leaves bare patches in the field which attract thrips.

Management

- Grow resistant varieties like Kadiri 3, Kadiri 4, Vemana, ICGS-11, etc.
- Adopt spacing of 15x15 cm.
- Intercropping with Bajra.
- Spray [monochrotophos@1.6ml/lt](#) or dimethoate@2ml/lt

Kalahasti malady

Tylenchorhynchus brevilineatus

Economic importance

It was first reported in 1975-76 from Kalahasthi area of **Chittoor** district. The losses range from 20-60%.

Symptoms

Small, black or brownish yellow lesions appear on the pegs, pod stalks and on young developing pods. The margins of the lesions are slightly elevated because of the proliferation of host cells around the lesion. Pod stalks are much reduced in length and in advanced stages of the disease the entire pod surface becomes discoloured. Discolouration is also seen on roots. Affected plants are stunted and greener than normal foliage. The size of the seeds in the infected pods is reduced. The disease is severe in sandy soils or light soils and occurs in the same area year after year.



Disease cycle

P.I: Nematodes present in the soil or on the pods.

S.I: Nematodes spread through irrigation water, rain water and during ploughing

Management

- Soil treatment with aldicarb and carbofuran is effective in reducing soil population.
- Use resistant varieties like **Tirupathi-3** (TCGS 320), **Kalahasthi** (TCGS 1518) and **Prasuna**.
- Deep ploughing and leaving fallow during summer
- Apply neem cake @2.5t/ha or FYM@10t/ha or Poultry manure@5t/ha
- Apply carbofuran granules once in 4 years at 4 kg a.i. (133 kg) per hectare 25-30 days after sowing along with irrigation water.
- Apply Sebuphos 10G granules at 40kg/ha, 30 days after sowing in between rows followed by irrigation.
- The disease incidence is less in groundnut fields sown after **rice** or **marigold**.

LECTURE 22

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.

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 (20-25⁰C), high relative humidity, excessive rainfall and cloudy weather favour the disease.

Management

- Treat the seeds with Captan or Thiram@0.25% or Carbendazim@0.1%
- Hot water treatment at 53⁰C for 30 minutes gives good control of the disease.
- Spray twice with [Mancozeb@0.25%](#) or Thiophanate methyl@0.25% or Carbendazim@0.1%

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.

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 perennates 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.



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 persistent 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°C), 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 Monocrotophos (0.03%) or 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.

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 30⁰C 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 or treat the seeds with carbendazim@0.1% or Thiram at 4g/kg.
- Apply farm yard manure or green leaf manure at 10t/ha or neem cake 250 kg/ha.
- Spot drench with Carbendazim at 0.5 g/liter.
- 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.

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. It is internally seed-borne and secondary spread through rain splash.

Management

- Keep the field free of infected plant debris.
- Seed treatment with hot water at 52⁰C for 10 minutes.
- Steep the seed in Agrimycin 100 (250 ppm) or streptomycin suspension (0.055) for 30 minutes.
- Spray twice with Streptomycin sulphate or Oxy-tetracycline hydrochloride at 100g/ha at 15 days interval.

LECTURE 23

DISEASES OF CASTOR (*RICINUS COMMUNIS*)

Wilt

Fusarium oxysporum f.sp. ricini

Symptoms

The disease appears in patches. Plants are attacked at all growth stages. When seedlings are attacked, cotyledonary leaves turn to dull green colour, wither and die subsequently. Diseased plants are sick in appearance. Necrosis of leaves starts from margins spreading to interveinal areas and subsequently to the entire leaf. All lower leaves droop and drop off leaving behind only a few top leaves. Subsequently plants die. Sometimes a cluster of purple coloured sporodochia develops on the stem and superficial cracks are noticed on the stem. Split open stems show brownish discolouration and white cottony mycelial growth much prominently in the pith region. The fungus is seed borne.

Pathogen

The fungus produces macroconidia, microconidia and chlamydospores. Macroconidia are falcate shape, hyaline and 5-9 celled. Microconidia are hyaline, thin walled, unicellular and ovoid. The dark walled chlamydospores are also produced.

Disease cycle

The fungus survives in the soil in the infected plant debris. It is also seed-borne and primary infection occurs through infected seeds or through chlamydospores in soil. The secondary infection may be caused by conidia disseminated by rain splash and irrigation water.

Management

- Select disease free seeds for planting
- Rogue out and burn disease affected plants and crop debris regularly
- Follow crop rotation for 2-3 years with non-host plants like pearl millet, finger millet or other cereals.
- Follow intercropping with redgram
- Seed treatment with *Trichoderma viride*@4g/kg and Thiram@3g/kg seed or carbendazim@2g/kg seed.
- Multiplication of 2 kg *T. viride* formulation by mixing in 50 kg FYM. Sprinkle water and cover with polythene sheet for 15 days and then apply between rows of the crop.
- Cultivate wilt resistant varieties, viz., **Jyothi**, **Jwala** and hybrids, viz., DCH 32, DCH 177, DCH 519, GCH 4, GCH 5 and GCH 6.

Root rot/Charcoal rot

Macrophomina phaseolina

Symptoms

Sudden wilting of plants in patches under high soil moisture stress coupled with high soil temperatures is a common symptom. The plants show signs of water shortage. Within a week, the leaves and petiole droop down and within a fortnight the infected plants dry up. Dark brown lesions are seen on the stem near the ground level. The taproot shows signs of drying and root bark sheds off easily. Fruiting bodies (pycnidia) of the fungus are seen as minute black dots on woody tissues and in pith region. In severe infection entire branch or top of the branch withers away. Young leaves curl inwards with black margins and drop

off later. Such branches die-back. Diseased plants flower prematurely. Incidence at maturity causes spike blight. Seed development is affected.

Disease cycle

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

Favourable conditions

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

Management

- Burn crop debris containing the sclerotia of the fungus.
- Seed treatment with *Trichoderma viride*@4g/kg seed or carbendazim@1g/kg seed.
- Crop rotation with cereals
- Provide irrigation at critical stages of crop growth
- Soil drenching with carbendazim@0.1%, 2-3 times at 15 days interval.
- Grow tolerant and resistant varieties / hybrids like Jwala, GCH-4, and GCH-6.

Grey mold/Grey rot/Blossom blight

Botrytis ricini (Sexual stage: *Sclerotinia ricini*)

Economic importance

The disease poses problem when rains occur during capsule formation and also due to prolonged wet weather.

Symptoms

Symptoms of the disease can be seen on leaves, stem, flowers and capsules, being prominent on spikes. Initially water soaked lesions form on the male flowers at the base of the spike. These flowers rot and are covered by characteristic grey or ash coloured growth of the fungus. Subsequently the disease spreads upward infecting all flowers and capsules which are covered by the fungus thereby involving the entire spike. This is followed by development of cottony white growth which later converts into grey colour due to sporulation. The infected capsules rot. Blue spots of different sizes appear on the side branches and laterals of the spike. Yellowish drops of liquid exude from these portions which are covered by fluffy grey fungal growth. Affected portions break off at the point of infection.

Infection at flowering results in flower rot and affects seed filling. Infected spikes become sterile without capsules. Infected capsules rot and shed off. Infection spreads to the seed also on which black sclerotia develop. Leaves which are in contact with the diseased spikes are also infected on which irregular light brown spots with marked borders consisting of greyish fungal growth develops.

Pathogen

Conidiophores long, slender, branched, septate, apical cells enlarged or rounded bearing clusters of conidia on short sterigmata. Entire structure resembles a grape bunch. Conidia are hyaline or ash coloured, grey in mass, one-celled, globose to ovoid.



Disease cycle

The fungus survives through sclerotia on infected seed and crop debris.

Favourable conditions

Night temperatures below 22⁰C followed by rains highly favour the disease spread.

Management

- Adjust sowing time in such a way that crop maturation occurs during dry season
- Adopt wider spacing (90 x 60cm)
- Remove diseased spikes and destroy them
- Grow varieties like Jwala with non-spiny capsules and less compact inflorescence.
- Seed treatment with carbendazim@3g/kg
- Spray carbendazim / Thiophanate methyl @0.1% before the onset of cyclonic rains based on weather forecast followed by second spray soon after rains have receded.
- Application of 20kg urea and 10kg of muriate of potash after removal of diseased panicles may be useful for the growth of panicles that subsequently develop.

Bacterial leaf spot

Xanthomonas campestris pv. ricini

Symptoms

All the above ground parts are attacked by the bacterium. On cotyledons and leaves, water soaked, angular spots are produced. Leaf symptoms are first noticed at the tip which extends to center becoming irregularly angular, dark brown to jet black in colour. Diseased leaves become blighted and plants defoliate. Diseased areas consist of bacterial exudation as small beads on both the surfaces. Elongate dark lesions may also develop on petioles and young branches. The bacterium is **seed borne**.

Management

- Remove and destroy the infected plant debris.
- Hot water treatment of seeds at 50-60⁰C for 10 minutes.
- Spray streptomycin@500ppm or [paushamycin@0.025%](#) in combination with [COC@0.3%](#)

Seedling blight

Phytophthora parasitica

Economic importance

The disease was first reported from Pusa in the year 1909. An average loss of 10% occurs in crop stand due to this disease.

Symptoms

The disease appears as circular, dull green patch on both the surface of the cotyledonary leaves. It later spreads and causes rotting. Under humid conditions, infection spreads to stem and causes withering and death of seedling due to destruction of growing tip. In

mature plants, the infection initially appears on the young leaves and spreads to petiole and stem causing black discoloration and severe defoliation. On older leaves, spots are round to irregular and show alternate yellow and brown concentric zones with yellowish green halo on the upper surface. Affected leaves are blighted and shed prematurely. Under moist conditions, a whitish fungal growth is found on the under surface of the spots.

Pathogen

The fungus produces non-septate and hyaline mycelium. Sporangiohores emerge through the stomata on the lower surface singly or in groups. They are unbranched and bear single celled, hyaline, round or oval sporangia at the tip singly. The sporangia germinate to produce abundant zoospores. The fungus also produces oospores and chlamydospores in adverse seasons.

Disease cycle

The pathogen is soil borne. The fungus may survive through resistant chlamydospores and spreads by zoospores carried by rain water.

Favourable Conditions

Continuous rainy weather, low temperature (20-25⁰C), low lying and ill drained soils.

Management

- Remove and destroy infected plant residues.
- Avoid low-lying and ill drained fields for sowing.
- Treat the seeds with Metalaxyl at 3g/kg or *T. viride* at 4g/kg.
- Soil drenching with [Metalaxyl@0.2%](#) or [COC@0.3%](#)
- Give need based spray of [COC@0.3%](#) to avoid further spread of the disease.

Rust

Melampsora ricini

Economic importance

This is a common disease of castor in India and other countries. The disease usually develops after the south west monsoon and is severe during November to December.

Symptoms

Minute, orange-yellow coloured, raised pustules appear with powdery masses on the lower surface of the leaves and the corresponding areas on the upper surface of the leaves are yellow. Often the pustules are grouped in concentric rings and coalesce together leading to drying of leaves.



Pathogen

The fungus produces only uredosori in castor plants and other stages of the fungus are unknown. Uredospores are two kinds, one is thick walled and other is thin walled. They are elliptical to round, orange-yellow coloured and finely warty.

Disease cycle

The fungus survives in the self sown castor crops in the off season. It can also survive on other species of *Ricinus*. The fungus also attacks *Euphorbia obtusifolia*, *E.geniculata*, and *E.marginata*. The infection spreads through air-borne uredospores.

Management

- Rogue out the self-sown castor crops and other weed hosts.
- Spray [Mancozeb@0.25%](#) or [Tridemorph@0.1%](#) or dust fine Sulphur powder at 25kg/ha.

LECTURE 24

DISEASES OF SUNFLOWER (*HELIANTHUS ANNUUS*)

Leaf blight

Alternaria 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.



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

The fungus survives on seed, host debris and weed hosts. 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 Thiram or Carbendazim at 2 g/kg.

- Spray twice or thrice with zineb or Mancozeb at 0.2% or carbendazim@0.1% 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.

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

The pathogen survives in the volunteer sunflower plants and in infected plant debris in the soil as teliospores. The disease spreads by wind-borne uredospores.

Favourable conditions

Weather parameters like temperatures of 25.5 to 30.5⁰C 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 Mancozeb or Zineb@0.2%, 2-3 times at 10 days interval. The first spray should be given as soon as the disease is noticed or 35 DAS.

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.

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 perennates 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.

Management

- Remove and destroy infected crop debris.
- Spray wettable [sulphur@0.3%](#) or [Calixin@0.1%](#)

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.



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

The fungus survives as a saprophyte in host debris and other crop residues. 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 Thiram or 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 Mancozeb 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.



Pathogen

The pathogen produces dark brown to black sclerotia.

Disease cycle

The fungus survives as **sclerotia** in soil and plant debris. 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.



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

Primary infection of the crop occurs through soil borne oospores. 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.

Disease cycle

The virus is transmitted through sap, seed and white flies, *Bemesia tabaci*. The virus can survive in **amaranthus**.

Management

- Rouging of infected plants
- Spray **Triazophos** 1ml or **Monochrotophos** 1.5 ml per litre of water.

Sunflower necrosis virus (SND)

Tobacco streak virus

Economic importance

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.



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.

LECTURE 25

DISEASES OF SAFFLOWER (*CARTHAMUS TINCTORIUS*)

Leaf blight

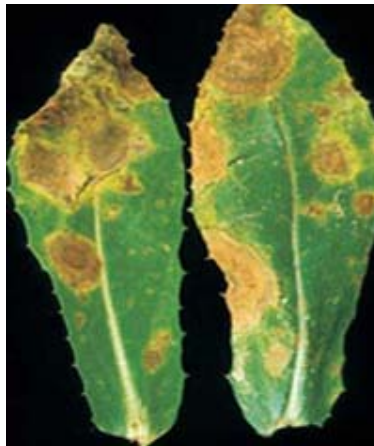
Alternaria carthami

Economic importance

It is the most destructive disease and appears in a severe form wherever safflower is grown.

Symptoms

Dark brown lesions measuring 2-5mm in diameter are first found on hypocotyls and cotyledons. The disease is severe on leaves and occasionally attacks stem and flowers. Minute brown to dark brown spots with concentric rings of 1-2mm appear on leaves. The centre of the spot is light brown with a dark brown margin. Elongated black lesions can be seen on the petiole and stem. The fungal infection on flower buds leads to drying and shedding. Seeds also may be affected. Dark sunken lesions are produced on the testa.



Pathogen

The mycelium of the fungus is sub-hyaline initially and become brown coloured on maturity. The conidiophores are stout, erect, rigid, unbranched, septate and arise singly or in clusters. The conidia are 3-11 celled with irregular shape, light brown in colour with a long beak.

Disease cycle

The fungus is externally seed-borne and also survives in plant debris. The disease spread is through windblown conidia.

Management

- Collect and destroy infected plant debris
- Treat the seeds with Thiram or Captan at 3g/kg or Carbendazim@0.1%
- Hot water treatment of seed at 50⁰C for 30 minutes
- Spray Mancozeb or zineb@0.2% or carbendazim@0.1%.

Wilt

Fusarium oxysporum f.sp. carthami

Symptoms

In seedling stage cotyledonary leaves show small brown spots either scattered or arranged in a ring on the inner surface and they may be shrivelled or rolled or curved. Symptoms become apparent when plants are in 6-10 leaf stage as yellowing of leaves followed by wilting, Epinasty and vascular browning. Symptoms develop in acropetal succession. In older plants the lateral branches on one side may be killed while the remainder of the plant remains free from the disease. Infected plants produce small sized flower heads which are partially blossomed. Most of the ovaries fail to develop seeds or they may form blackish, small, distorted chaffy seeds.



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.

Disease cycle

The fungus survives in seed, soil and infected plant debris. The primary spread is by soil-borne chlamydospores and also by seed contaminant. The secondary spread in the field is through irrigation water and implements.

Favourable conditions

The disease is severe in acidic soils with high nitrogen and warm moist weather with a temperature of 15-20⁰C is conducive for disease development.

Management

- Avoid growing safflower in low lying areas
- Collection and destruction of plant debris.
- Follow crop rotation with sorghum
- Grow wilt resistant / tolerant hybrids DSH 129, NARI-NH-1 and varieties A1, PBNS-40 and NARI-6 in endemic areas.
- Treat the seeds with Thiram or Captan at 3g/kg or carbendazim@0.1% or *T. viride* @10g/kg seed

Rust

Puccinia carthami (*Puccinia calcitrapae* var. *centaureae*)
or *P. verruca* or *Aecidium carthami*

Economic importance

Rust is mainly restricted to A.P, M.P and U.P. The disease causes considerable yield loss if the infection starts early in the crop growth.

Symptoms

The fungus attacks cotyledons, young leaves, tender stems and underground parts. Infection of the cotyledons is seen as yellow discoloration accompanied by drooping and wilting. The pustules (uredosori) are chestnut brown in colour, erumpent and scattered throughout the leaves. Later in the season, black teliosori are formed on the same spots. Seedlings sometimes die suddenly without exhibiting symptoms in the aerial parts. Infection on the hypocotyl causes **hypertrophy** of the tissues due to accumulation of mycelium between cells. Stem girdling occurs in older plants. The rust pustules also appear on tap root and lateral roots.



Pathogen

The fungus is an obligate parasite with **autoecious** life cycle in safflower. Uredia and telia are produced and pycnial and aerial stages are unknown. Uredospores are single celled, light brown coloured and echinulate. Teliospores are globose to broadly ellipsoid, two celled, chestnut brown in colour, thick walled with hyaline pedicels.

Disease cycle

The fungus remains on the **seeds** and infected crop debris in the soil as teliospores for more than a year. The fungus also produces uredial and telial stages in the collateral host *Carthamus oxyacantha* and this also serves as primary source of infection in addition to dormant teliospores in soil. The secondary spread occurs through wind-borne uredospores.

Management

- Grow resistant varieties like **Sagaramuthyalu**, **Manjeera** and APRR-3.
- Treat the seeds with Thiram or Captan@3g/kg or Carbendazim@2g/kg.
- Remove and destroy the plant debris in the soil.
- Rogue out the collateral host.
- Spray wettable sulphur or Mancozeb@0.2%

Mosaic

Cucumber mosaic virus (CMV)

Symptoms

In CMV infected safflower plants, young leaves show irregular yellow or light green patches alternating with normal green areas. Leaves may become blistered and distorted and infected plants are stunted. In few plants primary leaves are produced, forming a rosette of leaves exhibiting mosaic mottling and from the centre of this the axis bearing secondary leaves is produced.

Disease cycle

The virus can infect a number of wild and cultivated plants and is transmitted by aphid, *Myzus persicae*.

Management

- Rogue out and destroy infected plants
- Spray systemic insecticides like Monochrotophos 1.5ml or dimethoate 2ml for the control of aphid vectors.

LECTURE 26

MUSTARD

White rust

Albugo candida or *A. cruciferarum*

Economic importance

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. Sporangiohores 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

The fungus over-summers through cleistothecia as ascospores or as mycelium on volunteer host plants. 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%](#) or [Dinocap@0.1%](#) or [tridemorph@0.1%](#)

Alternaria leaf spot

Alternaria brassicae and *A. brassicola*

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*.

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

The pathogen survives in the infected crop debris as dormant mycelium. The secondary spread is mainly by air-borne conidia.

Management

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

LECTURE 27 & 28

RED GRAM (*Cajanus cajan*)

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

Economic importance

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 lose 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 25⁰C 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. Primary infection is from oospores and 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 25⁰C 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. The primary spread is by soil-borne chlamydospores and also by seed contaminant. Chlamydospores remain viable in soil for 8-20 years. 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

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

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, **cankorous** 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°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.

Management

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

LECTURE 29

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**. 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. The secondary spread is through irrigation water, cultural operations and implements.

Favourable Conditions

High soil temperature (Above 25⁰C), 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 of 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

The fungus survives as uredospores in the legume weed *Trigonella polycerata* during summer months and serve as primary source of infection. The spread is through wind-borne uredospores.

Management

- Destroy weed host.
- Dust Sulphur at 20 kg/h or spray [Mancozeb@0.25%](#)

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 black 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 10⁰C and day temperature of 20⁰C, 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. The primary spread is from seed-borne pycnidia and plant debris in the soil. 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 Thiram 2g or Carbendazim 2 g /kg.
- Exposure of seed at 40-50⁰C reduced the survival of *A. rabiei* by about 40-70 per cent. Spray with [Carbendazim@0.1%](#) or [Chlorothalonil@0.3%](#).

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

The pathogen survives in the soil in infected host debris as sclerotia for several years. The secondary spread is through farm implements, irrigation water and rain splash.

Favourable conditions

Maximum ambient temperatures above 30⁰C, minimum above 20⁰C, and moisture stress favour disease development.

Management

- Treat the seeds with Carbendazim or Thiram at 2 g/kg or 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 30

BLACK GRAM (*Vigna mungo*) **and** **GREEN GRAM** (*Vigna radiata*)

Powdery mildew *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. Primary infection is usually from ascospores from perennating cleistothecia. 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. Primary infection is by the basidiospores developed from teliospores. Secondary spread is by wind-borne uredospores. The fungus also survives on other legume hosts.

Favourable Conditions

Cloudy humid weather, temperature of 21-26⁰C and nights with heavy dews favour the disease.

Management

- Remove and destroy the infected plant debris.
- Spray Mancozeb@0.3% or Tridemorph@0.1% or Wetttable 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

The fungus survives on diseased plant debris in soil and on **seeds**. 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

The fungus is seed borne and can survive on host debris for two years. 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

Protomyces 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 *Protomyces patelii*. The fungus is related to smut fungi of the genus *Tilletia* because it produces relatively large, opaque 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 intercalary. 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.

Protomyces produces finely punctate (rough) chlamydospores which are formed terminally on the mycelium and helps in survival of the fungus.

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 within a 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 produce pycnidiospores (conidia) which are thin walled, hyaline, single celled and elliptical.

Disease cycle

The fungus survives in the infected debris and also as a facultative parasite in soil. The primary spread is through seed-borne and soil-borne sclerotia. The secondary spread is through seed-borne and soil-borne sclerotia. The secondary spread is through pycnidiospores which are air-borne.

Favourable Conditions

Day temperature of 30°C 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 *Pseudomonas fluorescens* @ 10g/kg of seed.
- Apply farm yard manure or green leaf manure (*Gliricidia maculata*) at 10 t/ha or neem cake 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

The bacterium is seed borne and grows through perennial vines. 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 streptomycin 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

The virus survives in the weed hosts and other legume crops. 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 periodically.
- 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

The virus is **seed-borne** and primary infection occurs through infected seeds. 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°C 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

- Perpetuates through seed which fall onto the ground
- 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 31

SOYBEAN (*Glycine max*)

Rust

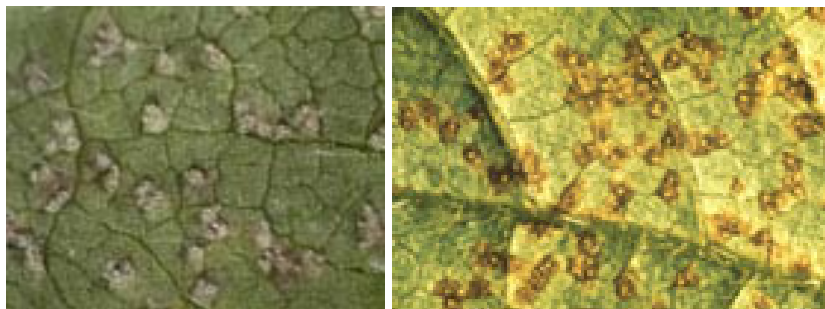
Phakopsora pachyrhizi

Economic importance

This disease is the most destructive disease of soybean. It causes yield losses of 10-65% in Uttar Pradesh, 35% 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

The pathogen survives as teleutospores in crop debris. Secondary spread is through wind borne uredospores.

Favourable Conditions

The disease is favoured by temperature of 18 to 23°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

The virus is seed borne and is transmitted by aphids.

Management

- Use virus free seed from healthy crop.
- Rogue out infected plants and burn them.
- Spray [monochrotophos@1.5ml/lt](#) or 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

The bacterium survives in crop residue and seed

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 45 and 55 DAS with a mixture of [Blitox@0.2%](#) + Streptocycline@250ppm effectively control the disease.

LECTURE 32

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

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