B.Sc. Ag
V Sem

Diseases of Field and Horticultural Crops and their Management

Credit - 3(2+1)

As per ICAR
5TH Dean Syllabus

COURSE MATERIAL FOR PATH 371

DISEASES OF FIELD CROPS AND THEIR MANAGEMENT

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LECTURE 1

DISEASESW 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 secondatum.* 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 ^{0}C or less than 26 ^{0}C), more number of rainy days, longer duration of dew, cloudy weather, slow wind movement and availability of collateral hosts.

Forecasting

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

Management

- Use of seeds from a disease free crop
- 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

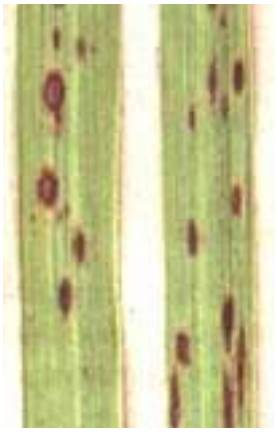
Helminthosporium oryzae (Syn: *Drechslera oryzae*)

(Sexual stage: *Cochliobolus miyabeanus)*

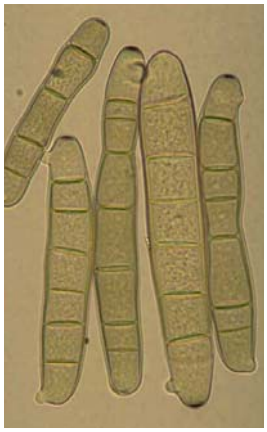
In India, this disease is the principal cause of Bengal famine of 1942-43. The first report of the disease in India was made by Sundararaman from Madras in 1919, and now is reported from all of the rice growing states. Under highly favourable conditions, the disease causes a reduction in yield ranging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

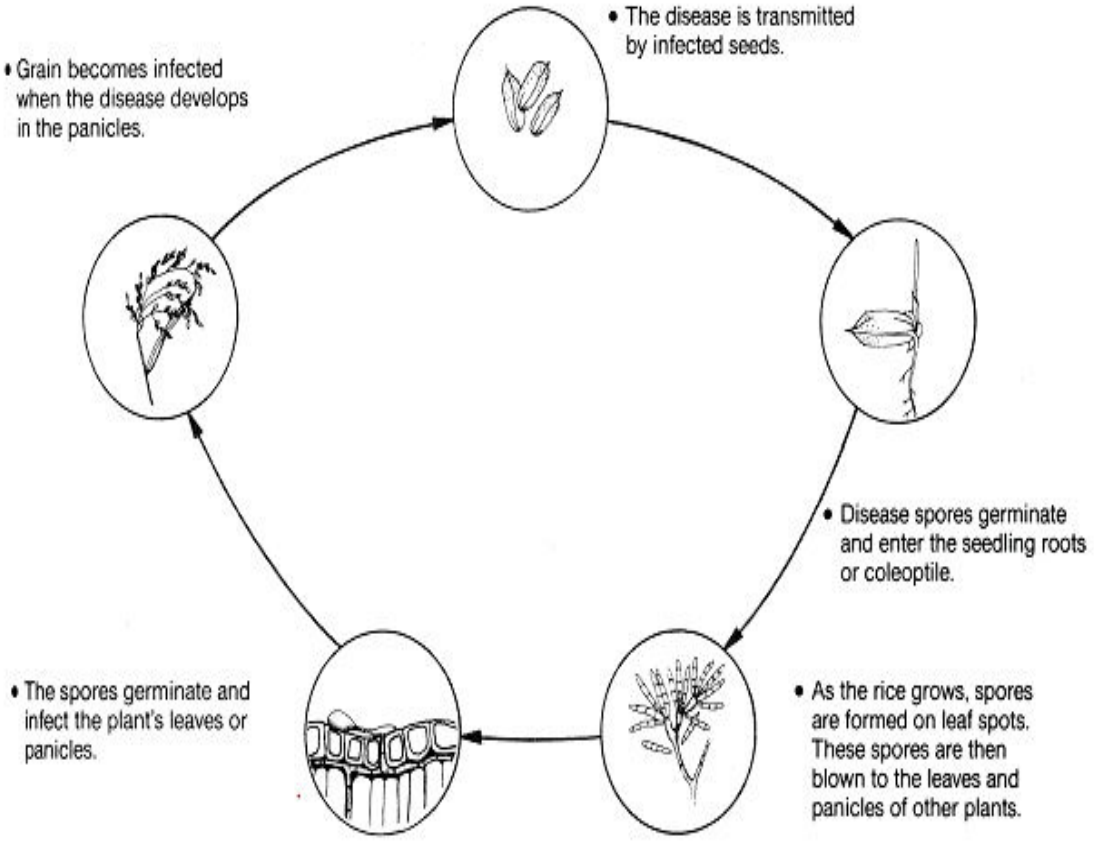
Etiology

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

Disease cycle

The fungus overwinters mainly in the infected plant parts. It is not soil borne. The infected seeds are the most common source of primary infection. Diseased seeds

(externally seed borne) may give rise to the seedling blight, the first phase of the disease. The young seedlings show infection symptoms soon after germination. Pale yellowish-brown spots appear on the coleoptiles, spreading to cover the other tissues of the seedling. The fungus reproduces on the spots and is disseminated by air currents. The fungus also survives on collateral hosts like *Digitaria sanguinalis, Leersia hexandra, Echinochloa colonum, Pennisetum typhoides, Setaria italic*a and *Cynodon dactylon*.
The symptoms of potassium deficiency are somewhat similar to that of brown spot making it often difficult to ascribe the symptoms to fungus attack or nutrient deficiency.



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

Management
- Use 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^{O}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 coalesing with each other to cover entire tillers from the water line to the flag leaf. The presence of several large lesions on a leaf sheath usually causes death of the whole leaf, and in severe cases all the leaves of a plant may be blighted in this way. The infection extends to the inner sheaths resulting in death of the entire plant. Older plants 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

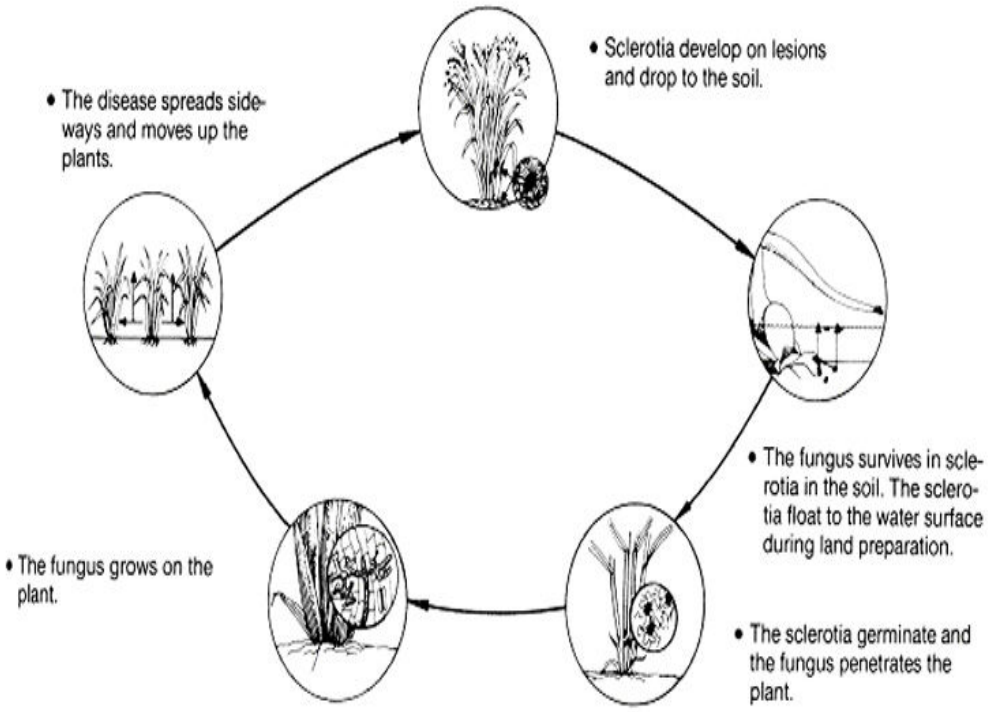
The fungus produces usually long cells of septate mycelium which are hyaline when young, yellowish brown when old. It produces large number of globose **sclerotia**, which are initially white, later turn to brown or purplish brown.

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 ^{0}C), closer planting and heavy doses of nitrogenous fertilizers.



Management

- Avoid excess doses of fertilizers.
- Adopt optimum spacing.
- Eliminate weed hosts.
- Apply organic amendments.
- Avoid flow of irrigation water from infected fields to healthy fields.
- Deep ploughing in summer and burning of stubbles.
- Grow disease tolerant varieties like **Shiva** (WGL 3943)
- Spray Propiconazole@0.1% or Hexaconazole@0.2% or Validamycin@0.2%

- Seed treatment with *Pseudomonas fluorescens* @ of 10g/kg of seed followed by seedling dip @ of 2.5 kg of product/ha dissolved in 100 litres and dipping for 30 minutes.
- Soil application of *P.fluorescens* @ of 2.5 kg/ha after 30 days of transplanting (This product should be mixed with 50 kg of FYM/Sand and then applied.
- 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

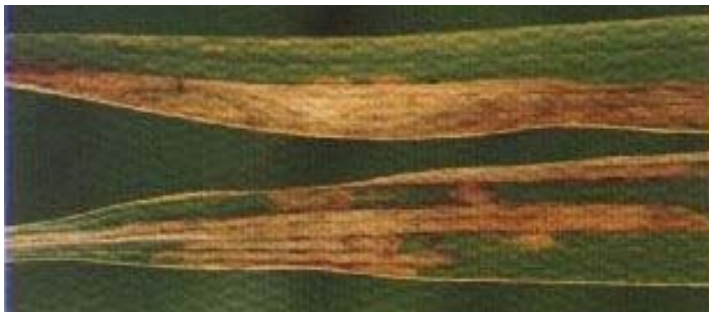
The disease was first observed in Japan (1884). In Indonesia, **Kresek** disease was reported to kill young seedlings completely in 1950. In India, BLB was first reported in 1959. A severe outbreak of the disease occurred in Bihar and Uttar Pradesh in 1963. In the tropics the disease is usually referred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

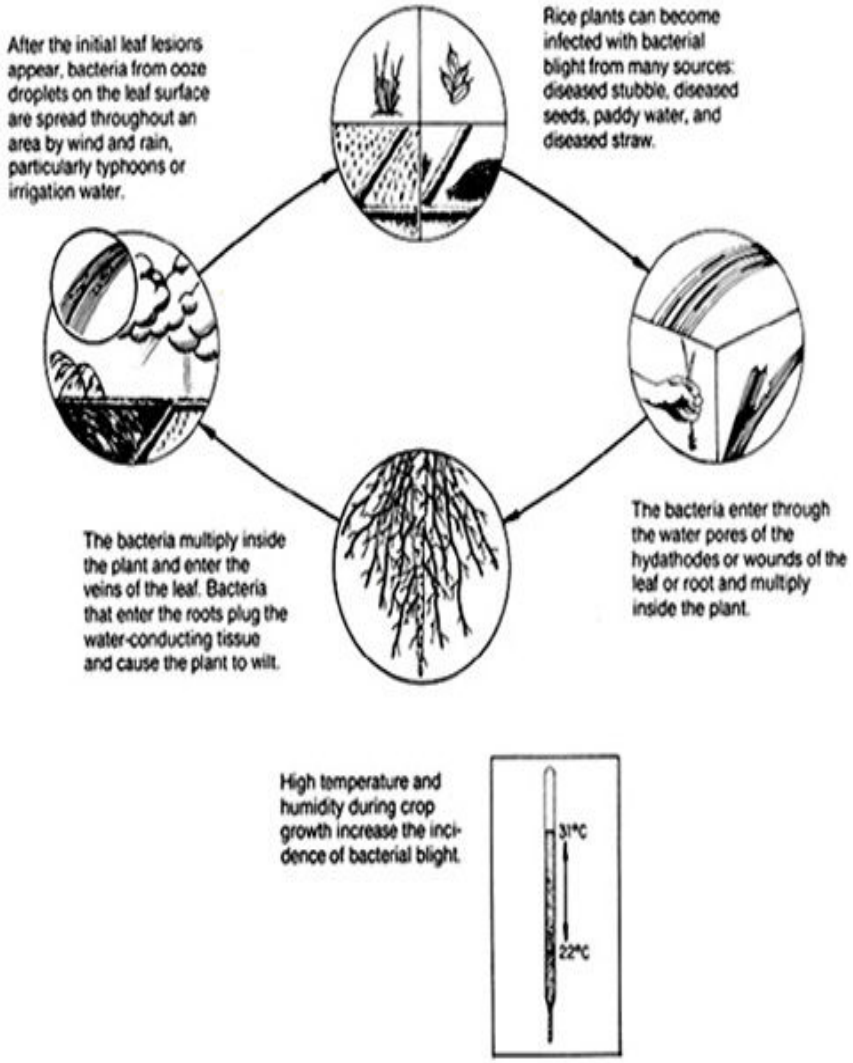
The bacterium enters the plant through water pores (**hydathodes**) along the edges of the leaf and through injuries in roots or leaves. It does not enter through stomata. BLB is primarily a vascular or **systemic** disease. Bacterial cells move along the vascular tissues causing wilting. Rain storms 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 25-30^{0}C and application of excessive nitrogen, especially late top dressing.

Management

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



- Remove and destroy weed hosts.
- Soaking seeds for 8 hrs in Agrimycin (0.025%) followed by hot water treatment for 10 minutes at 52-54 ^{0}C eradicates the bacterium in the seed
- Spray Streptocycline (250 ppm) along with copper oxychloride (0.3%)

Bacterial leaf streak

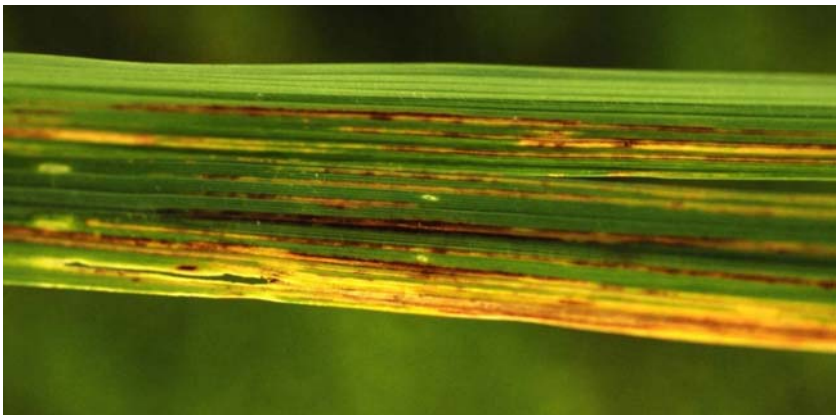
Xanthomonas campestris p.v. oryzicola

Economic importance

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

Symptoms

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

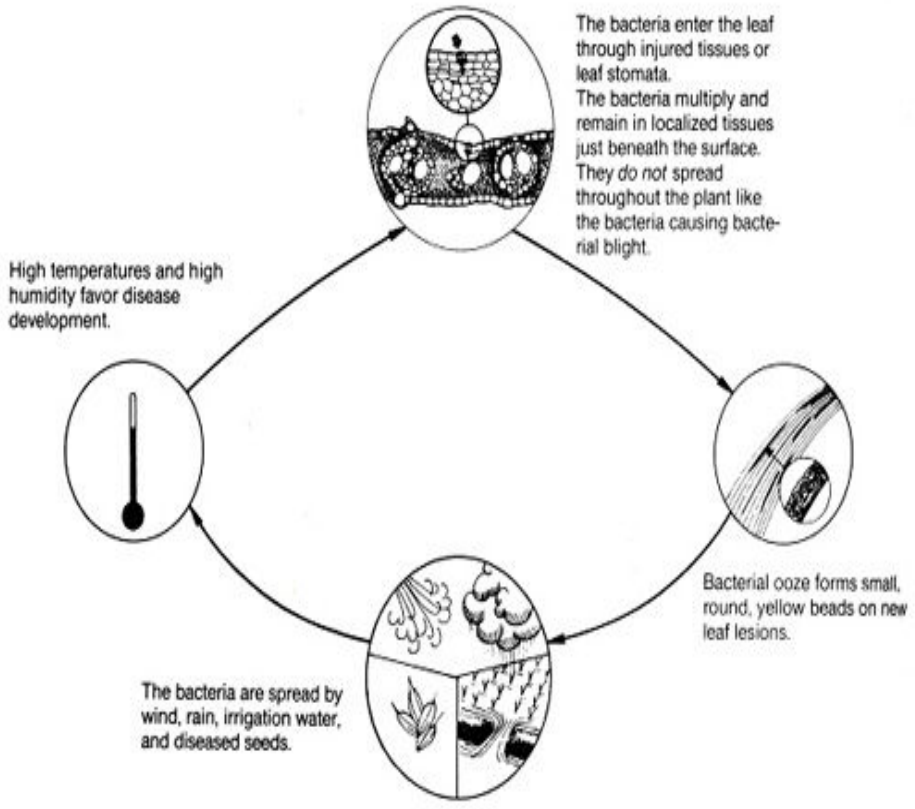


Etiology

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

Disease cycle

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 ^{0}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 hudred and thousands of hectares in Phillippines.

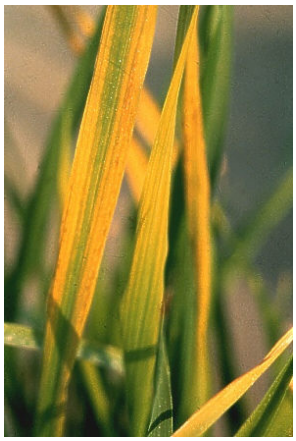
Symptoms

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

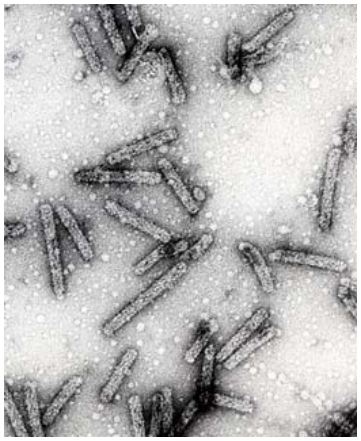
Tungro infected plants can be chemically identified by **Iodine Test.** Ten cm long leaf tip is cut in the early morning before 6 A.M. and dipped in a solution containing 2g Iodine and 6 g Potassium Iodide in 100 ml of water for 30 minutes. Tungro infected leaves show dark blue streaks.

Etiology

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



Yellow 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 filed
- 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.

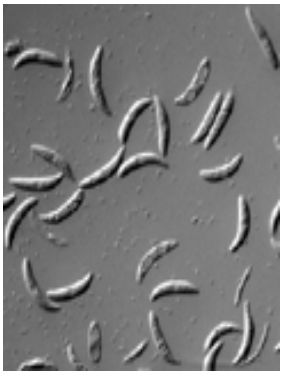
Symptoms

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^{0}C and high humidity aggravates the disease.

Management

- Destruction of infected plant debris and collateral hosts
- Crop rotation with non-host crops
- Grow resistant varieties like SPV 162, CSV 17, Texas Milo and Tift sudan etc.

- 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. **Teliopores** develop later sometimes in the old uredosori or in teliosori, which are darker and longer than the uredosori. The pustules may also occur on the leaf sheaths and on the stalks of inflorescence.

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^{0}C favours teliospore germination and a spell of rainy weather favours the onset of the disease.

Management
- Grow resistant varieties like CSH 5, SPV 13, 81, 126, PSH 1, CSV 17, etc.
- Remove and destroy the alternate host *Oxalis corniculata.*
- Spray the crop with 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 ^{0}C) and cloudy weather during anthesis encourages disease spread

rapidly causing severe losses in hybrid seed production. Male sterile lines are highly susceptible.

Management

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

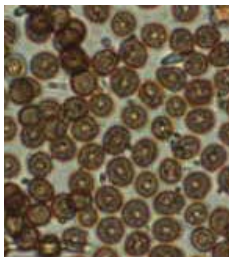
Economic importance

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

Disease cycle

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

Management

- Use disease free seeds.
- Grow resistant varieties like T 29/1, PJ 7K, PJ 23K, Nandyal and Bilichigan.
- Treat the seed with fine sulphur powder @0.5% or 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 35^0C and moisture stress conditions preceding crop maturity and application of more nitrogenous fertilizers.

Management

- Thin plant population should be maintained in problematic areas (60,000 plants/ha)
- The infected plants along with trash should be collected and burnt immediately
- Avoid moisture stress at flowering
- 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. Sporangiophores emerge through the stomata in single or in clusters which are stout and dichotomously branched with pointed sterigmata. On each sterigmata a single hyaline, globose thin walled and non-papillate sporangium is formed which germinates directly by a germ tube without any zoospores. Oospores are typically produced abundantly in parallel bands between fibro vascular strands of the shredded leaf tissue which are three walled, more or less round, thick walled and golden yellow in colour.

Disease cycle

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

Favourable Conditions

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

Management

- Destroyal of affected plants by burning before oospore formation, reduce the inoculum potential
- Crop rotation with other crops like pulses and oilseeds.
- Grow tolerant varieties like CSH 2, CSV 5, SPV 101, 165 and 190.
- Seed treatment with Metalaxyl (Apron 35 SD) @4g/kg seed
- Spray Metalaxyl (Ridomyl MZ) @0.2% 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 35^0C and soil moisture of 30 per cent is favourable for Striga infection.

Management

- Hand weeding of the parasites before flowering
- Crop rotation with cowpea, groundnut and sunflower
- Mixing of **ethrel** with soil triggers germination of Striga in the absence of host. After germination, Striga can be removed and destroyed.
- Spray **Fernoxone** (sodium salt of 2, 4-D) or Agroxone (MCPA) at 450g/500 liters of water 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^{O}C provided with free water on the leaf. Infection takes place early in the wet season.

Management

- Crop rotation
- Grow resistant hybrids like DHM-1
- Treat the seeds with 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 symtoms.

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 (35-45^{0}C). 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 (37^0C) at the time of silking. Imbalanced fertilizer application and high plant density influence disease prevalence and severity.

Management
- Long crop rotation with crops that are not natural host of the fungus.
- Field sanitation
- Irrigate the crops at the time of earhead emergence to maturity.
- Treat the seeds with 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 20-25^{0}C 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^{O}C.

Management
- Selection of seed from healthy crop
- Collect diseased plants, especially before oospores are formed, and burn them
- Summer deep ploughing
- Rogue out infected plants.
- Prolonged crop rotation
- Grow resistant varities like **WCC 75**, PHB 10, ICMH 451 ICTP 8203, Mallikarjuna, HB-1, HB 5 and PHB 14
- Grow tolerant varieties like MBH 118, CM 46, Balaji composite, Nagarjuna composite, Visakha composite, New vijaya composite, RBS 2, etc.
- Treat the seeds with Metalaxyl (Apron 35SD)@6g/kg 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 germpores, sparsely echinulated and pedicellate. Teliospores are dark brown in colour, 2 celled, cylindrical to club shaped, apex flattered, broad at top and tapering towards base.

Favourable Conditions
Closer spacing, presence of abundant brinjal plants and other species of *Solanum,* viz., *S.torvum, S. xanthocarpum* and *S. pubescens, S. panduriforme.* The uredial stage also occurs on the species of *Pennisetum*, including *P. leonis, P. purpureum, P. orientale, P. spicatum* and *P. polystachyon*.

Disease cycle
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^{0}C during flowering period, favour the disease development.

Management
- Adjust the sowing date so that the crop does not flower during September when high rainfall and high relative humidity favour the disease spread.
- Immerse the seeds in 10 per cent common salt solution and remove the floating sclerotia.
- Eradication of collateral hosts

- Grow resistant varieties like PHB 10, 14; Co 2, 3 and Bajra 24.
- Spray with 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 rain 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 (20^0C) and day (30^0C) temperatures with high relative humidity (92-95%) and rain or continuous drizzles favour the disease development. Presence of collateral hosts like bajra, wheat, barley and oats.

Management

- Destruction of collateral hosts and infected plant debris
- Treat the seeds with Captan or Thiram@3g/kg or Carbendazim at 2 g/kg.
- Grow resistant varieties like Ratnagiri, Padmavati, Gowtami and Godavari
- Spray withCarbendazim@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 7-11µ 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

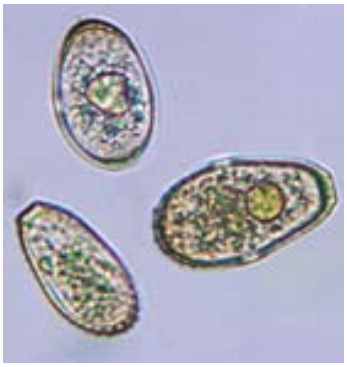
Economic importance

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

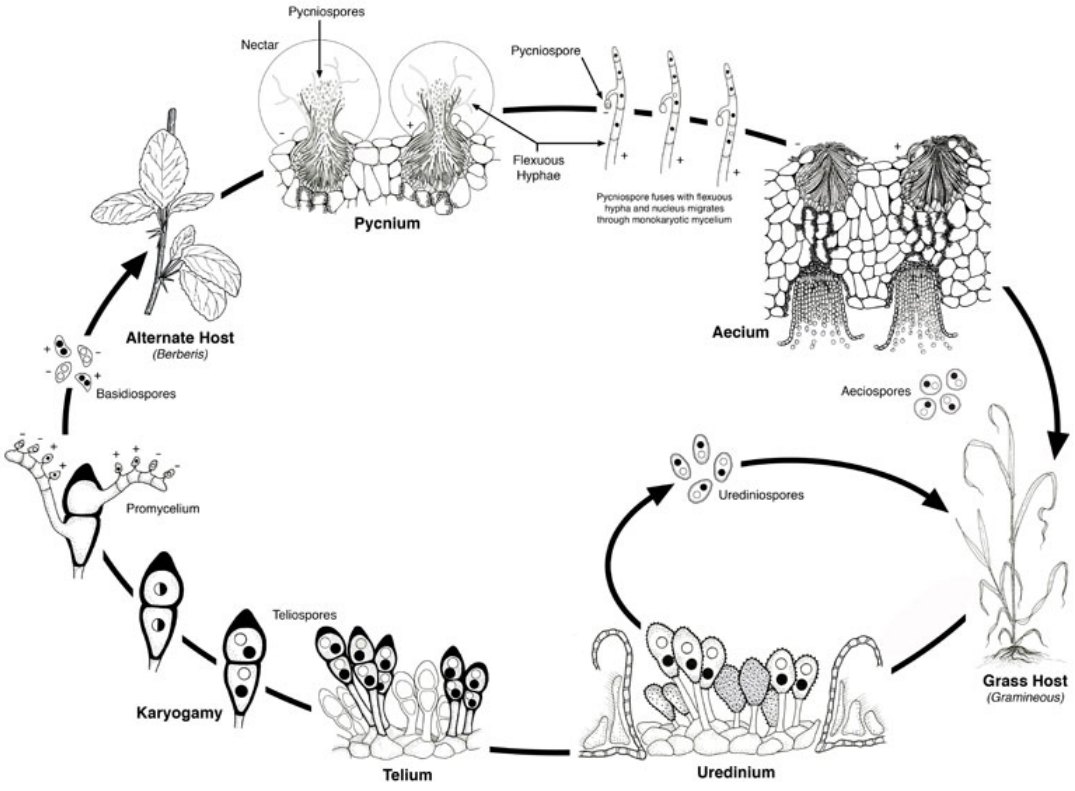
Black stem rust is **heteroecious** full cycle rust. It requires more than one host species to complete its life cycle. The uredial and telial stages occur on wheat, barley and some grasses and the pycnial and aecial stages on the species of ***Berberis*** (Barbery) and ***Mahonia***, the alternate hosts. 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 barbery plants play a role in USA, Europe and Australia, where as in India they are not known to play any role in the perpetuation of the fungus. The source of inoculum for black rust comes from south, i.e., **Nilgiri** and **Pulney** hills. In plains of North India during summer months the uredospores cannot survive because of the high temperatures. The possibilities of the fungus surviving on ratoon tillers or **self sown wheat** plants, late and **off season wheat** crops and certain grasses growing in cool areas particularly in the foot hills of Himalayas in the North, the Nilgiris and Pulney hills in the South appear to be great.

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



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

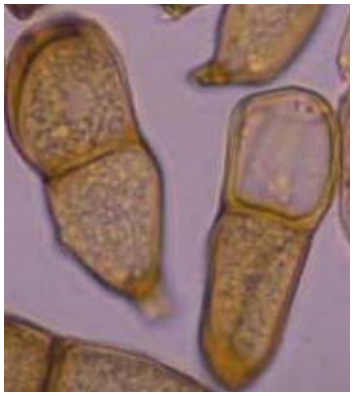
Economic importance

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 oversummer in other countries. The role of *Thalictrum* is not clear in India. In early January, the rust gets well established in the foot hills of Himalayas and also in the plains of Tamil Nadu and Karnataka in the South. The first 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 striformis

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 catharaticus, Bromus japonicus*** and ***Hordeum murinum*** also serve as primary source of inoculum. Secondary infection is by wind borne uredospores. There are about 40 races in the world including 13, 14, 19, 20, 24 and 31 A which are wide spread in India.

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

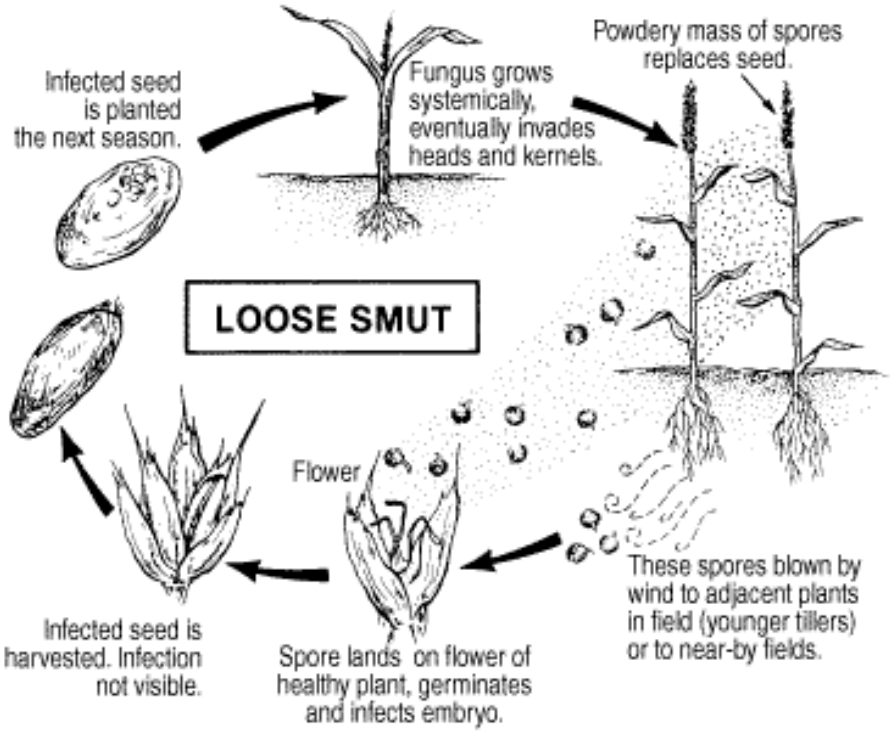
Loose smut is one of the major diseases on wheat. There was loose smut epidemic during 1970-75 in Punjab, Haryana and Western U.P. In Sonalika, the incidence was 5 to 6%. Incidence is more in North than in southern parts of India. Country wide loss is about 2-3% in yield.

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 ^{0}F or 52^{0}C for about 10 minutes. Dry the seed in shade before sowing.
- ***Solar seed treatment*** (**Luthra and Sattar**, 1934): Soak the seed in water for 4 hours (8 AM to 12 Noon) and expose the seed to the hot sun for 4 to 5 hours (from 12 Noon to 5 PM) on cement or rocky surface. This can be practiced in the areas where the summer temperatures are high (42-44^{0}C)
- ***Anaerobic seed treatment*** (USA): Soak the seeds for 2-4 hours in water between 60-70^{0}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)

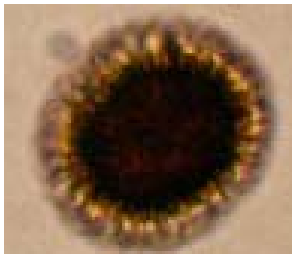
Economic importance

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 sac of teliospores. As the grains mature the outer glumes spread and the inner glumes expand, exposing the bunted grains. The bunt balls are first enclosed by the pericarp but when it bursts the masses of bunt spores are exposed. The bunt affected plants emits a foul smell which is mainly due to the presence of **Trimethyl amine**.

Bunted grains



Teliospore

Allantoid sporidia

Pathogen

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

Disease cycle

The teliospores in soil germinate producing primary sporidia. The sporidia become air borne and deposit on leaves of host plants. Under high humid conditions they produce a secondary crop of secondary sporidia (allantoids). If boot emergence stage coincides with drizzle, the secondary sporidia get washed down to sheath. The sporidia germinate on glumes to enter epidermal cells to penetrate ovary. The sporidial germ tubes penetrate stomata in rachis, glumes, lemma and palea. The disease progresses systemically to other florets within an infected spikelet. The infection mostly starts from the embryonal end and spreads **along the grain suture**. The hyphae grow through the base of the glumes into sub-ovarian tissue and enter pericarp through funiculus. Hyphae of the pathogen proliferate, remain restricted to pericarp and produce teliospores terminally. In severe cases grain is reduced to black shiny sack of teliospores. The embryo and endosperm are not colonized. The pericarp ruptures during threshing and teliospores deposit in soil and adhere to the surface of the seed.

Favourable conditions

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

Management

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

- Seed treatment with copper carbonate or 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 ^{0}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^{0}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 winkling, twisting and various other distortion of the leaves, stem and produce small round galls on the leaves. The infected plants are shorter and thicker than healthy plants. In the distorted earheads dark galls are found in place of kernels.

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

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

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 looses 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 28^0C, high atmospheric temperature of 30-40^0C, 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 52-56^0C 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 loose 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^{0}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 ***(Myllocerus 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 20^0C 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

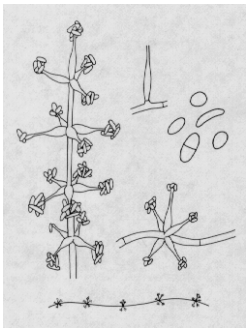
Economic importance

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^{0}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^{O}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 (20-30^{0}C) 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.***

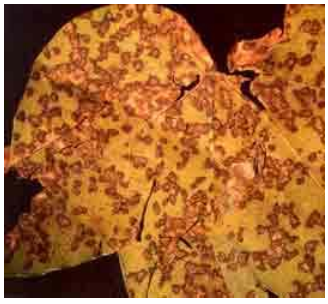
Favourable Conditions
Prolonged rainfall at the time of boll formation and close planting predispose the disease.

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 25-28^{0}C 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)*

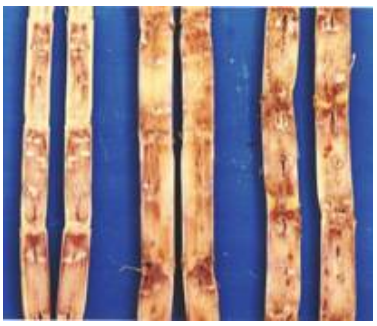
Economic importance

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, velvetty 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 velvetty **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 52^0C for 30 min or 50^0C for 2 hours followed by steeping in 0.1% carbendazim solution.
- Setts can be treated with aerated steam at 52^0C for 4 to 5 hours and by moist hot air at 54^0C 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 50^0C for 2 hours.
- Grow resistant varieties like **Co 6806** and **Co 62175**

Wilt

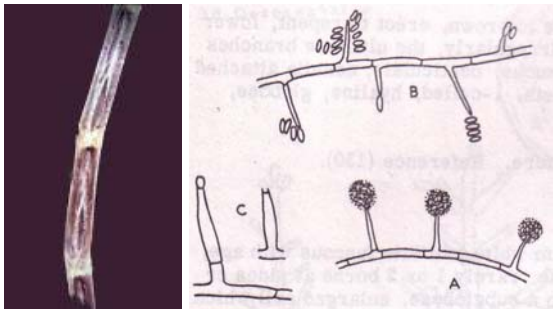
Cephalosporium sacchari

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^{0}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^{0}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 auxillary 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 mm 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

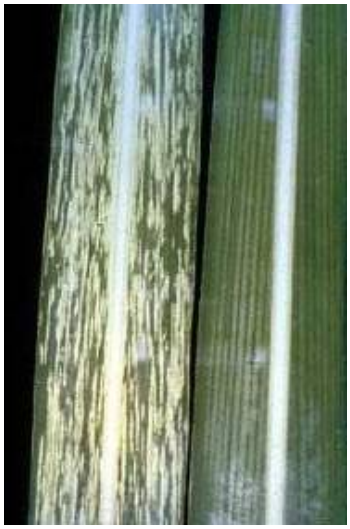
- Plant disease free setts.
- Remove and burn the infected clumps periodically.
- Avoid ratooning in problem areas
- Hot Water Treatment (HWT) of setts at 52^0C for 30min or Aerated Steam Therapy (AST) at 50^0C for 1 hr followed by steeping in fungicidal solution of carbendazim@0.05% for 15 minutes.
- 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 12-15µm. 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 vulgar****e, Pennisetum americanum, Eleusine indica, Setaria lutescens, Echinochloa crusgalli, Stenooaphorum secondatum, Digitaria didactyla etc.,* which serve as potential sources of virus inoculum. The disease mainly spreads through sap and **aphids**, viz., *Rophalosiphum 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: 52^0C for 20 minutes on the first day, 57.3^0C for 20 minutes on the second day and 57^0C for 20 minutes on the third day or Aerated Steam Therapy (AST) at 56^0C 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

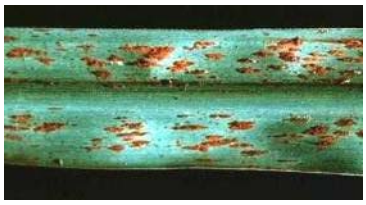
- Select the setts from disease free field.
- Remove and burn the clumps showing the disease.
- Sterilization of cutting knives with lysol or any other antiseptic solution.
- Hot air treatment of setts at 54^0C for 8 hours or hot water treatment at 50^0C for 2 hrs or aerated steam treatment at 50^0C 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 30^0C, 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 discolouration. 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 the 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 24^0C) and low soil temperature of about 20^0C.

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/2.5m^2)
- 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 20-30^{0}C, 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

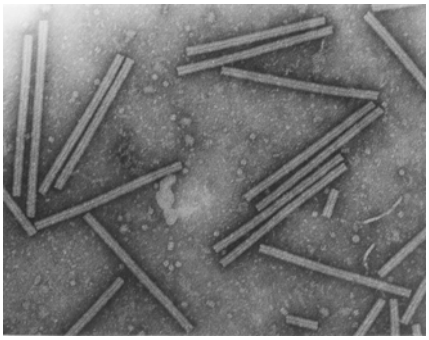
Economic importance

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 90^0C 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 30^{th}, 40^{th} and 50^{th} 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

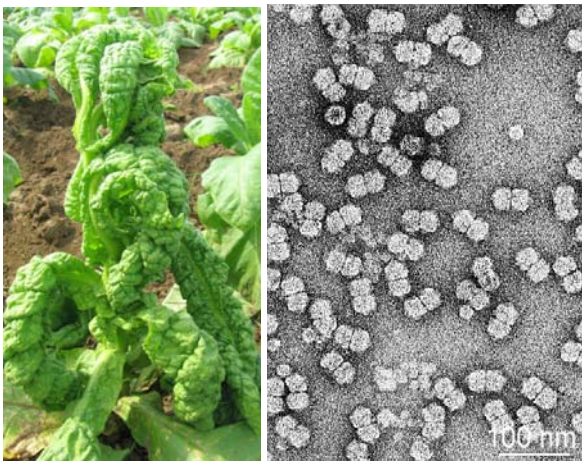
Tobacco leaf curl virus or Nicotiana Virus 10 (Ruga tabaci)

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 berkeleyii)*
*(*Syn: *Cercosporidium personatum*)

Economic importance

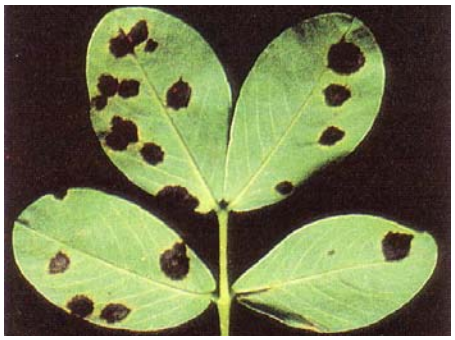
This is the most destructive disease of groundnut occurring where ever groundnut is cultivated. In India this disease occurs in all the groundnut areas causing severe damage, particularly when the plants are affected early in the season. The loss in pod production may be sometimes as high as 50%. The **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^{0}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

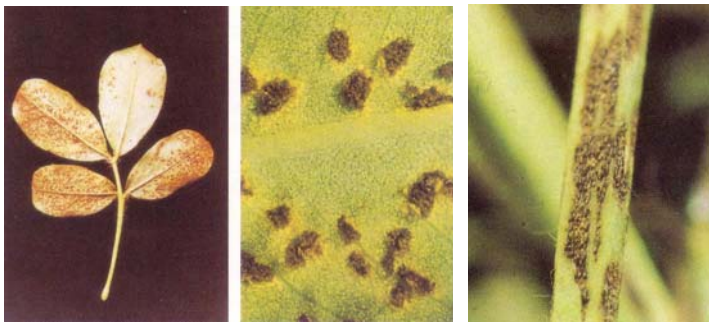
Puccinia arachidis

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 (20-25^{0}C) 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 PBND than late sown crop (late June)
- Higher incidence where plant population is less (<23 plants/m^2) as against optimum population (33 plants/m^2). 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/m^2) as against optimum population (33 plants/m^2). 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 (20-25^{0}C), 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 53^{0}C 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 perenneates through cleistothecia in the infected plant debris in soil. The ascospores from the cleistothecia cause primary infection. The secondary spread is through wind-borne conidia.

Favourable Conditions

Dry humid weather and low relative humidity favours the disease.

Management

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

Phyllody

Phytoplasma

Economic importance

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

Symptoms

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

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

Disease cycle

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

Favourable conditions

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

Management

- Remove all the reservoir and weed hosts.
- Delay sowing in the endemic areas to reduce the vector population and thereby the disease.
- Avoid growing sesamum near cotton, groundnut and grain legumes.
- Rogue out the infected plants periodically.
- *Sesamum mulayanum* is the resistant source to the pathogen.
- Spray 2-3 times with 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 30^0C and above and prolonged drought followed by copious irrigation.

Management
- Treat the seeds with *Trichoderma viride* at 4g/kg or *Pseudomonas fluorescens* 10 g/kg 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 52^0C for 10 minutes.
- Steep the seed in Agrimycin 100 (250 ppm) or streptocycline 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 35^0C 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 22^{0}C 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 50-60^{0}C for 10 minutes.
- Spray streptocycline@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. Sporangiophores 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 (20-25^{0}C), 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 30.5^{0}C with RH of 86-92% favours rust disease severity. The incidence of rust increases with age, the maximum being on 75 days old plants.

Management

- Remove and burn the infected plant debris in the field.
- Remove the volunteer sunflower plants.
- Crop rotation for 3 years
- Grow tolerant variety like BSH-1.
- Spray 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 perenneates through cleistothecia in the infected plant debris in soil. The ascospores from the cleistothecia cause primary infection. The secondary spread is through wind-borne conidia.

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, *Bamesia 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

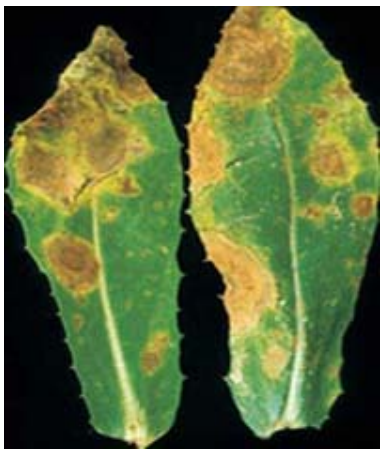
Alternaria carthami

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 50^0C 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 15-20^{0}C 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. Sporangiophores are dichotomously branched with sterigmata which are pointed with acute angles usually of equal length. Oval and non-papillate sporangia are produced over the pointed sterigmata. Sporangia always germinate by germ tube and behave as conidia.

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

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

Powdery mildew
Erysiphe cruciferarum

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

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

Disease cycle
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 loose turgidity, and become desiccated. Lesions girdle the affected main stems or branches which break at this point and foliage above the lesion dries up. When conditions favour the pathogen, it is common for many plants to die. Pigeonpea plants that are infected by blight, but not killed often produce large galls on their stems especially at the edges of the lesions. The pathogen infects the foliage and stems but not the root system.


Pathogen

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

Favourable Conditions

Cloudy weather and drizzling rain with temperatures around 25^0C favour infection that requires continuous leaf wetness for 8 hours to occur. Warm and humid weather following infection results in rapid disease development and plant death. Soils with **poor drainage**, low lying areas, heavy rain during the months of July-September favours the disease. Pigeonpeas are usually not infected after they are 60 days old.

Disease cycle

The fungus survives in the soil and plant debris in the form of oospores, and dormant mycelium. 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 25^0C 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, **cankerous** lesions appear on the stem. Leaf spots do not usually cause defoliation. Cankers can cause stems to break, but the broken part usually attaches to the plant. Stems often break at the point where the primary leaves are attached. Often, the affected plants do not break, and the stem cankers increase in size until they are 15-25 cm long. In cases of severe infection the affected branches dry.


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

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

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

Management

- Treat the seeds with Carbendazim or Thiram at 2 g/kg or treat the seeds with *Trichoderma viride* at 4 g/kg or *Pseudonomas fluorescens* @ 10g/kg of seed.
- Apply heavy doses of organic manure or green manure.
- Follow 6-year crop rotation with non-host crops.
- Grow resistant cultures like **Kranthi** (ICCC 37), **Swetha** (ICCV-2), ICCV 10, Avrodhi, G 24, C 214, BG 244, Pusa 212 and JG 315.

Rust

Uromyces ciceris-arietini

Symptoms

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

Pathogen

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

Disease cycle

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 block dots. The stem and petioles usually break at the point of infection due to girdling. If the main stem is girdled at the collar region, the whole plant dies.

Pathogen

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

Favourable Conditions

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

Disease cycle

The fungus survives in the infected plant debris as pycnidia. The pathogen is also **externally** and **internally** seed-borne. 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 40-50^0C 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 30^0C, minimum above 20^0C, 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 21-26^{0}C 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 Wettable sulfur@0.3%, immediately on the set of disease and repeat after 15 days.
- Grow tolerant black gram cultivar like **LBG 648**.

Cercospora leaf spot

Cercospora canescens

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

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

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

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

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

Symptoms
Symptoms appear from 3 weeks after sowing. Small light yellow spots appear on older leaves, enlarge gradually turning into angular black spots. When infection is severe, several such spots coalesce resulting in drooping, drying and defoliation. Yields are greatly reduced due to poor pod set and reduction in seed size.

Pathogen

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

Protomycopsis produces finely punctuate (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 with in week. Dark brown lesions are seen on the stem at ground level and bark shows shredding symptom. The affected plants can be easily pulled out leaving dried, rotten root portions in the ground. The rotten tissues of stem and root contain a large number of black minute sclerotia.

Pathogen

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

Disease cycle

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

Favourable Conditions

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

Management

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

Bacterial leaf spot

Xanthomonas phaseoli

Symptoms

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

Disease cycle

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 streptocycline solution for 30 minutes before sowing.
- Spray twice with paushamycin or plantomycin 100 mg in combination with 3 g of COC per liter at an interval of 12 days.

Yellow mosaic

Mungbean yellow mosaic virus

Economic importance

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

Symptoms

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

Disease cycle

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

Leaf crinkle

Leaf crinkle virus

Symptoms

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

Disease cycle

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

Cuscuta

Symptoms

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

Survival and spread

- 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

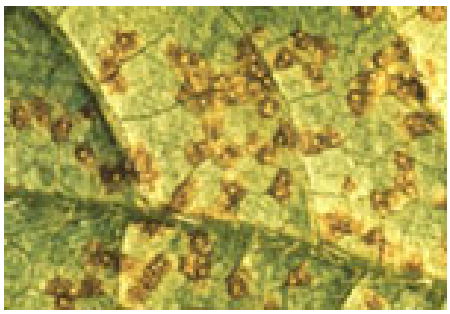
Phakopsora pachyrhizi

Economic importance

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

Symptoms

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

Disease cycle

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^{0}C and R.H of 80%.

Management

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

Soybean mosaic

Soybean mosaic virus

Symptoms

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

Disease cycle

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 45and 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

Disease of Horticultural Crops

& Their Management

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Lecture 01 - Diseases of Citrus

Gummosis: ***Phytophthora parasitica, P. palmivora,P. citrophthora***

Symptoms

The symptoms appear as yellowing of leaves, followed by cracking of bark and profuse gumming on the surface. The main source of infection is infected planting material. As a result of severe gumming, the bark becomes completely rotten and the tree dries owing to girdling effect. Prior to death, the plant usually blossoms heavily and dies before the fruits mature. In such cases, the disease is called foot rot or collar-rot.

Pathogen

Aseptate, intercellular & intracellular hypha. Sporangia are ovoid or ellipsoid. Sporangium attached with the sporangium at the right angles sporangia germinate to release zoospore.

Favourable conditions

Prolonged contact of trunk with water as in flood irrigation; water logged areas and heavy soils.

Mode of spread and survival

Soil inhabitants, Sporangia spread by splashing rain water, irrigation water and wind.

Management

Preventive measures like selection of proper site with adequate drainage, use of resistant rootstocks and avoiding contact of water with the tree trunk by adopting ring method of irrigation are effective. Alternatively the disease portions are scraped-out with a sharp knife and the cut surface is disinfected with Mercuric chloride (0.1%) or Potassium permanganate solution (1%) using a swab of cotton. Painting 1 m of the stem above the ground level with Bordeaux helps in controlling the disease. Also spraying and drenching with Ridomil MZ 72@ 2.75 g/l or Aliette (2.5 g/l) is effective in controlling the disease.

Scab/Verucosis : ***Elsinoe fawcetti***

Symptoms

The lesions in early stages appear on the underside of the leaves as small semi-translucent dots, which finally become sharply defined pustular elevations. In later stages, leaves often become distorted, wrinkled, stunted and deformed. On the fruit, lesions consist of corky projections, which often break into scabs. The opposite surface corresponding to the warty growth shows a circular depression with a pink to red center.

Pathogen

Ascostroma are simple, innate, intra or sub epidermal, partially erumpent at maturity, small pulvinate to crustose. Asci are ovoid. Ascospares are 1-3 septate oblong to elliptical and hyaline to yellowing conidia are produced in acervuli. Conidia are hyaline, ablong, elliptical with two minute droplets of their ends.

Mode of Spread and Survival

The pathogen survive in off season as ascospores and spreads through Conidia.

Management

The diseased leaves, twigs and fruits should be collected and destroyed. Spraying of Carbendazim 0.1% is quite effective

Canker : *Xanthomonas campestris* pv *citri*

Symptoms

Acid lime, lemon and grapefruit are affected. Rare on sweet oranges and mandarins. Affects leaf, twig and fruits. In canker, leaves are not distorted.Lesions are typically circular with yellow halo; appear on both sides of leaf, severe in acid lime (difference from scab) When lesions are produced on twigs, they are girdled and die. On fruits, canker lesions reduce market value.

Pathogen

It is Gram negative, non spore forming, aerobic bacteria. It is rod shaped, forms chains and capsules and is motile by one polar flagellum.

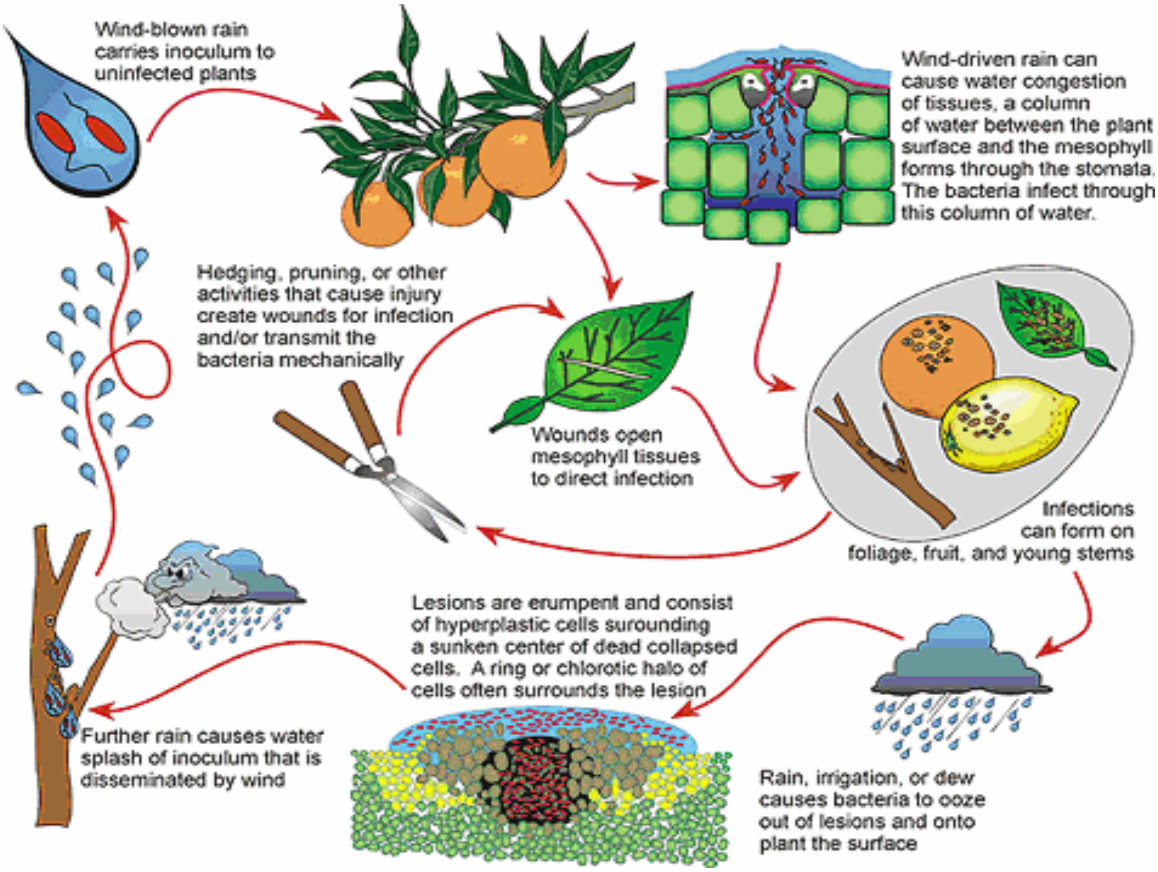
Favourable conditions

Free moisture for 20 minutes, 20-30°C.

Mode of survival and spread

Wind and rain splashes. Survives in infected leaves for 6 months. Injury caused by leaf miner helps the entry of the bacterium.

Disease Cycle



Management

Streptomycin sulphate 500-1000 ppm; or Phytomycin 2500 ppm or Copper oxychloride 0.2% at fortnight intervals. Control leaf miner when young flush is produced. Prune badly infected twigs before the onset of monsoon.

Tristeza or quick decline : *Citrus tristeza virus* **(CTV)**

Symptoms

Lime is susceptible both as seedling or buddling on any root stock. But mandarin and sweet orange seedlings or on rough lemon, trifoliate orange, citrange; Rangpur lime root stocks tolerant; susceptible root stocks are grapefruit and sour orange.

In sweet orange or mandarin on susceptible root stocks, leaves develop deficiency symptoms and absise. Roots

decay, twigs die back. Fruit set diminishes; only skeleton remains. Fine pitting of inner face of bark of sour orange stock. Grapefruit and acid lime are susceptible irrespective of root stock. Acid lime leaves show large number of vein flecks (elongated translucent area). Tree stunted and dies yield very much reduced. Fruits are small in size. Use of infected bud wood and *Toxoptera citricida* (aphid) is the important vector.

Pathogen

Citrus tristeza virus is long, flexuous rod and measure 2000x 12nm in size. Three strains *viz.*, mild, severe and seedling yellow are reported.

Mode of spread

Use of infected bud wood *Toxoptera citricida* (aphid) is the important vector.

Management

For sweet orange and mandarin, avoid susceptible root stocks. For acid lime, use seedling preimmunised with mild strain of tristeza.

Exocortis of scaly butt: *Viroid*

Symptoms

Affects only Rangpur lime, trifoliate orange and citrange root stocks. Vertical cracking and scaling of bark in the entire, root stock. Extreme stunting of plant.

Pathogen

Viroid is free RTVA without protein coat.

Mode of Spread and Survival

Transmission normally occurs through infected bud, wood, and contaminated tools. Not through vector and seed.

Management

Spray with any one of the systemic insecticide to control the aphid vector. Use virus-free certified bud wood; use tolerant stocks like rough lemon Periodically wash budding knife with disodium Phosphate solution.

Greening: *Liberobactor asiaticum* (Phloem limited bacteria)

Symptoms

This disease affects almost all citrus varieties irrespective of root stock. Stunting of leaf, sparse foliation, twig die back, poor crop of predominantly greened, worthless fruits. Sometimes only a portion of tree is affected.A diversity of foliar chlorosis. A type of mottling resembling

zinc deficiency often predominates. Young leaves appear normal but soon assume on outright position, become leathery and develop prominant veins and dull olive green colour.

Green circular dots on leaves. Many twigs become upright and produce smaller leaves. Fruits small, lopsided with curved columella. The side exposed to direct sunlight develops full orange colour but the other side remain dull olive green. Low in juice and soluble solids, high in acid. Worthless either as fresh fruit or for processing. Seeds poorly developed, dark coloured, aborted.

Pathogen

Rickettsia like organisam

Mode of spread

Infected budwood; psyllid vector-*Diaphorina citri*

Management

Control psyllids with insecticides. Use pathogen free bud wood for propagation. 500 ppm tetracycline spray, requires fortnightly application.

Lecture 02 - Diseases of Mango (2 Lectures)

Anthracnose: *Colletotrichum gloeosporioides*

Symptoms:

The disease appears on young leaves, stem, inflorescence and fruits. Leaves show oval or irregular, greyish-brown spots which may coalesce to cover larger area of the leaf. The affected leaf tissues dry and shred. Leaves on infected petioles droop and fall. On young stem, grey-brown spots develop. These enlarge and cause girdling and drying of the affected area. The disease appears on young leaves, stem, inflorescence and fruits.

Often, black necrotic areas develop on the twigs from the tip downwards causing a dieback. In humid weather, minute, black dots develop on the floral organs. The infected flower-parts ultimately shed resulting in partial or complete deblossoming. Latent infections of fruit are established before harvest. The ripening fruits show typical anthracnose. Black spots appearing on skin of the affected fruits gradually become sunken and coalesce.

Pathogen

Mycelium septate and coloured. Conidia Single celled, hyaline, small and elongated.

Mode of survival and spread

On dried leaves, defoliated branches mummified flowers and flower brackets. Contact with diseased fruit during transport and storage. The secondary spread is through airborne conidia.

Favourable conditions

Temperature of 25°C and Relative Humidity 95-97%

Disease cycle

The survival of pathogen in detached diseased twigs and leaves lying on surface of soil and in diseased twigs attached to the tree. They successfully reproduced the disease by inoculating leaves, petioles, stems and fruits. The optimum temperature for infection was found

to be 25˚C. The disease spreads rapidly in the rainy season. Cloudy and misty weather during flowering favors damage to the infected floral parts.

The pathogen causes severe leaf spotting. The appearance of spots in more concentration at the stem-end and sometimes in stripes down the sides of the fruits suggested distribution of spores by rain water over surface of the fruit. The fungus can enter the pores of green fruits. The latent infection of mature fruits may take place through lenticels. The fungus apparently infects the fruit while it is green and develops in flesh during ripening.

Management

Spray *P. fluorescens* (FP 7) at 3 weeks interval commencing from October at 5g/like on flower branches. 5-7 sprays one to be given on flowers and bunches. Before storage, treat with hot water, (50-55°C) for 15 minutes or dip in Benomyl solution (500ppm) or Thiobendazole (1000ppm) for 5 minutes

Powdery mildew: *Oidium mangiferae (Acrosporum mangiferae)*

Symptoms

Powdery mildew is one of the most serious diseases of mango affecting almost all the varieties. The characteristic symptom of the disease is the white superficial powdery fungal growth on leaves, stalk of panicles, flowers and young fruits. The affected flowers and fruits drop pre-maturely reducing the crop load considerably or might even prevent the fruit set. Rains or mists accompanied by cooler nights during flowering are congenial for the disease spread.

Pathogen

Mycelium is ectophytic. Conidiophores short, hyaline and conidia single celled -barrel shaped, produced in chain. Fungus is odium type.

Mode of survival and spread

Survives as dormant mycelium in affected leaves. Secondary spread by air borne conidia.

Disease Cycle

Spores blown wind from infected areas readily adhere to hairy, unopened flowers near tip of the inflorescence and germinate in five to seven hours. Fungus grows rapidly during cloudy weather accompanied with heavy morning mist. Warm, humid weather and low night temperatures favour dissemination of the pathogen. Overall disease development is favoured by high humidity.

Management

Dusting the plants with fine sulphur (250-300 mesh) at the rate of 0.5 kg/tree. The first application may be soon after flowering, second 15 days later (or) spray with Wettable sulphur (0.2%), (or) Carbendazim (0.1%),(or) Tridemorph (0.1%),(or) Karathane (0.1%).

Mango malformation : *Fusarium moliliforme var. subglutinans*

Symptoms

Three types of symptoms: bunchy top phase, floral malformation and vegetative malformation. In bunchy top phase in nursery bunching of thickened small shoots, bearing small rudimentally leaves. Shoots remain short and stunted giving a bunchy top appearance. In vegetative malformation, excessive vegetative branches of limited growth in seedlings. They are swollen with short internodes forming bunches of various size and the top of the seedlings shows bunchy top appearance. In malformation of inflorescens, shows variation in the panicle. Malformed head dries up in black mass and persist for long time. Secondary branches are transformed into number of small leaves giving a witches broome appearance.

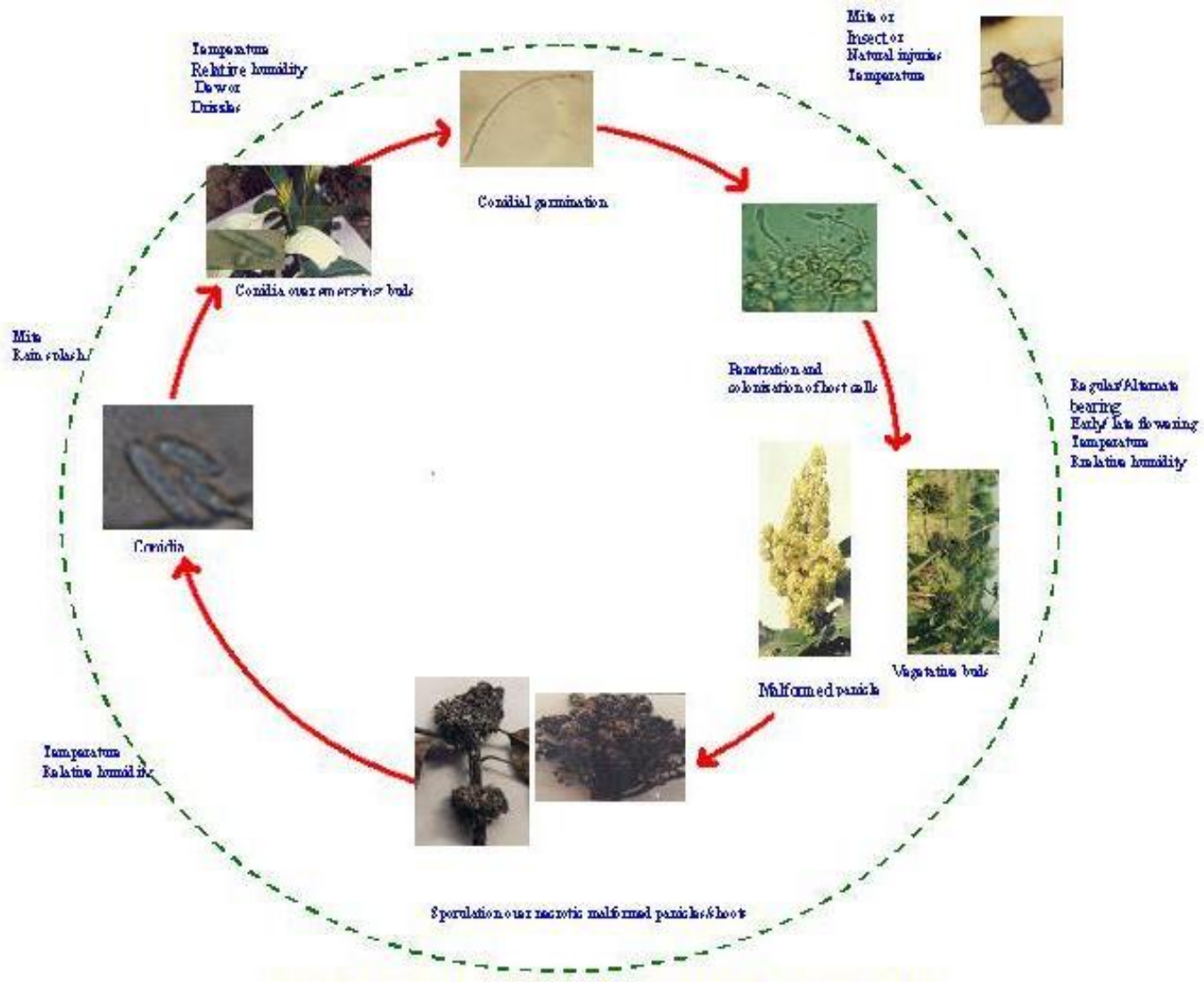
Pathogen

Micro conidia are one or 2 celled, oval to fusiform and produced from polyphialides. Macro conidia are rarely produced. They are 2 -3 celled and falcate. Chlamydospores are not produced.

Mode of spread

Diseased propagatives materials.

Disease Cycle



Management

Diseased plants should be destroyed .Use of disease free planting material. Incidence reduced by spraying 100-200ppm NAA during October. Pruning of diseased parts along the basal 15-20 cm apparently healthy portions. This is followed by the spraying of Carbendazim (0.1%) or Captafol (0.2%).

Stem end rot: *Diplodia natalensis*

Symptoms

The dark epicarp around the base of the pedicel. In the initial stage the affected area enlarges to form a circular, black patch. Under humid atmosphere extends rapidly and turns the whole fruit completely black within two or three days. The pulp becomes brown and somewhat softer. Dead twigs and bark of the trees, spread by rains

Pathogen

The fungus produces brown to black, globose to sub globose, pyriform, erumpent pycnidia that are ostiolate. They are 120-155x370-465 micron meter. Two types of conidia are produced within a pycnidium. One is hyaline, thin walled and unicellular.Tha another one is thick walled and bicelled with four to six longitudinal striations.

Mode of spread and survival

The fungus persists in infected plant parts which serve as source of inoculum.

Management

Prune and destroy infected twigs and spray Carbendazim or Thiophanate Methyl(0.1%) or Chlorathalonil (0.2%) as fortnightly interval during rainy season.

Red-rust: *Cephaleuros virescens*

Symptoms

Algae attacks foliage and young twigs. Rusty spots appear on leaves, initially as circular, slightly elevated, coalesce to form irregular spots. The spores mature fall off and leave cream to white valvet texture on the surface of the leaves.

Pathogen

Cephaleuros virescens after a period of vegetative growth develops its reproductive structures.Sporangia formed directly on the thallus are sessile and thick walled with orange pigments. hey are formed singly on the vegetative filaments. When the sporangia are riped the con tents are converted into Zoospores and liberated through an opening in the wall. The Zoospores are orange in colour, ovoid and swim actively by means of cilia.

Management

Bordeaux mixture (0.6%) or Copper oxychloride 0.25%

Grey Blight : *Pestalotia mangiferae*

Symptoms

Brown spots develop on the margin and at the tip of the leaf lamina. They increase in size and become dark brown. Black dots appear on the spots which are acervuli of the fungus. Survive on mango leaves for over a year. Spreads through wind borne conidia. Heavy infection is noticed during the monsoon when the temperature is 20-25˚C and high humidity.

Pathogen

Acervuli seen as minute black dots on affected portion. Mycelium is colored and septate. Conidia five celled middle three cells are colored and the end cells are hyaline Slender 3-5 appendages are produced at the apex of the spore.

Mode of survival and spread

Survive on mango leaves for over a year. Spreads through wind borne conidia.

Favourable conditions

Heavy infection is noticed during the monsoon when the temperature is 20-25˚C and high humidity.

Management

Remove and destroy infected plant parts. Spraying copper oxychloride 0.25 Mancozeb 0.25% or Bordeaux mixture 1.0%.

Sooty mould : *Capnodium mangiferae*

Symptoms

The fungi produce mycelium which is superficial and dark. They row on sugary secretions of the plant hoppers. Black encrustation is formed which affect the photosynthetic activity. The fungus grows on the leaf surface on the sugary substances secreted by jassids, aphids and scale insects.

Favourable conditions

The fungus grows on the leaf surface on the sugary substances secreted by Jassids,Aphids and scale insects.

Management

Management should be done for insects and sooty moulds simultaneously. Controlling of insect by spraying systemic insecticides like Monocrotophos or methyl dematon .After that spray starch solution (1kg Starch/Maida in 5 litres of water. Boiled and dilute to 20 liters) .Starch dries and forms flake which are removed along with the fungus.

Lecture 03 - Diseases of Banana (2 Lectures)

Panama disease :*Fusarium oxysporum* f. sp*cubense*

Economic Importance

The first major disease which attacked banana was called Panama disease from the area where it first became serious. Banana wilt is a soil-borne fungal disease and gets entry in the plant body through roots and wounds caused by nematodes. It is most serious in poorly drained soil. Disease spreads through infected suckers.

Symptoms

Yellowing of the lower most leaves starting from margin to midrib of the leaves. Yellowing extends upwards and finally heart leaf alone remains green for some time and it is also affected. The leaves break near the base and hang down around pseudostem. Longitudinal splitting of pseudostem. Discolouration of vascular vessels as red or brown streaks. The fungus spreads through use of infected rhizomes Continuous cultivation results in build up of inoculum.

Pathogen

Mycelium is septate, hyaline and branched. Fungus produces micro, macro conidia and also chlamydospores. Micro conidia - Single celled or rarely one septate hyaline elliptical or oval. Macro conidia - Sickle shaped hyaline, 3-5 septate and tapering at both ends. Chalamydospores - Thick walled, spherical to oval, hyaline to slightly yellowish in colour.

Mode of spread and survival

The pathogen is soil borne. It survives in soil as chlamydospores for longer periods. The primary spread of the disease is through infected rhizomes and secondary spread is through irrigation water. Continuous cultivation results in build up of inoculum.

Management

Avoid growing of susceptible cultivars *viz.*, Rasthali, Monthan, Red banana and Virupakshi. Grow resistant cultivar Poovan. Since nematode predispose the disease pairing and prolinage wit Carbofuran granules. Corm injection of 3 ml of 2% Carbendezim injected in the corm by making a hole to a depth of 10cm with 45 0 angle on 5 th and 7 th month as mentioned earlier.

Moko disease : *Pseudomonas solanacearum / Burkholderia solanacearum*

Symptoms

Leaves become yellow and progress upwards. The petiole breaks and leaves hang. When it is cut open discolouration in vascular region with pale yellow to dark brown colour. The discolouration is in the central portion of the corm. Internal rot of fruits with dark brown discoloration. When the pseudostem is cut transversely bacterial ooze can be seen.

Pathogen

It is rod shaped, gram negative bacterium with one polar flagellum.

Mode of spread and survival

The pathogen is soil borne, it survives in susceptible hosts like banana and Heliconia spp.

Management

Eradicate infected plant. Expose soil to direct sunlight. Use of clean planting material. Fallowing and crop rotation is advisable.Disinfection of pruning of tools. Providing good drainage.

Tip over or Heart rot: *Erwinia carotovora* subsp. *carotovora*

Symptoms

The base of the pseudostem and upper portion of the corm are affected and leads to rotting. Young 1-3 month old plantation susceptible during summer months.

Management

Plant disease free suckers. Remove infected plants and destroy. Drench with Methoxy ethyl mercuric chloride (Emisan-6) 0.1 / or Sodium hypohlorite 10% or Bleaching powder 20g /litre/tree.

Sigatoka disease : *Mycosphaerella musicola (Cercospora musae*)

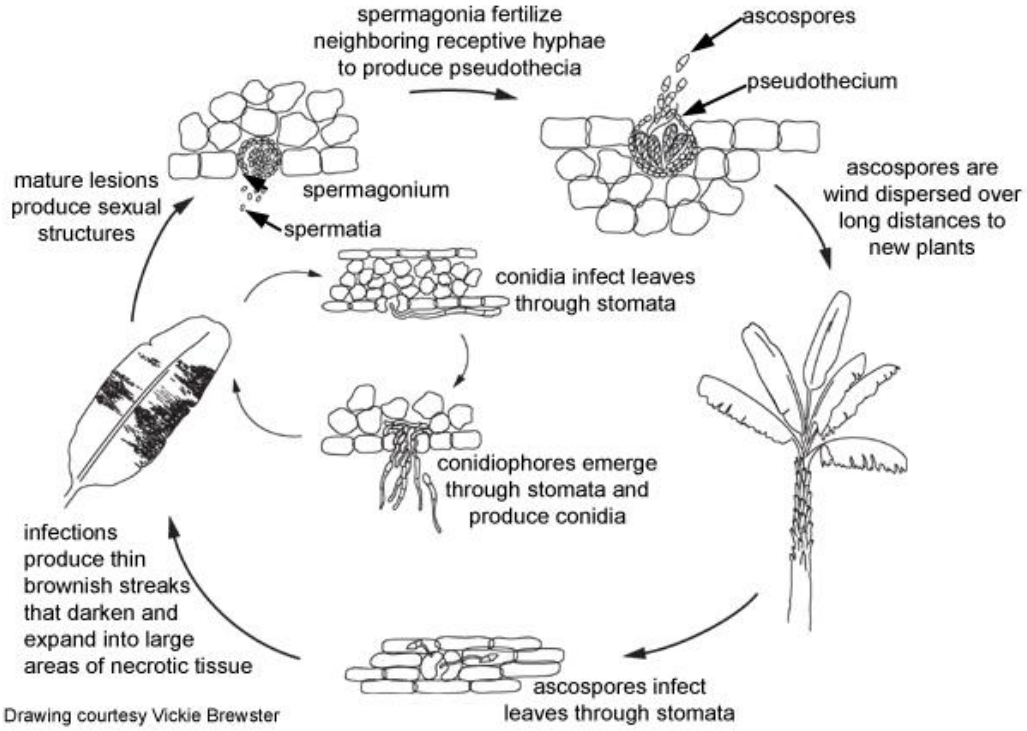
Symptoms

On leaves small light yellow or brownish green narrow streaks appear. They enlarge in size becomes linear, oblong, brown to black spots with dark brown brand and yellow halo. Black specks of fungal fruitification appear in the affected leaves. Rapid drying and defoliation of the leaves.

Pathogen

Conidia are elongated, narrow and multi septate and measure 20 – 80 x 2-6micron meter. Perithecia are dark brown to black and asci are oblong, clavate and measure 28.8- 36.8x8.0-10.8 micron meter. Ascospores are one septate, hyaline, obtuse with upper cell slightly broader.

Disease Cycle



Management

Removal and destruction of the affected leaves. Spray Propiconazole + Carbendazim 0.1% or Chlorothalonil 0.25%. Add wetting agent such as teepol or sandovit added at the rate of 1ml/lit of water.

Cigar end Rot *(Verticillium theobromae, Trachsphaera fructigena* and *Gloeosporium musarum)*

Symptoms

A black necrosis spread from the perianth into the tip of immature fingers**.** The rotted portion of the banana finger is dry and tends to adhere to fruits (appears similar to the ash of a cigar).

Pathogen

Conidiophores are usually solitary or in small groups. Conidia are hyaline, oblong to cylindrical. They are borne at the end of tapering phialides, aggregated into rounded, mucilaginous translucent heads.

Control:

Removal of pistil and perianth by hand 8-10 days after bunch formation and spraying the bunch with Dithane M -45 (0.1%) or Topsin M (0.1%) controls the disease effectively. Minimising bruising; prompt cooling to 14°C; proper sanitation of handling facilities reduce the incidence in the cold storage.

Anthracnose*:* *Gloeosporium gloeosporioides*

Symptoms:

The skin at the distal ends of the fingers turn black shrivels. The fungus produces masses of conidia which form a pinkish coat. The entire fruit and bunch is affected in severe cases. Sometimes main stalk of bunch diseased. The bunch becomes black and rotten. Acervuli produces cylindrical conidiophores, hyaline, septate, branched. Conidia hyaline, non-septate, oval to elliptical.

Pathogen

Acervuli are usually rounded or sometimes elongated, erumpent. Conidiophores are cylindrical, tapered towards the apex, hyaline and septate. Conidia are hyaline, aseptate, oval to ellipitical in shape.

Mode of spread and survival

The spread of the disease is by air borne conidia and numerous insects which frequently visit banana flowers also spread the disease.

Management

Post harvest dipping of fruits in Carbendazim 400 ppm, or Benomyl 1000 ppm, or Aureofunginsol 100 ppm.

Freckle or Black Spot: *Phyllostictina musarum*

Symptoms

Minute raised dark brown spots appear with black dots in the centre on leaves and fruits. On the fruits the pathogen is confined to the skin. The fungus produces pycnidium which are dark. conidiophores simple, short, elongate. Conidia are byline, single celled ovoid. Fungus survives in infected plant debris. Conidia spread by rain water and wind.

Pathogen

The fungus produces pycnidia and pycnidiospores. Pycnidiospores are needle shape, hyaline and multi septate.

Management

Spray Copper oxychloride 0.25%. Add wetting agent such as teepol or sandovit added at the rate of 1ml/lit of water.

Banana bunchy top: *Banana bunchy top virus*

Economic Importance

The disease is covered by domestic quarantine regulations. Losses were estimated to be Rs.4 crores every year and 100% loss occurs if infected suckers are planted.

Symptoms

Subsequent leaving show the same symptoms and are dwarfed. Dark broken bands of green tissues on the veins, leaves and petioles. Plants are extremely stunted. Leaves are reduced in size marginal chlorosis and curling. Leaves upright and become brittle. Many leaves are crowded at the top. Branches size will very small. If infected earlier no bunch will be produced. The disease is transmitted primarily by infected suckers.

Mode of spread

Secondary spread is through the aphid vector *Pentalonia nigronervosa*

Management

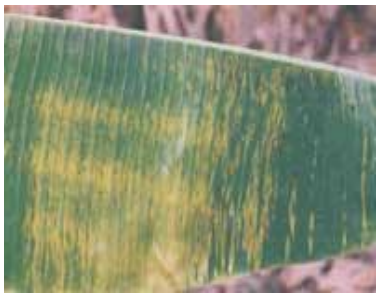
Select suckers from disease free areas. Control vector by spraying methyl demoton 1 ml/l.or Monocrotophos, 2 ml/l.or Phosphomidon 1 ml / lit. or Injection of Monocrotophos 1 ml / plant (1 ml diluted in 4 ml). Infected plants are destroyed using 4ml of 2, 4, D (50g in 400 ml of water).

Infectious chlorosis: *Cucumber mosaic virus*

Economic Importance

Infectious chlorosis or heart rot of banana is caused by Cucumber Mosaic Virus (CMV) has recently become serious, the disease has been recorded from 20 to 80 per cent in Poovan cultivar.

Symptoms



Chlorotic or yellow linear discontinuous streaks on leaves, upward curling of leaves, twisting and bunching of leaves at the crown, erectness of newly emerged leaves. Sometimes heart rot symptom also appear. Diseased plants are dwarf, do not produce bunches. The virus spreads through infected suckers and aphid vectors -*Aphis gossypii*

Management

Destroy infected plants. Use disease free suckers. Control vector by spraying systemic insecticide 0.1%

Lecture 04 - Diseases of Grapes

Downy mildew: *Plasmopara viticola*

Symptoms

Irregular, yellowish, translucent sports on the upper surface of the leaves. Correspondingly on the lower surface, white, powdery growth on leaves. Affected leaves become, yellow, brown and gets dried. Premature defoliation. Dwarfing of tender shoots. Brown, sunken lesions on the stem. White growth of fungus on berries which subsequently becomes leathery and shrivels. Later infection of berries result in soft rot symptoms. No cracking of the skin of the berries.

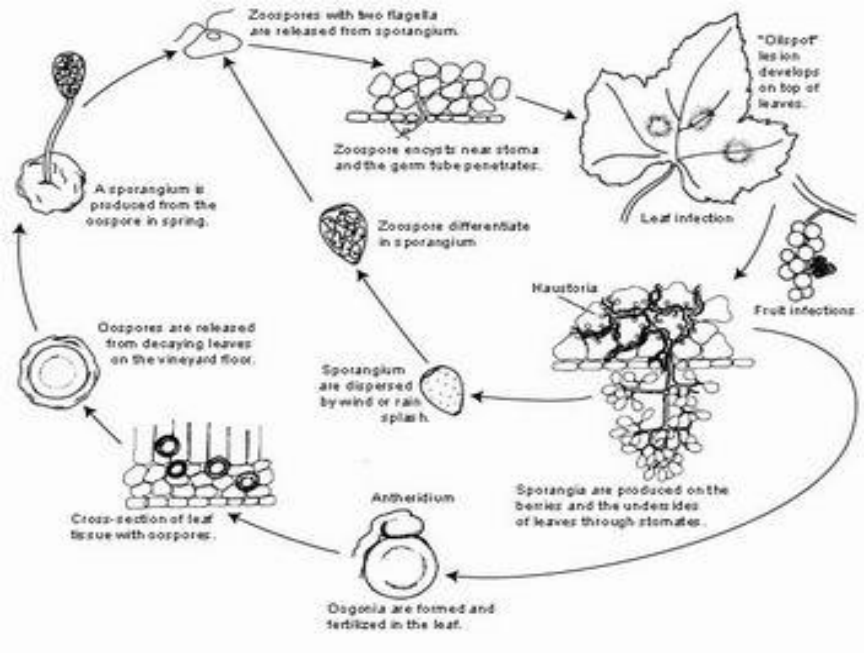
Pathogen

Mycelium is intercellular with spherical haustoria, coenocytic, thin walled and hyaline. Sporangiophores arise from hyphae in the sub stomatal spaces. It branched at right angle to the main axis and at regular intervals. Secondary branches arise from lower branches.The sporangia are thin walled, oval or lemon shaped. The Zoospores are pear shaped, biflagellate and 7 – 9 micron meter. The oospores are thick walled.

Mode of Spread and Survival

Through sporangia by wind, rain etc. As oospores present in the infected leaves, shoots and berries. Also as dormant mycelium in infected twigs. Optimum temperature: 20-22°C. Relative humidity: 80-100 per cent.

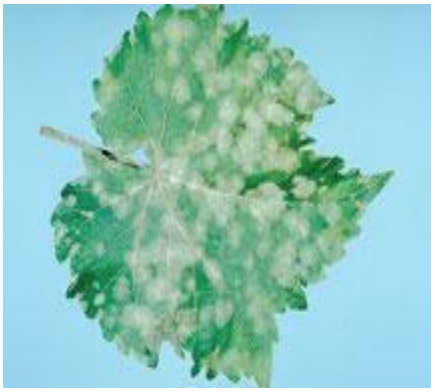
Disease Cycle



Management

Spray Bordeaux mixture 1 % or Metalaxyl + Mancozeb 0.4 %.

Powdery mildew : *Uncinula necator*



Symptoms

Powdery growth mostly on the upper surface of the leaves.Malformation and discolouration of affected leaves. Discolouration of stem to dark brown. Floral infection results in shedding of flowers and poor fruit set. Early berry infection results in shedding of affected berries. Powdery growth is visible on older berries and the infection results in the Cracking of skin of the berries.

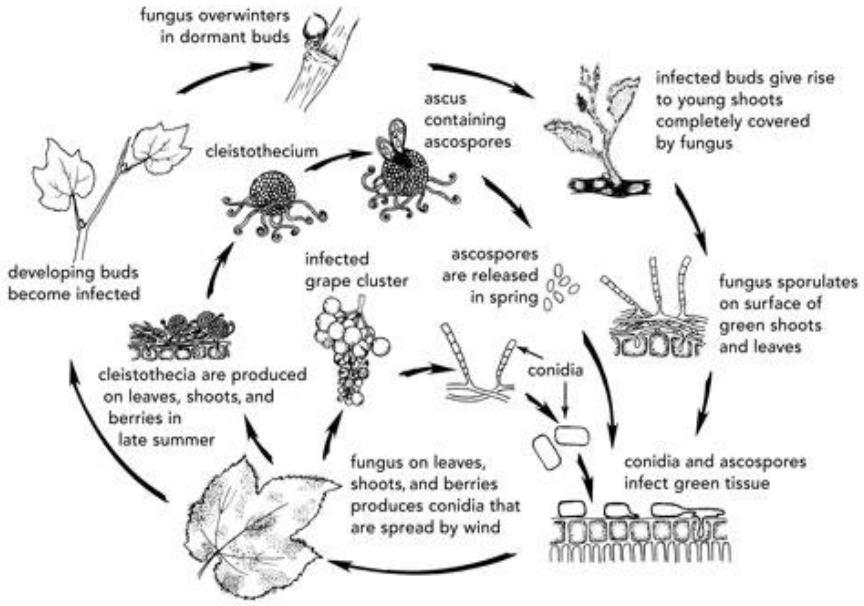
Pathogen

White growth consists of mycelium, conidiophores and conidia. Mycelium is external, septate and hyaline. Conidiophores are short and arise from external mycelium. Conidia are produced in chain. They are single celled, hyaline and barrel shaped. The fungus is oidium type.

Mode of Spread and Survival

It spread through air-borne conidia**.** Through dormont mycelium and conidia present in the infected shoots and buds. Sultry warm conditions with dull cloudy weather, highly favourable.

Disease Cycle



Management

Spray Inorganic sulphur 0.25 % or Chinomethionate 0.1 % or Dinocap 0.05 %.

Bird's Eye Spot/Anthracnose: *Gloeosporium ampelophagum* (*Elsinoe amphelina)*

Symptoms

The disease appears first as dark red spots on the berry. Later, these spots are circular, sunken, ashy-gray and in late stages these spots are surrounded by a dark margin which gives it the "bird's-eye rot" appearance. The spots vary in size from 1/4 inch in diameter to about half the fruit. The fungus also attacks shoots, tendrils, petioles, leaf veins, and fruit stems. Numerous spots sometimes occur on the young shoots. These spots may unite and girdle the stem, causing death of the tips. Spots on petioles and leaves cause them to curl or become distorted.

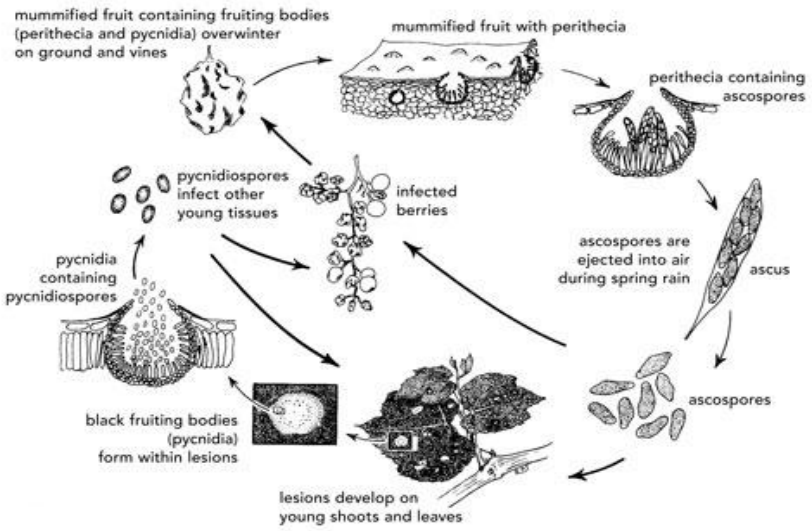
Pathogen

Mycelium is septate and dark colored. Conidia single celled oval and hyaline.

Mode of Spread and Survival

Seed-borne-infected vine, cuttings and air-borne conidia. As dormant mycelium in the infected stem-cankers. Warm wet weather. Low lying and badly drained soils.

Disease Cycle



Management

Removal of infected twigs.Copper oxychloride 0.2% or Mancozeb 0.25%

Lecture 05 - Diseases of Pomegranate and Papaya

Pomegranate

Cercospora fruit Spot: *Cercospora* sp.

Symptom

The affected fruits showed small irregular black spots, which later on coalesce, into big spots.

Management

The diseased fruits should be collected and destroyed. Two to three spray at 15 days interval with Mancozeb 0.25%.

Leaf Spot or Blight: *Colletotrichum gloesporioides; Pseudocercospora punicae; Curvularia lunata* and *Cercospora punicae*)

Symptom

The disease is characterized by appearance of small, irregular and water-soaked spots on leaves.Affected leaves fall off.

Pathogen

Conidiophores are olivaceous brown, short, fasciculate, sparingly septate. Conidia are hyaline to pale olivalceous cylindric and septate.

Mode of spread and survival

The pathogen spread through wind borne conidia.

Management

Spraying Mancozeb 0.25 % at 15 days interval gives good control of the disease.

Alternaria fruit spot: *Alternaria alternata*

Symptom

Small reddish brown circular spots appear on the fruits. As the disease advances these spots, coalesce to form larger patches and the fruits start rotting. The arils get affected which become pale and become unfit for consumption.

Management

All the affected fruits should be collected and destroyed. Spraying Mancozeb 0.25 % effectively controls the disease.

Fruit Rot (*Aspergillus foetidus):*

The symptoms are in the form of round black spots on the fruit and petiole. The disease starts from calyx end and gradually the entire fruit shows black spots. The fruit further rots emitting a foul odour.

Management

The disease can be controlled by spraying of Bavistin (0.5%), Dithane M-45 (0.25%) or Dithane Z-78 (0.25%) at an interval of 10-15 days from the onset of flowering.

Papaya

Stem rot / Foot rot – *Pythium aphanidermatum*

Symptoms

Water soaked spot in the stem at the ground level which enlarge and griddle the stem. The diseased area turns brown or black and rot. Terminal leaves turn yellow droop off. The entire plant topples over and dies. Forward by rain. *R. solani* is favoured by dry and hit weather. Common in 2-3 year old trees.

Pathogen

Mycelium is septate, brown and much branched. The sclerotia are black, spherical to irregular shape and produced in abundance.

Management

Seed treatment with Thiram or Captan 4 g/kg or Chlorothalonil.

Drenching with Copper oxychloride 0.25 % or Bordeaux mixture 1% or Metalaxyl 0.1%.

Powdery mildew – *Oidium caricae*

Symptoms

While mycelia growth appear on the upper surface of the leaf, flower stalks and fruit. Seven attak causes yellowing and defiation of leaves.

Pathogen

It is an obligate parasite. The mycelium is hyaline, septate and haustoria develop in epidermal cells. Conidia are hyaline.

Mode of spread and survival

The pathogen spread through wind borne conidia.

Management

Spray Wettable Sulphur 0.25% or Dinocap 0.05% or Chinomethionate 0.1% or Tridemorph 0.1%.

Papaya ring spot – Papaya ring spot virus

Symptoms

Vein clearing, puckering and chlorophyll leaf tissues lobbing in. Margin and distal parts of leaves roll downward and inwards, mosaic mottling, dark green blisters, leaf distortion which result in shoe string system and stunting of plants. On fruits circular concentric rings are produced. If affected earlier no fruit formation.

Pathogen

The virus particles are rod shaped and thermal inactivation point of the virus lies between 54 and 60˚C.

Mode of spread

Vectored by aphids *Aphis gossypii, A. craccivora* and also spreads to cucurbits not through seeds.

Management

Raise papaya seedlings under insect-proof conditions. Plant disease free seedlings. Raise sorghum / maize as barrier crop before planting papaya. Rogue out affected plants immediately on noticing symptoms. Do not raise cucurbits around the field.

Leaf curl – *Papaya leaf curl virus*

Symptoms

Curling, crinkling and distortion of leaves, reduction of leaf lamina, rolling of leaf margins inward and downward, thickening of veins. Leaves become leathery, brittle and distorted. Plants stunted. Affected plants does not produce flowers and fruits.

Mode of spread

Spread by whitefly *Bemisia tabaci***.**

Management

Uproot affected plants. Avoid growing tomato, tobacco near papaya. Spraying with systemic insecticides to control the vector.

Anthracnose – *Colletotrichum gloeosporioides*

Symptom

If affect leaf and stem on erotic spots are produced. On fruit initially brown superficial discoloration of the skin develops which are circular and slightly sunken. Then they coalesce in which sparse mycelial growth appear on the margins of a spot. Under humid condition salmon pink spores are released. Fruits mummified and deformed.

Mode of spread

Infection is caused by fruit from field. Secondary spread by conidia by rain splashes

Management

Spray with Carbendazim 0.1% (or) Chlorothalonil 0.2% or Mancozeb 0.2%.

Lecture 06 - Disease of Guava

Powdery mildew – *Oidium caricae*

Symptoms

While mycelia growth appear on the upper surface of the leaf, flower stalks and fruit. Seven attak causes yellowing and defiation of leaves.

Pathogen

It is an obligate parasite. The mycelium is hyaline, septate and haustoria develop in epidermal cells. Conidia are hyaline.

Mode of spread and survival

The pathogen spread through wind borne conidia.

Management

Spray Wettable Sulphur 0.25% or Dinocap 0.05% or Chinomethionate 0.1% or Tridemorph 0.1%.

Papaya ring spot – Papaya ring spot virus

Symptoms

Vein clearing, puckering and chlorophyll leaf tissues lobbing in. Margin and distal parts of leaves roll downward and inwards, mosaic mottling, dark green blisters, leaf distortion which result in shoe string system and stunting of plants. On fruits circular concentric rings are produced. If affected earlier no fruit formation.

Pathogen

The virus particles are rod shaped and thermal inactivation point of the virus lies between 54 and 60˚C.

Mode of spread

Vectored by aphids *Aphis gossypii, A. craccivora* and also spreads to cucurbits not through seeds.

Management

Raise papaya seedlings under insect-proof conditions. Plant disease free seedlings. Raise sorghum / maize as barrier crop before planting papaya. Rogue out affected plants immediately on noticing symptoms. Do not raise cucurbits around the field.

Leaf curl – *Papaya leaf curl virus*

Symptoms

Curling, crinkling and distortion of leaves, reduction of leaf lamina, rolling of leaf margins inward and downward, thickening of veins. Leaves become leathery, brittle and distorted. Plants stunted. Affected plants does not produce flowers and fruits.

Mode of spread

Spread by whitefly *Bemisia tabaci***.**

Management

Uproot affected plants. Avoid growing tomato, tobacco near papaya. Spraying with systemic insecticides to control the vector.

Anthracnose – *Colletotrichum gloeosporioides*

Symptom

If affect leaf and stem on erotic spots are produced. On fruit initially brown superficial discoloration of the skin develops which are circular and slightly sunken. Then they coalesce in which sparse mycelial growth appear on the margins of a spot. Under humid condition salmon pink spores are released. Fruits mummified and deformed.

Mode of spread

Infection is caused by fruit from field. Secondary spread by conidia by rain splashes

Management

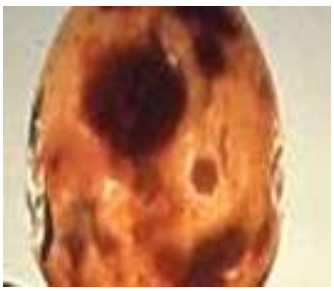
Spray with Carbendazim 0.1% (or) Chlorothalonil 0.2% or Mancozeb 0.2%.

Lecture 06 - Disease of Guava and Sapota

Guava

Anthracnose: *Colletotrichum gloeosporioides*

Symptoms



Symptoms of this disease are observed on mature fruits on the tree. The characteristic symptoms consist of sunken, dark colored, necrotic lesions. Under humid conditions, the necrotic lesions become covered with pinkish spore masses. As the disease progresses, the small sunken lesions coalesce to form large necrotic patches affecting the flesh of the fruit.

Pathogen

Conidia are hyaline, aseptate, oval to elliptical condiophore is cylindrical. Acervulli are dark brown to black.

Mode of Spread and Survival

The conidia are spread by wind or rain.

Management

Spray Mancozeb 0.25%.

Guava rust: *Puccinia psidii*

Symptoms

The pathogen can affect foliage, young shoots, inflorescences and fruit of guava. Typical symptoms associated with this disease include distortion, defoliation, reduced growth and if severe, mortality. On fully expanded leaves, dark bordered, roughly circular brown lesions with yellow halos develop.

Management

Control of guava rust is based on the use of fungicides. Scouting fields for onset of disease or during the times of year when environmental conditions are favorable for pathogen infection are recommended so that proper and timely fungicide applications can

Lecture 07 - Disease of Apple (2 Lectures)

Scab –*Venturia inaequalis*

Symptoms

Symptom appears on leaves and fruits. On lower side of the leaf lesion appear as olivaceous spots which turn dark brown to black and become velvety. On young foliage, the spots have a radiating appearance with a feathery edge. On older leaves the lesions are more definite in outline. The lesion may form a convex surface with corresponding concave area on the opposite side. In severe infection leaf blade curved, dwarfed and distorted. Fruits show small, rough, black circular lesions. The centre of the spots become corky and on mature fruits, yellow halo is seen around the lesions.

Pathogen

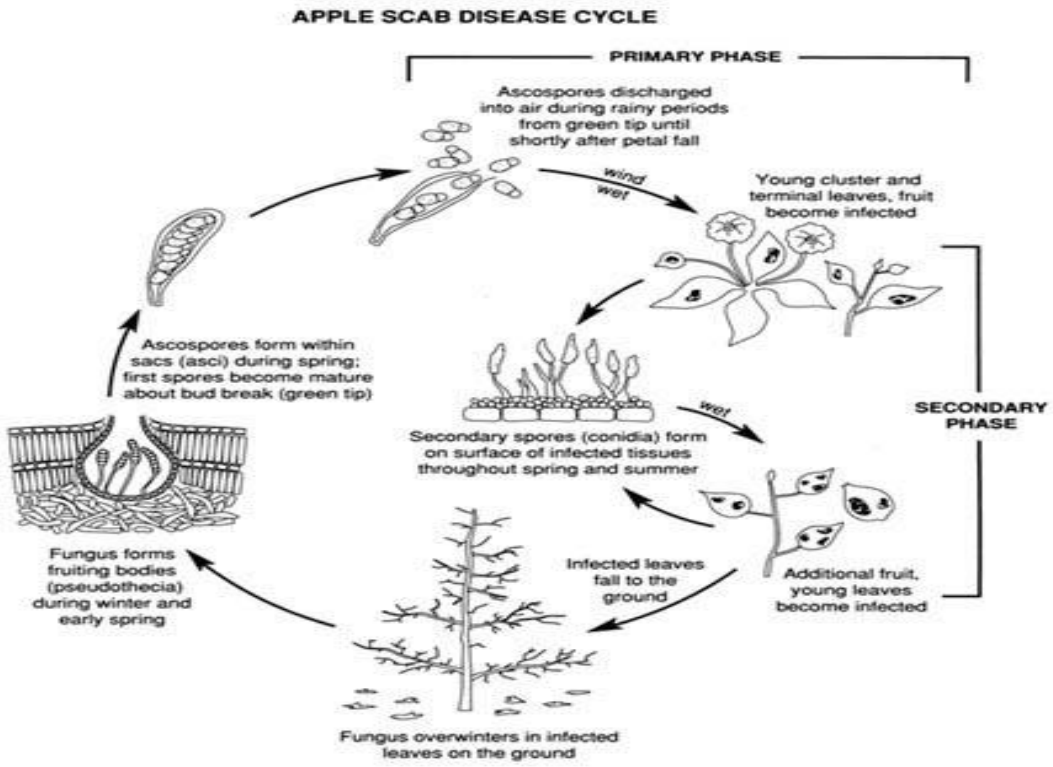
The mycelium is internal. Ascospores are two celled, greenish, grey or yellowish in color.

Mode of Spread and Survival

Pseudothecia formed in autumn and winter mature in spring to produce ascospores, the chief inoculum for primary infection. The secondary spread is through conidia.

Disease cycle

This disease, caused by the fungus *Venturia inaequalis* (anamorph *Spilocaea pomi*), may be quite severe when rainy, cool weather occurs in the spring. Fungal spores are produced in early spring on dead, fallen apple leaves about the time buds begin to develop. These spores are splashed by rain and blown by wind to land on developing plant tissue and initiate infections. After spots appear on the newly formed leaves, more spores are produced that spread infection to other parts of the tree. Again, rainy weather greatly encourages spore spread and infection during the secondary phase of spore production. The fungus over winters on fallen leaves.



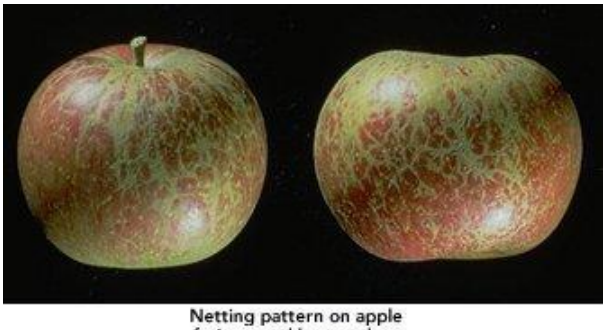
Clean cultivation, collection and destruction of fallen leaves and pruned materials in winter to prevent the sexual cycle. Spray Tridemorph 0.1% before flowering. Spray Mancozeb 0.25 % at bearing stage. Spray 5 % urea prior to leaf fall in autumn and 2 % before bud break to hasten the decomposition of leaves.

S.No	Tree stage	Fungicide/100lit
1	Silver tip to given tip	Captafol 200 gm (or) Captan 300 g or Mancozeb 400 g
2	Pink bud or 15 days after 1 st spray	Captan 250 g or Mancozeb 300 g
3	Petal fall	Carbendazim 50 g
4	10 days later	Captan 200 g.or Mancozeb 300g
5	14 days after fruit set	Captofol 150 g

Add stickers – teepol or triton 6 ml/10 lit of spray fluid.

Powdery mildew – *Podosphaera leucotricha*.

Symptom



Netting pattern on apple fruit caused by powdery mildew infection.

Malformation of apple leaves caused by powdery mildew.

Powdery mildew may be found on buds, blossoms, leaves, twigs, and fruit. In spring, infected flower buds open 5-8 days later than healthy buds. The buds are killed or distorted. Symptoms first appear in the spring on the lower surface of leaves, usually at the ends of branches. Small, whitish felt-like patches of fungal growth appear and quickly cover the entire leaf. Diseased leaves become narrow, crinkled, stunted and brittle, which results in their drying out and fall. The fungus spreads rapidly to twigs, which stop growing and become stunted. In some cases the twigs may be killed back. Leaves and blossoms from infected buds will be diseased when they open the next spring. Infected blossoms shrivel and produce no fruit. Fruit symptoms are not usually seen unless the disease has built up to high levels on susceptible cultivars. The fruit surface may become russetted or discolored, and dwarfed. Heavily mildewed trees are weakened, and are more susceptible to other pests and winter injury. It is the only fungal apple disease that is capable of infecting without wetting from rain or dew. In nurseries the fungus may spread to all developing leaves and cause stunting of vegetative terminal growth.

Pathogen

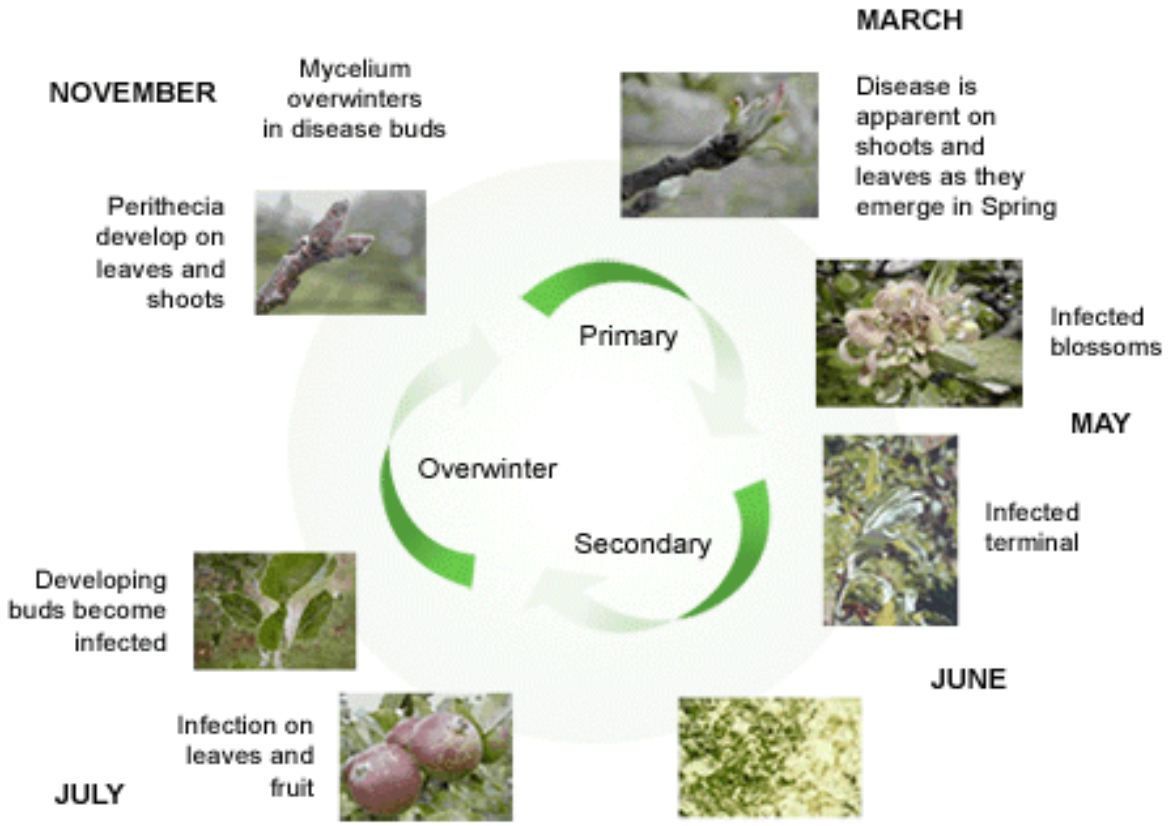
Powdery mildew is caused by, *Podosphaera leucotricha*, an ascomycetous heterothallic fungus. Conidia are ellipsoidal, truncate and hyaline. Perithecia are subglobose, are densely gregarious, and rarely scattered, and have apical and basal appendages. The asci in the perithecia are oblong to subglobose. Eight ascospores are present in the ascus. The fungus over winters as fungal strands (mycelium) in vegetative or fruit buds which were infected the previous season.

Mode of Spread and Survival

The fungus overwinters in the form of mycelium in diseased vegetative buds and fruits. Secondary spread is through wind borne conidia.

Disease Cycle

The mildew fungus over winters mainly as mycelium in dormant blossom and shoot buds produced and infected the previous growing season. Conidia are produced and released from the unfolding leaves as they emerge from infected buds at about tight cluster stage. Conidia germinate in the high relative humidity usually available on the leaf surface at 10-25˚C with an optimum of 19-22˚C. Germination does not occur in free moisture. Early-season mildew development is affected more by temperature than by relative humidity. Abundant sporulation from over wintering shoots and secondary lesions on young foliage leads to a rapid buildup of inoculum. Secondary infection cycles may continue until susceptible tissue is no longer available. Since leaves are most susceptible soon after emergence, infection of new leaves may occur as long as shoot growth continues. Fruit infection occurs from pink to bloom. Over wintering buds are infected soon after bud initiation. Heavily infected shoots and buds are low in vigor and lack winter hardiness, resulting in a reduction of primary inoculum at temperatures below -24˚C.



Management:

Spray Dinocap 0.05% or Chinomethionate 0.1%

Fire blight- *Erwinia amylovora*

Symptom

The initial symptom usually occurs on leaves, which become water soaked, then shrivel turn brownish to black in colour and fall or remain hanging in tree. The symptom spread to twigs. Terminal twigs wilt from tip to downward and also spread to branches.Fruits becomes water soaked, turns brown, shrivels and finally becomes black. Oozing may be seen in the affected area.

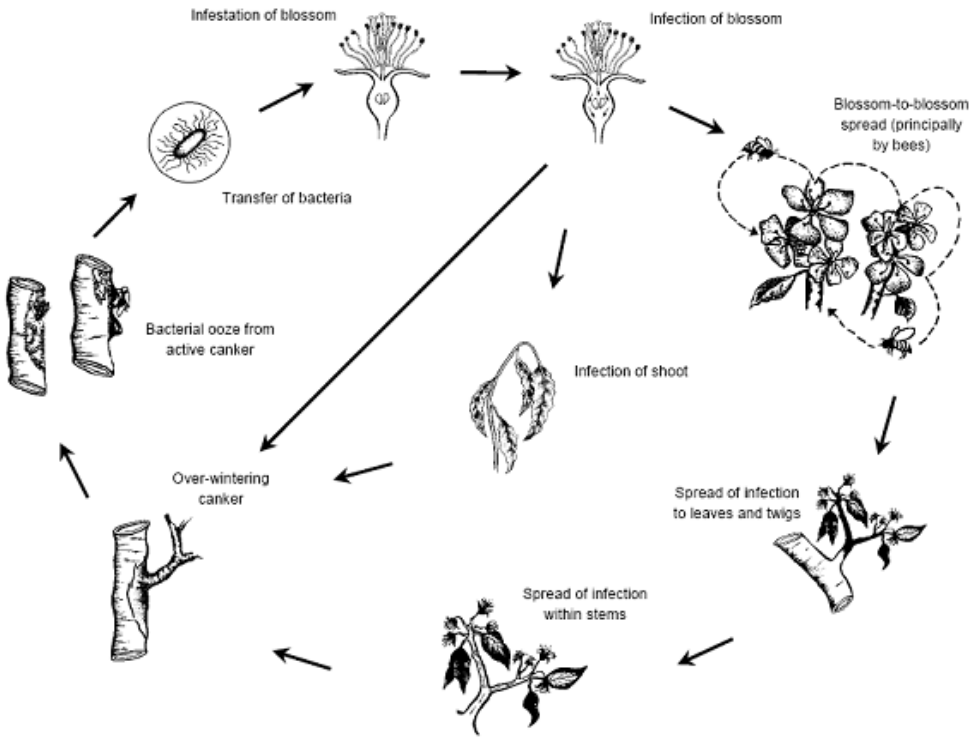
Pathogen

The bacterium is rod shaped and motile by peritrichous flagella. Bacterium occurs usually singly but pairs or chais of 3 of 4 bacteria also exist. Each bacterial cell is enclosed in a capsule.

Mode of Spread and Survival

The bacterium overwinters at the margin of cankers formed during previous season. They survive most often in large branches and seldom in twigs less than 1 cm in dia. Flies, Wasps and honey bees and rain splashes spread the bacteria into freshly wounded tissues inside the leaf. Young tender twigs are infected by bacteria through their lenticels, through wounds made by various agents and through insects carrying bacteria and feeding on the twigs.

Disease Cycle

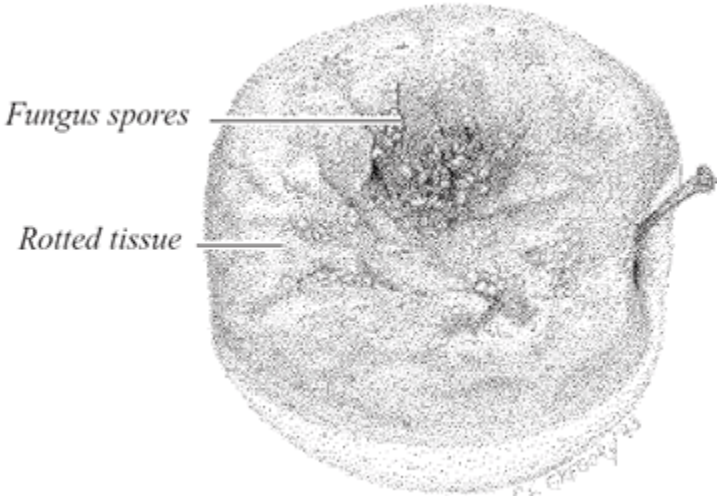


Management

Removal and destruction of affected parts. Removal of blighted twigs. Spray with Streptomycin 500 ppm.

Soft rot – *Penicillium expansum*

Symptom



Young spots starts from stem end of the fruit as light brown watery rot. As the fruit ripens area of the rotting increases, skin becomes wrinkled. A peculiar musty odour is emitted

under humid condition a bluish green sporulating growth appears. Infection take place by wounds in the skin caused by insects and during handing in storage and transport.

Pathogen

Conidiophores give rise to 1-3 main branches. They in turn produce crowdwd whorls of branchlets. Conidia are formed in chains. Conidia are green or bluish green in mass**.**

Mode of Spread and Survival

The fungal spores are spread by air. Mycelium can infect through bruisedor wounded fruits in close contact. Infection of the fruit usually take place through wounds in the skin, such as wounds caused by insect bites, careless picking, rough handling during washing, grading , packing, transit and storage. Infection is also through lenticels.

Disease cycle

Spores of the soft rot fungus are present almost everywhere and can survive long periods of unfavorable conditions. Bulk bins, field crates, pack house lines, and storage rooms are usually contaminated. Injuries to fruit, especially during picking and handling operations, are the primary points of entry. At ordinary temperatures, infected fruit can rot in 2 weeks or less.

Management

Careful handling of fruits without causing any wounds. Dipping the fruits aureofunginsol @ 500 ppm for 20 min gives best control.

Bitter rot – *Glomerella cingulata*

Symptom

Faint, light brown discolouration beneath the skin develops. The discolouration expands in a cone shape. The circular, rough lesions become depressed. The lesions increased and covers entire areas of fruits. Diny black dots appear beneath the cuticle which gives rise to acervuli . Pink masses of spores are found arranged in defined rings.

Disease Cycle

The fungus over winters in mummified fruit, in cracks and crevices in bark, and in cankers produced by the bitter rot fungus or by other diseases, such as fire blight. Jagged edges of broken limbs are also ideal sites. The bitter rot fungus is one of the few rot organisms that can penetrate unbroken skin of fruit. Although penetration is direct, wounds can be colonized rapidly by the fungus. Spores are waterborne and are released during rainfall throughout the growing season. Fruit infection can occur early but is more common from mid to late season. Often, the first infections appear in cone-shaped areas within the tree beneath mummies or a canker. Factors which determine the time of appearance of bitter rot are the maturity of fruit, temperature and humidity, and the presence of disease in the area. The optimum conditions for disease development include rainfall, relative humidity of 80 to 100 percent, and warm temperatures. Infection can occur in as little as five hours at 26˚C.

Management

Spray Mancozeb 0.25 % in field. Treatment with Mancozeb 0.25 % to check the disease in storage.

Lecture 08 - Diseases of Chilli

Damping off: *Pythium aphanidermatum*

Symptoms:

Seedlings killed before emergence. Water soaking and shrivelling of stem. Factors favouring infection:Moist soils poordrainage 90-100% R.H soil temperature 20°C.

Pathogen

Mycelium is hyaline, coenocytic and zoosporangia are lobed and branched. Zoospores are biflagellate and oogonia are spherical with smooth walled.Antheridia are monoclinous, intercalary or terminal. Oospores are aplerotic, single with thick wall.

Mode of spread and survival

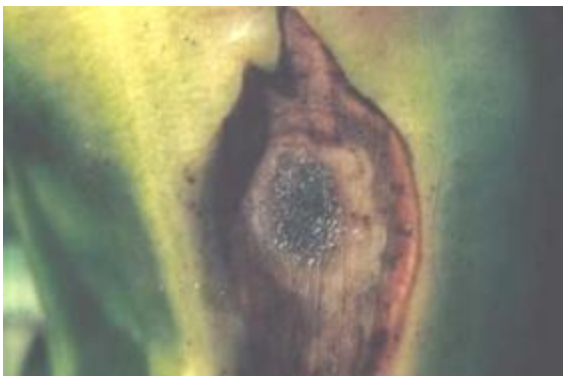
The pathogen is soil borne. Zoospores spread through irrigation water. The disease spreads to main field by planting infected seedlings.

Management

Soil drenching with Copper oxychloride 0.25%

Fruit Rot and Die Back- *Colletotrichum capsici*

Symptoms:



As the fungus causes necrosis of tender twigs from the tip backwards the disease is called die-back Infection usually begins when the crop is in flower. Flowers drop and dry up. There is profuse shedding of flowers. The flower stalk shrivel and dry up. This drying up spreads from the flower stalks to the stem and subsequently causes die-back of the branches and stem and the branches wither. Partia1lly affected plants bear fruits which are few and of low quality. On the surface of the soil the necrotic areas are found separated from the healthy area by a dark brown to black band.

Pathogen

The mycelium is septate and inter and intra cellular. Conidia in mass appear pinkish. They are borne singly at the tip of conidiophores.

Mode of spread and survival

The fungus is seed borne and the secondary infection is by air borne conidia ans also by rain. The disease spreads rapidly by wind blown rains during rainy season. Flies and other insects are found responsible for dissemination of the spores from one fruit to another. The fungus may not survive long in soil, but may survive on the dead twigs stored under dry conditions. Seeds from badly diseased fruits may also carry the primary inoculum.

Management

Use of disease-free seeds is important in preventing the disease. Seed treatment with Thiram or Captan 4g/kg is found to be -effective in eliminating the seed-borne inoculum. Good control of the disease has been reported by three sprayings with Ziram O. 25% Captan 0.2% or miltox 0.2%. Chemicals like wettable sulphur 0.2%, copper oxychloride 0.25% and Zineb 0.15% not only reduced the disease incidence but also increased the yield of fruits. The first spraying should be given just before flowering and the second at the time of fruit formation. Third spraying may be given a fortnight after second spraying.

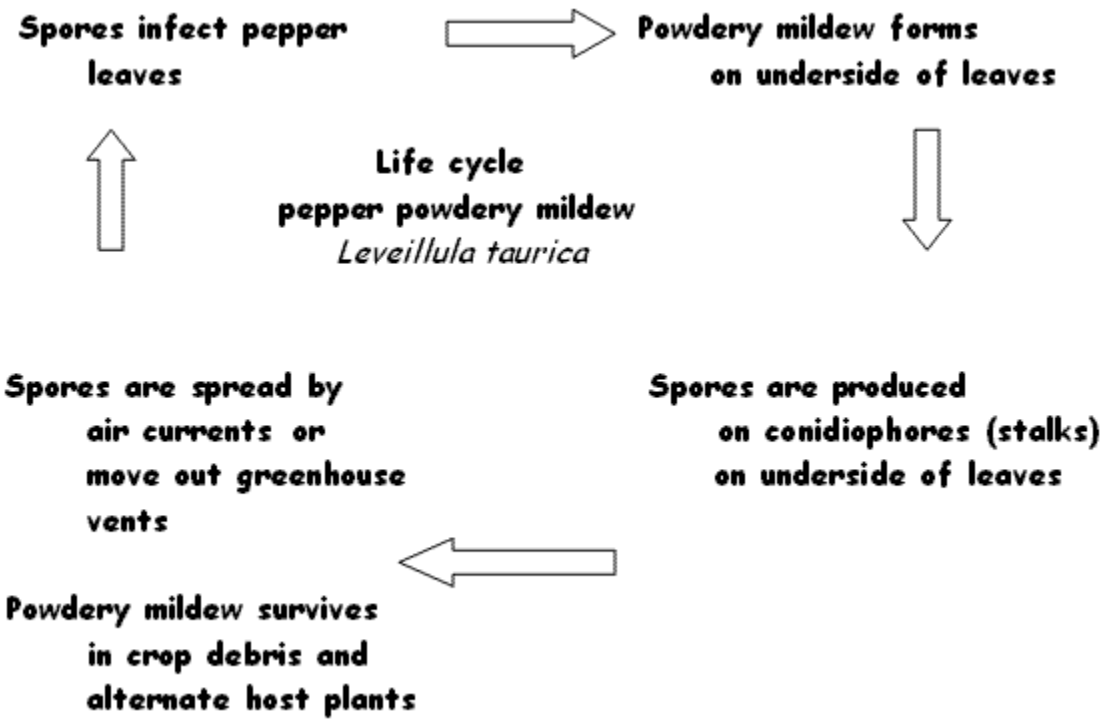
Powdery mildew: *Leveillula taurica*

Symptoms

Shedding of foliage. White powdery growth on lower side of leaves.

Disease cycle

The powdery mildew disease cycle (life cycle) starts when spores (known as conidia) land on a chilli leaf. Spores germinate much like a seed and begin to grow into the leaf. chilli powdery mildew parasitizes the plant using it as a food source. The fungus initially grows unseen within the leaf for a latency period of 18-21 days. Then the fungus grows out of the breathing pores (stomates) on the under surface of the leaf, producing spores which are borne singly on numerous, fine strands or stalks (conidiophores). These fungal strands become visible as white patches or mildew colonies on the under side of the leaf. Repeated cycles of powdery mildew can lead to severe outbreaks of powdery mildew that economically damage the crop.



Management

Spray Wettable sulphur 0.25% or Dinocap (Karathane) 0.05%

Bacterial leaf spot: *Xanthomonas campestris pv. vesicatoria*

Symptoms

The leaves exhibit small circular or irregular, dark brown or black greasy spots. As the spots enlarge in size, the centre becomes lighter Surrounded by a dark band of tissue. The spot coalesce to form irregular lesions. Severely affected leaves become chlorotic and fall off. Petioles and stems are also affected. Stem infection leads to formation of cankerous growth and wilting of branches. On the fruits round, raised water soaked spots with a pale yellow border and produced. The spots turn brown developing a depression in the centre wherein shining droplets of Bacterial cozen may be observed.

Mode of spread and survival

The disease is primarily seed borne. It spreads in the nursery and is further disseminated with infected transplants. Spattering rains are the chief means of dissemination. The bacterium subsists in infected debris.

Management

Seed treatment with 0.1% mercuric chloride solution for 2 to 5 minutes is effective. Seedlings may be sprayed with Bordeaux mixture 1. Per cent or copper oxychloride 0.25%. Spraying with streptomycin should not be done after fruits begin to form. Field sanitation is important. Also seeds must be obtained from disease free plants.

Cercospora leaf spot :*Cercospora capsici*

Symptoms

Leaf lesions typically are brown and circular with small to large light grey centers and dark brown margins. The lesions may enlarge to 1cm or more in diameter and some times coalesce. Stem, petiole and pod lesions also have light grey centers with dark borders, but they are typically elliptical. Severely infected leaves drop off prematurely resulting in reduced yield.

Pathogen

Stromata are well developed. Conidiophores are 30- 60 x 4.5 – 5.5 micron meter. Conidia are subhyaline to coloured, acicular to obculate.

Mode of spread and survival

Primary source of infection are infected seeds, volunteer plants and infected plant debris. Secondary spread is through air borne conidia.

Management

Spray twice at 10-15 days interval with Mancozeb 0.25% or Chlorothalonil (Kavach) 0.1%.

Fusarium wilt :*Fusarium oxysporum f.sp.capsici*

Symptoms

Fusarium wilt is characterised by wilting of the plant and upward and inward rolling of the leaves. The leaves turn yellow and die. Generally appear localised areas of the field where a high percentage of the plants wilt and die, although scattered wilted plants may also occur. Disease symptoms are characterised by an initial slight yellowing of the foliage and wilting of the upper leaves that progress in a few days into a permanent wilt with the leaves still attached. By the time above - ground symptoms are evident, the vascular system of the plant is discoloured, particularly in the lower stem and roots.

Pathogen

Mycelium is grayish white. Microconidia are formed singly, hyaline and cylindrical. Macro conidia are cylindrical to falcate. Chlamydospores are globose to oval and rough walled.

Management

Use of wilt resistant varieties. Drenching with 1% Bordeaux mixture or Blue copper or Fytolan 0.25% may give protection.· Seed treatment with 4g Trichoderma viride formulation or 2g Carbendazim per kg seed is effective. Mix 2kg T.viride formulation mixed with 50kg FYM,

sprinkle water and cover with a thin polythene sheet. When mycelia growth is visible on the heap after 15 days, apply the mixture in rows of chilli in an area of one acre.

Leaf curl

Leaves curl towards midrib and become deformed. Stunted plant growth due to shortened internodes and leaves greatly reduced in size. Flower buds abcise before attaining full size and anthers do not contain pollen grains. The virus is generally transmitted by whitefly. So control measures of whitefly in this regard would be helpful.

Mosaic Viruses

Light green and dark green patches on the leaves. Stunted plant growth during early stages. Yellowing, chlorotic ring spots on leaves and fruits.

Management of viral diseases

Control measures are not known for majority of viral diseases. Hence, mechanical, cultural methods are mostly recommended. The infected plants should be uprooted and burnt or buried to avoid further infection. Avoid monoculture of chilli crop. Selection of healthy and disease - free seed. Suitable insecticidal sprays reduce the incidence of viral diseases, since majority of viral diseases are transmitted by insect vectors. Soaking seeds in a solution containing 150 g Trisodium orthriphosphate per litre of water for 30 minutes inhibits seed - borne inoculum.

Treated seed should be washed with fresh water and dried before sowing. Nursery beds should be covered with nylon net or straw to protect the seedlings from viral infection. Raise 2-3 rows of maize or sorghum as border crop to restrict the spread of aphid vectors. Apply Carbofuran 3G @ 4-5 Kg/acre in the mainfield to control sucking complex and insect vectors selectively. If it is not possible spray the crop with systemic insecticides. Like Monocrotophos 1.5 ml or Dimethoate 2ml of Acephate 1g per litre of water. Collect and destroy infected virus plants as soon as they are noticed.

Bacterial soft rot -*Erwinia carotovora* subsp. *Carotovora*

Symptoms

The fleshy fruit peduncle is highly susceptible and is frequently the initial point of infection. Both ripe and green fruit may be affected. Initially, the lesions on the fruit are light to dark-colored, water-soaked, and somewhat sunken. The affected areas expand very rapidly, particularly under high temperatures, and tissues lose their texture. In later stages, bacterial ooze may develop from affected areas, and secondary organisms follow, often invading the rotted tissue. Post-harvest softening of stem end of fruit. The affected fruit hang from the plant like a water-filled bag.

Conditions for Disease Development

The bacteria may persist in fields where peppers are rotated with other susceptible crops such as cabbage and potato. The bacteria may be present as a contaminant on the surface of pepper seed. The bacteria can be transmitted by drainage water, irrigation water, or by sprinkler irrigation, but a wound is necessary for infection to occur. Wounding often arises from rough handling of plants during weeding, or due to a strong wind, or from insect feeding. European and Asiatic corn borers may introduce bacteria into the fruit peduncle of pepper during feeding. A high rate of nitrogen fertilization is associated wit increased susceptibility to soft rot. Warm, moist weather is also highly favorable for infection.

Management

Use chlorinated wash water to reduce populations of soft rot bacteria and to reduce the risk of infection during washing. This will not reduce soft rot development in fruit infected with the bacterium prior to harvest. Allow fruit to dry thoroughly. During packing and storage, the fruit should be kept clean and maintained in a cool, dry place

Alternaria Rot- *Alternaria sp.*

Symptoms

The fungus is reported to enter wounds (sunscald or punctures). Dusty black spores on fruit spots are characteristic. In most instances this disease follows blossom-end rot, but it also follows injuries, chilling, and other decays. On the fruit, large greenish-brown to brown lesions covered, with grayish-brown mold are produced. Similar lesions on the lower-part of the fruit are characteristic of *Alternaria* rot following blossom-end rot. The larger lesions may show

alternating light and dark-brown concentric zones. Shipping peppers under standard refrigeration will check the development of this rot, but when the fruit is removed from refrigeration the decay will advance rapidly at moderate to warm temperatures.

Pathogen

Hyphae are septate, branched, light brown becoming darker with age and inter and intra cellular. Conidiophores emerge through stomata. Conidia are single and muriform.

Mode of spread and survival

Infected seeds, volunteer plants and infected plant debris are primary source of infection.

Management

Pre storage dry heat

The effectiveness of a prestorage dry heat treatment and hot water dip in reducing storage rots of capsicum caused by *Alternaria alternata*. Treatment with hot air at 38˚C for 48-72 h or hot water at 50˚C to 53˚C for 2 to 3 min, resulted in reduction in the pathogenicity and development of these pathogens in inoculate peppers.

Lecture 09 - Diseases of Brinjal

Bacterial Wilt: *Pseudomonas solanacearum*

Symptoms

Bacterial wilt symptoms on leaf surface Wilting, stunting, yellowing of the foliage and finally collapse of the entire plant are the characteristic symptoms of the disease. Lower leaves may droop first before wilting occurs. The vascular system becomes brown. Bacterial ooze comes out from the affected parts. Plant show wilting symptoms at noontime will recover at nights, but die soon.

Pathogen

The bacterium is non acid fast, non spore forming, non capsulated and motile by a polar flagellum. The bacterium produces acid but no gas in dextrose, sucrose, lactose and glycerol. Starch hydrolyzed with slight liquefaction of gelatin.

Mode of spread and survival

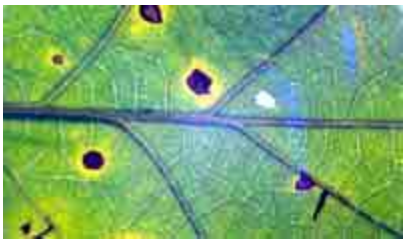
The bacterium infects banana, chillies, fennel, ginger, potato, radish, tomato etc., the bacterium though a non spore former is found to be alive and viable for more than 16 months under laboratory conditions. The pathogen is found to be alive in the infected plant debris for about 10 months. Presence of root knot nematode, *Meloidogyne javanica* increases the wilt incidence.

Management

Use resistant variety .Crop rotation with cruciferous vegetables such as cauliflower help in reducing the disease incidence. Fields should be kept clean and effected parts are to be collected and burnt. Spray Copper fungicides to control the disease (2% Bordeaux mixture.). The disease is more prevalent in the presence of root knot Nematodes, so control of these nematodes will suppress the disease spread.

Cercospora Leaf Spot :*Cercospora solani -melongenae, C. solani*

Symptoms



The leaf spots are characterized by chlorotic lesions, angular to irregular in shape, later turn grayish-brown with profuse sporulation at the centre of the spot. Severely infected leaves drop off prematurely, resulting in reduced fruit yield.

Pathogen

The fungus produces stromata which are globular. Conidiophores in mass are medium dark and slightly olivaceous brown in colour and paler towards the tip. Conidia are sub hyaline to pale olivaceous.

Mode of spread and survival

The disease is spread by air borne conidia.

Management

Pant Samrat variety is resistant to both the leaf spots. Diseases can be managed by growing resistant varieties. Spraying 1 per cent Bordeaux mixture or 2 g Copper oxychloride or 2.5 g Zineb per litre of water effectively controls leaf spots.

Alternaria leaf Spot :*Alternaria melongenae, A. solani*

Symptoms

Cracks appearing in leaf spot. The two species of *Alternaria* occur commonly, causing the characteristic leaf spots with concentric rings. The spots are mostly irregular, 4-8 mm in diameter and may coalesce to cover large areas of the leaf blade. Severely affected leaves may

drop off. *A. melongenae* also infects the fruits causing large deep-seated spots. The infected fruits turn yellow and drop off prematurely.

Pathogen

Mycelium is septate, branched, light brown to dark brown. It is inter and intra cellular. Conidiophores emerge through stomata and dark colored. Conidia are single celled, muriform, beaked and produced in chains. The conidia are with 5-10 transverse septa and a few longitudinal or oblique septa.

Mode of spread and survival

The disease is spread by wind borne conidia.

Management

Spraying 1 per cent Bordeaux mixture or 2 g Copper oxychloride or 2.5 g Zineb per litre of water effectively controls leaf spots.

Little Leaf of Brinjal

Economic Importance

This disease of brinjal was reported from India in 1938 and as far as known it occurs only in India and Sri Lanka. In almost all the states of the country it has become a serious problem facing brinjal cultivation. The yield loss is hundred per cent in the diseased plants.

Symptom

The characteristic symptom is the smallness of the leaves. The petioles are so short and the leaves appear to be sticking to be stem. Such leaves are narrow, soft, smooth and yellow. Newly formed leaves are much more shorter. The internodes of the stem are also shortened.

Axillary buds get enlarged but their petioles and leaves remain shortened. This gives the plant a bushy appearance. Mostly, there is no flowering but if flowers are formed they remain green. Fruiting is rare.

Pathogen

Little leaf was first considered a disease caused by a virus. In 1969 it was attributed to a mycoplasma-like organism, closely related to aster-yellows and curly top.

It is a sap transmissible disease. The organism has been transmitted to *Datura*, tomato and tobacco. It occurs in nature on *Datura fastuosa* and *Vinca rosea*. Natural transmission is through a vector, *Cestius phycytis* (*Eutettix phycytis*) while *Empoasca devastans* is a less effective vector. Perennation of the organism is through its weed hosts.

Mode of spread and survival

The disease is transmitted by leaf hoppers, *Hishimonas phycitis* and *Empoasca devastans* and grafting. *E. devastans* is less effective vector. Perennation of virus is through weed host. This disease has a very wide host range.

Management

The severity of the disease can be reduced by destruction of affected plants and spraying of insecticides. New crop should be planted only when diseased plants in the field and its neighbourhood have been removed.

Methyldemeton 25 EC	2 ml / litre
Dimethoate 30 EC	2 ml/ litre
Malathion 50 EC	2 ml/litre

has been recommended for vector control.

Although mycoplasmas are reported to be suppressed by tetracyclines field application of this method has not yet been recommended. Varietal resistance has not been systematically studied. Cultivars such as Pusa Purple Cluster, Arka Sheel, Aushy, Manjari Gota and Banaras Giant show moderate resistance to resistance in the field. Other cultivars found tolerant to the disease are Black Beauty, Brinjal Round and Surati.

Damping off: *Pythium aphanidermatum, Pythium indicum, , Phytophthora parasitica, Rhizoctonia solani* and *Sclerotium rolfsii.*

Symptom

Sudden collapsing of the seed lings occur in the seed bed. The seedlings are attacked at the collar region and the attacked seedlings are toppled down. The disease spreads through fungi present in the soil. The disease spreads through fungi present in the soil.

Management

The disease can be controlled by seed treatment with agrosan or ceresin @2gm/kg of seed.

Tobacco mosaic virus (TMV)

Symptoms

Mosaic mottling of leaves and stunting of plants are the characteristic symptoms of potato virus Y Mosaic symptoms are mild in early stages but later become severe. Infected leaves are deformed, small and leathery. Very few fruits are produced on infected plants. The important symptom produced by tobacco mosaic virus is conspicuous mottling of leaves. Leaves also develop blisters in advanced cases. Severely infected leaves become small and misshapen. Plants infected early remain stunted. PVY is easily sap transmitted.

It is transmitted in the field through aphids, *Aphis gossypii* and *Myzus persicae* and perpetuates on weed hosts like *Solanum nigrum* and *S.xanthocarpum.* TMV is transmitted by sap, contaminated implements and clothes, soil debris and hands of labour. It can perpetuate on many cultivated plants like cucurbits, legumes, pepper, tobacco, tomato and weed hosts. The virus survives in plant debris in soil.

Management

Destroy all weeds and avoid planting cucumber, pepper, tobacco, tomato near brinjal seed beds and field. Wash hands with soap and water before working in seed beds. Prohibit smoking or chewing of tobacco who are handling brinjal seedlings. Spray insecticides like Dimethoate 2 ml/litre or Metasystox 1 ml/litre of water to control the insect vectors.

Collar rot :*Sclerotium rolfsii*

Symptoms

The disease occasionally occurs in serious form. The lower portion of the stem is affected from the soil borne inoculum (sclerotia). Decortications is the main symptom. Exposure and necrosis of underlying tissues may lead to collapse of the plant. Near the ground surface on the stem may be seen the mycelia and sclerotia. Lack of plant vigour, accumulation of water around the stem, and mechanical injuries help in development of this disease.

Management

Seed treatment with 4 g of *Trichoderma viride* formulation per kg seed will help in reducing the disease. Spraying with Mancozeb @ 2g/Litre of water. Collection and destruction of diseased parts and portions of the plant.

Fruit rot :*Phomopsis vexans*

Symptoms

Affects all above the ground plant parts. Spots generally appear first on seedling stems or leaves. Girdle seedling stems and kill the seedlings. Leaf spots are clearly defined, circular, up to about 1 inch in diameter, and brown to gray with a narrow dark brown margin. Fruit spots are much larger, affected fruit are first soft and watery but later may become black and mummified. Center of the spot becomes gray, and black pycnidia develop.

Pathogen

Pycnidia with or without beak are found in the affected tissue. They are globose or irregular. Conidiophores in the pycnidium are hyaline, simple or branched. Conidia are hyaline, one celled and sub cylindrical. Ascospores are hyaline, narrowly ellipsoid to bluntly fusoid with one septum.

Mode of spread and survival

The fungus survives in the infected plant debris in the soil. It is seed borne. The spores are spread by rain splashes. The fungus spreads through implements and insects.

Management

Seeds should be dipped in hot water at 50˚C for 30 min. spraying with difolation 0.2% or captan 0.2% in the nursery and field at 7 – 10 days interval controls the disease. Deep summer ploughing, three year crop roation and collection and destruction of diseased plant debris are some of the other control methods. Spraying the crop in the field with zineb 0.2% or Bordeaux mixture 0.8% is effective in controlling Phomopsis blight.

Lecture 10 - Diseases of Bhendi

Bhendi

Cercospora Leaf Spots:*Cercospora malayensis,*

C. abelmoschi

Symptoms

In India, two species of *Cercospora* produce leaf spots in bhendi. C. *Malayensis* causes brown, irregular spots and *C. abelmoschi* causes sooty black, angular spots.Both the leaf spots cause severe defoliation and are common during humid seasons.

Pathogen

Conidiophores are pale to medium olivaceous brown, multiseptate, some times branched, geniculate and irregular. Conidia are obclavate to cylindric, olivaceous brown and straight to curved.

Mode of spread and survival

The fungus survives in the diseased crop material.

Management

Spraying Mancozeb 0.25 % control the disease.

Fusarium wilt*:* *Fusarium oxysporum f.sp. vasinfectum*

Symptoms

The conspicuous symptom is a typical wilt, beginning with a yellowing and stunting of the plant, followed by wilting and rolling of the leaves as if the roots were unable to supply sufficient water. Finally, the plant dies. If a diseased stem is split lengthwise, the vascular bundles appear as dark streaks. When severely infected, nearly the whole stem is blackend.

Pathogen

Macroconidia are 3- 5 septate formed on sporodochia and pionnotes. In mass conidia appear buff or salmon orange in color. Macroconidia are fusiform and curved inward at both ends. The base is pedicellate. Microconidia are septate. Terminal and intercalary chlamydospores are broadly ovate.

Mode of spread and survival

The fungus is soil borne.

Management

Treat the seeds with Mancozeb @ 3g/kg seed. Drench the field with Copper oxy chloride @ 0.25%.

Powdery mildew: *Erysiphecichoracearum*

Symptoms

Powdery mildew is very severe on bhendi. Greyish powdery growth occurs on the under as well as on the upper surface of the leaf causing severe reduction in fruit yield.

Pathogen

Conidia are single celled, hyaline, barrel shaped and in long chains. Cleistothecia are globose and dark brown myceloid appendages. The asci are pedicellate, ovate or ellipsoid. The number of ascospores is usually 2 rarely 3 per ascus. The ascospores are single celled, hyaline and oval to sub cylindrical

Management

Spary inorganic sulphur 0.25% or Dinocap 0.1% 3 or 4 times at 15 days interval.

Vein-Clearing/Yellow Vein Mosaic :*Bhendi yellow vein mosaic virus*

Symptoms

Yellowing of the entire network of veins in the leaf blade is the characteristic symptom. In severe infections the younger leaves turn yellow, become reduced in size and the plant is highly stunted. The veins of the leaves will be cleared by the virus and intervenal area becomes completely yellow or white. In a field, most of the plants may be diseased and the infection may start at any stage of plant growth. Infection restricts flowering and fruits, if formed, may be smaller and harder. The affected plants produce fruits with yellow or white colour and they are not fit for marketing.

Pathogen

The virus particles are 16 – 18nm in diameter.

Mode of spread

The virus is spread by whitefly.

Management

By selecting varieties resistant to yellow vein mosaic like Parbhani Kranti, Arka Abhay, Arka Anamika, and Varsha Uphar, the incidence of the disease can be minimised. The virus is transmitted by the whitely (*Bemisia tabaci*,. Parbhani Kranti, Janardhan, Haritha, Arka Anamika and Arka Abhay can tolerate yellow vein mosaic. For sowing during the summer season, when the whitefly activity is high, the susceptible varieties should be avoided. Spraying monocrotophos 1.5 ml/litre of water can restrict the disease spread. Synthetic pyrethroids should not be used because it will aggravate the situation. It can be controlled by application of Chlorpyriphos 2.5 ml + neem oil 2 ml lit of water.

Phoma canker (*Phoma exigua)*

Water soaked lesion appear on fruits. Black spots with irregular margin Black area - pycnidial formation. 80-90% fruit loss post harvest rot of okra pods *rhizoctonia solani* in brazil. Completely rotted, the pod's typical greenish color turning brown and the infected tissues fully covered with mycelia. Internally, immature seeds and placenta infected. Diseased tissues were light brown to black. Externally, mycelia tend to be fluffy and lighter in color, forming a large number of dark sclerotia on the fruit surface.

Lecture 11 - Diseases of Potato (2 Lectures)

Late blight of potato: *Phytopthora infestans*

Symptom:

It affects leaves, stems and tubers. Water soaked spots appear on leaves, increase in size, turn purple brown& finally black colour White growth develops on under surface of leaves. This spreads to petioles, rachis& stems. It frequently develops at nodes. Stem breaks at these points and the plant topples over. In tubers, purplish brown spots and spread to the entire surface on cutting, the affected tuber show rusty brown necrosis spreading from surface to the center.

Pathogen

The mycelium is endophytic, coenocytic and hyaline which are inter cellular with double club shaped haustoria type. Sporangiophores are hyaline, branched intermediate and thick walled. Sporangia are thin walled, hyaline, oval or pear shaped with a definite papilla at the apex. The sporangium may act as a conidium and germinate directly to form a germ tube. Zoospores are biflagellate possess fine hairs while the other does not.

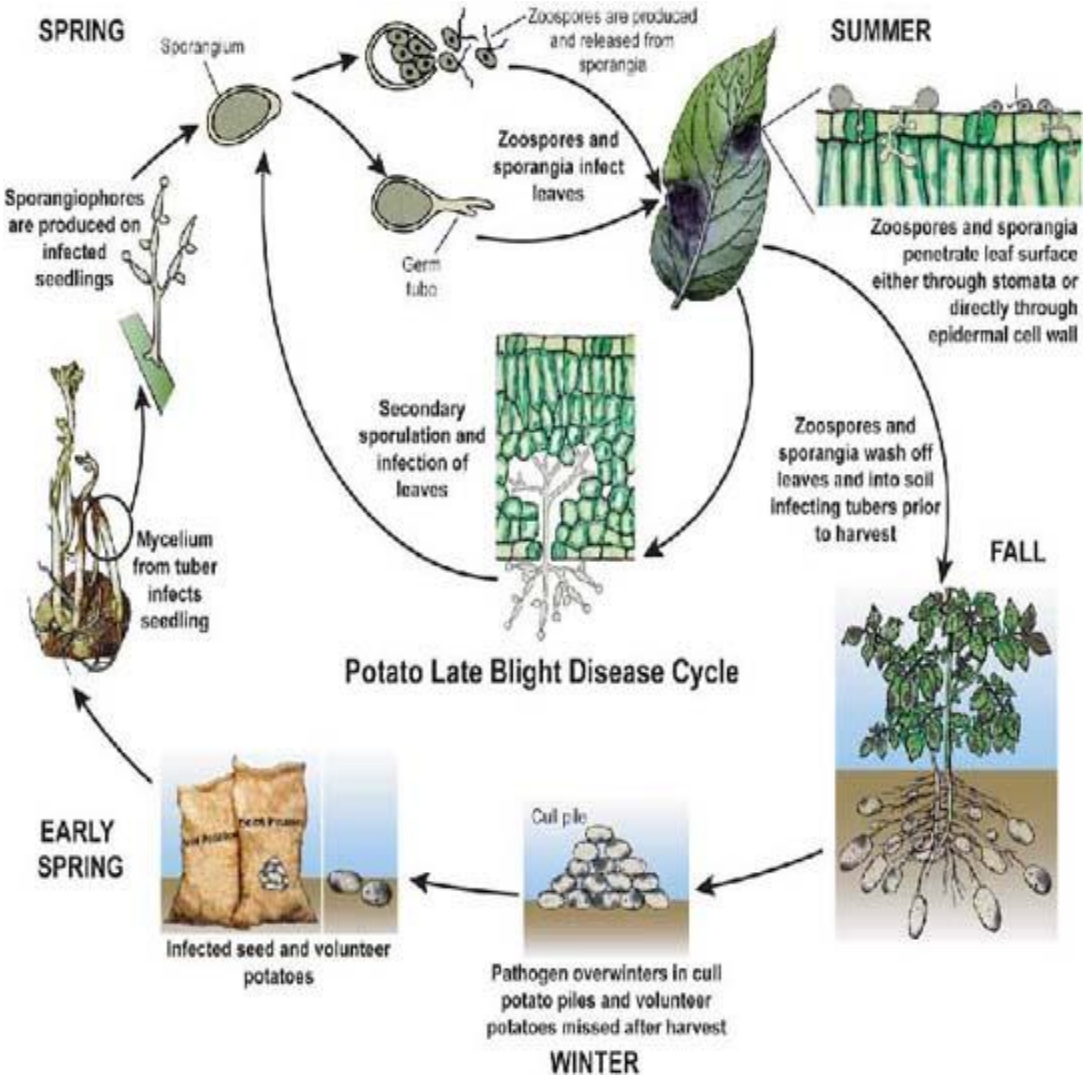
Mode of spread and survival

The infected tubers and the infected soil may serve as a source of primary infection. The diseased tubers are mainly responsible for persistence of the disease from crop to crop. The air borne infection is caused by the sporangia.

Favourable conditions

RH->90% , Temp.-10-25°C and Night temperature:10°C. Cloudiness on the next day

Rainfall at least 0.1mm, the following day.



Management

A regular spraying and dusting during the growing season give effective control. First spraying should be given before the commencement of the disease and subsequent should follow at regular interval of 10 -15 days. Protective spraying with mancozeb or zineb 0.2 % should be done to prevent infection of tubers. Destruction of the foliage few days before harvest is beneficial and this is accomplished by spraying with suitable herbicide. Tuber contamination is minimized if injuries are avoided at harvest time and storing of visibly infected tubers before storage. The resistant varities recommended for cultivation are Kufri Naveen, Kufri Jeevan, Kufri Alenkar, Kufri Khasi Garo and Kufri Moti.

Early blight: *Alternaria solani*

Symptoms

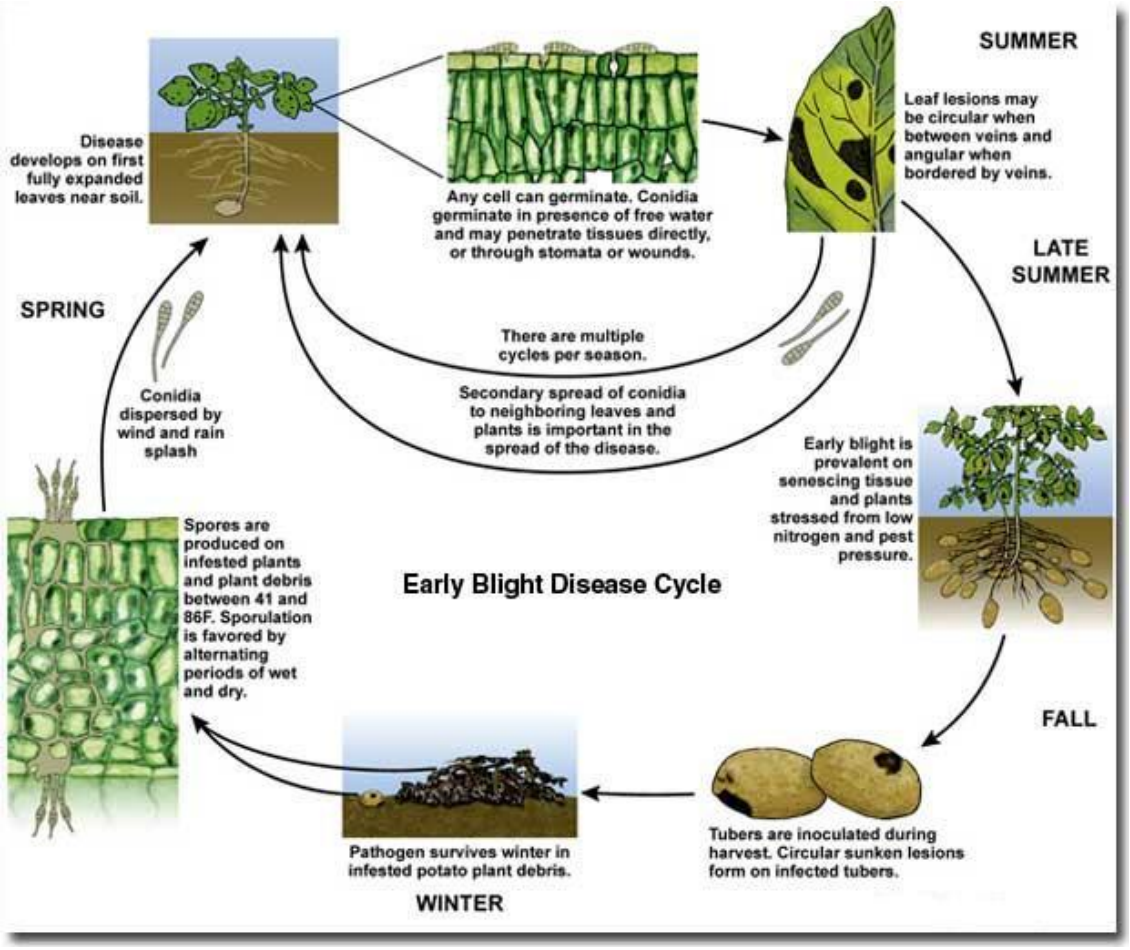
It is present in both hills & plains. Brown-black necrotic spot-angular, oval shape characterized by concentric rings .Several spot coalesce & spread all over the leaf. Shot holes on fruits.

Pathogen

Hyphae are light brown or olivaceous which become dark coloured with age. The hyphae are branched, septate and inter and intra cellular. The coniophores emerge through the stomata or between the epidermal cells. The conidia are club shaped with a long beak which is often half the long of the whole conidium. The lower part of the conidium is brown while the neck is colorless. The body of the conidium is divided by 5 – 10 transverse septa and there may or may not be a few longitudinal septa.

Favourable condition

Dry warm weather with intermittent rain .Poor vigor. Temperature: 25-30°C. Poorly manured crop.



Mode of spread and survival

The conidia and the mycelium in the soil or in the debris of the affected plants can remain viable for more than 17 months. These conidia or the new conidia found on the overwintered mycelium bring about the primary infection of the succeeding potato crop. Secondary infection is more important in the spread of the disease. The conidia formed on the spots developed due to primary infection are disseminated by wind to long distances. The conidia from the affected plant may also be disseminated to the adjoining plants by rain and insects.

Management

Disease free seed tubers should be used for planting. Removal and destruction of infected plant debris should be done because the spores lying in the soil are the primary source of infection. Very early spraying with Zineb or captan 0.2% and repeating it for every 15 – 20 days gives effective control. The variety Kufri Sindhuri possesses a fair degree of resistance.

Post-harvest tuber rots - *Sclerotium rolfsii*

Symptoms

Wilting is the initial symptom. Yellowish brown coloured Sclerotia appeared on the infected tuber. Rotting of the tuber. Milky white and floccose appearance of the tuber.

Pathogen

The mycelium is silky white and floccose. It is comprised of septate and branched hyphae. The branching take place just below the septum. The cells are large in size. Sclerotia of the fungus are white to begin with and become clove brown at maturity. They are globose and smooth surfaced.

Favorable condition

Optimum temperature 30-35ºC. Alternate period of wet and dry soil condition.

Mode of spread and survival

The mycelium and sclerotia of the organisam subsist in the soil and are responsible for the infection of the crop. The pathogen is disseminated with infected soil, in running water and on farm implements. Mycelium and sclerotia may also be carried to soil with the seed tubers. In dry soil scleritia can remain viable for more than two years.

Management

Treating seeds with mercury compounds after harvest reduces tuber rot. Treating the furrows at planting with PCNB @ 15kg/ ha reduces the disease icidence.Cultural practices like heavy earthing and irrigation at regular intervals can also check the disease. The disease is low in the variety Kufri Sindhuri. Among the Indian commercial cultivars, Kufri Bahar, Kufri Chamatkar, Kufri Jyothi, Kufri muthu and Kufri swarna are resistant. The disease can be controlled to a certain extent by growing non susceptible crops like corn and sorghum.

Black scurf- *R. solani*

Symptoms

Black speck, black speck scab, russet scab on tubers. At the time of sprouting dark brown colour appear on the eyes. Affected Xylem tissue causes to wilting of plants. Infected tuber contains russeting of the skin. Hard dry rot with browning on internal tissue. Spongy mass appear on the infected tuber. Seed tubers are source of spread. Moderately cool, wet weather and temp 23 °C are the favourable for the development of disease.

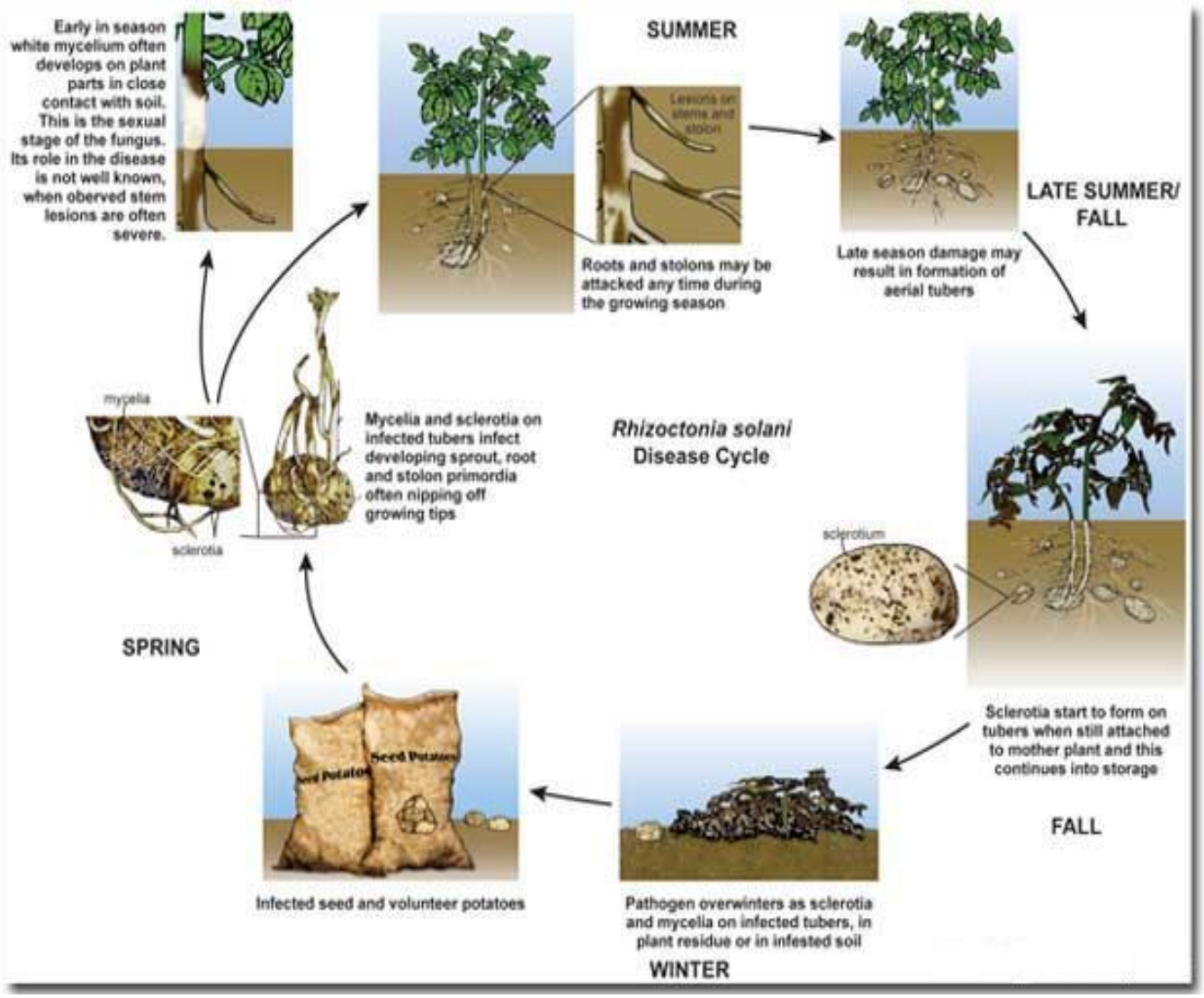
Pathogen

The mycelium is hyaline when young and brown at maturity. Hyphae are septate and branched with a characteristic constriction at their junction with the main hyphae. The branches arise at a right angle to main axis. Sclerotia are black. A basidium bears four sterimata each with a basidiospore at the end. The basidiospores are hyaline, elliptical to obovate and thin walled. They are capable of forming secondary basidiospores.

Mode of spread and survival

The fungus is capable of leading a saprophytic life on the organic material and can remain viable in the soil for several years. The sclerotia on the seed tubers is the principal source

of infection of the subsequent crop raised with these tubers. On return of favourable conditions the mycelium present in the soil may develop producing new hypae.

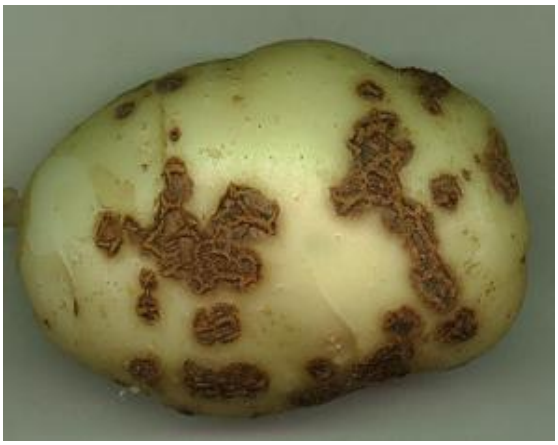


Management

Disease free seed tubers alone should be planted. If there is a slight infection of black scurf that can be controlled by treating seed tubers with mercuric chloride solution for 1.5 hr with acidulated mercuric chloride solution for 5 min. Treating the soil with pentachloroni trobenzene at the rate of 70 kg/ ha lowers the incidence of the disease, but it is too expensive and cumbersome. Well sporulated tubrs may be planted shallow to control disease. The disease severity is reduced in the land is left fallow for 2 years.

Common scab or corkey scab – *Streptomyces scabies*

Symptoms



Corkiness of the tuber periderm is the characteristics symptoms. 1/4 inch into the tuber surface are russette appearance. Slightly pitted on the infected tuber. Light brown to dark brown lesion appears on the infected tuber. Affected tissue will attract insects.

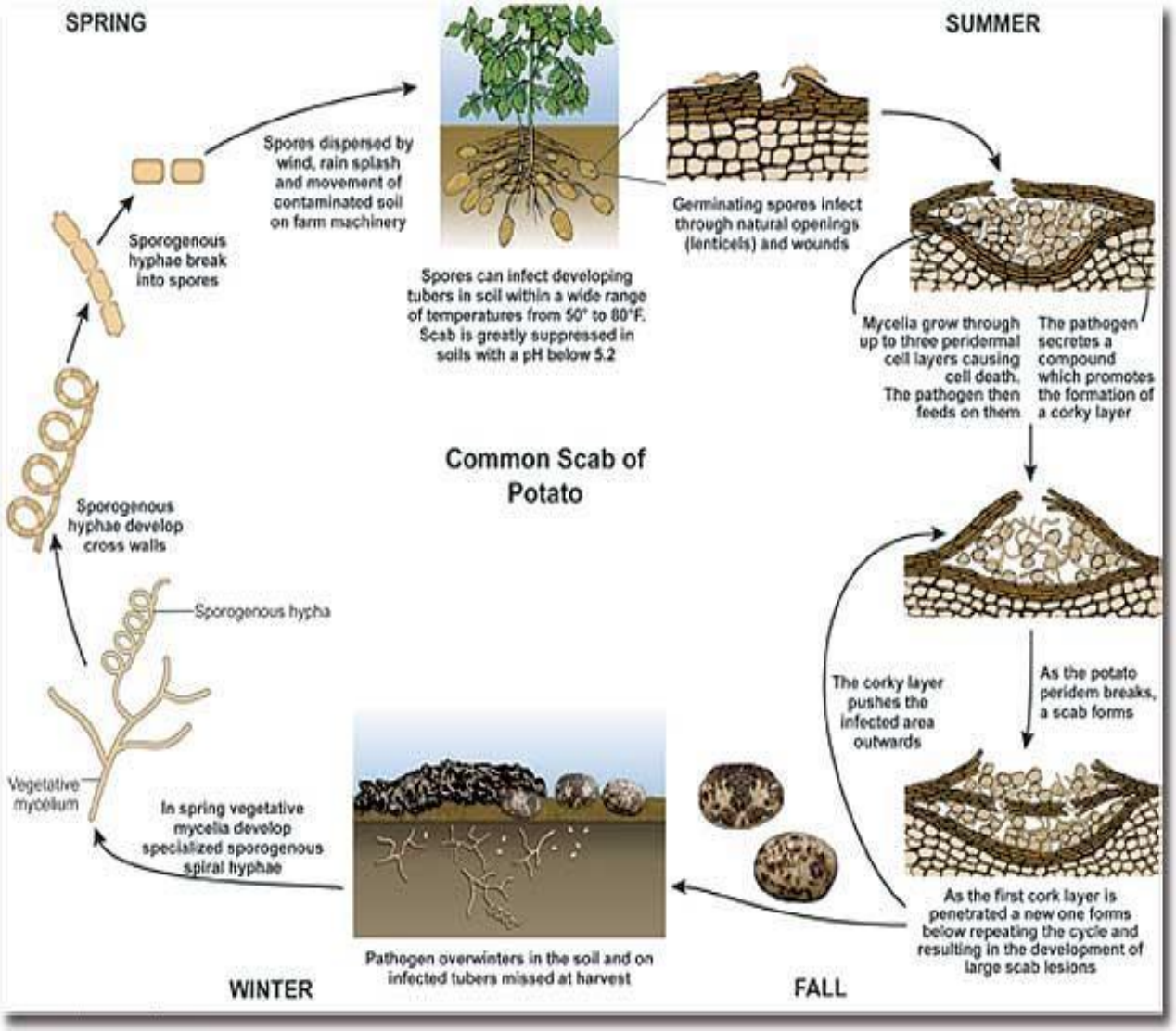
Pathogen

Aerial mycelium in pure culture has of prostrate branched threads. Sporogenous hyphae are spiral in form. Conidia are produced by the formation of septa at intervals along the hyphae, which contract to form narrow isthmuses between the cells. Conidia are roughly cylindrical and hyaline. The conidia can germinate even at higher temperatures. The growth of the organisam is good in slightly alkaline medium and is checked at pH 5.2.

Mode of spread and survival

It attacks cabbage, carrot, egg plant, onion, radish, spinach and turnip. The causal organism perpetuates in soil and infects the crop every year. Infected potato tubers serve as the main source of long distance spread of the disease. The pathogen may survive passage through digestive tract of animals and hence it may spread with farm yard manure.

Disease Cycle



Management

Only scab free seed potatoes should be planted as this will help in checking the spread of the inoculum and infection to be subsequent crop. Infection of the seed tubers can be removed by 1.5hrs dip in mercuric chloride 0.1% solution or by 2h dip in 1 part formaldehyde in 240 parts of water. This disease can be reduced by soil application of PCNB at the time of planting. Four to six years crop roation with alfalfa satisfactory under irrigated conditions. The disease incidence can be effectively reduced by green manuring the fields before planting potatoes. Common scab is severe in alkaline soil and application of alkaline fertilizers like calcium ammonium nitrate should be avoided.

Brown rot or Bangle blight -*Ralstonia solanacearum*

Symptoms

brown vascular bundle more severe infection

At the time tuber formation wilt is the main characteristic symptom. In leaf symptom - wilt, stunt and yellowing. Browning of xylem tissue. Eye buds are black in colour. Bacteria ooze coming on infected tuber surface and emits a foul odour.

Pathogen

G –ve, short rod, 1-4 flagella. Colonies are white to brown in colour

Favourable condition

Temp 25to 35ºC , RH above 50 % and PH 6.2-6.6 favours for the development of disease. Acid soil is not favourable.

Mode of spread and survival

Infected soil and seed tubers form the main source of the primary infection. Brown rot affected plant parts decay and release masses of bacteria in the soil where these may remain viable from season to season. The bacteria in the soil are disseminated by wind from one field to the other. The infection usually occurs through wounds in the root system.

Disease cycle

R. solanacearum is a soilborne and waterborne pathogen; the bacterium can survive and disperse for various periods of time in infested soil or water, which can form a reservoir source of inoculum. In potato, the brown rot pathogen is also commonly tuber borne. The bacterium usually infects potato plants through the roots (through wounds or at the points of emergence of lateral roots).

Under favorable conditions, potato plants infected with *R. solanacearum* may not show any disease symptoms. In this case, latently infected tubers used for potato seed production may play a major role in spread of the bacterium from infected potato seed production sites to healthy potato-growing sites. *R. solanacearum* can survive for days to years in infected plant material in soils, infested surface irrigation water, infected weeds, and infected potato washings and sewage. From these sources of inoculum, bacteria can spread from infested to healthy fields by soil transfer on machinery, and surface runoff water after irrigation or rainfall. Infected semi-aquatic weeds may also play a major role in disseminating the pathogen by releasing bacteria from roots into irrigation water supplies.

Soft rot- *Erwinia carotovora* subsp *caratovora*

Symptoms

Infection at two phases are black leg and soft rot. Black lesion appear on the base of the plant .Systemic and browning of infected tubers. Yellow appearance of the plant. Finally the plants wilt and die. Lenticels (water soaked brown rot). Rot and collapse of tubers. Soft, reddish or black ring appear on the infected tuber.

Pathogen

It is a gram negative rod shaped bacterium with 1 to 6 peritrichous flagella.

Mode of spread and survival

Infected tubers attract the flies (*Hymelia* and *Phorlin* sp). Spread through immature contaminated soil and tuber. Optimum temperature 21 to 29 ºC and RH 94%

Management

The pathogen is difficult to control because of long survival both on seed tubers and in soils. However using disease free seed tubers could minimize the disease incidence. Before planting the seed tubers are treated with Boric Acid (3% for 30 minutes) and dried in shade. The same treatment is repeated before the storage of the tubers.

The disease can be reduced by soil application of PCNB (30 kg/ha) at the time of planting. Following crop rotations with wheat, pea, oats, barley, lupin, soybean, sorghum and bajra checks the disease development. In plains, treatment of the seed tubers with TBZ + acetic acid + 0.05% Zinc Sulphate solution or Carbendazim 1% for 15 minutes effectively controls the disease. Soaking of tubers in Mercuric chloride 0.1% formalin.

Lecture 12 - Diseases of Cucurbits (2 Lectures)

Cucumber and squash

Vascular Wilt: *Erwinia tracheiphila*

Symptoms

Symptoms of the disease first appear on a single leaf which suddenly wilts and becomes dull green. The wilting symptoms spread up and down the runner sometimes as a recurring wilt on hot, dry days. Soon infected runners and leaves turn brown and die. The bacteria spread through the xylem vessels of the infected runner to the main stem, then to other runners. Eventually the entire plant shrivels and dies.

Less susceptible plants, such as certain squash varieties, may show dwarfing of growth before the wilt symptoms become apparent.

Creamy white bacterial ooze consisting of thousands of microscopic, rod-shaped bacteria may sometimes be seen in the xylem vascular bundles of an affected stem if it is cut crosswise near the ground and squeezed. This bacterial ooze will string out forming fine, shiny threads (like a spider's web) if a knife blade or finger is pressed firmly against the cut surface, then slowly drawn away about 1 cm.

Two cut stem ends can also be put together, squeezed, then separated to look for shiny strands of bacteria. The sap of a healthy plant is watery and will not string. Sometimes it helps to wait several minutes after cutting to perform the test. This technique is useful in field diagnosis to separate this disease from other vascular wilts. Beware, however, that the technique may not always work (i.e., no bacterial strings occur yet the plant is still infected). The test works better

for cucumbers than for muskmelons. Fruit may also show symptoms. Small water-soaked patches form on the surface. These patches eventually turn into shiny decayed spots on the fruit.

Pathogen

It is a motile rod with 4 – 8 peritrichous flagella and capsulated. Agar colonies are small, circular, smooth, glistening white and viscid.

Mode of spread and survival

The bacteria apparently overwinter in cucumber beetles and they appear to multiply in the beetle. The bacterium is not seed borne or soil borne. Bacteria in stems can survive for one month. Beetles prefer to feed on plants with bacterial symptoms than on healthy plants. Beetle can remain infective for atleast three weeks. Striped cucumber beetle and the 12- spotted cucumber beetle help in the spread of the bacterium.

Management

Larger plantings must be protected by insecticides. Some carbaryl (Sevin), malathion, or rotenone insecticides or combination products are registered to treat cucumber beetles. They will provide control of the beetles if applied when beetles first appear in the spring. Early control, beginning as soon as the plants emerge, is most important as a single beetle can introduce the bacteria. One to four generations of the beetle may occur on unprotected plants and applications of these insecticides at weekly intervals may become necessary. Apply a light even coating of the insecticide over the entire plant, especially where the stem emerges from the soil (that is where the beetles often congregate).

Scab: *Cladosporium cucumerinum*

Symptoms

Scab lesions appear on all parts of the vine that are above ground. The first symptoms appear as light water soaked or pale green spots on the leaves. These spots are numerous and appear on and between veins. Similar elongated spots develop on petioles and stems. Gradually, the spots turn grey to white and become angular.

The affected leaves near the tip of the vine may be stippled with dead and yellowish spots, stunted and crinkled. Fruits are infected at all stages of growth but is most susceptible while young. Fruit spots are grey, slightly sunken and about 2.0mm in dia.

Pathogen

Conidia are oblong, dark, mostly aseptate.

Mode of spread and survival

The fungus probably survives in old cucumber refuse or soil in cracks and on seed. It is disseminated by insects, clothings and tools.

Disease Cycle

The scab organism survives in soil on squash, melon, and pumpkin vines and reportedly may grow extensively as a saprophyte. The fungus may also be seed borne. It is disseminated on clothing and equipment and by insects. The conidia can survive long-distance spread in moist air. The most favorable weather conditions for disease development are wet weather and temperatures near or below 21°C. At 17°C the growing tips of young plants are killed. Conidia germinate and enter susceptible tissue within 9 hr. A spot may appear on leaves within 3 days, and a new crop of spores is produced by the fourth day.

Management

Crop rotation with corn once in 4 years. Grow resistant varieties like Highmoor and Maine no.2. Spray Mancozeb 0.2 %.

Musk melon and water melon

Gummy Stem Blight - *Mycosphaerella melonis*

Symptoms

Infected stems first appear water-soaked and then become dry, coarse and tan. Older stem lesions (dead tissue) reveal small black fruiting bodies (pycnidia) within the affected tissues. Large lesions girdle stems and plants wilt in the heat of the day. Stem lesions on melons exude a gummy, red-brown substance which may be mistaken for a symptom of Fusarium wilt.

Mode of spread and survival

The pathogen can be seed-borne and, thus, can spread by infected seedlings. The inoculum of the pathogen can also come from other cucurbitaceous host plants and weeds and infected plant debris in and around the facility. The pathogen produces two types of spores: asexually-produced pycniospores, and sexually-produced ascospores. Both types of spores are short-lived once they are released into the environment. However, the pathogen can survive up to 2 years as chlamydospores or mycelium on undecomposed, dry plant debris.

Disease Cycle

The gummy stem blight fungus is both seed- and soil-borne. The pathogen may be carried in or on infested seed. In the absence of host plants, the fungus can over winter for a year and a half or more on infected crop residue. The exact length of survival in the Northeast is currently being studied. The fungus survives as dormant mycelium or as chlamydospores (thick-walled modifications of the mycelium). In northern areas of the country in the spring, pycnidia are produced, giving rise to conidia, which serve as the primary inoculum. Conidia are released through a pore (ostiole) in the pycnidia and if moisture is high, conidia exude as "spore horns" containing thousands of conidia. Conidia vary in size, are short and cylindrical, with usually one septum near the middle, or they may be unicellular. Under moist conditions, they are readily dispersed by splashing water.

Both temperature and moisture are critical for germination, sporulation, penetration of conidia, and subsequent symptom development, but moisture (relative humidity over 85 percent, rainfall and duration of leaf wetness from 1 to 10 hours) has the greatest influence. The optimal temperature for symptom development varies depending on the cucurbit for watermelon 75° F is optimal, for cucumber 75-77° F, and for muskmelon 65° F. The optimal temperature for muskmelon reportedly is lower because its resistance increases at high temperatures.

This can be significant to determine when early-season disease scouting should be initiated for future control. Penetration by conidia is probably direct and does not need to occur through stomata or wounds. Wounding, striped cucumber beetles, and aphid feeding, along with

powdery mildew infection, predispose plants to infection. The additional nutrients provided by such injuries enhance gummy stem blight infection.

Management

Use of disease-free seed and transplants is essential to prevent serious crop losses. Periodic applications of fungicide like mancozeb @ 0.2% can help limit secondary infections, especially on fruits. Fall plowing and extended rotations with other crops can significantly reduce the amount of inoculum in infested fields.

Bacterial Wilt - *Erwinia tracheiphila*

Symptoms

On cucumber and melon, generally a distinct flagging of lateral and individual leaves occurs. Affected leaves turn a dull green. Sometimes wilting occurs on leaves that have been injured by cucumber beetles' feeding, but in many cases obvious feeding is not apparent. Leaves adjacent to the wilting leaves will also wilt, and eventually the entire lateral is affected. The wilt progresses as the bacteria move from the point of entry through the vascular system toward the main stem of the plant.

Eventually the entire plant wilts and dies. If you cut through the stem of an affected plant and squeeze both cut ends, a white, sticky exudate will often ooze from the water-conducting tissue of the stem. This exudate is composed of bacterial material that plugs the vascular system of the plant. Affected stems do not appear significantly discolored. Bacterial wilt is closely associated with either the striped or the spotted cucumber beetle. The bacteria over winter in the bodies of adult cucumber beetles. The beetles carry the bacteria when they emerge in the spring.

The bacteria are spread either through the feces of the beetle or from contaminated mouthparts. When the beetles feed on young leaves or cotyledons, they open entry points for the pathogen. Once inside the plant, the bacteria travel quickly through the vascular system, causing blockages that in turn result in wilting of the leaves. The disease progresses from plant to plant when a carrier beetle moves through the field or when clean beetles pick up the bacteria from a diseased plant and fly to healthy plants. Larvae are not known to carry the wilt organism.

Pathogen

It is a motile rod with 4 – 8 peritrichous flagella and capsulated. Agar colonies are small, circular, smooth, glistening white and viscid.

Mode of spread and survival

The bacteria apparently overwinter in cucumber beetles and they appear to multiply in the beetle. The bacterium is not seed borne or soil borne. Bacteria in stems can survive for one month. Beetles prefer to feed on plants with bacterial symptoms than on healthy plants. Beetle can remain infective for atleast three weeks. Striped cucumber beetle and the 12- spotted cucumber beetle help in the spread of the bacterium.

Management

In general, more bacterial wilt is seen on the edges of fields where beetles first encounter plants. Larger plantings must be protected by insecticides. Carbaryl, Malathion or rotenone insecticides or combination products are registered to treat cucumber beetles. They will provide control of the beetles if applied when beetles first appear in the spring. Early control, beginning as soon as the plants emerge, is most important as a single beetle can introduce the bacteria. One to four generations of the beetle may occur on unprotected plants and applications of these insecticides at weekly intervals may become necessary. Apply a light even coating of the insecticide over the entire plant, especially where the stem emerges from the soil (where the beetles often congregate).

Fusarium Wilt - *Fusarium oxysporum* f. sp. *melonis* attacks muskmelon and *Fusarium oxysporom* f. sp. *niveum* attacks watermelon.

Symptoms

Both fungi contribute to damping-off of seedlings, but most significant losses occur after young plants are infected in the field. Plants infected early in the season often produce no marketable fruits. Plants that begin to show wilt symptoms at or near maturity produce fewer and lower quality fruits. The first symptoms of Fusarium wilt are wilting and chlorosis (yellowing) of older leaves. The wilt is most evident during the heat of the day. Plants may appear to recover by morning, only to wilt again in the afternoon. Stem cracks and brown streaks often appear near the crown of the plant and are associated with a red-brown exudate. Fusarium wilt also causes vascular browning that is visible in stem cross-sections.

Mode of spread and survival

The wilt fungus is introduced to new areas on seed. It spreads by wind, equipment and workers. It can survive long periods in soil as chlamydospores and in association with melon plant residue.

Management

Planting resistant cultivars is the only reliable way to keep infested fields in production. Commercially acceptable resistant cultivars exist, but extremely high pathogen populations in the soil can overcome their resistance. Therefore, methods to reduce *Fusarium* populations in the soil also should be employed. These methods include extended rotations with crops other than cucurbits and fall plowing of severely infested fields.

Anthracnose *Colletotrichum orbiculare (= C. lagenarium)*

Symptoms

The diagnostic features of anthracnose vary with the host. Sunken, elongated stem cankers are most prominent on muskmelon, though leaf and fruit lesions also occur. Large lesions girdle the stems and cause the vines to wilt. Stem cankers are less obvious on cucumbers,

but leaf lesions are very distinct. Watermelon foliage affected by anthracnose appears scorched; sunken fruit lesions are easy to recognize. The anthracnose fungus over winters on diseased crop residue. There also reported that the pathogen is carried in or on cucurbit seed. In wet conditions each spring, the fungus releases airborne spores that begin new infections on vines and foliage. Anthracnose usually becomes established in mid-season, after the crop canopy has fully developed.

Mode of spread and survival

The fungus can infect muskmelon and watermelon in addition to cucumber. The pathogen survives the winter in infected plant residues. The fungus can also be associated with seed. As with most fungal diseases, long periods of leaf wetness favor disease development. Spores are splashed from leaf to leaf, and plant to plant, during irrigation or rain events. Several disease cycles can occur in a single growing season, resulting in defoliation of severely infected plants.

Management

Seed treatment with Carbendazim 2g/kg of seed. Spray Mancozeb 2g or Carbendazim 0.5g/lit.

Sudden Wilt

Symptoms

Unlike bacterial wilt, which can occur any time during the season, sudden wilt generally occurs late in the season and is closely associated with a heavy fruit load on the plant. Cucumbers and melons appear to be most sensitive to sudden wilt. Initial symptoms are a slight flagging of the plants in midday even when abundant moisture is present. This flagging will continue to worsen so that, by the third or fourth day, many of the plants are completely wilted. Disease progression is rapid, hence the name sudden wilt. After five to six days, all of the vines have melted down and only the immature fruits are left in the fields. Affected plants appear to lack feeder roots; other roots become slightly misshapen and thick. Currently it is thought that

sudden wilt is caused by a root rot complex involving *Pythium* sp., *Rhizoctonia solani* and *Fusarium* sp. that invade the roots and further colonize the root tissue. It is thought that stresses such as excess moisture and drought, prolonged periods of low temperatures (below 50 degrees F) and attack by the several viruses that commonly affect mellons and/or cucumbers individually or in combination weaken plants so that soil-borne pathogens can rapidly colonize the root systems.

Management

Good soil drainage and thin plant density reduces the incidence of disease. Destroy diseased plant debris. Soil application of *T.viride* @ 2.5 kg/ha with 50 kg FYM. Spray Mancozeb/ Copper Oxychloride at 2.5 g /lit or Carbendazim/ Thiophanate-methyl at 1 g /lit.

Powdery mildew - *Erysiphe cichoracearum*

Symptoms

It attacks muskmelons, squash, cucumbers, gourds, and pumpkins. It is evident as a superficial, powdery, grayish-white growth on upper leaf surfaces, petioles, and even main stems of infected plants. Affected areas turn yellow then brown and die. In dry seasons, powdery mildew can cause premature leaf drop and premature fruit ripening. Some early disease results from spores produced on over wintering cucurbit debris or weeds but the major source of disease inoculum is windblown spores from southern crops. Warm, dry weather conditions favor the development of powdery mildew.

Pathogen

The conidia measure 63.8 x 31.9 micron meter, the cleistothecia are globose which contain 10 – 15 asci. In each ascus, ascospores are two and are oval or sub cylindrical.

Mode of spread and survival

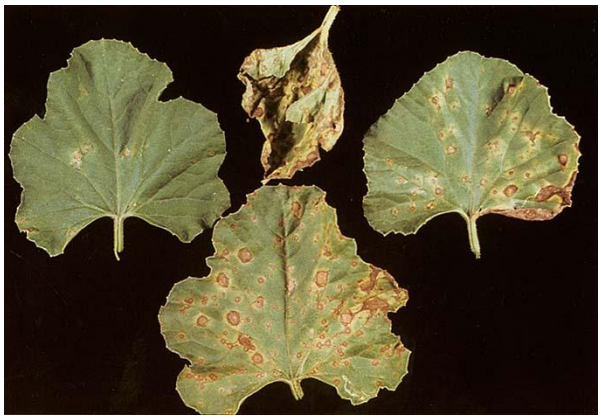
Perithecia developed on left over cucurbit crop in isolated areas serve as primary inoculum. Wild cucurbits harbour the conidial stage of the fungus and release conidia for primary infection to the spring or summer sown cucurbits. Conidia are spread by wind , thrips and other insects.

Management

Powdery mildew can be controlled by application of Wettable sulphur @ 0.2%.

Alternaria Blight - *Alternaria cucumerina*

Symptoms



It usually occurs on foliage during the middle of the growing season. The disease starts as small, yellow spots which enlarge to form concentric rings on the upper leaf surfaces. Muskmelons are more susceptible than other cucurbits to Alternaria blight.

Often muskmelon vines will be almost completely defoliated by this disease. The pathoegen also may cause fruit injury. *Alternaria cucumerina* may be carried in and on seed and can also overwinter in diseased plant debris or cucurbit weeds. Spores produced on infected foliage are spread by wind, rain, people, tools, etc. Plants weakened by lack of proper fertilizer or poor soils are more likely to be attacked than young, vigorously growing plants. Warm, wet weather favors development of Alternaria blight.

Pathogen

In water melon isolate, the conidia are 50.5 – 86.4 x 22.8 micron meter. Cross septa vary from 1 to 9 and longitudinal septa range from 1 to 4.

Mode of spread and survival

The fungus can survive as mycelium in refuse from diseased plants at least for one season and possibly two years in dry conditions. Fungus spores can survive in dry warm conditions for several months. Conidia are air borne.

Management

To control Alternaria blight, plant disease-free seed in fertile, well-drained soil, practice crop rotation with unrelated crops, destroy cucurbit weeds. Spray the crop with Mancozeb @ 2 g /lit.

Downy mildew - *Pseudoperonospora cubensis*

Symptoms

It occurs on cucumbers, squash, muskmelons, and pumpkins and less frequently on watermelons. On cucurbits other than watermelons, small, yellowish areas occur on the upper leaf surface. Later a more brilliant yellow color develops with the center of the lesion turning brown. Usually spots are angular because they are restricted by leaf veins. When leaves are wet, a downy, white-gray-light blue fungus growth can be seen on the underside of individual lesions. On watermelons, yellow leaf spots may be angular to non-angular and turn brown to black. Spores produced on the lower leaf surface are readily spread by the wind. Rainy, humid weather favors the development of downy mildew.

Pathogen

It is an obligate parasite. The mycelium is coenocytic and intercellular with small ovate or finger like haustoria. One to five sporangiosphores arise through the stomata. Sporangia are grayish to olivaceous purple, ovoid to ellipsoidal, thin walled with a distal papilla. Zoospores are 10 – 13 micron meter. Oospores are not common.

Mode of spread and survival

The pathogen survives on the diseased plant debris. In warm and humid climates, transmission from old to younger crops takes place all the year round. Where warm and dry summers alternate with cooler and wet winters, year round survival is possible on summer irrigated crops. They may overwinter as thick walled oospores. Sporangia are disseminated by wind. Cucumber beetles are reported to carry the sporangia.

Disease cycle

Pseudoperonospora cubensis is an obligate parasite requiring living host tissue to survive. It does not live in debris in the soil. Occasionally, under optimum environmental conditions, the pathogen may develop thick-walled spores called oospores that are resistant to low temperatures and dry conditions. This is rare and not considered an important source of inoculum. Infections in greenhouses likely originate from another type of spore (sporangia) that enters the facilities from the outside. Local field infections are usually established by spores carried by moist air currents blowing northwards from distant warmer regions where the fungus can over winter on plant material.

Moisture on the leaf surfaces is necessary for infection to occur. When spores land on a wet leaf surface, they can either germinate and infect through the breathing pores (stomates) on leaves or release many smaller spores, called zoospores, that swim in the film of water on leaves during humid or wet conditions, and enter and infect leaves through stomata. Optimum temperatures for infection range between 16°c and 22°c, with infection occurring more rapidly at the warmer temperatures. The periods of wetness needed for infection on cucumber leaves are about 12 hr at 10°c-15°c, 6 hr at 15°c-19°c, and 2 hr at 20°c. About 4-5 days after infection, new spores are produced and released into the air, primarily in the morning. Spores can quickly spread within the greenhouse via moist air currents, contaminated tools, equipment, fingers and clothing.

Management

Spraying with Metalaxyl 500 g or Metalaxyl + Mancozeb 1 kg/ha or Mancozeb 1 kg/ha.

Angular Leaf Spot - *Pseudomonas lachrymans*

Symptoms

Symptoms of the disease firsts appear as small, angular, water-soaked lesions on the leaves. When moisture is present, bacteria ooze from the spot in tear like droplets that dry and form a white residue on the leaf surface. Water-soaked areas turn gray or tan, die, and may tear away leaving irregular holes. Water-soaked spots may also appear on the fruit and are frequently followed by soft rot bacteria.

Pathogen

The bacterium is a rod with 1 – 5 polar flagella and forms capsule and a green fluorescent pigment in culture. The colonies on beef – peptone agar are circular, smooth, glistening, transparent and white.

Mode of spread and survival

Infected seeds may harbour the bacterium. They survive in soil or debris from diseased plants for two years. They spread by irrigation water.

Management

Angular leaf spot may be controlled by planting disease-free seed. Rotating with unrelated crops, keeping workers out of fields when foliage is wet and Spray 400ppm Streptomycin sulphate.

Gourds

Downy mildew: *Pseudoperonospora cubensis*

Symptoms

Symptoms resembling mosaic viz, pale green areas separated by dark green areas appear on upper surface of leaf. During wet season, corresponding lower surface is covered with faint purplish fungal growth. The entire leaf dries up quickly.

Pathogen

It is an obligate parasite. The mycelium is coenocytic and intercellular with small ovate or finger like haustoria. One to five sporangiosphores arise through the stomata. Sporangia are grayish to olivaceous purple, ovoid to ellipsoidal, thin walled with a distal papilla. Zoospores are 10 – 13 micron meter. Oospores are not common.

Mode of spread and survival

The pathogen survives on the diseased plant debris. In warm and humid climates, transmission from old to younger crops takes place all the year round. Where warm and dry summers alternate with cooler and wet winters, year round survival is possible on summer irrigated crops. They may overwinter as thick walled oospores. Sporangia are disseminated by wind. Cucumber beetles are reported to carry the sporangia.

Management

Use of bed system with wide spacing with good drainage and air movement and exposure to sun help to check the disease development. Spray with Moncozeb 0.2 % or Chlorothalonil 0.2% or Difolaton 0.2% or Ridomil MZ 72 0.1% Seed treatment with Apron SD 35 @ 2 g./kg. followed by spraying with Mancozeb 0.2% is effective in reducing the disease.

Powdery mildew: *Erysiphe cichoracearum*

Symptoms

Powdery mildew, is especially prevalent in hot dry conditions. White or brown mealy growth will be found on upper and lower surfaces and stems. Under severe infestations, the plant will be weakened and stunted.

Pathogen

The conidia measure 63.8 x 31.9 micron meter, the cleistothecia are globose which contain 10 – 15 asci. In each ascus, ascospores are two and are oval or sub cylindrical.

Mode of spread and survival

Perithecia developed on left over cucurbit crop in isolated areas serve as primary inoculum. Wild cucurbits harbour the conidial stage of the fungus and release conidia for primary infection to the spring or summer sown cucurbits. Conidia are spread by wind , thrips and other insects.

Management

The disease can be controlled by spraying Wettable sulphur 0.1%.

Mosaic: PRSV/CMV

Symptoms:

A virus distributed world wide, affecting most cucurbits but rarely affecting watermelon. New growth is cupped downward, and leaves are severely mottled with alternating light green and dark green patches. Plants are stunted, and fruits are covered with bumpy protrusions. Severely affected cucumber fruit may be almost entirely white.

Mode of spread and survival

It is transmitted by mechanical inoculation and by insect vectors, *Aphis gossypii* and *Myzus persicae.*

Management

The virus is readily transferred by aphids and survives on a wide variety of plants. Varietal resistance is the primary management tool, and eliminating weeds and infected

perennial ornamentals that may harbor the virus is critical. Spray with any one of the systemic insecticide.

Lecture 13 - Diseases of Crucifers (2 Lectures)

Beet root

Leaf Spot: *Cercospora beticola*

Symptoms

This is a commonly occurring disease on foliage of beet roots. High humidity usually favours the spread of this disease. Numerous small circular spots appear on the leaf surface. The spots increase in size, becoming brownish or purplish in color. Individual spots are usually circular but several may coalesce into larger areas of dead tissue. The spots dry up giving a shot-hole appearance to the leaves. In case of severe infection spots cover the entire leaf surface resulting in pre-mature death and dropping of the leaves. As leaves die, the crown becomes cone-shaped with a rosette of dead leaves at the base. Defoliation occurs throughout the growing season resulting in reduction in root size and yield. Older leaves are mostly affected.

Pathogen

Conidia are borne singly at the tip of conidiophores. They are hyaline, elongate, filiform and multiseptate. Perfect stage is not known.

Mode of spread and survival

The pathogen is carried with the seed. The chief overwintering inoculum is in infected plant debris, in which mycelium remain viable. The fungus can overwinter in debris from diseased plants, in weed hosts and in beet seeds. The fungus can survive 12 – 18 months. The conidia are disseminated chiefly by air. Insects, splashing water, cultivation tools, workers and irrigation water also spread of the disease. Moist weather is essential for sporulation.

Management

Removal and destruction of affected plants and practicing crop rotation are beneficial in controlling the disease. Spraying with Copper oxychloride (0.3 %) thrice at an interval of 15 days controls the disease effectively.

Downy Mildew: *Perenospora schachtti*

Symptoms

The disease is mostly prevalent during the cooler months. Symptoms appear as irregular greasy greyish areas on the leaves. Under moist conditions, these areas expand rapidly and a white powdery growth appears on the lower surface of the affected leaves. Affected leaf dries and shrivels quickly. Flower shoots on infected plants become stunted and distorted. The entire inflorescence has a compact appearance and excessive leaf development may give an appearance witches broom. The fungus survives on the crop residues in the soil and is also carried by the seed.

Pathogen

Peronospora produces sporangia abundantly on the cotyledons and is splashed from there to other plants. The sporangia germinate by means of a germ tube and not by zoospores.

Management

Preventive measures such as good field sanitation, crop rotation and use of resistant cultivars is recommended. Seed treatment with Thiram (2.5-3 g/kg of seed) protects the emerging seedlings from the disease attack. Spraying with Dithane Z-78 (0.3 %) thrice at an interval of 15 days is also recommended as an effective control measure.

Curly-top virus

Symptoms

External symptoms of curly top virus infection may appear in leaves, stems, flowers, fruits, or roots of infected plants. Generally, mottling is absent, but infected plant parts may become distorted through curling, twisting, rolling, stunting, etc.

Leaves become thickened and leathery. Curly top virus may impair both yield and quality of the root of an infected plant. Some of the most pronounced symptoms resulting from curly top virus attacks are internal and non-observable with the unaided eye. Such internal symptoms consist of death of the food conducting vessels, as well as of extreme variations from the normal in numbers and sizes of cells composing the plant tissues.

Pathogen

Beet curly top virus particals are 18 – 22 nm in dia. The thermal death point of the virus is 80˚C and longevity in vitro is 8 days.

Mode of spread and survival

The beet leaf hopper is the vector of BCTV. The first generation leafhoppers migrate out of the range lands to sugar beet fields, carrying the virus with them. Leafhoppers produce several generations each year, which migrate through susceptible crops spreading the virus. As the crops mature and dry, the leafhoppers move back into the over wintering areas in search of the winter host.

Leafhoppers acquire BCTV by feeding on infected host, either the winter host or crop plants. Leafhoppers are able to acquire the virus during very short feeding times. The leafhopper retains the ability to transmit BCTV for a month or more after acquisition. The vector may maintain the virus during its over-wintering period.

Management

Losses can be reduced by the use of resistant varieties; Adopting sanitary measures including the eradication of susceptible weeds and susceptible volunteer crop plants from a previous planting; Regulating the time of planting in order to avoid the main flights of the beet leafhopper; Use of barriers of trap crops and early removal and destruction of infected plants. Spraying malathion (2ml/litre of water) controls the population of beet leaf hoppers.

Beet Yellows: Virus

Symptoms

This disease is transmitted mainly through aphids. The important symptoms of the disease include yellow spots on the young leaves in the initial stages of infection. As the disease progresses, the leaves exhibit irregular yellow patches alternating with normal green colour of the leaves. The older leaves of infected plants become chlorotic, noticeably thickened, leathery and brittle. The foliage becomes abnormally red or yellow and often dies.

Pathogen

Beet yellow virus (BYV) and beet mild yellowing virus (BMYV) both can occur alone or together to result in yellows. Beet mild yellowing virus make the plants more susceptible to fungal attack (Powdery mildew).

Mode of spread and survival

The viruses are spread to healthy plants by aphids. Beet yellow virus persists in aphids for few hours, but once infected with beet mild yellowing virus and aphids remains infective for most of its life cycle. The main field vector is *Myzus persicae* but other aphids may spread the viruses, eg. The black bean aphid (Aphid fabae) can also spread BYV but not BMYV.

Management

Control measures include removal of infected plants and weeds from the field. The disease incidence can be minimized by controlling the population of aphids by spraying oxydemeton Methyl 25 EC (2ml/litre of water)

Purple Leaf of Beet virus

Symptoms

This viral disease is caused by a strain of tobacco mosaic virus (TMV). The infected plants are stunted and leaves have a tendency to stand erect and come closer, unlike the healthy

plants where the leaves are broad, long and profuse. Leaves of infected plants show an unusual intense purple colour, white the young emerging leaves show it prominently. Few leaves develop minute necrotic lesions all over the lamina.

Mode of spread

The virus is readily transmissible through sap.

Management

Removal and destruction of virus-infected plants and weed hosts helps in minimizing disease

Radish

Alternaria Blight :*Alternaria raphani*

Symptoms

The pathogen affects leaves, stem, pods and seeds. Symptoms of the disease first appear on the leaves of seed stem in the form of small, yellowish, slightly raised lesions. Lesions appear later on the stems and seed pods. Infection spreads rapidly during rainy weather, and the entire pod may be so infected that the style end becomes black and shriveled. The fungus penetrates in pod tissues, ultimately infecting the seeds. The infected seed fails to germinate.

Pathogen

A. raphani conidia are 70 – 115 x 14 – 18 micron in size.

Mode of spread and survival

It is seed borne. The fungi subsistas mycelium in the infected plant refuse. They also survive in susceptible weeds or perennial crops. The conidia are borne abundantly in moist atmosphere and are disseminated readily by air currents.

Management

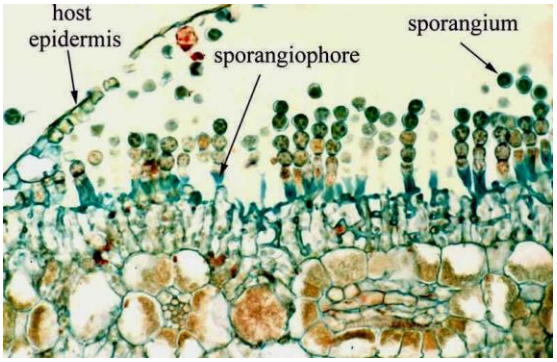
Spraying with Mancozeb 0.25 %

White Rust: *Albugo candida*

Symptoms

Disease attacks the leaves and flowering shoots. Affected flowering shoots get deformed and bear only malformed flowers. White powdery substance in patches is observed on the under surface of the leaves.

Pathogen



Here, Pathogen is an obligate parasite; Mycelium is intercellular producing knob shaped haustoria in the host cells. Each sporangium has 4 to 8 zoospores.

Mode of Spread and Survival

Over wintering may be through oospores in plant debris in the soil and mixed with seeds and perennial mycelium in weed hosts are primary source of inoculum.

Management

Regular spraying with Mancozeb 0.25 % effectively controls the disease.

Cauliflower

Downy Mildew: *Peronospora parasitica*

Symptoms

Downy mildew can cause much of a field of milk white cauliflower curds to develop superficial discolored spots that renders the disease damage heads unmarketable.

Pathogen

Conidiophores are erect, dichotomously branched; conidia are broadly oval, ellipsoidal and hyaline.

Mode of Spread and Survival

The fungus penetrates in the soil through oospores in hosts. Secondary spread of the disease is through water and wind borne conidia.

Management

Seed treatment with Metalaxyl (Apron 6g/kg). Foliar spray with Metalaxyl (Ridomil) @ 0.4 %

Wire stem: *Rhizoctonia solani*

Symptoms:

Wire stem can be a seed problem where cauliflower or other cruciferous transplants are grown crowded together in unsterilized soil or seedling beds. This disease makes the seedlings unsuitable for transplanting since many of the affected plants will die or grow poorly.

Pathogen

The fungus shows branching at right angles near the distal septum in young hyphae. Sclerotia are irregular, brown to black and 5mm in dia. The fungus produces both terminal and intercalary, barrel shaped chlamydospores. In the perfect stage basidia are produced on the host. They are barrel shaped, clavate and have four sterigmata. Basidiophores are hyaline and ellipsoid.

Management

Sterilized soil and seedbed drenches with Copper oxychloride 0.25% will give good disease control

Cabbage

Black leg: *Phoma lingam*

Symptoms

It is caused by *Phoma lingam* and occurs in most regions, specially in areas with rainfall during the growing period. The fungus is carried by the seed and hence it may occur from the early stage. Stem of the affected plant when split vertically, shows severe black discoloration of sap stream. Whole root system decays from bottom upwards. Frequently, the affected plants fall over in the field.

Pathogen

Pycnidia are flask shaped, dark coloured and sometimes with papillate ostiole. Ascocaeps are globose, & Ascospores are biseptate, ellipsoidal.

Mode of Spread and Survival

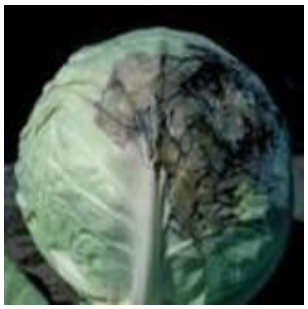
Phoma lingam can survive for up to four years in seed and three years in infected crop debris. The pathogen infects seedlings, forms pycnidia, and produces abundant amounts of spores which exude from the pycnidia in long coils and are splashed to nearby plants to initiate new infections. The disease is favored by wet, rainy weather.

Management

Seed infection can be prevented by spraying the seed plants with copper oxychloride or with an organo mercuric compound. Seed treatment with Captan or Thiram 4g/kg of seed, followed by seed treatment with *Trichoderma viride* 4g/kg. Pusa Drumhead, a cabbage cultivar has been reported to be tolerant under field condition.

Downy mildew: *Peronospora parasitica*

Symptoms



It may attack young plants and also at the seed production stage as being commonly observed in northern India in recent years, when high humidity prevails during seed production stage. The fungus when attacks the young seedlings, discoloration occurs and in severe cases the whole plant perishes. Purplish leaf spots or yellow brown spots on the upper surface of the leaf appear, while fluffy downy fungus growth is found on the lower surface.

Pathogen

It is an obligate parasite. It has large, finger shaped or clavate and branched haustoria. Conidiophores are erect and dichotomously branched. Sterigmata are long, slender and pointed. A single conidium is borne at the tip of each branch. Conidia are brodly oval, ellipsoidal and hyaline. Oogonium is spherical and hyaline. Oospores are globose and yellow in color.

Mode of Spread and Survival

The fungus attacks broccoli, cabbage, cauliflower, radish and turnip. The fungus perennates in the soil through oospores in roots or in old diseased plant parts and as contaminant with seeds. It also persists in perennial hosts. Secondary spread of the disease is through water and wind borne conidia.

Management

Seed treatment with Metalaxyl (Apron 6g/kg of seed). Foliar spraying with Metalaxyl (Ridomil) 0.4%.

Root rot: *Rhizoctonia solani*

Symptoms

Young plants show soft, water soaked lesion on the stem near soil level, the cotyledons wither and the plant eventually falls over and perishes. When infection occurs at a later stage of

growth, the lower part shows discoloration over a length of several centimeters, becomes hard and woody, and thinner than usual as the cortical tissue dies and this phenomenon is known as wire stem.

Pathogen

The fungus shows branching at right angles near the distal septum in young hyphae. Sclerotia are irregular, brown to black and 5mm in dia. The fungus produces both terminal and intercalary, barrel shaped chlamydospores. In the perfect stage basidia are produced on the host. They are barrel shaped, clavate and have four sterigmata. Basidiophores are hyaline and ellipsoid.

Management

Nursery beds: Soil drenching with Methyl bromide @ 1 kg/10 m 2 and covered with polythene sheet. Seed treatment with Captan/Thiram 4g/kg, followed by seed treatment with Trichoderma viride 4g/kg.

Black spot: *Alternaria* sp.

Symptoms

In older plants, leaves, petioles, and stems small, brown to black circular to slightly elongated spots appear. Sometimes the spots join together. It causes damage to cabbage heads and cauliflower curds after maturity and during seed production stage.

Pathogen

The fungal hyphae are branched, septate, inter and intracellular. Conidiophores arise singly or in groups of 2 to 12. They are simple, erect, cylindrical, slightly swollen at base, septate, pale, smooth and 90 x 5 to 8 mm. Conidia are formed in chains of 20 or more. They are cylindrical, muriform, tapering slightly towards the apex and the basal cell is rounded.

Mode of Spread and Survival

Pathogens are seed borne or the conidia are borne abundantly in moist atmosphere and are disseminated readily by air currents.

Management

First foliar spraying with Tridemorph 0.1% followed by spraying with Mancozeb 0.25% a month interval.

Club root: *Plasmodiophora brassicae*

Symptoms

Stunting and yellowing of plants. Leaves become yellowish and wilt on hot days. Club like swelling of root and root lets. Club root is particularly prevalent on soils with a pH below 7, whereas it has been observed that the disease is often less serious on heavy soils and on soils containing little organic matter.

Pathogen

Primary zoospores are anteriorly by flagellate which is of whiplash type. Secondary zoospores are smaller than primary zoospores.

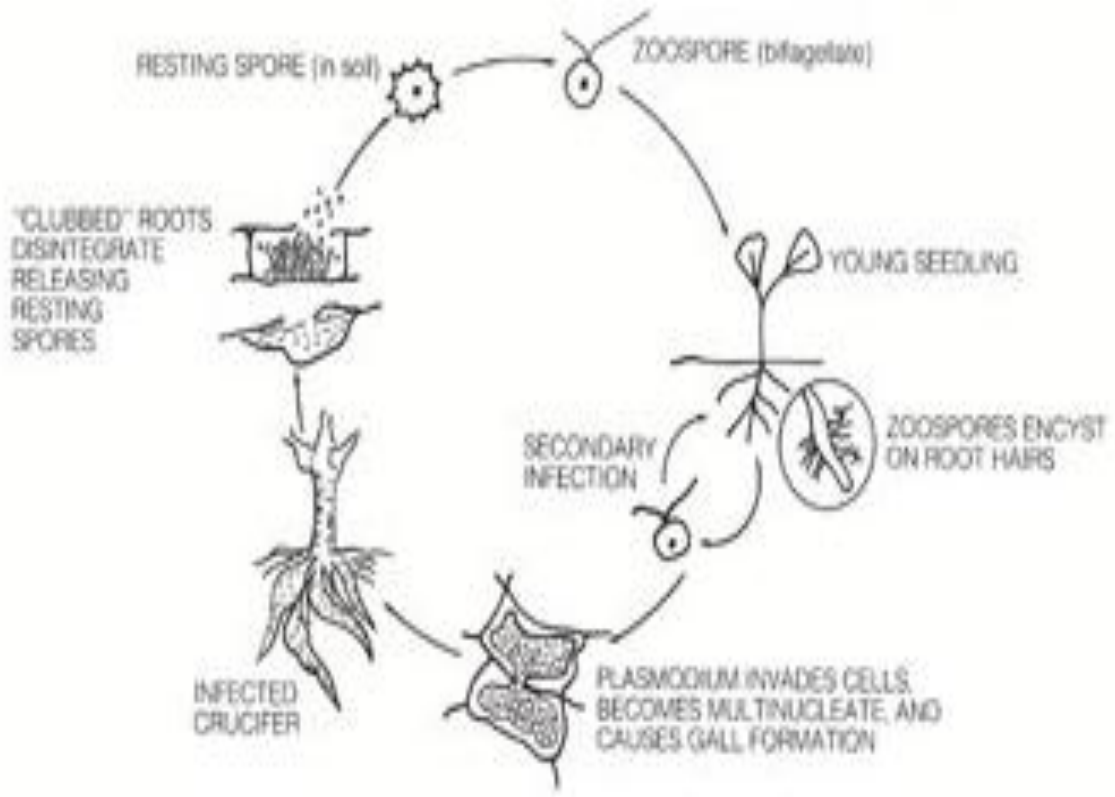
Mode of Spread and Survival

Fungus is soil borne and survival in the crop refuses in the form of minute resting spores for at least 10 years. Contaminated soil can be caused by wheel of implements, carts, tools and on the feet of human being.

Disease Cycle

P. brassicae is capable of surviving in the soil for 7-10 years or longer as resting spores. The resting spores of the fungus can be spread from field to field by infested soil, contaminated water supplies, infected transplants, infested soil on farm machinery, and even by roving animals such as cattle. When soil conditions dictate, the resting spores of the pathogen germinate to

produce zoospores, which are able to "swim" by means of flagella to infect susceptible plant root hairs. The germination of resting spores requires moist, acid soil and can occur over a wide temperature range of 12-27°C. Disease development is favored by high soil moisture and soil temperatures between 18-25°C. Although clubroot has been found in soils exhibiting a wide pH range from 4.5-8.1, the disease is primarily associated with acid soils. Within the infected plant roots, the organism develops rapidly, causing an increase in the number and size of cells, which results in "clubbing." During the development of the organism in the plant, new zoospores are produced; these are capable of infecting the same plant or adjacent plants and, thus, repeating the cycle. Eventually, resting spores are formed within the diseased plant tissue, and these are released into the soil when the plant roots disintegrate.



Management

Soil fumigation with Methly bromide 1kg/10m 2 followed by covering with plastic film. Seed treatment with Captan/Thiram 4g/kg, followed by *T.viride* 4g/kg. Application of lime 2.5 t/ha. Soil drenching with Copper oxychloride 0.25%.

Powdery mildew: *Erysiphe polygoni*

Symptoms

Initially, white tufts of mould arise on the upper surface of the leaves and later run together and the entire leaf becomes covered with greyish white mycelium.

Pathogen

Conidiophores are septate. The cleistothecia are sharp and globose.

Mode of Spread and Survival

The disease spread through water and wind borne conidia.

Management

Spary inorganic sulphur 0.25% or Dinocap 0.05%.

Bacterial diseases

Black rot: *Xanthomonas campestris* pv. *campestris*

Symptoms

The infection of the foliage results in yellow 'V' shaped spots arising along the margin which extend in the direction of the midrib. These spots are associated with a typical black discoloration of the veins. The infection extends through the xylem to the stalk and the vascular bundles turn black. In severe infection, the whole leaf shows discoloration and eventually falls off.

Pathogen

It is gram negative, short rod with rounded ends and non capsulated. It occurs singly, rarely in pairs and motile with single polar flagellum.

Mode of Spread and Survival

Black rot is spread rapidly during warm, humid weather, with an optimal temperature range of 27- 30°C at 80- 100% humidity. Once in the soil, the bacteria are spread by splashing rain and wind. Bacteria enter plants through wounds or natural openings at the leaf margins called hydathodes

Management

Seed treatment with Aureomycin 1000ppm for 30 min is effective in killing both the internally and externally seed-borne pathogen. Drenching the nursery soil with formaldehyde 0.5% helps in checking the disease. Application of bleaching powder at 10.0 to 12.5 kg/ha controls the disease.

Turnip

Alternaria Leaf Spot: *Alternaria* spp.

Symptoms

The pathogen affects leaves, stem, pods and seeds. Symptoms of the disease first appear on the leaves of seed stem in the form of small, yellowish, slightly raised lesions. Lesions appear later on the stems and seed pods. Infection spreads rapidly during rainy weather, and the entire

pod may be so infected that the styler end becomes black and shrivelled. The fungus penetrates in pod tissues, ultimately infecting the seeds. The infected seed fails to germinate.

Pathogen

The fungal hyphae are branched, septate, inter and intracellular. Conidiophores arise singly or in groups of 2 to 12. They are simple, erect, cylindrical, slightly swollen at base, septate, pale, smooth and 90 x 5 to 8 mm. Conidia are formed in chains of 20 or more. They are cylindrical, muriform, tapering slightly towards the apex and the basal cell is rounded.

Mode of Spread and Survival

Pathogens are seed borne or the conidia are borne abundantly in moist atmosphere and are disseminated readily by air currents.

Management

Spraying with Mancozeb 0.25 %

Carrot

Bacterial blight: *Xanthomonas campestris* pv.*carotae*

Symptoms

The bacterium causes irregular brown spot on leaves, dark brown streaks on petioles and a blighting of floral parts. Lesions on foliage begin as small yellow spots. Soon the centre of the spots they become dry and brittle with an irregular halo.

Pathogen

The bacterium is rod shaped and polar flagellum.

Mode of Spread and Survival

The bacterium is borne in and on seed from diseased seed plants. They also live in soil. Rain or irrigation water splashes bacteria from cotyledons or soil to young seedlings. Insects also carry the bacterium mechanically. Under rainy warm conditions, epidermis occur rapidly.

Disease Cycle

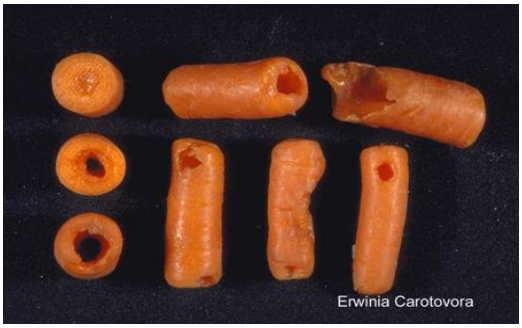
The carrot leaf blight pathogens survive on or in the seed and on diseased crop debris in the soil. The fungal pathogens produce spores that become airborne and are spread predominantly by wind. The bacterial pathogen is spread primarily by wind-driven rain or by irrigation water. Moisture is essential for infection by all blight organisms because bacterial cells and fungal spores require surface moisture and warm temperatures to germinate. The higher the temperature, the shorter the wet period required for infection. When temperatures are warm or when moisture in the form of rain, dew, or irrigation water is persistent, the threat of infection and rapid spread of leaf blight organisms is high.

Management

Spraying early with Copper oxychloride 0.25 %.

Bacterial soft rot: *Erwinia carotovora* sp. *Carotovora*

Symptoms



Cells become water soaked, the middle lamella is destroyed and the cells collapse into a soft, watery slimy mass.The rotted tissues are grey to brown. they may be accomplished by a foul odour. The decay develops most rapidly along the core of the root.

Pathogen

It is large, gram negative and motile with large peritrichous flagella.

Mode of Spread and Survival

Soil is the principal source of primary inoculum for stored carrots. Soil that contains debris from plants that were diseased the previous year is the most important inoculum source. The pathogen lives and multiplies within the soil. If soft rot occurs on carrot roots in fields, the inoculum source can be traced back to carrot foliage from which it moves directly down to the roots. Harvest bruises, freezing injury, fungus invasion and insect wounds offer penetration sites.

Management

Dipping in a solution of 1:500 of sodium hypochlorite before storage or transits reduce the disease.

Cercospora leaf spot: *Cercospora carotae*

Symptom

The first symptom usually appears as elongated lesions along the edge of the leaf segment. Non-marginal lesions appear as small, pin-point chlorotic spots which shows develop into a necrotic center surrounded by a diffuse chlorotic border. Coalescence of spots is common. linear dark lesions develop o n the petiole, sometimes girdling the latter and killing the leaf.

Pathogen

Conidiophores are interminate in growth and show scars where conidia attached. The conidia are slightly obclavate, hyaline and many celled.

Mode of Spread and Survival

The fungus subsists on seed and diseased crop residues. Stromatic masses in diseased tissues are the main source of survival from season to season. They produce conidia which are transmitted by wind or water.

Management

Seed treatment with Captan 4g/kg. Spraying at 10 days interval with Copper oxychloride or Mancozeb.

Sclerotinia Rot or White mold: *Sclerotinia sclerotiorum*

Symptom:

Mycelia growth and sclerotia (red arrow)

Carrots may show little or no damage incidence in the field but following washing and storage white mold outbreaks often occur on the stored roots. Only a small percentage of the roots may be initially infected but the fungus mycelium can move very rapidly from carrot to carrot. In a matter of weeks the whole storage container may become a mass of white mold and black sclerotia surrounding each and every carrot.

Management

Frequent inspection in storage, low temperatures, aeration and washing in a final water of 2-5 % diluted bleach solution may give adequate control (1 part bleach, (sodium hypochlorite) to 20 parts water.

Asparagus

Crown Rot & Seedling Blight: *Fusarium oxysporum* f. sp. *asparagi*

Symptoms

Crown rot coupled with winter injury can reduce newly seeded and established asparagus plantings by up to 50% or more in a year. Infected seedlings will exhibit stunting, yellowing and wilting of the foliage as the primary roots are rotted off. Established plants will produce spindly spears in the spring. Shoots become dwarfed, wilted and brown in color. Later in the season one or more shoots per crown appear stunted, turn yellow, then can wilt and die. Roots are also rotted and discolored.

Management

The disease is seed- and soil-borne. New plantings should be established on soil (well-drained, sand-loam soils are preferred) where asparagus has not been previously grown for at least five years. Use strong healthy plants (1 year crowns) to start a plantation and to ensure good

plant health by following good planting and growing procedures such as fertilization, insect and weed control and avoid over harvesting.

Purple Spot: *Stemphyllium vesicarium*

Symptoms

This disease can render the spears unmarketable by the presence of numerous purplish lesions or spots. The lesions are superficial, slightly sunken and purple. There can also be larger spots that are brown in the middle with a purple margin. Often these lesions will be more prevalent on one side of the spear that the other. On the asparagus fern there will be light brown lesions, up to 15mm long, with dark purple edges. In severe cases, defoliation and dieback can happen. Repeated defoliation can lead to a reduction in yield.

Management

Remove or bury crop residue in the fall to help limit infection.

Rust: *Puccinia asparagi*

Symptoms

Red or brown elongated spots appear on the shoots spears or needles of asparagus. Successive years if infestation reduces root vitality resulting in poor shoot development and death.

Management

Plant in areas with good air circulation and irrigate during the day so plants can dry out before evening.

Lecture 14 - Diseases of Tomato

Damping off: *Pythium aphanidermatum*

Symptoms

Damping off of tomato occurs in two stages, i.e. the pre-emergence and the post-emergence phase. In the pre-emergence the phase the seedlings are killed just before they reach the soil surface. The young radical and the plumule are killed and there is complete rotting of the seedlings. The post-emergence phase is characterized by the infection of the young, juvenile tissues of the collar at the ground level. The infected tissues become soft and water soaked. The seedlings topple over or collapse.

Mode of spread and survival

All the causal organisms are soil inhabitants and they build up in soil with the available hosts. Generally these pathogens have wide host range.

Management

Used raised seed bed. Provide light, but frequent irrigation for better drainage. Drench with Copper oxychloride 0.2% or Bordeaux mixture 1%. Seed treatment with fungal culture *Trichoderma viride* (4 g/kg of seed) or Thiram (3 g/kg of seed) is the only preventive measure to control the pre-emergence damping off. Spray 0.2% Metalaxyl when there is cloudy weather

Fusarium Wilt: Fusarium *oxysporum f. sp. lycopersici*

Symptom

The first symptom of the disease is clearing of the veinlets and chlorosis of the leaves. The younger leaves may die in succession and the entire may wilt and die in a course of few days. Soon the petiole and the leaves droop and wilt. In young plants, symptom consists of clearing of vein let and dropping of petioles. In field, yellowing of the lower leaves first and affected leaflets wilt and die. The symptoms continue in subsequent leaves. At later stage, browning of vascular system occurs. Plants become stunted and die.

Pathogen

Mycelium is septate and hyaline. They produce macro and micro conidia. Micro conidia are one celled, hyaline, ovoid to ellipsoid. Two races of pathogen have been identified.

Mode of spread and survival

The fungus is seed borne and soil borne. The fungus survives in the soil as chlamydospores or as saprophytically growing mycelium in infected crop debris for more than 10 years. One of the chief methods of its distribution is by seedlings raised in infected soil. Wind borne spores, surface drainage water and agricultural implements also help in distribution of the pathogen from field to field.

Management

The affected plants should be removed and destroyed. Spot drench with Carbendazim (0.1%). Crop rotation with a non-host crop such as cereals.

Early Blight : *Alternaria solani*

Symptoms

This is a common disease of tomato occurring on the foliage at any stage of the growth. The fungus attacks the foliage causing characteristic leaf spots and blight. Early blight is first observed on the plants as small, black lesions mostly on the older foliage. Spots enlarge, and by the time they are one-fourth inch in diameter or larger, concentric rings in a bull's eye pattern can be seen in the center of the diseased area. Tissue surrounding the spots may turn yellow.

If high temperature and humidity occur at this time, much of the foliage is killed. Lesions on the stems are similar to those on leaves, sometimes girdling the plant if they occur near the soil line. Transplants showing infection by the late blight fungus often die when set in the field. The fungus also infects the fruit, generally through the calyx or stem attachment. Lesions attain considerable size, usually involving nearly the entire fruit; concentric rings are also present on the fruit.

Pathogen

Mycelium is septate, branched, light brown which become darker with age. Conidiophores are dark colored. Conidia are beaked, muriform, dark colored and borne singly.

Mode of spread and survival

The pathogen is spread by wind and rain splashes. Under dry conditions it survives in infected plant debris in the soil for upto three years and is also seed borne.

Management

Removal and destruction of crop debris. Practicing crop rotation helps to minimize the disease incidence. Spray the crop with Mancozeb 0.2 % for effective disease control.

Septoria Leaf Spot:*Septoria lycopersici*

Symptom

The plant may be attacked at any stage of its growth. The disease is characterized by numerous, small, grey, circular leaf spots having dark border.

Pathogen

Mycelium is septate, branched, hyaline when young and darkens with age. Pycnidia are erumpent. Pycnidiospores are filiform, hyaline and septate.

Mode of spread and survival

The pathogen is spread by wind and rain splashes, insects and on the hands and clothings of tomato pickers. It survives from one season to the next on infested crop debris and also on

solanaceous weeds. The fungus also survives on or in the seed. Seed stocks contaminated with spores produce infected seedlings.

Management

Removal and destruction of the affected plant parts. Seed treatment with Thiram or Dithane M-45 (2 g/kg seed) is useful in checking seed borne infection. In the field spraying with Mancozeb 0.2 % effectively controls the disease.

Bacterial wilt: *Burkholderia solanacearum*

Symptom

This is one of the most serious diseases of tomato crop. Relatively high soil moisture and soil temperature favour disease development. Characteristic symptoms of bacterial wilt are the rapid and complete wilting of normal grown up plants. Lower leaves may drop before wilting. Pathogen is mostly confined to vascular region; in advantage cases, it may invade the cortex and pith and cause yellow brown discolouration of tissues. Infected plant parts when cut and immersed in clear water, a white streak of bacterial ooze is seen coming out from cut ends.

Pathogen

The bacterium is gram negative, rod shaped often occurs in pairs, motile with 1 – 4 flagella. The optimum temperature for growth is 30 - 37˚C.

Mode of spread and survival

The bacterium survives in soil and they spread through irrigation water and by transplanting of infected seedlings. The bacterium survives for 3 years in fallow and for a unlimited period in cultivated land. Chilli, egg plant, grount nut, potato and tobacco are alternative hostswhich help it to survive between tomato crops.

Management

Avoid damage to seedling while transplanting. Apply bleaching powder @ 10kg/ha. Crop rotations, viz., cowpea-maize-cabbage, okra-cowpea-maize, maize- cowpea-maize and finger millet-egg plant are reported effective in reducing bacterial wilt of tomato.

Bacterial Leaf Spot : *Xanthomonas campestris pv. vesicatoria*

Symptom

Moist weather and splattering rains are conducive to disease development. Most outbreaks of the disease can be traced back to heavy rainstorms that occur in the area. Infected leaves show small, brown, water soaked, circular spots surrounded with yellowish halo. On older plants the leaflet infection is mostly on older leaves and may cause serious defoliation.

The most striking symptoms are on the green fruit. Small, water-soaked spots first appear which later become raised and enlarge until they are one-eighth to one-fourth inch in diameter. Centers of these lesions become irregular, light brown and slightly sunken with a rough, scabby surface. Ripe fruits are not susceptible to the disease. Surface of the seed becomes contaminated with the bacteria, remaining on the seed surface for some time. The organism survives in alternate hosts, on volunteer tomato plants and on infected plant debris.

Pathogen

The bacterium is gram negative, short rod shaped and has a single, polar flagellum. Capsules are formed.

Mode of spread and survival

The pathogen survives in the diseased plant debris, volunteer plants. It is seed borne. The bacterium enters through stomata or injuries and lenticels. Secondary spread through rain splashes. Disease spreads to new areas through infected seeds and diseased transplants.

Management

Disease-free seed and seedlings should always be used and the crop should be rotated with non-host crops so as to avoid last years crop residue. Seed treatment with mercuric chloride (1:1000) is also recommended for control of disease. Spraying with a combination of copper and organic fungicides in a regular preventative spray program at 5 to 10 day intervals or Spraying with Agrimycin-100 (100 ppm) thrice at 10 days intervals effectively controls the disease.

Mosaic: *Tomato mosaic virus* **(TMV)**

Symptom

The disease is characterized by light and day green mottling on the leaves often accompanied by wilting of young leaves in sunny days when plants first become infected. The leaflets of affected leaves are usually distorted, puckered and smaller than normal. Sometimes the leaflets become indented resulting in "fern leaf" symptoms. The affected plant appears stunted, pale green and spindly. The virus is spread by contact with clothes, hand of working labour, touching of infected plants with healthy ones, plant debris and implements.

Pathogen

Virus paricles are rod shaped, not enveloped, usually straight and thermal inactivation point is 85 - 90˚C.

Mode of spread and survival

The virus is seed borne and upto 94% of seeds may contain the virus. The virus infection occurs during transplanting It is readily transmissible. Many solanaceous plants are susceptible to tomato mosaic virus. The virus is spread easily by man and implements in cultural operations or by animals and by leaf contact.

Management

Seeds from disease free healthy plants should be selected for sowing. Soaking of the seeds in a solution of Trisodium Phosphate (90 g/litre of water) a day before sowing helps to reduce the disease incidence. The seeds should be thoroughly rinsed and dried in shade. In the nursery all the infected plants should be removed carefully and destroyed. Seedlings with infected with the viral disease should not be used for transplanting. Crop rotation with crops other than tobacco, potato, chilli, capsicum, brinjal, etc. should be undertaken.

Leaf curl: *Tomato leaf curl virus* **(ToLCV)**

Symptom

Leaf curl disease is characterized by severe stunting of the plants with downward rolling and crinkling of the leaves. The newly emerging leaves exhibit slight yellow colouration and later they also show curling symptoms. Older leaves become leathery and brittle. The nodes and internodes are significantly reduced in size. The infected plants look pale and produce more lateral branches giving a bushy appearance. The infected plants remain stunted.

Pathogen

The virus particles are 80nm in diameter.

Mode of spread and survival

It is neither seed nor sap transmissible. But seeds from fresh fruits having infection may have the virus on the seed coat. The virus is transmitted by white fly, *Bemisia tabaci* and grafting. Even a single viruliferous insect is able to transmit the virus.

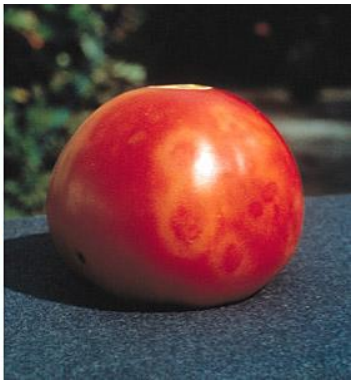
Management

Keep yellow sticky traps @ 12/ha to monitor the white fly. Raise barrier crops-cereals around the field. Removal of weed host. Protected nursery in net house or green house. Spray

Imidachloprid 0.05 % or Dimethoate 0.05% @ 15, 25, 45 days after transplanting to control vector.

Spotted wilt: *Tomato spotted wilt disease* **(TSWV),** *Groundnut bud necrosis virus*

Symptom

It causes streaking of the leaves, stems and fruits. Numerous small, dark, circular spots appear on younger leaves. Leaves may have a bronzed appearance and later turn dark brown and wither. Fruits show numerous spots about one-half inch in diameter with concentric, circular markings. On ripe fruit, these markings are alternate bands of red and yellow.

Pathogen

It is isometric particles of 70 – 90nm diameter. Thermal inactivation point is 40˚C.

Mode of spread and survival

The spotted wilt virus is transmitted through thrips (*Thrips tabaci*, *Frankliniella schultzi* and *F. occidentalis*).

Management

The affected plants should be removed and destroyed. Alternate or collateral hosts harboring the virus have to be removed. Raise barrier crops – Sorghum, Maize, Bajra 5-6 rows around the field before planting tomato. Spray Imidachloprid 0.05% or any systemic insecticide to control the vector.

Gray Mould: *Botrytis cinerea*

Symptoms

Lesion - a watery area with a light brown or tan-colored central region. Converted into a soft, watery mass within a few days. Skin is broken, the grayish mycelium and spore clusters develop within a few hours. Halo forms around the point of entry -small whitish rings approximately - develop on young green fruit. "Ghost spots" are usually single rings but may be solid white spots; the center of which contain dark-brown specks.

Pathogen

Mycelium is septate and branched, hyaline but become dark in color upon age. Conidiophores are branched and bear conidia at the apex. Conidia are continuous or one septate, oblong and dark.

Mode of spread and survival

High relative humidities are necessary for prolific spore production. Optimum temperatures for infection are between 65° and 75° F (18° and 24° C), and infection can occur within 5 hours. High temperatures, above 82° F (28° C), suppress growth and spore production.

Management

Spraying with Bordeaux mixture 1.0 % or mancozeb 0.2% is helpful in reducing the disease. Resistant varieties like Vetomold may be grown in area's where disease appears in an endemic form. Eurocross varities like Antincold, LMRI and Sapsford's No.1 are resistant.

Early Blight : *Alternaria solani*

Symptoms

The fruit become infected-through the calyx or stem attachment, either in the green or ripe stage. Concentric ring present on the fruit surface. Appear leathery and may be covered by a velvety mass of black spores. Infected fruit frequently drop, and losses of 50% of the immature fruit may occur.

Pathogen

Mycelium is septate, branched, light brown which become darker with age. Conidiophores are dark coloured. Conidia are beaked, muriform, dark colored and borne singly. In each conidium 5 – 10transverse and a few longitudinal septa are present.

Mode of spread and survival

The pathogen is spread by wind and rain splashes. Under dry conditions it survives in infected plant debris in the soil for upto three years and is also seed borne.

Management

Disease free seeds should be used for sowing. Seeds soaked in thiram 0.2% at 30˚C for 24h gives better protection. Seed treatment with thiram 2g/ kg gives good protection against seed borne infection. Three sprayings with difolatan 0.2% or mancozeb at fortnightly interval prevent the spread of the disease. Infected plant debris should be removed. Three year rotation with non solanaceous crop is recommended.

Bacterial Soft Rot and Hollow Stem: Erwinia *carotovora* pv. *carotovora*

Symptoms

Fruit -soft watery decay of fruit, starting at one or more points, as very small spots. Enlarge-very rapidly until the entire fruit -soft watery mass. Pathogen liquefies fruit tissue by breaking down the pectate "glue" that holds plant cells together Leakage-internal collapse resembling a shriveled water balloon. Bacteria -single-celled - rapidly multiply and spread-in water. During wet weather and High humidity, Heavy rain fall or irrigation. Warm temperatures in the 73 - 95 F. range

Phoma Rot: *Phoma destructive*

Symptoms

Distinguished from other rots by the black color of this spot .Small, black, pimple-like eruptions. Specks are the pycnidia or fruiting bodies of the fungus. Moderate temperature and high humidity.

Pathogen

The ascospores are irregularly arranged in two series. They are ellipsoid with obtuse ends, hyaline and guttulate. Pycnidia are solitary to gregarious and dark brown. Conidia typically biguttulate, straight and irregular.

Mode of spread

The pathogen is seed borne.

Management

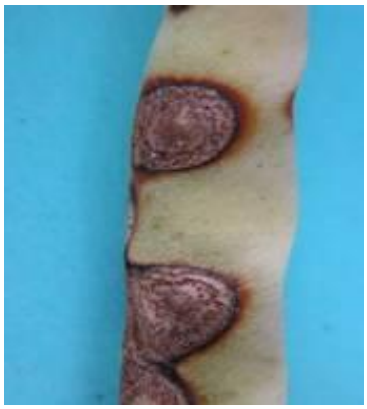
Seed treatment with organomercurial and spraying the crop with zineb 0.2% gives adequate protection against the disease.

Lecture 15 - Diseases of Beans

Beans

Anthracnose: *Colletotrichum lindemuthianum*

Symptoms



Bean pods with black, sunken lesions or reddish-brown blotches most likely have anthracnose, a fungal disease caused by *Colletotrichum lindemuthianum*. Black, sunken lesions about ½ inch in diameter develop on stems, pods and seedling leaves (cotyledons) but are most prominent on pods. A salmon colored ooze on lesions and the veins on lower leaf surfaces turns black. On lima beans, symptoms are sooty- appearing spots on leaves and pods. Anthracnose develops primarily during the spring and fall when the weather is cool and wet, and not during our hot, dry summers. Lima beans are particularly susceptible.

Pathogen

Mycelium is branched, septate , hyaline at first and dark colored with age. Acervuli develop beneath the cuticle. Conidia are borne on short conidiophores. Setae are few, brown and septate. Conidia are one celled, hyaline and cylindrical with rounded ends or with one end slightly pointed.

Mode of spread and survival

The fungus is seed borne and can survive from one season to another in debris from infected plant as well as in diseased seed. The fungus can remain alivein seeds even after the seeds are dead.

Disease Cycle

The fungus survives the winter primarily in bean seed. Survival in soil or in plant residue varies greatly, depending on environmental conditions. Moisture is required for development, spread, and germination of the spores as well as for infection of the plant. A prolonged wet period is necessary for the fungus to establish its infection. The time from infection to visible symptoms ranges from 4 to 9 days, depending on the temperature, bean variety, and age of the tissues. The fungal spores are easily carried to healthy plants in wind-blown rain and by people and machinery moving through contaminated fields when the plants are wet. Frequent rainy weather increases disease occurrence and severity.

Management

Prevent this disease by using certified disease-free seed for planting and removing all plant debris after harvest. Anthracnose can survive in the soil for two years on plant debris or be brought to the garden on infected seeds. Do not plant bean seeds in an area that had disease for two to three years. Avoid overhead watering and avoid splashing soil onto the plants when watering. Fungicide sprays of fixed copper are the only recommended chemical that can be used on lima beans for anthracnose control.

Bean Root Rots: *Rhizoctonia solani*, *Pythium, Fusarium solani*

Symptoms

Many fungi, including *Rhizoctonia solani*, *Pythium* species and *Fusarium solani,* form species *phaseoli,* live in the soil and will infect young seedlings or the seeds of bean plants. Seedlings fail to emerge after planting when the seeds rot in the soil or young seedlings may be stunted.

Plants are usually affected slightly above or below the soil line with a watery soft rot. Roots of the plant usually die and leaves turn yellow.

Management

Do not plant beans in low, poorly drained areas. Plant on raised beds. Plant after the soil has warmed to 69° F at a 4 inch depth. Reduce disease buildup in the soil by rotating locations in the garden where you plant bean or pea with other vegetables. Try to avoid injury to the root system, which often occurs during planting, through cultivation or due to a large population of nematodes in the soil. Remove crop debris immediately after harvest. Plant seeds previously treated with captan. Apply chemicals according to directions on the label.

Rust: *Uromyces appendiculaters*

Symptoms

Bean rust is mainly a disease of bean leaves that causes rust-colored spots to form on the lower leaf surfaces. Severely infected leaves turn yellow, wilt, and then drop off of the plant. Stems and pods may also be infected. This disease is caused by the fungus *Uromyces appendiculaters*. It affects most types of beans under humid conditions.

Pathogen

The fungus is autoecious, thus living its entire life on bean and long cycled rust. Uredia are brown and powdery. Uredospores are globose or ellipsoid. Spore wall is golden brown. Telia are formed on uredia and are dark brown or black.Teliospores are globose or broadly ellipsoid, pedicellate and one celled. Pycnia appear on the yellowish spots on the upper surface of the leaves.

Mode of spread and survival

The rust fungus is not seed borne, but can be disseminated locally by farm tools, insects, animals or other moving bodies. However wind is the principal agent for long distance spore dissemination.

Management

The fungus survives the winter in the soil, on plant debris and even on poles used the previous year. In gardens where rust has been severe, crop rotation is important. As plants begin to bloom, sulfur or chlorothalonil can be sprayed weekly on snap and green beans only. Do not apply chlorothalonil to lima (butter) beans. Wait seven days between spraying and harvest when using chlorothalonil on beans, and 14 days on Southern peas. Apply chemicals according to directions on the label.

Bacterial Blight: *Xanthomonas campestris* pv *phaseoli*

Symptoms

There are two widespread bacterial blights that affect most types of beans, common blight (*Xanthomonas campestris* pv *phaseoli*) and halo blight (*Pseudomonas syringae* pathovar *phaseolicola).* The stems, leaves and fruits of bean plants can be infected by either disease. Rain and damp weather favor disease development. Halo blight occurs primarily when temperatures are cool. Light greenish-yellow circles that look like halos form around a brown spot or lesion on the plant. With age, the lesions may join together as the leaf turns yellow and slowly dies. Stem lesions appear as long, reddish spots. Leaves infected with common blight turn brown and drop quickly from the plant. Common blight infected pods do not have the greenish-yellow halo around the infected spot or lesion. Common blight occurs mostly during warm weather.

Pathogen

The bacteria is gram negative rod, non capsulated and motile with single polar flagellum.

Mode of spread and survival

The pathogen is seed borne and the disease spread through wind splashed rains from diseased to healthy plants. In new area disease spreads through infected seeds.

Management

Both of these diseases come from infected seeds. The diseases spread readily when moisture is present. Avoid overhead watering and do not touch plants when the foliage is wet. The bacteria can live in the soil for two years on plant debris. Do not plant beans in the same location more frequently than every third year. Buy new seeds each year. Fixed copper can be applied at ten day intervals. Wait one day between spraying and harvest.

Mosaic Viruses

Symptoms

Mosaic viruses in which the leaves show sharply defined patches of unusual coloration may occur in beans. The causal agents of these symptoms may be nutrient imbalance or herbicide injury or result from infection by one of several viruses.

Pathogen

Cucumber mosaic virus

Mode of spread and survival

The virus occurs worldwide in many agricultural crops, ornamentals and weeds. Many of these plants serve as reservoirs for season to season survival of the virus. The virus is transmitted mechanically with ease as well as in seed and by aphids, especially *Myzus persicae* and *Aphis gossypii*.

Management

There are no recommended chemical controls for these problems. Many of these viruses are transmitted by aphids and are also transmitted through seed. For this reason it is unwise to save seeds from year to year.

Powdery Mildew: *Erysiphe polygonii*

Symptoms

Leaves are covered with patches of a whitish to grayish powdery growth. This disease is caused by the fungus *Erysiphe polygonii*. New growth appears contorted, curled or dwarfed and may turn yellow and drop. Pods are dwarfed and distorted. This is mostly a problem on fall beans. Powdery mildew is spread by wind and rain.

Pathogen

The mildew pathogen develops mycelial threads between a few cells near the epidermis and grows root like structures, haustoria that slowly withdraw food from the living plant tissue. After the fungus covers the upper and sometimes the lower leaf surface with fungus threads, the threads can produce many short multicellular fungus stalks, each of which bears a few spores resembling beads in a chain.

Mode of spread and survival

The fungus is capable of attacking different leguminous hosts and survives in conidial or perithecial form. The conidia are easily carried by wind, rain and insects. The spores are short lived and usually die in about 2 days if they do not reach a suitable host. When humididity is high and the leaf surface is dry, the spores germinate readily in few hours and the germ tubes enter the plant. Some strains produce sexual perithecia with asci which can remain alive from one season to the next.

Management

Avoid crowding plants by allowing adequate space between rows. On Southern peas, sulfur can be used. When the disease is first noticed, sprays or dusts of sulfur are recommended for use on snap and green beans only. Do not use sulfur on young plants. Apply chemicals according to directions on the label.

Cercospora Leaf Spot: *Cercospora* sp.

Symptoms

This fungal disease, caused by *Cercospora* species, occurs primarily on the lower leaves of plants as irregular, tan spots. Severe infection causes excessive leaf drop and stunting of the plant. Infection is worse during periods of extended rainfall, high humidity and temperatures between 75 to 85° F.

Management

Use disease-free seed for planting. Remove all debris in the garden after harvest. Do not plant beans in the same area for two to three years. There are no resistant varieties or recommended chemicals for this disease in the home garden.

Watery Soft Rot: *Sclerotinia sclerotiorum*

Symptoms

Small, soft, watery spots that are caused by the fungus *Sclerotinia sclerotiorum* occur on the stems, leaves and pods of beans. These spots enlarge rapidly under cool, moist conditions, and run together, girdling the stem. Infected pods turn into a soft, watery mass, before dying out and turning brown. Soon infected areas are covered by a white fungal growth.

Management

Improve air circulation between plants and rows. Too much fertilizer favors heavy vine growth, creating areas for the disease to develop. There are no recommended chemical controls for the home garden.

Lecture 16 - Diseases of Onion & Garlic

Basal Rot: *Fusarium oxysporum* f.sp. *cepae*

Symptoms

The leaves turn yellow and then dry up slowly. The affected plant shows drying of leaf tip downwards. The entire plant shows complete drying of the foliage. The bulb of the affected plant shows soft rotting and the roots get rotted. There will be a whitish mouldy growth on the scale. This disease can begin in the field and continue on in storage.

Pathogen

The fungus produces many chlamydospores which are thick walled resting spores and microconidia which are one celled and thin walled.

Mode of spread and survival

The pathogen is soil borne and the optimum temperatures for development are 28 - 32˚C. Infection occurs through the root either directly or through wounds.

Management

Growers must follow crop rotation and harvested bulbs must be thoroughly cured to reduce potential storage losses. Onions are very sensitive to low soil copper levels. In order to optimize crop production and disease susceptibility, additional soil copper fertility may be needed especially on mucky and sandy soils. Soil drenching with Copper oxychloride 0.25 %.

Downy mildew: *Peronospora destructor*

Symptoms

White downy growth appears on the surface of the leaves. Finally the infected leaves are dried up.

Pathogen

The sporangiophores are non septate, long and swollen at the base.Sporangia are pyriform to fusiform, attached to the sterigmata by their pointed end. These sporangia germinate by one or two germ tubes. The coenocytic mycelium is intercellular with filamentous haustoria. Oogonia are formed in the intercellular spaces.

Mode of spread and survival

The fungus attacks the seed stalks in a seed crop and has been found on and in the seed as mycelium but true seeds do not help in carry over of the fungus from one season to the next. The main sources of perennation are the diseased bulbs used for propagating the crop in many areas and oospores present in diseased crop residues. If infected bulbs are planted, the fungus grows up with the foliage produces sporangia and these spread the disease to other plants.

Disease Cycle

Dormant Period

It is believed that the DM fungus over winters primarily as mycelium in infected onions that remain in onion fields or in nearby cull piles. The pathogen also can over winter in perennial varieties of onion in home gardens. It is suspected that spores of the fungus that persist in the soil may directly infect the roots of young onion plants. These plants become systemically infected and serve as focal points for infection in commercial onion fields.

Primary Spread

When favorable environmental conditions occur, the over wintering fungal mycelium in systemically infected plants produces spores. After dissemination through the air, these spores infect the leaves of onion plants in commercial fields. Spores are formed at night when high humidity and temperatures of 4–25˚C occur, with an optimal temperature of 13˚C. The spores mature early in the morning and are disseminated during the day. Spores remain viable for about 4 days. Germination occurs in free water from 1–28˚C with an optimal range of 7–16˚C. Rain is not needed for infection if heavy dews occur continuously during the night and morning hours.

Secondary Spread

The mycelium of DM in leaves of infected onion plants in commercial bulb production fields produces a new crop of spores called conidia in cycles of approximately 11–15 days. As the upper portions of a leaf are killed, the fungus infects the next lower part of the leaf in each successive cycle of spore formation. Such cycles can be repeated several times until the leaf may

be completely killed. These repeated cycles of spore formation can result in severe and continued epidemics of DM if disease favorable environmental conditions persist.

Management

Three spraying with Mancozeb 0.2 % is effective. Spraying should be started 20 days after transplanting and repeated at 10-12 days interval.

Leaf Blight (Blast)*: Botrytis* spp.

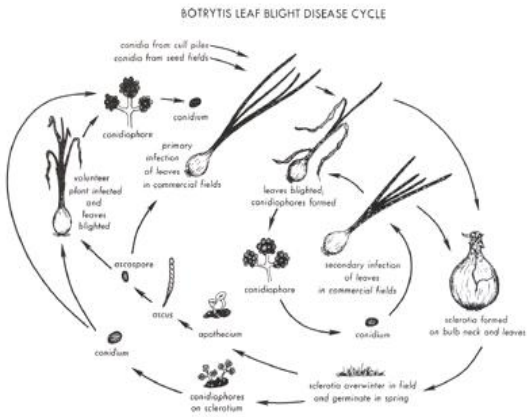
Symptoms

Botrytis is the major disease of onions in cool climate areas. Light infections do not affect yields but heavy infections causing major yield reductions can occur. Hundreds of white specks are seen on the foliage. The disease then spreads very rapidly and tops of the entire crop may be killed.

Pathogen

Botrytis is characterized by its conidiophores which present an appearance of grapebunch. The conidiophores are tall, erect and branches irregularly or dichotomously. They are dark and septate. The terminal cells swell to produce sporogenous ampullae. On each ampulla numerous conidia arise simultaneously on short denticles. The conidia are hyaline or tinted, aseptate and globose to ovoid.

Disease cycle



Dormant Period

The Botrytis leaf blight pathogen over winters as sclerotic (compact mass of fungi capable of surviving unfavorable environmental conditions). These are produced on infected onion bulbs left in cull piles, on mother bulbs stored for seed production, and on bulbs left in the field. The latter results in volunteer onion plants the following spring. Sclerotic also over winter directly in the soil and on leaves that persist as debris in commercial onion fields. The sclerotia are formed on infected leaves and the necks and upper portions of bulbs before or after harvest. Infected leaves may be raked or washed together and persist as leaf tissue debris in which many sclerotic can be found. Sclerotic in the soil result from the disintegration and decay of infected leaves on which sclerotic were formed.

Primary Spread

Sclerotic on onion bulbs in cull piles, on mother bulbs in seed fields, and on volunteer onion plants in commercial onion fields produce conidia (spores) that infect leaves on sprouted bulbs and onion plants in commercial fields. Sclerotic on the surface of the soil in commercial onion fields also produce conidia that can infect the leaves of nearby onion plants. Sclerotia on leaf debris produce conidia and also ascospores (sexual spores) that infect leaves of onion plants. Because ascospores are the result of sexual reproduction, they may serve as the source of new strains of the pathogen that are tolerant to fungicides used to control Botrytis leaf blight. The ability of sclerotic to germinate and produce conidia repeatedly (up to four times) results in the production of conidia over an extended period of time. Sclerotic on the bulbs of volunteer onions produce conidia that infect either leaves of the same plant or those of onion plants in commercial fields. In the absence of seed fields and cull piles, it is suspected that sclerotic in the soil and sclerotic on volunteer plants provide the primary source of inoculum for outbreaks of Botrytis leaf blight in commercial onion fields.

Secondary Spread

The dense, tangled growth of leaves that develop from bulbs in cull piles provides conditions (little air movement and high relative humidity) that are favorable for subsequent production of spores on dead leaf tissue. This results in secondary cycles of infection in the cull piles. Similar secondary cycles occur slightly later in seed fields. Conidia are blown from the seed fields and cull piles to commercial bulb production fields and the disease cycles continue.

Leaves of onion plants in commercial fields can be infected by conidia that develop from sclerotic in the soil.

These infected leaves also serve as secondary sources of inoculum once conidia are produced on the dead leaf tissue. Leaves of volunteer onion plants infected by conidia produced by sclerotic on the same plant also serve as a secondary source of inoculum. Leaves of onion plants grown for commercial production are repeatedly infected, and these serve as the source of inoculum for infecting new leaves throughout the growing season.

Management

Bulb treatment with Captan /Thiram 0.25%. Spraying of Maneb or Mancozeb or Chlorothalonil. Fungicides may be applied every 5 - 7 days for disease control.

Pythium root rot: *Pythium aphanidermatum*, *P. debaryanum and P. ultimum*

Symptoms

This disease causes seed rotting, pre-emergence damping off. The disease appears in circular patches in the field here and there. All the affected plants get killed. if the disease occurs prior to seed germination, it causes gappiness. The seeds or seed materials are killed before their establishment. The disease also occurs after establishment of the crop *i.e* after 15 to 30 days of sowing or planting. This stage is called post-emergence damping off. If the disease occurs very late, it causes stunting of the plant and rotting of the roots.

Pathogen

Pythium aphanidermatum

Epidemiology

The fungus is mainly soil borne. The disease is favoured by ill drained conditions in the field due to stagnation of water. High soil moisture or continuous rain may favour rapid development of the disease.

Management

Seed treatment with Thiram or Captan @ 4g/kg. The bulbs may be dipped in Thiram solution 0.25%. After sprouting, the root region of the plants along the rows should be given a soil drenching with Copper oxychloride 0.25%.

Smut: *Urocystis cepulae*

Symptoms

Black smut sori are seen at the base of the leaves and leaf surface. Black powdery mass is seen after rupturing of sorus wall.

Pathogen

The sori of urocystis cepulae contain dark coloured and powdery spore masses. The spores are found in permentant balls. Each ball consists of an enveloping cortex of tined, sterile, bladder like cells with one or two central dark coloured thick walled chlamydospores. The spores germinate by means of short promycelium while still in the ball.

Mode of spread and survival

The fungus remains viable for 15 years in infected soil by means of spore balls. It persists in soil as a saprophyte. Onion bulbs and onion transplants are important means of widespread distribution of the fungus. Implements also help in the spread. Wind borne soil and surface drainage water are important means of local dissemination.

Management

Seed treatment with Thiram or Captan @ 4g/kg. The bulbs may be dipped in Thiram solution 0.25%.

White Rot: *Sclerotium cepivorum*

Symptoms

The leaves become yellow and die-back and when the plants are pulled up, roots are found to be rotten and the base of the bulb covered with a white or grey fungal growth. Later, numerous small black spherical sclerotia are produced. The bulb of the onion completely rots.

Mode of spread and survival

The disease is worst in warm summers or in the case of winter onions during warm spell in autumn or spring. Sclerotia persist in soil for eight years. The primary inoculum consists of spherical small black sclerotia produced in infested fissure of *Allium* spp. during previous years. Scelerotia are transported from field to field by flood water.

Disease Cycle

The sclerotia that form on the decaying host will lay dormant until a host plant's root exudates stimulate germination, specifically root exudates that are unique to *Allium* spp. Cool weather is also needed for germination of sclerotia and hyphal growth. The soil moisture levels optimal for host root growth are also optimal for sclerotia germination. Mycelium will grow through the soil, and once it encounters a host root the fungus will form appresoria, structures whose purpose is to aid in the attachment and penetration of the host.

Mycelium can grow outwards from the roots of one plant to the roots of a neighboring plant, and it is by this method that the pathogen can move down a planted row. Sclerotia are formed on the decaying host tissue, and once the host tissue completely decays the sclerotia are free in soil. If the bulbs survive long enought to be placed into storage, the pathogen may continue to decay the bulbs if there is high humidity and low temperatures. If the bulbs are stored dry then the disease may not spread but bulbs infected in the field will continue to decay.

Management

Crop rotation and clean seed are the only effective control. Heavy manuring with organic manures reduces the disease in the crop. Seed dressing with Benomyl, Carbendazim or Thiophanate-methyl (100 to 150 g/kg seed) gives effective control.

Purple blotch: *Alternaria porri*

Symptoms

This disease occurs mainly at the top of the leaves, the infection starts with whitish minute dots on the leaves with irregular chlorotic areas on tip portion of the leaves. Circular to oblong concentric black velvety rings appear in the chlorotic area. The lesions develop towards the base of the leaf. The spots join together and spread quickly to the entire leaf area. The leaves gradually die from the tip downwards.

Pathogen

Alternaria porri mycelium is branched, coloured and septate. Conidiophores arise singly or in groups. They are straight or flexuous, sometimes geniculate.

Management

Disease free bulb should be selected for planting. Seeds should be treated with Thiram @ 4 g/kg seed. The field should be well drained. Three foliar sprayings with Copper oxychloride 0.25 % or Chlorothalonil 0.2 % or Zineb 0.2 % or Mancozeb 0.2 %.

Neck Rot *:Botrytis allii, B. squamosa and B. cinerea*

Symptoms

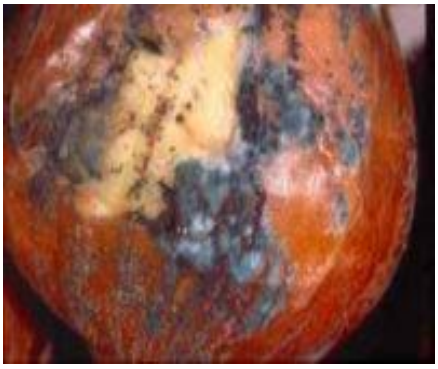
Symptoms usually appear after harvest, although infections originate in the field. Greatest epidemic development occurs when cool (50° to 75°F), moist weather prevails for some days before or during harvest. If the weather remains dry during harvest and curing, losses found in storage are usually small. Symptoms are first seen as a softening of the tissues around the neck of the bulb, or more rarely, at a wound. A definite margin separates diseased and healthy tissues. Infected tissues become sunken, soft, and appear brownish to grayish in color, as if they had been cooked. These symptoms progress gradually to the base of the bulb. Then the entire bulb may become mummified. Hard, irregularly shaped kernel-like bodies, sclerotia, may form between scales, especially at the neck region.

Mode of spread and survival

The fungi that cause neck rot survive the winter on previously infected onion debris in the soil, in cull piles and refuse dumps, and in trash in storage sheds.

Blue mould rot- *Penicillium* sp

Symptoms



Blue mold generally appears during harvesting and storage. Initial symptoms include water soaked areas on the outer surface of scales. Later, a green to blue green, powdery mold may develop on the surface of the lesions. Infected areas of fleshy scales are tan or gray when cut. In advanced stages, infected bulbs may disintegrate into a watery rot. Many species of *Penicillium* can cause blue mold. These fungi are common saprophytes on plant debris and senescent plant tissue.

Pathogen

Penicillium produces an enormous number of spores on a broom like conidiophore. Some of these spores are in the air at all times. They can be carried to long distances by wind. In moist air they germinate readily. Symptoms develop slowly on the bulbs.

Mode of spread and survival

Invasion of onion bulbs and garlic is usually through wounds, bruises, or uncured neck tissue. Once inside the bulb, the mycelium grows through the fleshy scales, eventually sporulating profusely on the surface of lesions and wounds. Optimum conditions include moderate temperatures 70° to 77°F (21° to 25°C) and high relative humidity.

Black mould- *Aspergillus niger*

Symptoms

Infection usually is through neck tissues as foliage dies down at maturity. Infected bulbs are discolored black around the neck, and affected scales shrivel. Masses of powdery black

spores generally are arranged as streaks along veins on and between outer dry scales. Infection may advance from the neck into the central fleshy scales. In advanced disease stages, the entire bulb surface turns black, and secondary bacterial soft rot may make the bulb soft and mushy. No external symptoms may be found with some bulbs.

Management

Seeds should be treated with Thiram @ 4 g/kg seed. The field should be well drained. Three foliar sprayings with Copper oxychloride 0.25 % or Chlorothalonil 0.2 % or Zineb 0.2 % or Mancozeb 0.2 %. Growers must follow crop rotation and harvested bulbs must be thoroughly cured to reduce potential storage losses. Soil drenching with Copper oxychloride 0.25 %

Lecture 17 - Diseases of Coconut and Oil palm

Coconut

Bud rot

Phytophthora palmivora

Symptoms

Palms of all ages are susceptible to the disease, but it is more severe in young palms of 5-20 years. The first indication of the diseases is seen on the central shoot of the tree (spindle). The heart leaf shows discolouration which becomes brown instead of yellowish brown. This is followed by drooping and breading off the heart leaf. With the progress of diseases, more number of leaves get affected with loss of lusture and turn pale yellow. The entire base of the crown may be rotten emitting a foul smell. The central shoot comes off easily on slight pulling.

The leaves fall in succession starting from the top of the crown. The leaf falling and bunch shedding continue until a few outer leaves are left unaffected. But within few months the infection leads to complete shedding of leaves, within subsequent wilt and death of the tree.

Pathogen

The fungus produces intercellur, non septate, hyaline mycelium. Sporangiophores are hyaline and simple or branched occasionally. The sporangiophores are hyaline, Thin walled, pear shaped with a prominent papillae. Sporangia releases reniform, biflagellate zoospores upon germination. The fungus also produces thick walled, spherical oospores. In addition, thick walled, yellowish brown chlamydospores are also produced.

Favorable Conditions

High rainfall, high atmospheric humidity (above 90 per cent), low temperature (18-20˚C) and wounds caused by tappper and Rhinoceros beetles.

Mode of Spread and Survival

The fungus remains as dormant mycelium in the infected tissues and also survives as chamydospores and oospores in crop residues in the soil. The diseases spread is mainly through air-borne sporangia and zoospores. Rainfall also helps in spreading the diseases. Insects and tappers also help in the spread of the inoculum from diseased trees.

Mangement

Remove and burn badly affected trees which are beyond recovery. If diseases is detected in early stage, remove the infected tissue thoroughly by cutting the infected spindle along with two leaves surrounding it and protect the cut portion with Bordeauex paste. Give prophylactic spray with 1% Bordeaux mixture to all the healthy plams in the vicinity of diseases one and also before onset of monsoon rains.

Basal Stem Rot (Thanjavur wilt / Bole rot)

Ganoderma lucidum

Symptoms

The trees in the age group of 10-30 years are easily attacked by the pathogen. The fungus is soil-borne and infects the roots. The most usual symptoms are yellowing, withering and drooping of the outer fronds which remain hanging around the trunk for several months before shedding. The younger leaves remain green for sometime and later turn yellowish brown. The new fronds produced become successively smaller and yellowish in colour which do not unfold

properly. Soft rot occurs in the bud with a bad newly formed leaves wither away. More often the spindle is blown off leaving the decapitated stem.

The wilting plants also show bleeding patches near the base of the trunk. A brown gummy liquid oozes out from the cracks in the tree which slowly result in the death of outer tissues. As the infection advances, fresh bleeding patches appear above the old once, up to 3-5 meters height. The decay of the basal portion occurs slowly and tree succumbs to the diseases in 2-3 years. In the advanced stages of infection, the fungus produces fruiting body (Bracket) along the side of the basal trunk. The roots of wilting trees show discoloration and severe rotting.

Pathogen

The fungus produces a semi circular basidiocarp (bracket), which is attached to the tree with a stalk. The bracket is very big about 10-12 cm diameter and woody. The upper surface is tough, shining, light to dark brown or almost black with concentric furrows. The lower surface is white and soft with numerous minute pores. These pores represent the opening of the hymenial tubes, which are lined with basidia and basidio-spores. Basidiospores are oval, brown and thick walled.

Favourable Conditions

Trees grown in sandy loam and sandy soils, water logging during severe rains, low soil moisture content during summer months and damages caused by weevils and beetles.

Mode of Spread and Survival

The fungus is soil-borne and survives in the soil for long time. The primary infection is through basidiospores in the soil, which attack roots. The irrigation water and rain water also help in the spread of the fungus.

Management

Remove and burn severely infected trees which are beyond recovery. Isolate the diseased trees by digging a trench all around to check further spread. Irrigate the palms at least once in a fortnight during summer months. Apply heavy doses of farm yard manure or compost for green manure at 50 Kg/tree/year along with 5 kg of neem cake. Drench the soil near the tree with 40 litres of 1 per cent Bordeaux mixture at quarterly interval for thrice a year and repeat after 2-3 years. Apply Aureofunginsol 2g+Copper sulphate 1g in100 ml of water or Tridemorph 2ml/100 ml of water through stem injection or root feeding at quarterly intervals for one year.

Stem bleeding

Theilaviopsis paradoxa

(*Ceratocystis paradoxa*)

Symptoms

The characteristic symptom is the exudation of reddish brown fluid from the cracks in the stem. The fluid trickles down to several feet on the stem and the exudates dries up forming a black crust. The tissues below the cracks turn yellow and decay. As the disease progresses, more area underneath the bark gets decayed and the bleeding patch extends further up. The vigour of the tree is affected and nut yield is reduced. The tree is not killed out right but become uneconomical to maintain. In extreme cases, the trees may become barren and die.

Pathogen

The fungus produces two type of conidia. Macroconidia are produced on conidiophores singly or in chains. They are spherical and dark green in colour. Microcondinia are produced endogenously inside the long cells ruptures when mature and release the microcondia in long chain. Microconidia (endoconidia) are thinwalled, hyaline and cylindrical in form. *C. paradoxa* also produces hyaline perithecia with a long neck base is ornamented with knobbed appendages and ostiole is covered by numerous pale-brown, erect, tapering hyphae. Asci are clavate and ascospores are hyaline ad ellipsoid.

Favourable Conditions

Copious irrigation or rainfall followed by drought, shallow loamy soils or laterite

soil with clay or rock layer beneath the soil, poor maintenance of gardens and damages by *Diocalandra* and *Xyleborus* beetles.

Mode of Spread and Survival

The fungus survies in the infected plant debris and soil as perithecia and conidia. The spread is mainly through wind-borne conidia. The irrigation and rain water also help in the disease spread. The beetles which feed on the diseased plants also help in transmission.

Management

Maintain the gardens properly with adequate fertilization. Scoop out the diseased tissue with a portion of healthy tissues, burn the exposed tissue and apply molten coal tar followed by swabbing Bordeaux paste. When stem bleeding is observed in association with *Ganoderma,* follow root feeding or stem injection technique. Irrigate during the summer months.

Root wilt disease (Kerala wilt)

Phytoplasma

Symptoms

Palms of all ages are found infected by the pathogen. The important diagnostic symptom is "flaccidity" of leaves i.e. they curve abnormally inwards, resembling the ribs of mammals. Yellowing of leaves and marginal necrosis of leaflets are also conspicuously. Wilting of leaves from middle whorl to outward and shedding of buttons and immature nuts occur. The size of mature nuts are small with thin kernel. The crown size also gets reduced in advanced stages and trees remain unproductive.

The roots show rotting symptoms, which rot from tip backwards. The older roots show cracks and blotches and cortex turns brownish black resulting in drying in flakes. The root wilt affected palms become highly susceptible to leaf rot disease caused by *Bipolaris halodes.* Occurrence of leaf rot independent of root wilt is very rare. The first symptom is blackening and shrivelling of the distal ends of leaflets in the central spindle and in some of the young leaves. Later the affected portion breaks off in bits giving the leaf a fan-like
appearance. This rotting hastens the decline of the palms.

Pathogen

The disease is caused by Phytoplsama which is frequently identified in the phloem tissues of infected trees.

Favourable Conditons

Sandy and sandy loam soils, severe floods and abundance of lace wing bug
Stephanitis typia.

Mode of Spread and Survival

The severely infected plants serve as primary sources of inoculum. The MLO is transmitted by the lace wing bug *Stephanitis typicuc* from diseased to healthy palms.

Management

Remove all severely infected and uneconomic palms and replant with healthy hybrid seedling like CDO X WCT or WCT X CDO. Remove all the juvenile (young) palms showing symptoms irrespective of its intensity. Spray the leaves with 0.01 per cent Monocroptophos. Apply balanced doses of fertilizers (1kg Urea, 1.7kg Super phosphate, 1.7kg Muriate of potash and 3kg Magnesium sulphate per palm per year in two splits, 1/3 during April-May and 2/3 during September-October for rainfed palms and in 4 splits during January, April, July and October for irrigated palms).

Apply 50kg of farmyard manure/palm/year. Grow green manure crops n basin and incorporate at the time of fertilizer application. Control the leaf rot disease by spraying 1%Bordeaux mixture or 0.3% Mancozeb. Irrigate the palm during summer months at the rate of 600-900 litres of water/basin once in 4 to 6 days. Avoid water logging by providing proper drainage during rainy seasons. Raise crops in the inter space and maintain the milch cows to recycle the manure and other organic wastes to increase the nut yield in affected gardens.

Grey leaf blight

Pestalotia palmarum

Symptoms

Initially symptoms develop only on the outer whorl of leaves, especially in older leaves. Minute yellow spots surrounded by a greyish margin appear on the leaflets. Gradually, the centre of the spots turns to greyish white with dark brown margins with a yellow halo. Many spots coalesce into irregular grey necrotic patches. Complete drying and shrivelling of the leaf blade occur giving a blighted or burnt appearance. Large number of globose or ovoid black acervuli appear on the upper surface of leaves.

Pathogen

The fungus produces conidia inside the acervuli. The acervuli are black in colour, cushion shaped and sub epidermal and break open to expose conidia and black sterile structures, setae. The conidiophores are hyaline, short and simple, bear conidia at the tip singly. The conidia are five celled, the middle three cells are dark coloured, while the end cells are hyaline with 3-5 slender, elongated appendages at the apex of the spore.

Favourable conditions

Ill drained soils, soils with potash deficiency, continuous rainy weather for 4-5 days and strong winds.

Mode of Spread and Survival

The fungus remains in the infected plant debris in soil. The disease is spread through wind-borne conidia

Management

Remove and burn the infected, fallen leaves periodically. Apply heavy doses of potash. Improve the drainage conditions of the soil. Spray the crown with 0.25 per cent copper oxychloride or 1 per cent Bordeaux mixture before the onset of rains.

Oil palm

Anthracnose: *Botryodiplodia palmarum*

Symptoms

This disease occurs in the nursery. It is recognized by regular or irregular brown to black leaf blotches surrounded by yellow haloes, which develop along the margin, centre or tip of the leaves. It causes heavy seedling loss.

Management

The disease can be controlled by spraying Mancozeb or Captan at the rate of 200 g/100 litres of water. Copper fungicides should not be used because of the extreme susceptibility of oil palm seedlings to copper burn (scorching).

Lecture 18 - Diseases of Mulberry

Mulberry

Root rot- *Macrophomina phaseolina*

Symptoms

Sudden wilting, withering of leaves and affected plants fail to sprout after pruning and dry up completely. Affected plants can be pulled out easily. Rotting of primary and secondary roots, rotten roots turn black and roots contain large number of black sclerotia. Decay of root bark.

Management

Uprooting the infected plant and the stump and root portions are burnt. Application of Neem cake @ 1 tonne/ha in four split doses. Application of antagonist *Bacillus subtilis* @ 25 g/plant. Application of antagonist fungus *Trichoderma viride* @ 25 g/plant. Drenching the soil with carbendazim @ 10 ml of 1% concentration per plant

Stem canker- *Lasiodiplodia (Botryodiplodia) theobromae*

Symptoms

Nursery

Failure of cuttings to sprout. Sudden withering and death of sprouts. Discolouration and drying of stems and buds above the soil. Rotting and peeling of bark on stem below the soil surface. Black mycelial threads seen below infected bark and black eruptions on the bark of the infected stem portion.

Grown-up plants

Greyish brown discolouration of the bark at the cut ends of the stem. Delayed sprouting, death of buds and sprouts, black eruptions on the bark in the infected region and death of plants. The above symptoms can be observed a few days after the plants are pruned.

Management

Planting in winter months is avoided. Pre-treatment of cuttings with carbendazim @ 4g/l for a period of 12 h. After pruning, the cut surfaces of the stems should be dressed with a spray/smear of carbendazim @ 4g/l.

Leaf rust - *Cerotelium fici*

Symptoms

Presence of small, irregular reddish to rusty brown spot on older leaves on lower surface. Leaves become yellowish and wither off prematurely.

Management

Providing wider spacing. Spraying carbendazim @ 500-625 g/ha

Leaf spot- *Cercospora moricola*

Symptoms

Brownish circular or irregular leaf spots in the initial stage, enlarge, coalesce and form shot holes in later stage. Severely affected leaves become yellowish and fall off prematurely.

Management

Spraying carbendazim @ 500-625 g/ha

Powdery mildew - *Phyllactinia corylea*

Symptoms

Initially, white powdery patches on lower surface of leaves are seen which later cover the entire leaf surface. Later turn black to brown in colour. Infected leaves turn yellow and fall off. High humidity (>70%) and low temperature (24-26˚C) favour outbreak of the disease.

Pathogen

The fungus produces ectophytic mycelium. It gets nutrition from the host through haustoria sent into the mesophyll tissues. Conidiophores are erect, long and hyaline, which cut off oval shaped conidia at their tips. Cleistothecia are flat, sphere shaped, papillate and bear asci inside.

Mode of spread and survival

The fungus spreads through conidia or ascospores.

Providing wider spacing. Growing resistant varieties like MR1, MR2 and China White Spraying Carbendazim @ 500-625 g/ha. Releasing yellow lady bird beetles and white spotted lady bird beetles, since they feed on the mildew fungus.

Bacterial blight- *Pseudomonas mori*

Symptoms

Numerous irregular water soaked patches on the lower surface of leaf. Leaves become curled, rotten and turn brownish black in colour. Black longitudinal lesions are seen on the bark of young shoots. Yellowing and defoliation.

Management

Uprooting and burning. Spraying 0.1 per cent of Streptomycin or Streptocyclin (safe period is 15 days).

Root knot nematode *Meloidogyne incognita*

Symptoms

Growth and yield of plants affected. Stunted plants, marginal necrosis and yellowing of leaves, necrotic lesions on the root surface. Formation of characteristic knots or galls on the roots. Wilting of plants.

Management

Deep ploughing in summer. Applying neem cake @ 1000 kg/ha. Applying Carbofuran 3G @ 30 kg/ha/year in four split doses (safe period is 50 days).

Lecture 19 - Diseases of Betelvine

Foot rot or Leaf rot or wilt - *Phytophthora parasitica* var. *piperina*

Symptoms

The fungus attacks the vines at all stages of crop growth. Initial symptom is sudden wilting of vines. The affected vines show yellowing and drooping of the leaves from tip downwards. The leaves become dull due to loss of lustre. The affected plant dry up completely within 2 or 3 days. The succulent stem turns brown, brittle and dry as stick. The lower portion of the stem near the soil level shows irregular black lesions upto second or third internode. The diseased intermodes undergo 'wet rot' and the tissue become soft, slimy with a fishy odour. The roots of the affected plants also show extensive discolouration and rotting.

In the young crop, the fungus produces 'Leaf rot' symptoms. The leaves near the soil region show circular to irregular water soaked spots, often starting from the edge. The spots rapidly enlarge and cover a part or whole of the leaf blade, which shows rotting. The leaves turn brown to dark brown or dirty black and defoliation occurs. The leaves with in 2-3 feet height of the vine show the leaf rot symptom.

Pathogen

The fungus produces hyaline, non septate mycelium. The sporangia are thinwalled, hyaline ovate or learn shaped with papillae, measuring 30-40 X 15-20um. Zoospores, which are liberated from the sporangia, are kidney-shaped and biflagellate. Oospores are dark brown, globose and thick walled.

Favourable Conditions

September to February months with high atmospheric humidity and low night temperature (23˚C and below) are highly favorable.

Mode of Spread and Survival

The fungus is soil-borne and survives as facultative saprophyte in the infected plant debris and in the soil as oospores and chlamydospores. The fungus mainly spreads from field to field through irrigation water. The secondary spread is through sporangia and zoospores disseminated by splash irrigation and wind-borne rains.

Management

Select were matured (more one year old) seed vines from fields. Soak the seed vines in Streptocycline 500 ppm + Bordeaux mixture 0.05 per cent solution for 30 minutes. Apply 150 kg N/ha/year through neemcake (75 kg N) and 100 kg P2 O5 through Super phosphate and 50 kg Muriate of potash in 3 split doses, first at 15 days after lifting the vines and second and third dose at 40-45 days interval. Apply shade dried Neem leaf or *Calotrophis* leaves at 2t/ha in 2 split doses and cover it with mud. Collect and destory the infected vines and leaves. Regulate irrigation during the cold weather period. Drench the soil with 0.5 per cent Bordeaux mixture at 500 ml/hill during the cool weather period (October-January) at monthly intervals.

Sclerotium foot rot and wilt - *Sclerotium rolfsii*

Symptoms

The vines of all stages are susceptible to the disease. The infection usually starts at the collar region. Whitish cottony mycelium is seen on the stem and roots. The stem portion shows rotting of tissues at the point of attack and the plants show dropping of leaves and withering finally dry up.

Pathogen

The fungus produces white to grey mycelium which have profuse branching. Sclerotia are spherical smooth and shiny. Brown coloured mustard like sclerotia are seen on the infected stem and soil near the vines.

Favourable Conditions

May-July months with high temperature of 28-30˚C

Mode of Spread and Survival

The fungus is soil-borne and grow saprophytically in the dead plant tissue in soil. The fungus also survives as sclerotia in the infected plant debris in the soil for more than one year. The sclerotia spreads through irrigation water. The pathogen also survives on other hosts like chilli groundnut and brinjal.

Management

Remove the affected vines along with the roots and burn. Apply more of soil amendments like neemcake, mustard cake or farmyard manure. Drench the soil with 0.1 per cent Carbendazim.

Powdery mildew- *Oidium piperis*

Symptoms

The disease affects the crop at all stages of its growth and infection is mainly noticed on tender shoots and leaves. Whitish powdery growth is seen on both the surface of leaves which later enlarges and cover the major portion of the leaves. The affected tender shoots and buds are deformed and shrivelled and margins of leaves tum inwards. When the disease advances, the whitish growth turns to brown blotches and in servere cases, the leaves turn yellow and defoliation occurs.

Pathogen

The fungus is ectophytic and produces profusely branched, hyaline and septate hyphae on the surface of the leaves. The conidiophores are short, club shaped, non-septate and hyaline and produce conidia in chains. Conidia are single celled, hyaline elliptical, and borne over short conidiophore.

Favourable Conditions

Dry humid weather during the months of May-July.

Mode of Spread and Survival

The fungus survives in the infected crop residues in the soil. The primary infection is from soil-borne inoculum. The secondary spread in the field is through wind-borne conidia and carried through splash irrigation.

Management

Collect and burn the infected leaves. Spray 0.2 per cent Wettable Sulphur or dust Sulphur at 25 kg/ha after plucking the leaves.

Anthracnose- *Colletotrichum piperis*

Symptoms

The leaves show small black circular spots initially which later enlarge and develop to a size of 2 cm in size, become concentric and covered with a yellow halo. The affected leaves turn pale yellow and dry up with large black dots in the centre of the spots. Black, circular lesions may develop on the stem, enlarge rapidly and gridle the stem resulting in withering and drying.

Pathogen

The fungus produces large number of acervuli containing short, hyaline conidiophores and block coloured setae. The conidia are single celled, hyaline and falcate.

Mode of Spread and Survival

The fungus remain in the infected plant debris in the field. The primary infection is through the soil-borne conidia, spread by rainwater splash or splash irrigation. The secondary spread in the field is aided by air-borne conidia.

Management

Collect and destory the infected vines and leaves. Spray 0.2 per cent Ziram or 0.5 per cent Bordeaux mixture after plucking the leaves.

Bacterial leaf spot or stem rot - *Xanthomonas campestris* p.v. *betlicola*

Symptoms

The disease initiates as tiny, brown water soaked specks on the leaves surrounded by a yellow halo, which enlarge later and become necrotic and angular, mostly confined to interveinal areas. The infected leaves loose their lustre, turn yellow, show withering and fall off. Under wet weather condition, infection spreads to stem showing small elongated black lesions on lower nodes and inter nodes. These lesions increase in size in both directions and blackening may spreads to the length of several nodes. The stem tissues become weak and break easily at the infected nodes and the vine show withering and drying.

Pathogen

Bacterium is a small rod with a single polar flagellum. It is Gram negative and non-spore forming.

Favourable Conditions

Cloudy weather with intermittent rains and high relative humidity. Two to 3 years old vines are highly susceptible.

Mode of Spread and Survival

The bacteria which are viable in the infected vines and leaves serve as a primary source of inoculum. Rain splashes and splash irrigation water help in the secondary spread.

Management

Remove and burn the infected vines and stubbles in the field. Regulate irrigation during cold weather season. Spray Streptocycline 400 ppm+Bordeaux mixture 0.25 per cent at 20 days intervals, after plucking the leaves.

Lecture 20 - Diseases of Coffee

Coffee

Coffee leaf rust - *Hemileia vastatrix*

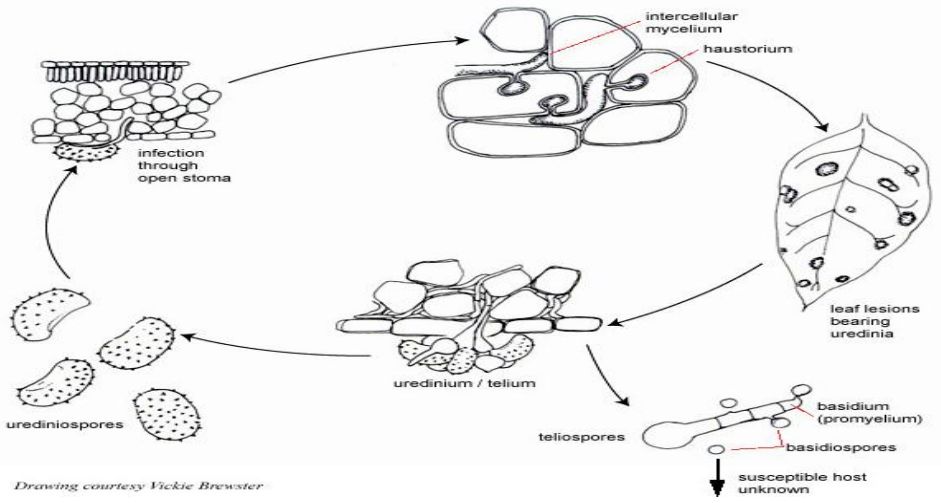
Symptoms

Small pale-yellow spots on the lower surface of infected leaves, orange-yellow spore mass appears, defoliation and die-back. Results in serious crop loss and causes fluctuations in production.

Pathogen

The mycelium is intercellular and sends haustoria into the cells. The mycelium sends out erumpent stalks through stomata which bear the uredospores. The uredospores are reniform or orange segment like in shape. The convex side of the spores are echinulated and the lower side is smooth and measure 26 – 40 x 20 – 30 micron meter. The telial stage succeeds the uredial stage in the later stage.

Disease Cycle



Mode of spread and survival

One lesion produces 1.5 lakhs uredospores which are spread by rain splash and wind. Many animals (insects, birds etc.,) can also carry spores over long distances. Infection requires the presence of water for uredospores germination and only occurs through stomata, which are on the underside of the leaf.

Management

Three applications of 0.5% Bordeaux mixture for susceptible varieties.

Black rot (*koleroga roxia*)

Economic Importance

In India it occurs in Karnataka and Tamil Nadu. In south India the disease is severe only in those areas growing with *C. arabica*. It is influenced by south west monsoon period from June – Sep.

Symptoms

Blackening and rotting of affected leaves, young twigs and berries. Affected leaves get detached and hang down by means of slimy fungal strands. Defoliation and berry drop occur.

Pathogen

The hyphae are hyaline when young and turn light brown with age. Fructifications arise with numerous basidia and basidiospores. Basidia are simple, oval rounded or pyriform. Basidiospores are hyaline, elongated, rounded at one end, slightly concave on one side. At a later stage the fungus forms sclerotia or hyphal clumps by repeated branching of short cells.

Mode of spread and survival

The pathogen penetrates the leaves through the stomata on the lower side and the hyphae invade intercellularly in the palisade tissue. The fungus mostly spreads by contact from leaf to leaf through the vegetative mycelium. The pathogen spread through infected plant debris. Mycelium lies in twigs throughout year.

Management

Remove and burn affected parts. Apply 1% Bordeaux mixture close to the south westerly monsoon if needed. Centre the coffee bushes, regulate the overhead canopy.

Berry blotch

Symptoms

Necrotic spots on the exposed surface of green berries enlarge and cover the major portion. Fruit skin shrivels and sticks fast.

Pathogen

Cercospora coffeicola conidiophores are short, fasiculate and olivaceous. Conidia are subcylindrical, hyaline, 2-3 septate and 40-60x 3.5 micron meter in size.

Mode of spread and Survival

The pathogen is seed borne and conidia are spread by wind.

Management

Spray 1% Bordeaux mixture during june and late august, maintain medium shade overhead.

Damping off / Collar rot – *Rhizocotonia solani*

Symptoms

It caused pre emergence damping off and post emergence damping off. In post emergence damping off collar region near soil level is infected leading the rotting of tissue and death of seedlings.

Mode of spread and survival

The disease is soil borne

Management

Soil drenching with Copper oxychloride 0.25%.

Die back or Anthranose – *Collectorichum coffeanum*

Symptoms

On leaves circular to grayish spots of 2-3 m in dia. On berries small dark coloured sunken spots are farmed. Beans become brown. Die back also occurs.

Mode of spread and survival

The fungus occurs as a saprophyte on dead tissue on the outer layer of the bark, which provides the major source of inoculum. It release large numbers of water borne conidia during the wet season. Conidia are spread by rain water percolating through the canopy and rain splash can disperse conidia between trees. Long distance dispersal occurs primarily by the carriage of conidia on passive vectors such as birds, machinery etc.

Management

Spraying Mancozeb 0.25%

Lecture 21 - Diseases of Tea

Blister blight – *Exobasidium vexans*

Symptoms

Small pale or pinkish circular spots appear on leaves and attain a size of 2.5 cm diameter. The spots in the upper surface of the leaf becomes light brown in color and depressed while in under surface of leaf it bulges farming a blister like swelling. Lower budget portion is covered with a white powdery growth of fungus. When many spots coursers, curling of leaves will occur. When it spreads to young succulent stems affected portion are withered. The leaf yield is reduced vigor of the tea bush is affected.

Pathogen

The mycelium is confined to the blistered areas on the leaves. They are septae and collect in bundles below the lower epidermis. Later by rupturing the epidermisa continuous layer of vertical hyphae are projected on the surface of spot. The fungus produces two kind of spores viz.,the conidia and basidiospores. The conidia are most abundant, borne singly at the tip of long stalks. Basidia are formed on the surface in large number but never form a continuous hymenium.

Mode of spread and survival

The fungus completes its life cycle in 11-28 days and several generations of spores are produced in a season. It produces conidia and basidiospores in the same blister. Spores are air borne. The perpetuation of the fungus appears to be form the pre existing infected bushes.

Management

Removal and destruction of the affected portion. Spraying with Copper oxychloride 0.25 % in effective. Spray with 210 g of COC + 210 g nickel chloride/ha at 5 days interval from June – September and 11 days interval in October – November gives economic control. Spraying with

systemic insecticides like Atemi 50 SL at 400 ml/ha (or) Baycor (300 EC) at 340 ml/ha a weekly interval is also effective. Chlorotalonil, Bayleton, tridemorph is also effective. Tridemorph at 340 and 60 ml/ha is satin factory under mild and moderate rainfall condition.

Black rot

Symptoms

Small dark brown irregular spots appear on leaf. They coalesce to produce a dark brown patch which eventually covers the whole leaf and drop off. Before the leaf turns black the lower surface assumes a white powdery appearance.

Pathogen

Corticium invisum and *C. theae*

Mode of spread and Survival

Basidiospores carried by workers. The disease develops rapidly when temperature is high and air is humid. At the beginning of rainfall they germinate and produce hyphae which start fresh infection.

Epidemiology

Occur in nursery shaded with Crotalaria. Basidiospores germinate only in wet weather or when leaves are covered with dew.

Management

Prune in December end, remove the prunings immediately, burn after drying. Collect all dead and dried leaves. Spray a copper fungide in third week of April.

Red rust: C*ephaleurus mycoidea*

Symptoms

Orange yellow, circular patches appear on upper surface of leaves. The spots become brown and dry up. When it affects the given stem it hardens prematurely.

Pathogen

C*ephaleurus mycoidea* also attacks *Tephrosia* sp. and *Desmodium gyroides* grown as green manure and shade.

Epidemiology

Rainy season is best suited for propagation of algae.

Management

Removal of infected portion and spraying with Copper oxychloride 0.25 %

Black root: *Rosellina areuata*

Symptoms

The fungus originate from the dead heaped leaves of 5 – 7.5 above the soil level. From there if spreads to roots region of tea bushes. When bark is removed star like growth of mycelium can be seen. At the surface of the soil the mycelium surrounds the stem and kills the bank for the length of 7.5 – 10.0 cm. A swollen ring of tissue is formed round the stem above the dead patch.

Pathogen

The fungus produces two kinds of fructification, a conidial stage and a perithecial stage. The conidia are borne on short bristle like stalks. The perithecia are black and spherical. They bear asci which in turn bear ascospores.

Mode of spread

The disease is spread by wind

Management:

Removal and destruction of infected plant. Clean cultivation with out fallen leaves

Dig a drench around the infected bush to provide sunlight in the drench which prevent the spread of mycelium.

Lecture 22 - Diseases of Rose

Black spot- *Diplocarbon rosae*

Economic Importance

Black spot of rose is a serious problem in chill and cold climate of temperate regions. The disease causes marked reduction in the size and number of flowers.

Symptoms

Black lesions with feathery margins surrounded by yellow tissue are found on the leaves. Infected leaves drop prematurely. Purple/red bumpy areas on first year canes may be evident. Plants may be weakened due to defoliation and reduced flower production may be observed.

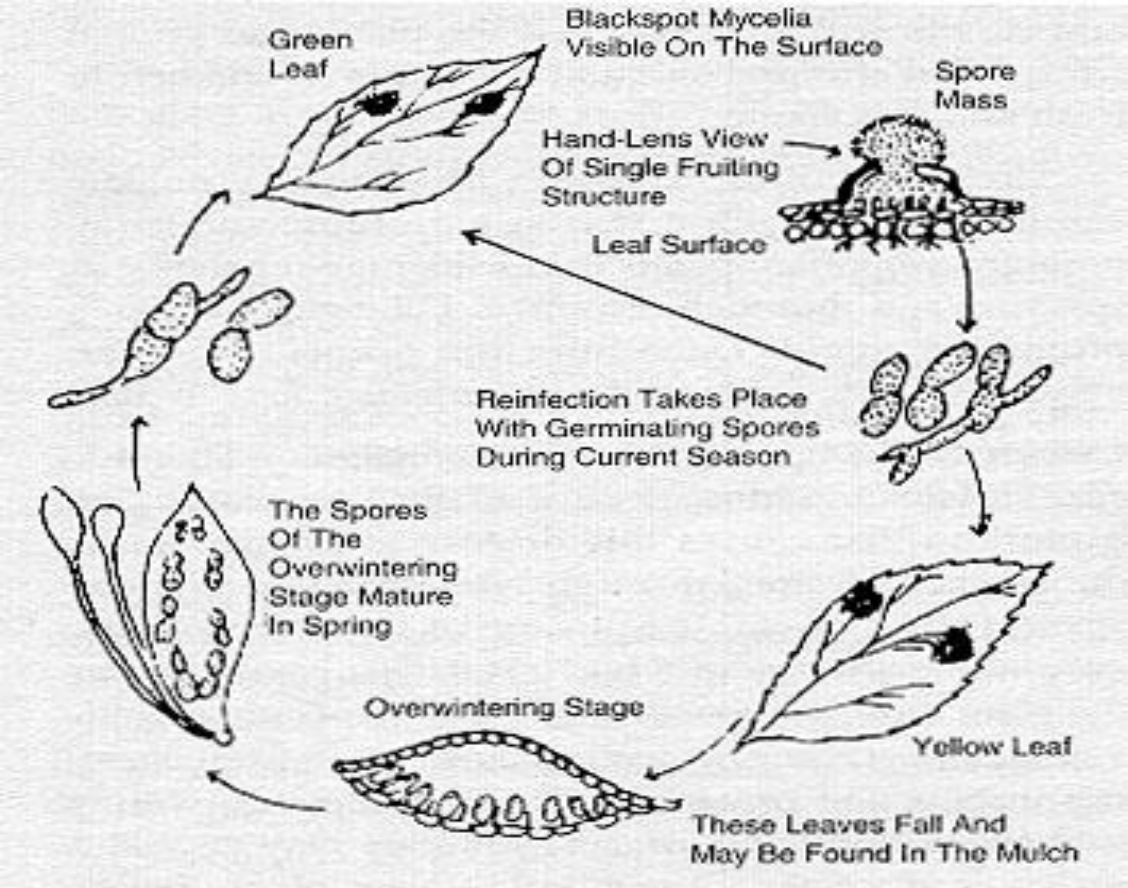
Pathogen

The vegetative body of the fungus consists of two parts *viz*., the subcuticular mycelium and the internal mycelium. The fungus produces acervuli on the central part of the tar spots as blister like projections. Asci are discoid, sub epidermal, erumpent and 84 to 224 micron meter in diameter. Stroma is thin. Conidiophores are hyaline short and cylindrical. Conidia are hyaline, two celled, fusiform or allantoid to obclavate, upper end round, base narrow, guttulate, 18 – 25 x 5 – 6 micron meter.

Mode of spread and survival

The fungal spores are spread primarily by splashing rain or water. Germination of the spores and infection occur when free water remains on the leaf surface for a period of 6 hours or longer. Leaf spots develop within 5 to 10 days.

Disease Cycle



Management

Cultural-Roses should be planted where the sun can quickly dry the night's dew. Space roses far enough apart for good air circulation Avoid overhead watering and keep foliage as dry as possible.Remove infected canes and burn diseased leaves. Spraying with Mancozeb (or) Chlorothalonil 0.2% (or) Benomyl 0.1% or a copper dust .

Powdery mildew – *Sphaerotheca pannosa*

Economic Importance

It is one of the widely distributed disease of rose. Powdery mildew is prevalent during Oct – Jan in south India and Dec- Feb in North India.

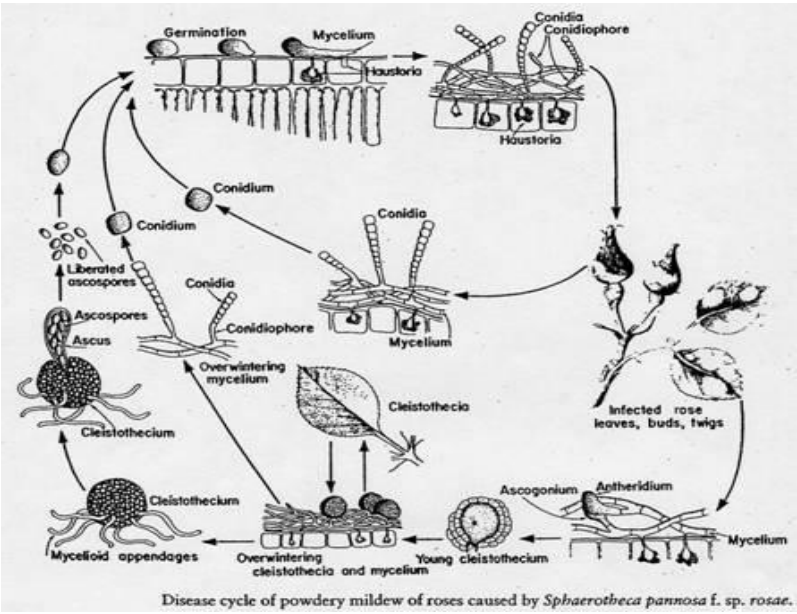
Symptoms

The symptom appears as grayish-white powdery substance on the surfaces of young leaves, shoots and buds. Infected leaves may be distorted, and some leaf drop may occur. Flower buds may fail to open, and those that do may produce poor-quality flowers. It can occur almost anytime during the growing season when temperatures are mild (70 - 80 °F) and the relative humidity is high at night and low during the day. It is most severe in shady areas and during cooler periods.

Pathogen

Mycelium is white , septate, ectophytic and sends globose haustoria into the epidermal cells of the host. Conidiophores are short and erect. Conidia are one celled, oblong, minutely verrucose with many large fat globules and 22.5 – 29.0 x 12.9 to 14.5 micron meter. Cleistothecia are formed towards the end of the season on the leaves, petals, stems and thorns. Cleistothecia are with simple myceloid appendages. Each ascus contains eight ascospores.

Disease Cycle



Disease cycle of powdery mildew of roses caused by *Sphaerotheca pannosa* f. sp. *rosae*.

Mode of spread and survival

The fungus over winters as mycelium in dormant buds and shoots which are not entirely killed. Either conidia or ascospores serve as primary inoculum. The secondary spread is through wind borne conidia.

Management

Collection and burning of fallen leaves. Spray with Wettable sulphur 0.3% (or) Dinocap 0.07% (or) Carbendazim 0.1% 2-3 sprays at 15 days interval is effective. Sulphur dust at 25 kg/ha. Use of sulphur at higher temperature conditions will be phytotoxic.

Die back – *Diplodia rosarum*

Economic Importance

In India it was first reported in 1961 from Delhi. Now it occurs in all the rose growing areas.

Symptoms

Drying of twigs from tip down wards. Blackening of the twigs. The disease spreads to root and causes complete killing of the plants.

Pathogen

The fungus produces round, black pycnidia which bear spores. The pycnidiospores are dark coloured and two celled. Perithecia are immersed in the host tissue and are surrounded by a pseudostroma. Ascospores are ellipsoidal or fusoid, hyaline, two celled with the septum in or near the middle.

Mode of spread and survival

The fungus persists in dead twigs and the stalks of the withered blooms.

Management

Pruning should be done so that lesions on the young shoots will be eliminated. Apply chaubatia pastic in the pruned area. Spray with COC 0.2% (or) Difolatan 0.2% (or) Chlorothalonil 0.2% (or) Mancozeb 0.2%

Rust – *Phragmidum mucronatum*

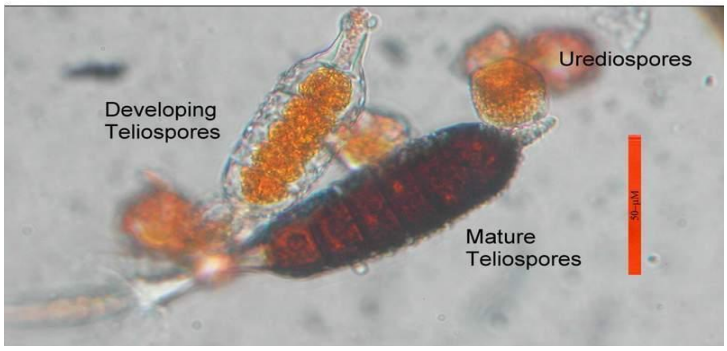
Economic Importance

Rose rust is restricted to higher altitudes. It occurs in Jammu and Kashmir, Himachal Pradesh, Punjab, Tamil Nadu and Uttar Pradesh. Outbreaks of rust disease was reported from Udaipur district of Rajasthan in the variety Chaiti Gulab.

Symptoms

Damage to lemon yellow pustules appear on lower surface of the leaves and stems. Then the color changes to blackish red. The affected leaves turn yellow deformed and fall prematurely. Die back symptom also appear due to weakening of the plant.

Pathogen



Phragmidum mucronatum on rosa sp. Aecidiospores are verrucose, orange yellow, 24 – 25 x 18- 21 micron meter.They are surrounded in the aecidium by club shapped paraphyses. Uredospores are ellipsoid or ovate, echinulate, orange yellow and 21 – 28 x 14 -20 micron meter. The uredospores are borne on short pedicels and are surrounded by paraphyses. Teleutospores are dark coloured, cylindrical, 6- 8 celled with a pointed papilla and 65 – 120 x 30 – 40 micron meter.

Management

Collection and burning of fallen leaves. Spray with Carboxin 0.1% or Wettable sulphur 0.25% or Captan 0.2%

Lecture 23 - Diseases of Chrysanthemum

Wilt -*Fusarium oxysporum* f.sp. *chrysanthemi*

Symptoms

Initial symptoms are in the form of yellowing and browning of leaves. Affected leaves die from the base of the plant upward. Infected plants are stunted and often fail to produce flower. Wilting may cause rotting of root or the base of the stem.

Mode of spread

The fungus is soil borne. The disease spreads through cuttings.

Management

Drenching the soil with or Carbendazim 0.1% is effective. Before planting dipping the rooted cuttings in a solution of Thiram @1.5g/litre of water. Since the disease spreads mostly through cuttings, it is important to use disease free planting material. Disease can further be minimized by following strict sanitation; periodical monitoring; crop rotation and roguing of infected plants.

Rust -*Puccinia chrysanthemi*

Symptoms

The disease symptoms are in the form of brown blister-like swellings, which appear on the undersides of leaves. These burst open releasing masses of brown, powdery spores. Severely infected plants become very weak and fail to bloom properly.

Management

Early removal of infected leaves/plants helps to prevent the further spread of the disease. Spraying the plants with Karathane @ 0.025% or Wettable Sulphur @ 0.3 % is effective in controlling the disease.

Septoria Leaf Spot -*Sepotria chrysanthemella*

Symptoms

Leaf spots occur during cool-wet periods of the rainy season. Since the pathogens are spread through rain splashes the lowermost leaves get infected first. Serious infection may result in premature withering of the leaves; the dead leaves hang to the stem for some time. When flowering starts the infection occurs on flower buds, which rot completely.

Pathogen

Pycnidia are numerous, amphigenous, sub epidermal, globose or lens shaped. Conidia are hyaline, filiform, straight or flexuous often curved or worm like.

Mode of spread and survival

Infected debris in the soil appeared to be primary source of infection or systemic infection carried through suckers. The fungus do not infect other members of the family compositae and is specific to chrysanthemum cultivars only.

Management

This disease can be controlled by spraying Carbendazim 0.1% six times at 15 days intervals from the end of July or spraying Benomyl (0.1%) followed by Captafol (0.2%) Destruction of disease debris and avoiding excessive irrigation is recommended.

Powdery Mildew -*Oidium chrysanthemi*

Symptoms

Infection is more severe in older plants under humid conditions. The growth of the fungus on the leaves appears as powdery coating. Infected leaves turn yellow and dry out. Infected plants remains stunted and fail to flower.

Management

Disease can be effectively controlled with Sulphur fungicides or Captan (0.2%). Good ventilation and proper spacing for free circulation of air is recommended.

Lecture 24 - Diseases of Jasmine and Crossandra

Jasmine

Cercospora leaf spot – *Cercospora jasminicola*

Economic Importance

In India, the disease was first reported in 1946. Now it's known to be widely distributed.

Symptoms

Circular to irregular reddish brown spots of 2-8 mm dia appear on the surface of the leaves. Later the spots become irregular covering larger areas of the leaves.

Pathogen

Stromata are pale to dark brown, globular, filling stomatal openings. Fascicles are mostly dense. Conidiophores are pale olivaceous brown, narrow, sparingly septate and straight or sinuous. It has bluntly rounded tip and are 2 to 4 x 5 to 25 micron meter. Conidia are pale to pale olivaceous obclavate cylindric, indistinctly septate and straight to mildly curved. Its base is obconically truncate and tip is subobtuse and 20 to 66 x 2 to 4 micron meter.

Mode of spread and Survival

It attacks all species of Jasminum. The disease spreads through wind borne conidia.

Management

Spraying with Mancozeb 0.25% (or) Carbendazim 0.1%

Alternaria leaf blight – *Alternaria jasmine, A. alternate*

Symptoms

In the leaves dark brown spots appear. On fumed condition the spots enlarges covering larges area causing blighting of leaves. Concentric rings can be seen the lesions. The disease also affects stem, petiole and flowers.

Mode of spread and Survival

The disease spreads through wind borne conidia.

Epidemiology

The disease attacks Jathi malli (*J. grandiflorum*) and mullai (*J. auriculatum*). The disease is severe during winter months (Oct-Dec). In certain areas the disease is noticed even upto February.

Management

Collection and removal of fallen leaves. Spray with Copper oxychloride 0.25% or Mancozeb 0.25%

Collar rot and Root rot – *Sclerotium rolfsii*

Symptoms

Plants at all stages are infected. First the older leaves become yellow followed by younger leaves and finally death of the plant. In the root black discoloration can be seen. On the infected tissues and stem surface white strands of mycelia and mustard like sclerotia are seen.

Management

Soil drenching with Copper oxychloride 0.25%. Heavy application of FYM with *Trichoderma viride*

Phyllody – *Phytoplasma*

Symptoms

Leaves become small malformed and bushy. In the place of flowers green leaf like malformed flowers are formed.

Mode of spread

The disease is transmitted by grafting and whitefly, *Dialeurodes kirkaldii*.

Management

Selection of cuttings from healthy plants. Spraying insecticide to control the vector.

Lecture 25 - Diseases of Crossandra

Wilt: *Fusarium solani*

Economic Importance

In India it was first reported from Tamil Nadu in 1976. The disease is formed in both air black and sandy loam soil and losses upto 80 % of plants has been reported.

Symptoms

Wilt is observed in patches. In the field the disease is observed one month after transplanting. Leaves of infected plants become pale and droop. Margin of the leaves show pinkish brown discoloration. The discoloration spreads to the midrib in a period of 7 to 10 days. Stem portion gets shriveled. Dark lesions are noticed on the roots extending upto collar region which result in sloughing off the cortical tissue.

Mode of spread and Survival

Chlamydospores survive in soil and they are spread by irrigation water.

Epidemiology

Incidence is more in the presence of root lesion nematode, *Pratylenchus delatrei* and *Helicotylenchus dihystera*.

Management

Affected plants should be pulled out and destroyed to reduce the disease. The nematode can be controlled by soil application of Phorate at the rate of 1 g/plant on 10th day of transplanting. Soil drenching with Carbendazim 0.1 per cent or Copper oxychloride 0.25 per cent on 30 days interval controls the disease. The treatment may be repeated after 3 to 4 weeks if needed.

Stem rot: *Rhizoctonia solani*

The pathogen also causes pre-emergence damping off, Brown to black lesions develop on stem just above soil level and result in girdling of the stem. The lesions extend to the upper part of the stem. The lesions extend to the upper part of the stem and result in collapse of seedlings. The roots are also rotted.

Management

Drenching with Fosesty1-A1 has been found effective in the control of the disease.

Leaf blight: *Colletotrichum crossandrae*

Symptoms

The symptoms of leaves consist of the development of brownish, depressed necrotic areas surrounded by reddish and slightly raised margins. Initially the spots appear as brownish specks but become darker as they expand. The lesions are more prominent on lower leaves and confined to the margins. Infected leaves roll up, shrivel and drop off, leaving a barren stem with a whorl of young leaves at the top.

Management

Spraying with benomyl 0.1% (or) Mancozeb 0.2% (or) Carbendazim 0.1%

Alternaria leaf spot: *Alternaria amaranthi* var. *crossandrae*

Symptoms

This disease was first reported from Tamil Nadu during 1972. Infected leaves show small, circular or irregular yellow spots on the upper surface. They soon enlarge turn brown and develop dark brown concentric rings. Infected leaves become yellow and drop off prematurely.

Management

Spraying with Benomyl 0.1% (or) Mancozeb 0.2% (or) Carbendazim 0.1%.

B.Sc. Ag
V Sem

Diseases of Field and Horticultural Crops and their Management

Credit - 3(2+1)

As per ICAR
5TH Dean Syllabus