

B. Sc. (Hons.) Degree Programme

Department of Plant Pathology

PAT 301- Diseases of Field and Horticultural Crops I

(2017 Syllabus) - Theory

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1. Etiology, symptoms, mode of spread, survival, epidemiology and integrated management of Diseases of rice

Fungal Diseases in Rice

Crop: Rice

Disease Name: Blast

Introduction:

Causal organism: *Pyricularia oryzae* (Syn: *P. grisea*, *Magnaporthe grisea*)

Symptoms: The fungus attacks the crop at all stages of crop growth from seedling to late tillering and ear heading stage. Symptoms appear on leaves, nodes, rachis, and glumes. On the leaves, the lesions appear as small bluish green flecks. The lesions soon enlarge under moist weather to form the characteristic spindle shaped spots with grey centre and dark brown margin (Leaf blast). The spots coalesce as the disease progresses and large areas of the leaves dry up and wither. Spots also appear on sheath. Severely infected nursery and field appear as burnt. Black lesions appear on nodes girdling them. The affected nodes may break up and all the plant parts above the infected nodes may die (nodal blast). During flower emergence, the fungus attacks the peduncle and the lesion turns to brownish-black which is referred to as rotten neck/neck rot/ panicle blast (neck blast). In early neck infection, grain filling does not occur while in late infection, partial grain filling occurs. Small brown to black spots may also be observed on glumes of the heavily infected panicles. The pathogen causes yield losses ranging from 30-61 per cent depending upon the stages of infection.

Rice blast disease- Symptoms



Pathogen: The mycelium is hyaline to olivaceous and septate. Conidia are produced in clusters on long septate, olivaceous conidiophores. Conidia are pyriform to ellipsoid, 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* producing perithecia. The ascospores are hyaline, fusiform, 4 celled and slightly curved.

Favourable conditions: Epidemiology: Intermittent drizzles, cloudy weather, more or rainy high relative humidity (93-99 per cent), low night temperature (between 15-20 C or less than 26 C), availability of collateral hosts and excess dose of nitrogen.

Mode of spread and survival: The disease spreads primarily through airborne conidia since spores of the fungus present throughout the year. Mycelium and conidia in the infected straw and seeds are major sources of inoculum. Irrigation water may carry the conidia to different fields. The fungus also survives on collateral hosts viz., *Panicumrepens*, *Digitariamagrinata*, *Brachiariamutica*, *Leersiahexandra* and *Echinochloacrusgalli*.

Management:

Grow resistant to moderately varieties CO47, IR 20, ADT36, ADT39, ASD 18, IR64 and. Avoid cultivation of highly susceptible varieties viz., IR50 and TGM6 in disease favourable season.

Remove and destroy the weed hosts in the field bunds and channels. Treat the seeds with Captan or Thiram or Carbendazim or Tricyclazole at 2 g/kg or *Pseudomonas*

fluorescens @ 10g/kg of seed. Spray the nursery with Carbendazim 50 WP 25 g or Edifenphos 50 EC 25 ml for 20 cent nursery. Spray the main field with Edifenphos 500 ml or Carbendazim 250 g or Tricyclazole 75 WP 500 g or Iprobenphos (IBP) 500 ml /ha.

Disease Name: Brown Spot or Sesame leaf spot

Introduction:

Causal organism: *Helminthosporium oryzae* (Syn: *Drechslera oryzae*)

(Sexual stage: *Cochliobolus miyabeanus*)

Symptoms: The fungus attacks the crop from seedling to milky stage in main field. Symptoms appear as minute spots on the coleoptile, leaf blade, leaf sheath, and glume, being most prominent on the leaf blade and glumes. The spots become cylindrical or oval, dark brown with yellow halo. Later becoming circular. The several spots coalesce and the leaf dries up. The seedlings die and affected nurseries can be often recognised from a distance by scorched appearance. Dark brown or black spots also appear on glumes leading to grain discolouration. It causes failure of seed germination, seedling mortality and reduces the grain quality and weight.

Pathogen : *H. oryzae* produces brown septate mycelium. Conidiophores may arise singly or in small groups. They are geniculate, brown in colour. Conidia are usually curved with a bulged center and tapered ends. They are pale to golden brown colour and are 6-14 septate. The perfect stage of the fungus is *C. miyabeanus*. It produces perithecia with ascii containing 6-15 septate, filamentous or long cylindrical, hyaline to pale olive green ascospores. The fungus produces terpenoidphytotoxins called ophiobolin A, (or Cochliobolin A), ophiobolin B (or cochliobolin B) and ophiobolin I. Ophiobolin A is most toxic. These breakdown the protein fragment of cell wall resulting in partial disruption of integrity of cell.



Favourable Conditions

Temperature of 25-30 C with relative humidity above 80 per cent are highly favourable. Excess of nitrogen aggravates the disease severity.

Mode of Spread and Survival

Infected seeds and stubbles are the most common source of primary infection. The conidia present on infected grain and mycelium in the infected tissue are viable for 2 to 3 years. Airborne conidia infect the plants both in nursery and in main field. Maximum flight of conidia takes place at a wind velocity of 4.0 - 8.8 hr. Minimum temperature of 27 –28 C, Relative humidity of 90-99% and rainfall of 0.4 -14.4 mm favoured the dispersal of the conidia to maximum extent. The fungus also survives on collateral hosts like *Leersia hexandra*, and *Echinochloa colonum*.

Management

Field sanitation-removal of collateral hosts and infected debris from the field. Use of slow release nitrogenous fertilizers is advisable. Grow tolerant varieties viz., Co44,

Bhavani. Use disease free seeds. Treat the seeds with Thiram or Captan at 4 g/kg. Spray the nursery with Edifenphos 40 ml or Mancozeb 80 g for 20 cent nursery. Spray the crop in the main field with Edifenphos 500 ml or Mancozeb 1 kg when grade reaches 3. If needed repeat after 15 days.

Disease name: Sheath blight

Introduction:

Causal organism: *Rhizoctonia solani* (*Sexual stage: Thanetophorus cucumeris*)

Symptoms

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

Sheath blight symptoms



Pathogen

The fungus produces septate mycelium which are hyaline when young, yellowish brown when old. It produces large number of spherical brown sclerotia. Favourable Conditions

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

Favourable conditions:

- presence of sclerotia or infection bodies floating on the water and presence of the disease in the soil
- Relative humidity from 95 to 100%

- Temperature from 28-32 °C
- High levels of nitrogen fertilizer
- High seeding rate or closing plant spacing
- Frequent rain

Mode of Spread and Survival

The pathogen can survive as sclerotia or mycelium in dry soil for about 20 months but for 5-8 months in moist soil. Sclerotia spread through irrigation water. The fungus has a wide host range.

Management

Apply organic amendments viz., neem cake @ 150Kg/ha or FYM 12.5 tonnes/ha. Avoid flow of irrigation water from infected fields to healthy fields. Deep ploughing in summer and burning of stubbles.. Spray Carbendazim 250 g /ha. Soil application of *P.fluorescens* @ of 2.5 kg/ha after 30 days of transplanting (The product should be mixed with 50 kg of FYM/Sand and applied). Foliar spray at 0.2% at boot leaf stage and 10 days later.(1 Kg/ha).

Disease name: Sheath rot

Introduction:

Causal organism: *Sarocladium oryzae* (Syn : *Acrocylindrium oryzae*)

Symptoms

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 panicles rot and abundant whitish powdery fungal growth is formed inside the leaf sheath.

Sheath rot symptoms



Pathogen

The fungus produces whitish, sparsely branched, septate mycelium. Conidia are hyaline, smooth, single celled and cylindrical in shape.

Favourable Conditions

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

Mode of Spread and Survival

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

Management

Spray Carbendazim 250g or Edifenphos 500ml or mancozeb 1 kg /ha at boot leaf stage and 15 days later. Soil application of gypsum (500 kg/ha) in two splits. Application of NSKE 5% or neem oil 3 % or Ipomoea or prosopis leaf powder extract 25 Kg/ha. First spray at boot leaf stage and second 15 days later.

Disease name: Grain discolouration

Causal organism: *Drechslera oryzae*, *D. rostratum*, *D.tetramera*, *Curvularia lunata*, *Trichocomis padwickii*, *Sarocladium oryzae*, *Alternaria tenuis*, *Fusarium moniliforme*, *Cladosporium herbarum*, *Epicoccum purpurascens*, *Cephalosporium sp.*, *Phoma sp.*, *Nigrospora sp.*

Symptoms

The grains may be infected by various organisms before or after harvesting causing discoloration, the extent of which varies according to season and locality. The infection may be external or internal causing discoloration of the glumes or kernels or both. Dark brown or black spots appear on the grains. The discoloration may be red, yellow, orange, pink or black, depending upon the organism involved and the degree of infection. This disease is responsible for quantitative and qualitative losses of grains.

Grain discolouration symptoms



Pathogen:



Phoma sp.

Curvularia lunata

Favourable Conditions:

High humidity and cloudy weather during heading stage.

Mode of Spread and Survival:

The disease spreads mainly through air-borne conidia and the fungus survives as parasite and saprophyte in the infected grains, plant debris and also on other crop debris.

Management:

Pre and post-harvest measures should be taken into account for prevention of grain discolouration. Spray the crop at boot leaf stage and at 50% flowering with Carbendazim + Mancozeb(1:1) @ 0.2%. Store the grains with 13.5-14% moisture content.

Disease name: False smut

Introduction:

Causal organism: *Ustilaginoidea virens*

Symptoms

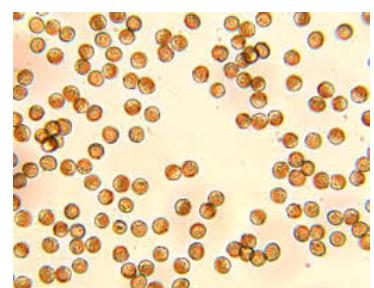
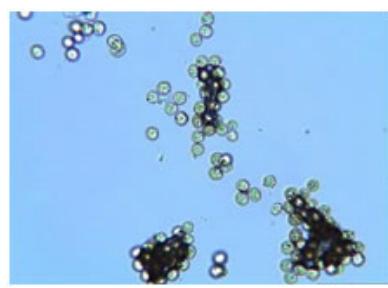
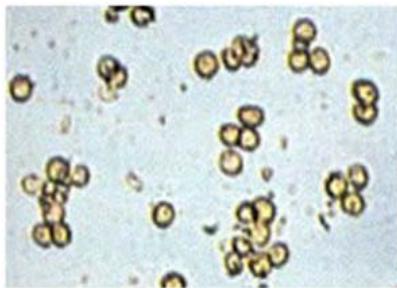
A sporadic disease. The fungus transforms individual ovaries/grains into greenish spore balls of velvety appearance. Only a few spikelets in a panicle are affected.

PATHOGEN:

Chlamydospores are formed as spore balls which are spherical to elliptical, warty and olivaceous.

Favourable conditions:

Rainfall and cloudy weather during flowering and maturity.



Management:

Preventive measures:

Use of disease-free seeds that are selected from healthy crop. Seed treatment with carbendazim 2.0g/kg of seeds. Control insect pests. Split application of nitrogen is recommended. Removal and proper disposal of infected plant debris.

Cultural methods:

Among the cultural control, destruction of straw and stubble from infected plants is recommended to reduce the disease. Early planted crop has less smut balls than the late planted crop. At the time of harvesting, diseased plants should be removed and destroyed so that sclerotia do not fall in the field. This will reduce primary inoculum for the next crop. Field bunds and irrigation channels should be kept clean to eliminate alternate hosts. Excess application of nitrogenous fertilizer should be avoided. Proper Destruction of straw and stubble.

Chemical methods:

Spraying of copper oxychloride at 2.5 g/litre or Propiconazole at 1.0 ml/litre at boot leaf and milky stages will be more useful to prevent the fungal infection. Seed treatment with carbendazim 2.0g/kg of seeds. At tillering and preflowering stages, spray Hexaconazole @ 1ml/lit or Chlorothalonil 2g/lit.

Disease name: Udbatta disease

Introduction:

Causal organism: *Ephelis oryzae* (Sexual stage: *Balansia oryzae-sativa*)

Symptoms

Symptoms appear at the time of panicle emergence. The entire ear head is converted into a straight compact cylindrical black spike like structure since the infected panicle

is matted together by the fungal mycelium. The spikelets are cemented to the central rachis and the size is remarkably reduced. The entire spike is covered by greyish stroma with convex pycnidia immersed inside.



Pathogen

Pycnidiospores are hyaline, needle shaped and 4-5 celled.

Favourable conditions:

- Warm temperature and high humidity
- Early stage of planting from maximum tillering to panicle initiation.

Management

The pathogen is internally seed borne. Hot water seed treatment at 45 C for 10 min. effectively controls the disease. Removal of collateral hosts *Isach neelegans*, *Eragrostis tenuifolia* and *Cynodon dactylon*.

Disease name: Foot rot or Bakanae disease

Introduction:

Causal organism: *Fusarium moniliforme* (Sexual stage: *Gibberella fujikuroi*)

Symptoms

Infected seedlings in nursery are lean and lanky, much taller and die after some time. In the main field, the affected plants have tall lanky tillers with longer internodes and aerial adventitious roots from the nodes above ground level. The root system is fibrous and bushy. The plants are killed before earhead formation or they produce only sterile spikelets. When the culm is split open white mycelial growth can be seen.



Pathogen

Fungus produces both macro and microconidia. Microconidia are hyaline, single celled and oval. Macroconidia are slightly sickle shaped, and two to five celled. The fungus produces the phytotoxin, fusaric acid, which is non-host specific.

Mode of Spread and Survival

The fungus is externally seed-borne.

Management

Treat the seeds with Thiram or Captan or Carbendazim at 2 g/kg.

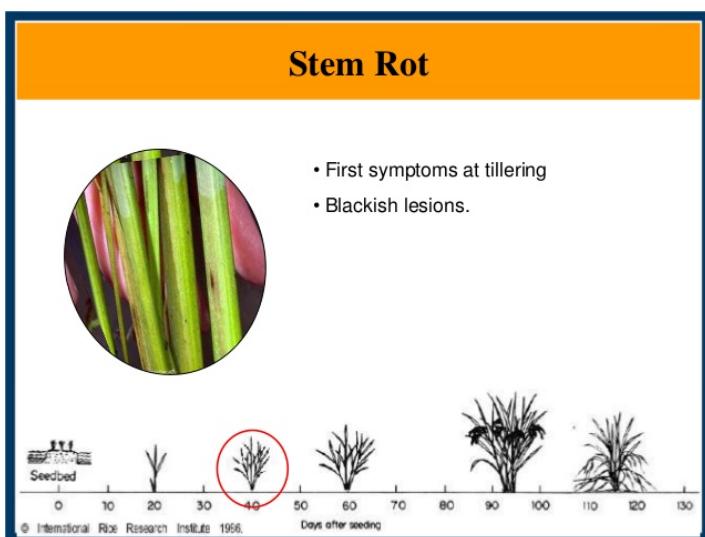
Disease name: stem rot

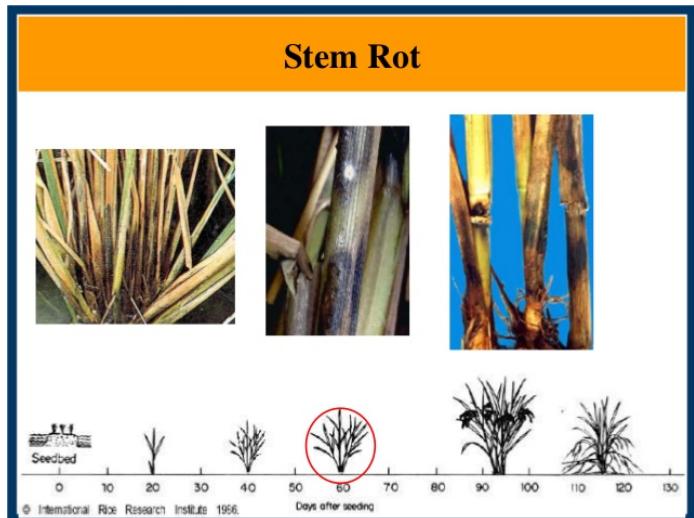
Introduction:

Causal organism: *Sclerotium oryzae* (Sexual stage: *Magnaporthe salvinii*)

Symptoms

Small black lesions are formed on the outer leaf sheath and they enlarge and reach the inner leaf sheath also. The affected tissues rot and abundant small black sclerotia are seen in the rotting tissues. The culm collapses and plants lodge. The sclerotia are carried in stubbles after harvest.





Pathogen

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

Stem Rot

Causal agent

Sclerotium oryzae (A. Cattaneo, Italy 1876)

Geographical distribution:
Irrigated rice growing areas worldwide

Favourable Conditions

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

Mode of Spread of Survival

The sclerotia survive in stubbles and straw that are carried through irrigation water.

Management

Deep ploughing in summer and burning stubbles to eliminate scleritria. Use of balanced application of fertilizer. Avoid flow of irrigation water from infected to healthy fields. Draining irrigation water and letting soil to dry.

Disease name: Stackburn disease

Introduction:

Causal organism: *Trichocomis padwickii* (Syn: *Alternaria padwickii*)

Symptoms:

Leaves and ripening grains are affected. On leaves circular to oval spots with dark brown margins are formed. The center of the spot turns light brown or white with numerous minute dots. On the glumes reddish brown spots appear. The kernels may shrivel and become brittle.

Pathogen

Conidia are elongated with a long beak at the tip, 3 to 5 septate, thick walled and constricted at the septa.

Management

Treat the seeds with Thiram or Captan or Mancozeb at 2g/kg. Hot water treatment at 54 C for 15 minutes is also effective. Burn the stubbles and straw in the field.

Bacterial Diseases of Rice

Crop: Rice

Disease Name: Bacterial Leaf Streak

Introduction:

Bacterial leaf streak occurs in areas with high temperature and high humidity. It is transmitted through seeds and infected stubbles to the next planting season. It can affect the plant during early stages, from maximum tillering to panicle initiation. Mature rice plants can easily recover from leaf streak and have minimal grain yield losses.

Causal organism: *Xanthomonas oryzae* pv. *oryzicola*

Symptoms:



Linear lesions



Water soaked lesions

Fine translucent streaks are formed on the veins and the lesions enlarge lengthwise and infect larger veins and turn brown. On the surface of the lesions, bacterial ooze

comes out and form small yellow band-like exudates under humid conditions. In severe cases the leaves dry up.

Pathogen Character:

The bacterium is aerobic, gram negative, non spore forming, rod with size ranging from $1-2 \times 0.8-1.0 \mu\text{m}$ with monotrichous polar flagellum. Bacterial colonies are circular, convex with entire margins, whitish yellow to straw yellow colored and opaque.

Favourable conditions/Epidemiology:

- ❖ Clipping of tip of the seedling at the time of transplanting
- ❖ Heavy rain, heavy dew, flooding, deep irrigation water
- ❖ Severe wind and temperature of 25-30 C
- ❖ Application of excessive nitrogen, especially late top dressing

Mode of spread and survival:

It is transmitted through seeds and infected stubbles to the next planting season.

The pathogen spreads through irrigation water and also through rain storms.

Management:

Cultural method

1. Burn the stubbles.
2. Use optimum dose of fertilizers.
3. Avoid clipping of tip of seedling at the time of transplanting.
4. Avoid flooded conditions.
5. Remove weed hosts periodically.

Chemical Method

1. Spray Streptomycin sulphate and tetracycline combination 300g + Copper oxychloride 1.25 Kg/ha.

Biological control

Grow resistant cultivars like IR 20 and TKM 6.

Reference Books or links

<http://www.knowledgebank.irri.org/training/fact-sheets/pest-management/diseases/item/bacterial-leaf-streak>

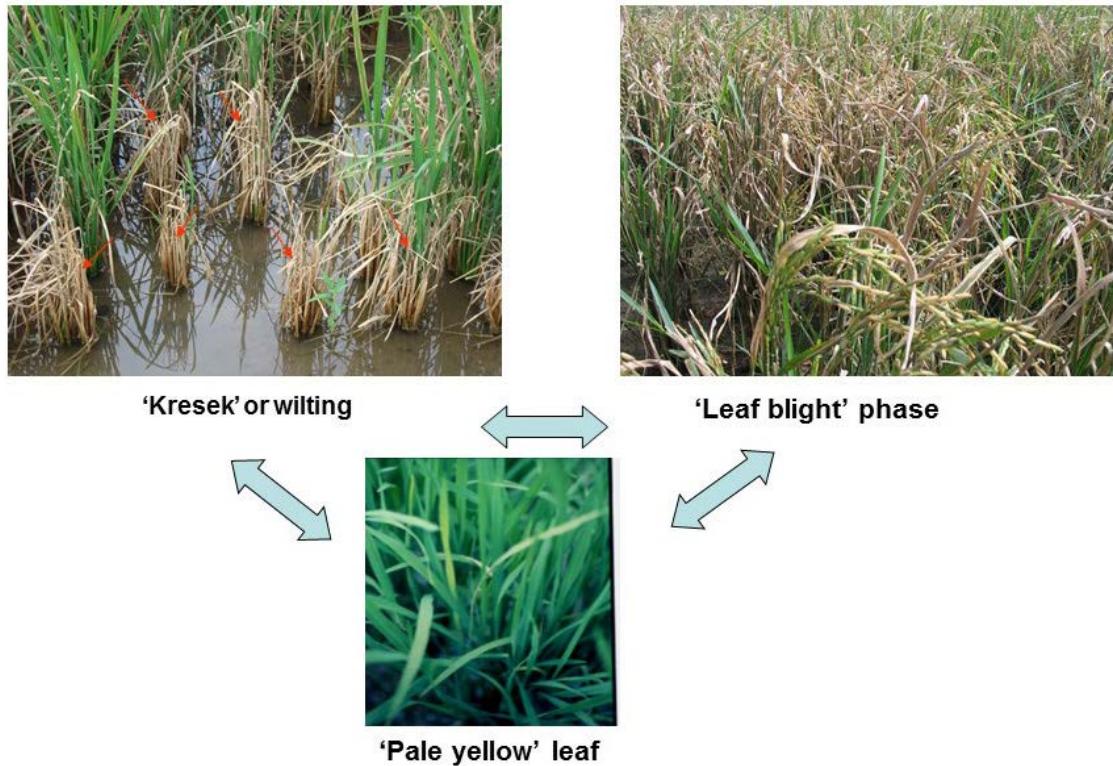
Disease Name: Bacterial Leaf Blight (BLB)

Introduction: The threatening nature of BLB on rice production was recognized only when TN1 and IR8, the first generation of semi dwarf high-yielding modern rice varieties (HYV), were attacked by the disease that it became epidemic in the 1960s. Like other bacterial diseases of rice, BLB is most prevalent in the wet season and in lowland ecosystems. In subtropical regions where double-cropping of rice is common, BB occurs in both rice crops. In temperate regions where most rice is irrigated, the disease is common from July to October during the rainy months, especially after heavy rainstorms or typhoons.

Causal organism: *Xanthomonas oryzae* pv. *oryzae*

Symptoms:

Bacterial blight syndrome exhibits three types of symptoms: leaf blight, kresek (the seedling blight or wilt phase), and the pale-yellow leaf. The disease has been referred to as “bacterial leaf blight” to indicate that the “leaf blight” phase of the syndrome is the most distinct and commonly observed symptom.



The disease is usually noticed at the time of heading but it can occur earlier also. Seedlings in the nursery show circular, yellow spots in the margin, that enlarge, coalesce leading to drying of foliage. **“Kresek”** symptom is seen in seedlings, 1-2 weeks after transplanting. The bacteria enter through the cut wounds in the leaf tips, become systemic and cause death of entire seedling.

In grown up plants water soaked, translucent lesions appear 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 advances, the lesions cover the entire lamina which turns white or straw coloured. 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. If the cut end of leaf is dipped in water, it becomes turbid because of bacterial ooze.

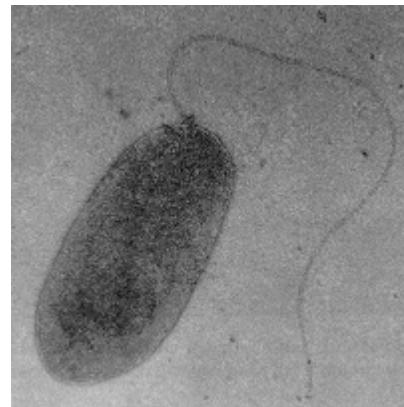
Pathogen Character:

The bacterium is aerobic, gram negative, non spore forming, rod with size ranging from $1-2 \times 0.8-1.0 \mu\text{m}$ with monotrichous polar flagellum. Bacterial colonies are circular, convex with entire margins, whitish yellow to straw yellow colored and opaque.

Bacterium

Favourable conditions/Epidemiology:

- ❖ Clipping of tip of the seedling at the time of transplanting
- ❖ Heavy rain, heavy dew, flooding, deep irrigation water
- ❖ Severe wind and temperature of 25-30 C
- ❖ Application of excessive nitrogen, especially late top dressing



Mode of spread and survival:

The infected seeds as a source of inoculum may not be important since the bacteria decrease rapidly and die in the course of seed soaking. The pathogen survives in soil and in the infected stubbles and on collateral hosts *Leersia* spp., *Plantago major*, *Paspalum dictum*, and *Cyanodon dactylon*. The pathogen spreads through irrigation water and also through rain storms.

Management:

Cultural method

6. Burn the stubbles.
7. Use optimum dose of fertilizers.
8. Avoid clipping of tip of seedling at the time of transplanting.
9. Avoid flooded conditions.
10. Remove weed hosts periodically.

Chemical Method

2. Spray Streptomycin sulphate and tetracycline combination 300g + Copper oxychloride 1.25 Kg/ha.

Biological control

Grow resistant cultivars like IR 20 and TGM 6.

Reference Books or links

<http://www.knowledgebank.irri.org/decision-tools/rice-doctor/rice-doctor-fact-sheets/item/bacterial-blight>

Rice- Viral diseases

Rice Tungro disease

Economic importance

Penyakitmerah which has been known in Malaysia since 1938 was identified as Tungro in 1965. The **mentak** disease of Indonesia is also identified as Tungro. Tungro is commonly found in Bangladesh and India. In India, it is seen in states of West Bengal, Kerala and other parts of India. Tungro is one of the most widely distributed and most destructive diseases in tropical Asia. The loss was estimated during 1940 as 30% or 1.4 million hectares annually. In Thailand a severe epidemic occurred in 1966 affecting more than 3 lakh hectares. An outbreak of Tungro in 1971 affected hundred and thousands of hectares in Philippines. In TamilNadu, the disease was first noticed in Thiruvallur district during 1942.

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 discolouration and interveinal chlorosis. Yellow discolouration is commonly seen in "Japonica" varieties, while "Indica" varieties show orange discolouration. Yellowing starts from the tip of the leaf and may extend to the lower part of the leaf blade. Third leaf from the top most infected plants are taller than other leaves. 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. Sterile panicles are higher than the normal panicles. The infected plants have few spikelets and panicles are small with discoloured grains. RTSV may be symptomless or exhibit mild symptoms.

Detection techniques

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. ELISA and PCR detection facilities are available.

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 RTBV RTSV

discolouration

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

Disease cycle

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 semi-persistent manner. Vectors picks up virus particle within 7 minutes of feeding and transmitted to healthy plants. Males and females nymphs of the insect can transmit the disease in the vector particles are non- circulative and non- propogative.

Favourable factors

Presence of viral sources, vectors. It affects all the stages of crop plants. High temperature favourable for multiplication of vector. So transmission rate and disease spread is very severe during June- July, August- September and March- April.

Management

- Field sanitation
- Summer deep ploughing and burning of stubbles.
- Destroy weed hosts of the virus and vectors.
- Avoid raising nursery near lamp post
- Apply 20 kg of neem cake for 20 cents as basal dose
- Foliar spraying of neem seed extract 5%
- Install light trap @ one/ha
- Install yellow sticky trap @ 12/ha
- Grow disease tolerant cultivars like MTU 9992, 1002, 1003, 1005, Suraksha, Vikramarya, Bharani, IR 36, IET 2508, RP 4-14, IET 1444, IR50 and Co45.
- Control the vectors in the nursery by application of carbofuran granules @170 g/cent, 10 days after sowing and @10kg/ac in main field
- Spray Thiomethoxam 25 WDG 100g/ha or Imidacloprid 17.8 SL 100ml/ ha at 15 and 30 days after transplanting to control leaf hoppers.

Rice Grassy stunt disease - *Rice grassy stunt tenuivirus*

Symptoms

Plants are markedly stunted with excessive tillering and an erect growth habit. Leaves become narrow, pale green with small rusty spots. May produce a few small panicles which bear dark brown unfilled grains.

Pathogen

Rice grassy stunt tenuivirus, flexuous, filamentous 950-1350nm long x 6nm wide, ssRNA genome

Disease cycle Disease spreads by the brown plant hopper, *Nilaparvata lugens*, in a persistent manner having a latent period of 5 to 28 days in the vector. Ratoon crop and presence of vector perpetuate the disease from one crop to other.

Rice dwarf- *Rice dwarf virus*

Symptoms

Infected plants show stunted growth, reduced tillering and root system. Leaves show chlorotic specks turning to streaks along the veins. In early stage of infection no ear heads are formed.**Pathogen**

- The virus is spherical, 70nm diameter with an envelope, dsRNA genome.

Disease cycle

Spreads by leafhopper feeding by *Nephrotettix cincticeps*, *Recllia dorsalis* and *N. nigropictus* in a persistent manner. The transmission is transovarial through eggs. Gramineous weeds *Echinochloa crusgalli* and *Panicum miliaceum* serve as source of inoculum.

Management

- Destory weed host that serve as source of inoculum

- Spray Thiomethoxam 25 WDG 100g/ha or Imidacloprid 17.8 SL 100ml/ ha to control vectors.

Rice ragged stunt disease – *Rice ragged stunt virus*

- Formation of ragged leaves with irregular margins, vein swelling, enations on leaf veins may be formed
- Stunting of plants, delayed flowering, production of nodal branches and incomplete emergence of panicles.

Pathogen

- Spherical virus ([Figivirus](#)), 65 nm diameter, dsRNA genome

Disease Cycle

Spreads through brown planthopper, *Nilaparvata lugens* transmitted in a persistent manner. Multiplies in the vector, latent period of 3 to 35 days, but not transmitted congenitally

Rice Stripe Mosaic Virus

RSMV-infected cultivated rice varieties exhibited slight dwarfing, striped mosaicism, stiff, crinkled or even twisted leaves, an increased number of tillers, delayed heading, cluster-shaped shortening of panicles and mostly unfilled grains. Slight differences in symptom occurrence time were observed under different environmental conditions.

Pathogen and vector

Rice stripe mosaic virus (RSMV) is a tentative new *Cytorhabdovirus* species in family *Rhabdoviridae* transmitted by the leafhopper *Recilia dorsalis*.

Management

- Spray Thiomethoxam 25 WDG 100g/ha or Imidacloprid 17.8 SL 100ml/ ha at 15 and 30 days after transplanting to control leaf hoppers.

Rice Phytoplasma diseases

Crop: Rice

Disease Name: Rice Orange leaf (ROL)

Introduction: ROL was first observed in northern Thailand in 1960. It was widely endemic in the Philippines. In orange leaf-infected rice plants, phytoplasma bodies occur in the phloem tubes. It is transmitted by zig zag leaf hopper in persistent manner.

Causal organism: *Candidatus phytoplasma*

Vector: zigzag leafhopper, *Recilia dorsalis*

Symptoms: Rice plants infected with ROL show orange discoloration beginning at the lower leaves and starting from the leaf tip. The discoloured leaves roll longitudinally and eventually wilt. Rice seedlings inoculated by viruliferous vectors show orange stripes on the outer margin or on one side of the leaf blade near the tip of the leaf 14 to 21 days after the inoculation. Leaves of infected plants show one or more well-defined orange stripes parallel to the midrib or along the veins and turn from light green or orange to nearly yellowish brown starting at the tip. As the disease progresses, the orange color becomes pronounced. The leaves gradually roll inward and dry. Affected plants tend to develop less tillers and poor root systems. Plants show stunting and generally wilt before the flowering stage. Plants infected at the later growth stages develop panicles with high sterility. Infected plants may show diseased tillers and normal looking tillers on one plant.



Favourable conditions/Epidemiology: ROL is generally sporadic and does not cause conspicuous yield loss.

Mode of spread and survival: *R. dorsalis* acquires ROL Phytoplasma in a 3-hour access feeding and become infective after an incubation period of 19-27 days at 17°C, 13-23 days at 25.7°C, and 6-15 days at 28.5°C. Viruliferous *R. dorsalis* transmitted ROLP to rice seedlings in a 30-min access feeding.

Management:

Cultural method

1. Remove weed hosts periodically.
2. Crop rotation
3. Plough the stubbles as soon as the crop is harvested to prevent the survival of orange leaf pathogen during offseason.

Chemical Method:

Vector control with any one of the following insecticides

Thiamethoxam 25 WDG 100g/ha (or) Imidacloprid 17.8 SL 100ml/ha spray at 15 and 30 days after transplanting. The vegetation on the bunds should also be sprayed with the insecticides.

Reference Books or links: 1. Rice diseases IRRI on line source, Vol. II.

Crop: Rice

Disease Name: Rice Yellow Dwarf (RYD)

Introduction: Yellow dwarf of rice (RYD) characterized by chlorosis of leaves and plant stunting occurs widely in Asia. It is generally sporadic and occasionally reaches epidemic proportions in areas under intensive cropping systems.

Causal organism: *Candidatus phytoplasma*

Vector: rice green leafhopper *Nephotettix cincticeps*, *N. virescens* and *N. nigropictus*

Symptoms:

Infected plants showed general chlorosis, pronounced stunting, and profuse tillering. Chlorotic leaves are uniformly pale green or pale yellow. The discoloration first appears on newly developing young leaves and all the succeeding leaves show chlorosis, become soft, and droop slightly. Profuse tillering and plant stunting appear on plants 60-90 days after the inoculation. Infected seedlings are generally alive until maturity. Infected plants produce no panicles or, if produced, plants bear a few, small panicles with mostly unfilled grains. After the rice harvest, infected ratoons develop new leaves with chlorosis and yellowing symptoms. In temperate regions, infected ratoons do not overwinter except in warm regions in warm years.



Favourable conditions/Epidemiology: RYDP is pleomorphic with an average diameter ranging from 200 to 800 μm and bounded with unit membranes 8 nm in thickness. RYD is generally sporadic in the field. It requires a very long incubation period in rice plants for symptom expression and also in the leafhopper vectors to gain infectivity especially in cool seasons. So, dispersal of RYD from primarily infected rice plants to surrounding plants is very slow in the field. The disease cycle of RYD is rather fragile and easily interrupted either in single or double cropping rice fields.

Mode of spread and survival:

Beside rice, *Oryza cubensis*, *Alopecurus aequalis*, and *Glyceria acutiflora* can be infected with RYD. In India, plants of *Oryza barthii*, *O. glaberrima*, *O. longistaminata*, and *O. rufipogon* exposed to RYD-viruliferous GLHs developed symptoms. RYDP is not transmitted through seeds in rice or through the soil. RYD is not mechanically transmissible in rice. RYD was transmitted in persistent manners by GLH.

Management:

Cultural method

1. Remove weed hosts periodically.
2. Crop rotation

Chemical Method: Vector control with any one of the following insecticides

Thiamethoxam 25 WDG 100g/ha (or) Imidacloprid 17.8 SL 100ml/ha spray at 15 and 30 days after transplanting. The vegetation on the bunds should also be sprayed with the insecticides.

Reference Books or links: 1. Rice diseases IRRI on line source, Vol. II.

2. Etiology, symptoms, mode of spread, survival, epidemiology and integrated management of Diseases of maize and sorghum

MAIZE or Corn DISEASES

Smut diseases

Common Smut / Boil smut/ Blister Smut : *Ustilago maydis* (Syn. *Ustilago zeae*)

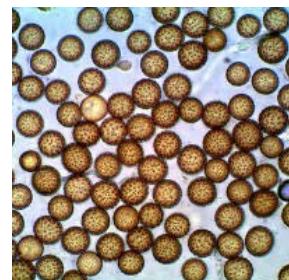
Introduction

Ustilago is a genus of smut fungi. Smuts are called so due to their black spores that resemble soot or smut. Spores masses replace the grain such as wheat, maize, sorghum etc. It is a facultative parasite. There are 400 species of this genus. Its various species infect the ovaries of grasses some time destroying the whole ears and producing a black powdery mass of spores. All species of the *Ustilago* complete their life cycle on the host and therefore known as autoecious parasite.

Symptoms

The most apparent symptoms are tumor-like galls that vary in size from less than 1 cm to more than 30 cm in diameter. All meristematic tissues are susceptible to infection. Galls are found most frequently on ears, tassels, stalks, nodal shoots, and mid-ribs of leaves. Even though galls may form on many above-ground parts of the plant, infection is local (Not systemically).

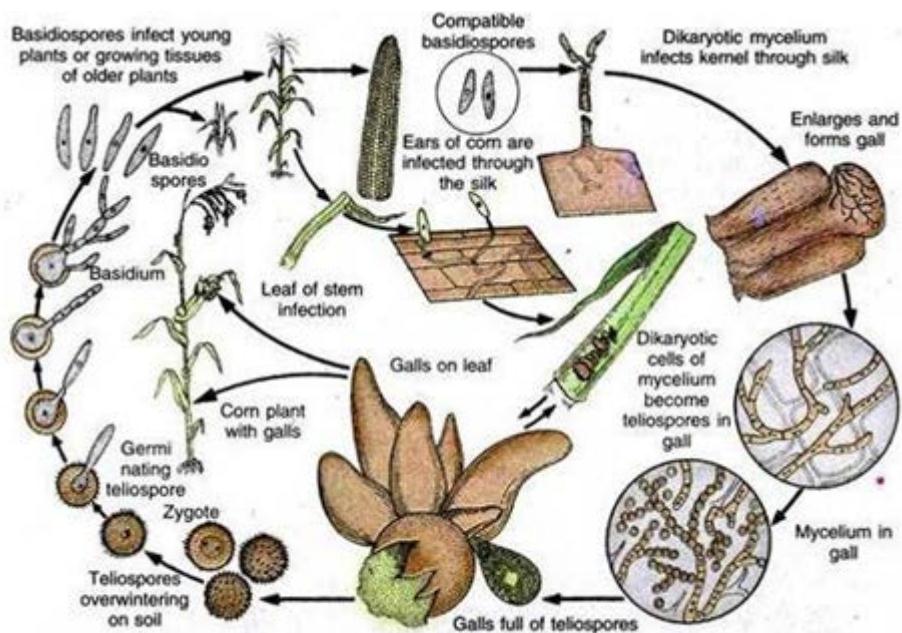
Smut galls consist of fungal and host tissues. Young galls are white, firm and covered with a semiglossy periderm membrane. As galls begin to mature, interior tissue becomes semifleshy and streaks of black tissues occur as teliospores begin to form. With further maturation, galls become a mass of powdery teliospores and the periderm ruptures releasing the spores.



Pathogen

The life cycle of *U.*

maydis includes three distinct stages. Diploid teliospores are formed in galls on infected hosts and are the overwintering propagules of the fungus. They are spherical to ellipsoid, olive-brown to black in colour and heavily echinulate, i.e., covered with tiny spines. When a teliospore germinates, it forms a septate promycelium, undergoes meiosis, and forms haploid sporidia (also called basidiospores) that usually have a single nucleus.



Ustilago maydis. Diagrammatic disease cycle.

Favourable Conditions

Wounding; plant stress that affects pollination

Mode of Spread and Survival

Spores may be spread by wind or splashing rain/irrigation water onto plants where they may infect through wounds or silks. The fungus overwinters as diploid teliospores in crop debris or soil. The smut spore retains its viability for two years. The fungus is externally seed-borne and soil-borne.

Management

crop rotation, sanitation, seed treatments, application of foliar fungicides, modification of fertility. Field maize hybrids are somewhat less susceptible to the disease compared to sweet corn hybrids. If common smut is a consistent problem in sweet corn, switching to a hybrid with more resistance can reduce disease incidence.

Huitlacoche - edible galls of *Ustilago maydis*

Since the fungus that causes common smut is closely related to other fungi that produce mushrooms, the smut galls themselves have some of the same characteristics that make them desirable for eating. In Mexico, common smut galls are commonly eaten as a delicacy and referred to as *huitlacoche* (or sometimes *cuitlacoche* or Mexican and/or maize truffles). Smutted ears are sold for more than 30 times the value of healthy ears in some Mexican produce markets. In the United States, restaurants now offer some selections on their menus and canned cuitlacoche is available in some popular grocery stores.



2. Head Smut: *Sphacelotheca reilina*

Symptoms

- fungus causing head smut infects plants systemically
- Symptoms are usually noticed on the cob and tassel.
- Large smut sori replace the tassel and the ear. Sometimes the tassel is partially or wholly converted into smut sorus.
- Infected ears and tassels may be completely replaced with smut sorus filled with large quantities of black, dusty teliospores
- The smutted plants are stunted in growth, produce little yield and remain greener than that of the rest of the plants. Fungal growth progresses into actively growing meristematic Tissue and the seedling plants become systemically infected, though infection is only obvious in flowering plants and later in the mature ears with the development of large amounts of teliospores.



Favourable Conditions

Cool and dry weather that delays seedling growth promotes initial infection of seedling roots by the head smut fungus. Warm (22-26° C) and relatively dry soil conditions favour fungal infection as well.

Epidemiology

The fungus survives as thick-walled dark brown to black spiny teliospores in the soil for several years, but not like common smut, the head smut teliospores are not contained within conspicuous fleshy galls.

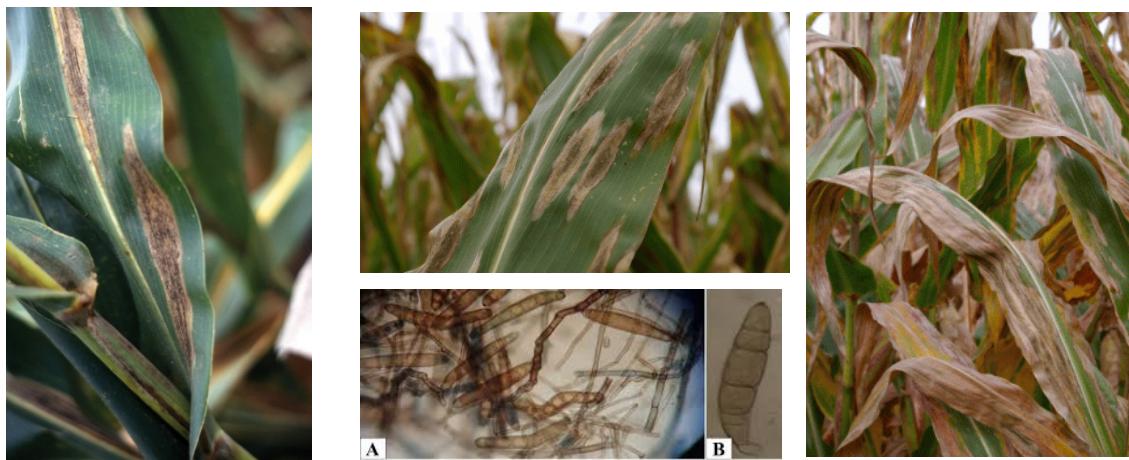
Management

Crop rotation, sanitation, seed treatments, application of foliar fungicides, modification of fertility. Hybrids vary in their susceptibility/resistance to the fungus that causes head smut.

3. Turcicum Leaf blight (TLB) - *Exserohilum turcicum*

Anamorph of *Setosphaeria turcica*

Introduction: Northern corn leaf blight (NCLB) is a ubiquitous foliar wilt disease that threatens maize production worldwide (Welz and Geiger, 2000). The disease is caused by the hemibiotrophic fungus *Exserohilum turcicum* which favors a high-humidity and cool temperature environment. The pathogen is the fungus *Exserohilum turcicum* (syn. *Helminthosporium turcicum*). It overwinters on corn leaf debris. Under favorable conditions, fungal infection manifests itself as large and irregularly emerging lesions that destroy the entire foliage.



Symptoms

Leaf Blight is the long cigar-shaped grey-green to tan-coloured lesions on the lower leaves.

- The tan lesions of northern corn leaf blight are slender and oblong tapering at the ends ranging in size between 1 to 6 inches.
- Lesions run parallel to the leaf margins beginning on the lower leaves and moving up the plant. They may coalesce and cover the entire leaf.
- Spores are produced on the underside of the leaf below the lesions giving the appearance of a dusty green fuzz.
- As the disease develops, the lesions spread to all leafy structures, including the husks. The lesions may become so numerous that the leaves are eventually destroyed causing major reductions in yield due to lack of carbohydrates available to fill the grain. The leaves then become greyish-green and brittle, resembling leaves killed by frost.

Yield losses can reach as high as 30-50% if the disease establishes itself before tasseling.

Pathogen

Mycelium appeared septate, branched and brownish while the Conidiophores was simple, cylindrical and septate. Variations were observed on isolates pigmentation, growth pattern and **conidia** sizes. Conidia of the fungus are olive grey and spindle shaped, curved, elongated and measuring 5 x 20 µm with one to nine septa. Conidiophores are simple, cylindrical and olivaceous brown [14]. Germination of *E. turcicum* conidia is bipolar and occurs 3-6 hours after inoculation.

Germ tubes are 20-150 µ long and in general, grow at an angle rather than parallel to the veins of the leaf . It has a single conidium formed terminally on the conidiophore, which resumes growth to produce new conidium at the new tip . Conidia has a hilum that protrudes distinctly from the conidia to bluntly rounded basal cells useful for identification.

Favourable Conditions

- Wet humid cool weather typically found later in the growing season.

Mode of Spread and Survival

- Spores of the fungus that causes this disease can be transported by wind long distances from infected fields. Spread within and between fields locally also relies on windblown spores

The conidia are transformed into thick-walled resting spores called chlamydospores. During warm, moist weather in early summer, new conidia are produced on the old corn residue, and the conidia are carried by the wind or rain to lower leaves of young corn plants.

Management:

Regular field monitoring will help detect diseases on time : Monitor field at least every four weeks from whorl through dent stages

Cultural Control : Infected maize stubbles are source of infection for next growing season. Therefore, stubbles should be ploughed in right after harvesting for decomposition.: Avoid planting in the same areas If possible

Chemical Control E Spray propiconazole @2ml/litre water. This fungicide had both curative and systemic activity and is used against both TLB and GLS. Use sandovit sticker@2ml/litre water.The best time to spray is 3 weeks before flowering and silking or as soon as lesions appear.

Maydis leaf blight: *Cochliobolus heterostrophus* (Drechs.) Drechs.

[anamorph = Bipolaris maydis (Nisikado) Shoemaker;

synonym = *Helminthosporium maydis* Nisikado]

Maydis leaf blight is a serious foliar disease of maize distributed widely in maize-producing areas throughout the world. It is also known as Southern leaf blight (SLB).

Maydis leaf blight is found in all tropical and temperate maize growing regions where the growing season is characterized by warm and wet conditions.

Symptom

Lesions are generally:

- from 1/8 to 1/4-inch-wide by 1/8 to 1 inch long
- tan in colour
- rectangular to oblong in shape
- usually found on leaves
- variable, making identification more difficult than for other diseases
- Lesion type may depend on hybrid genetics
- Lesions usually develop first on lower leaves and work up the plant



Pathogen

There are two races of the pathogen. Race O normally attacks only leaves. Lesions are tan, somewhat rectangular in shape, and have reddish-brown margins. Race T attacks leaves, husks, stalks, leaf sheaths, shanks, ears, and cobs. Conidia are olivaceous brown, spindle-shaped, and taper to round ends and measure 15-20 × 70-160 µm in size. They are 5 to 11 septate and are characterized by bipolar germination. The asexual stage (conidial stage) of the fungi is the most important source of inoculum, both during the season and at the start of the season. Perithecia have been reported in the field at the junction of leaf sheath and leaf blade (Schenck and Stelter, 1974). They are usually erumpent, black, no differentiated sterile setae, ascigerous part subglobose or more frequently somewhat ellipsoidal, usually 0.4 -0.6 µm diameter. The sexual stage is characterized by production of ascospores (typically 4 to 8) in cylindrical, hyaline ascii. Ascospores are thin walled 6-7 × 130-340 µm in size, dark, and have 5 to 9 septa (Holliday, 1980).

Management:

- Genetic resistance
- Crop rotation to reduce corn residue level and help break disease cycle
- Tillage to encourage breakdown of crop residue
- Fungicide application

Rust: There are three types of rusts affecting corn plant namely –
common rust caused by *Puccinia sorghi*,
southern corn rust (SCR)/*Polysora* Rust caused by *Puccinia polysora*
tropical rust caused by *Physopellazeae*
common **Rust:** *Puccinia sorghi*

Symptom

Minute flecks are appeared on both sides of leaves. Small round, tan, Brown pustules appear on leaves. Looks circular to elongate golden brown or cinnamon brown, powdery, erumpent pustules appear on both leaf surfaces. As the crop matures brownish black pustules containing dark thick walled two celled teliospores develop. In severe cases infection spreads to sheaths and other plant parts.



pathogen

- Heterocephalous rust
- Uredospores single celled, echinulate and yellowish brown. 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.
- Alternate hosts - *Oxalis europea*, *O. corniculata* and *O. stricta*.
- Basidiospores infect *Oxalis corniculata* (alternate host) where pycnial and aecial stages arise after infection. Aeciospores carried by wind and infect maize.

Favourable condition: Cool, warm and moist weather (15-25°C)

Mode of Spread :

- Primary spread through alternate host
- secondary spread through wind borne uredospores.

Management:

Remove the alternate host - Oxalis

Collect the remains of the crop and destroy by burning or burying, and weeds around maize

The use of resistant varieties

Fungicides have been used against both common and southern rust (Foliar application of Tebuconazole @ 0.1% at 35 and 50 DAS or chlorothalonil or mancozeb). sprays commence when there are on average six pustules per leaf.

Polysora rust /Southern corn rust : *Puccinia polysora*

Polysora rust (Southern corn rust) is a major disease of maize in tropical and subtropical regions worldwide and is particularly destructive in late planted or late maturing maize hybrids. Unlike common rust, Polysora rust is most severe in warm growing conditions

Symptoms



Early lesions on leaves are small and circular to oval, often with a prominent light green to yellow halo. These lesions develop into light orange to cinnamon-red, circular to oval (0.2-2mm long) raised pustules, which are densely scattered over the upper leaf surfaces (Fig. 1). Eventually these pustules rupture through the epidermis to reveal masses of powdery spores that are responsible for secondary cycles of the disease, giving the leaves a rusty appearance. Towards plant maturity, pustules become dark brown to black with teliospores production. This brownish-black spore stage is formed in concentric rings around the initial pustules.

Symptoms of Polysora rust are very similar to common rust (*P. sorghi*). However, the two can be distinguished, as pustules of Polysora rust occur primarily on the upper leaf surfaces, whereas pustules of common rust occur abundantly on both leaf surfaces. The uredia of *P polysora* are circular and light cinnamon-brown scattered over the whole leaf surface compared with those of *P sorghi*, which are circular to elongate and chocolate-brown forming in a localised band pattern.

Table I. A comparison of corn rust characteristics (Figure 5).

| <i>Characteristic</i> | <i>Common Rust</i> | <i>Southern Rust</i> |
|---------------------------------|---|--------------------------------|
| Causal agent | <i>Puccinia sorghi</i> | <i>Puccinia polysora</i> |
| Pustule location | Both leaf surfaces | Mainly upper leaf surface |
| Pustule color | Brick red, golden brown to cinnamon brown | Light cinnamon-brown to orange |
| Pustule distribution | Sparingly scattered | Dense clusters |
| Predominant urediniospore shape | Round | Oblong to ellipsoid |
| Optimal temperatures | 61-77°F | 77-82°F |
| Management | Resistance, fungicide application, and planting early | |

Management

Resistant Hybrids, Early Planting and Fungicidal applications (Propiconazole)

Downy mildews

Among the various maize diseases, downy mildews are considered to be the major diseases. In origin, the downy mildews are “old world” diseases that now are very damaging and prevalent on the “new world crop” – maize (Shaw, 1975). None of the downy mildew diseases originated on maize (Shaw, 1975) but they possessed the ability to attack maize when maize was introduced from the new world to old world.

Heavy losses (as high as 100 per cent) in maize due to downy mildew pathogens have been recorded in Philippines, Taiwan, Indonesia, Thailand, India. Twenty one species of downy mildew pathogens have been reported to attack the gramineae family (Shaw, 1975). Of these, ten species of fungi belong to three genera (seven species of *Peronosclerospora*, one species of *Sclerospora* and two species of *Sclerophthora*) have been reported to cause different types of downy mildews in maize.

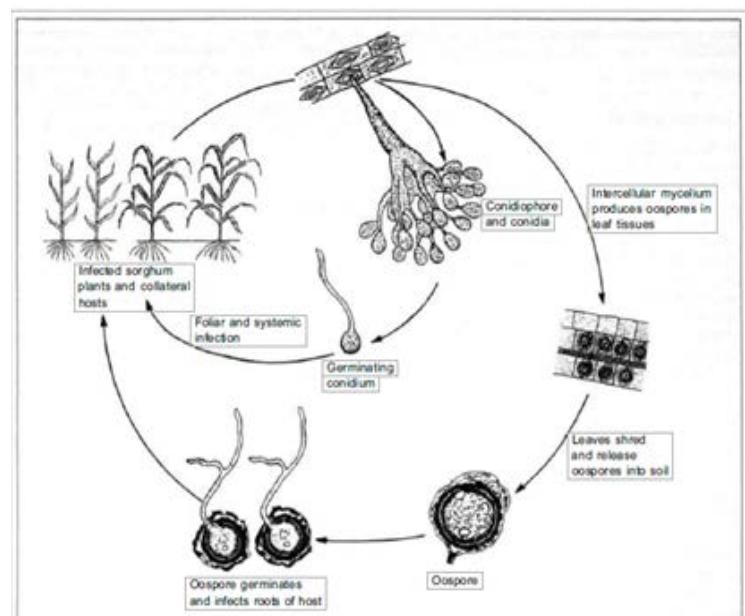
Downy mildews such as Sorghum downy mildew, Philippine downy mildew, Sugar cane downy mildew, Brown stripe downy mildew and more recently identified Rajasthan downy mildew were reported from different agroecological regions in India (Payak, 1975a; Siradhana et al., 1980).

| Sl. No. | Common name | Scientific name | Geographic distribution |
|----------------|--------------------|---------------------------------|--------------------------------|
| 1. | Java DM | <i>Peronosclerospora maydis</i> | Indonesia |
| 2. | Leaf splitting DM | <i>P. miscanthi</i> | Philippines, Taiwan |

| | | | |
|-----|----------------|------------------------------------|---|
| 3. | Philippine DM | <i>P. philippine</i> | India, Indonesia, Nepal, Philippines, Thailand |
| 4. | Sugar cane DM | <i>P. sacchari</i> | Australia, Fiji Islands, India, Japan, Nepal, New Guinea, Philippines, Taiwan, Thailand |
| 5. | Sorghum DM | <i>P. sorghi</i> | Asia, Central America, Europe, North America, South America, Africa |
| 6. | Spontaneum DM | <i>P. spontaneae</i> | Philippines |
| 7. | Rajasthan DM | <i>P. heteropogoni</i> | Rajasthan (India) |
| 8. | Green ear DM | <i>Sclerospora graminicola</i> | World wide |
| 9. | Crazy top | <i>Sclerophthora macrospora</i> | World wide |
| 10. | Brown strip DM | <i>S. rayssiae</i> var <i>zeae</i> | India, Nepal, Pakistan, Sikkim, Tailand |

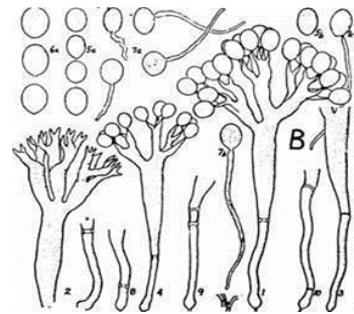
Sorghum downy mildew

- *Peronosclerospora sorghi*



Sorghum downy mildew, caused by *Peronosclerospora sorghi* (Weston and Uppal) C. G. Shaw, is a serious disease of both maize (*Zea mays* L.) and sorghum (*Sorghum bicolor* (L.) Moench) in the tropics and subtropics and is the most widely distributed of the major downy mildew diseases of either crop (Pupipat, 1975).

Sorghum Downy Mildew (SDM) is particularly prevalent in the Peninsular India, in the states of Karnataka, Tamil Nadu and Andhra Pradesh reportedly causing yield losses of 30 per cent and higher. It infects both maize and sorghum in warm and humid areas of the world (Frederiksen, 1980). The pathogen infects the roots primarily by oospores and the leaves by conidia and reaches the meristem causing systemic infection.



Crazy top Downy Mildew - *Sclerophthora macrospora*



Symptoms

Systemic infection in maize seedlings is characterized by chlorosis which normally appears 10-14 days after sowing. The leaves of infected plants tend to be narrower and more erect than those of healthy plants. Plants infected early usually die approximately four weeks after infection. In late infected plants, the chlorosis may be more noticeable on the lower half of the leaf which is often called half-leaf symptom. This chlorosis gradually covers the entire leaf surface at later stage. Under warm humid conditions, a white downy growth is produced on the lower leaf surface sometimes on both surfaces also. This growth is a combination of conidia and conidiophores. In maize, leaf shredding is rare, but it is common in sorghum. At flowering stage of growth, infected plants produced a bushy top, referred to a crazy

top in place of the tassel. Systemically infected maize plants generally do not form cob. In some cases when cobs formed, these are small and poorly filled
 Collateral host : johnsongrass (*Sorghum halepense* (L.) Pers.) and shattercane (a feral *S. bicolor*) are infected in the field by *P. sorghi* (Warren et al., 1974). These hosts, which produce oospores and conidia, serve as potential primary sources of inoculum in the early spring to infect maize or sorghum.

Pathogen

| | | |
|---|---|-----------------------------|
| Mycelium | - | Non-septate, inter cellular |
| Parasitism | - | Systemic and obligate |
| Conidia and Conidiophores: | | |
| Conidial formation usually begins at 0100—02 00 h during favourable periods. Air temperature, relative humidity, wind, light and the presence of dew on plants affect the time and amount of conidial production and release of spores. After conidial production and dispersal, germination occurs through one or more germ tubes and subsequent penetration occurs within a few hours . Conidiophores are erect, fragile, hyaline, 180–300 µm long, usually dichotomously branched 2–4 times and emerge through stomata on the lower sides of leaves. 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. Conidia are hyaline, oval to spherical, 15–26.9 × 15–28.9 µm, and borne | | |
| Oospores: | | |
| In advanced stages, are formed which are spherical, thick walled and deep brown. | | |

Mode of spread

- Primary spread through oospores in soil and seed-borne
- Secondary spread by conidia/sporangia

Favorable condition: Temperature: 24-26 C, Continuous drizzling rainfall, relative humidity more than 80 %

Epidemiology: Conidia of *P. sorghi* are produced in large numbers, they are thin walled, ephemeral, and can cause the rapid build up of an epidemic. Oospores are tough walled, long-lived, and provide a perennating stage for the pathogen, as well as a mechanism for long-distance transport

Management: Keep traffic out of SDM affected areas and minimize movement in adjacent areas Stop irrigating affected areas to healthy areas

Ensure that planting seed materials from SDM

Drying seed to less than 14% moisture content reduces or eliminates seed-borne transmission. Crop Rotations with more than three years between sorghum or maize crops, control of alternative grass hosts within the paddock will reduce soil-borne infection. Potential Time of planting: Delaying planting until April or until the onset of the monsoon season (Siradhana et al., 1980) reduced infection rates

Deep tillage of infected plant material, where spores were buried under at least 20 cm of soil Selective removal of symptomatic plants from crops

Host-plant resistance is an effective method for the control of SDM. Numerous Hybrids and inbred resistant lines have now been developed for maize Metalaxyl, applied as a seed treatment and sprayed after planting, completely controlled both systemic infection and local lesions of *P. sorghi* on maize. Infected plants sprayed with metalaxyl after planting recovered and produced normal heads. Seed treatment with metalaxyl alone did not provide protection against late systemic infection on main shoots or nodal tillers, or against local lesions on leaves. In India, seed treatments of 1 g a.i kg⁻¹ of seed plus a foliar spray of 1 g a.i L⁻¹ 40 days after emergence (DAE) or foliar sprays of 2 g a.i litre⁻¹ at 10 plus 40, or 20 plus 50 DAE gave complete control of systemic SDM

PHILIPPINE DOWNY MILDEW OF MAIZE - *Peronosclerospora philippinensis*

Downy mildew on maize leaves is characterised by elongated chlorotic streaks with a downy growth of conidia and conidiophores (Figure 1). Symptoms first appear 3–6 days after infection as pale yellow to whitish discolourations on the leaf blade. Tassels may be deformed, and ears may be aborted. When the disease is severe, the infected plants are stunted and weakened, and may die within a month. When the attack is moderate, infected plants usually reach maturity but produce small, deformed ears



Rajasthan downy mildew : *Peronosclerospora heteropogoni*

Rajasthan downy mildew of maize incited by *Peronosclerospora heteropogoni* is a unique example of ontogenetic predisposition of a crop to a pathogen from wild grass host. Since its first record on maize in 1968, several outbreaks have been reported during 1973-80 and losses of up to 60 per cent have been recorded. The pathogen can infect maize, teosinte, *Heteropogon melanocarpus*, and *H. contortus*. The latter seems to be the natural reservoir of the pathogen. Some extent of variability has been observed among populations of *P. heteropogoni*. Young maize seedlings, 3-4 days after germination, are most susceptible to RDM. The mycelium has been observed in seeds collected from systemically infected plants, but the studies on seed transmission suggest that seed transmission may not be of much significance. The most conducive condition for disease development are temperatures 22.9 to 28.6° C and RH > 85.0 per cent

Post flowering stalk rot (PFSR)

Post flowering stalk rot (PFSR) complex is one of the most serious, destructive and widespread groups of diseases in maize. The disease causes internal decay and discoloration of stalk tissue, directly reducing yield by blocking translocation of water,

nutrient and can result in death and lodging of the plant during the cropping season. The term «stalk rot» is often used to include stalk breakage, stalk lodging, premature death of plant and occasionally root lodging. The PFSR is a complex of disease and difficult to characterize because a number of fungi, bacteria and nematodes are involved in the decay of pith

Causal agents of PFSR complex:

1. Fusarium stalk rot - *Fusarium verticillioides* (Saccardo)
2. Gibberella stalk rot - *Gibberella zeae* (Schwein) ptec;
3. Charcoal rot - *Macrophomina phaseolina* (Tassi) Goidanich
4. Diplodia stalk rot - *Stenocarpella maydis* (Berk) Sutton
5. Anthracnose stalk rot - *Colletotrichum graminicola* (Ces) Wils
6. Black Bundle diseases - *Cephalosporium acrimum*
7. Late wilt - *Cephalosporium maydis* (Samra, Sabet, and Hingorani)
8. Bacteria stalk rot : *Erwinia chrysanthemi* pv. *Zeae*

Among all of these pathogens of PFSR, Fusarium stalk rot, charcoal rot and late wilt are most prevalent and destructive Disease in Maize.

Fusarium stalk rot

Among the stalk rots of maize, Fusarium stalk rot, caused by *Fusarium verticillioides*. *Fusarium* stalk rot was observed in the plant age group of 55 to 65 days which coincides with tasselling and silking and immediately followed grain formation stage. At these stages the stem reserves are depleted and most of the carbohydrates are translocated to developing sinks and stalks are predisposed to the fungi.

The stalk rot usually occurs after flowering stage and prior to physiological maturity, which reduces yields in two ways:



i) affected plants die prematurely, thereby, producing lightweight ears having poorly filled kernels and ii) plants with stalk rot easily lodge, which makes harvesting difficult, and ears are left in the field during harvesting

Favorable condition: *F. verticillioides* is more common in regions with hot and dry growing conditions (Doohan et al, 2003), especially before or during pollination. The water stress at flowering and high soil temperature help in increasing of the magnitude of the stalk rot symptoms at post flowering stage of maize crop.

Charcoal rot

The charcoal rot of maize, caused by *Macrophomina phaseolina* (Tassi) Goid, is an important disease of this crop. The pathogen is reported to infect nearly 500 species of plants in tropical and subtropical countries (Figure 3; Ghaffar, 1988). The pathogen produces numerous black sclerotia on diseased plant parts, which are globular to irregular in shape. The fungus is composed of many strains, differentiated by sclerotial size and the presence or absence of pycnidia. *Rhizoctonia bataticola* is considered to be the sclerotial and mycelial stage of *M. phaseolina*



The stalk rot symptoms are observed during post flowering and pre-harvest stage

- The affected plants exhibit wilting symptoms.
- As the plants mature the fungus spreads into the lower internodes of the stalk, causing premature ripening, shredding and breaking at the crown.
- The stalk of the infected plants can be recognized by greyish streak. The pith becomes shredded and grayish black minute sclerotia develop on the vascular bundles.
- Shredding of the interior of the stalk often causes stalks to break in the region of the crown. The crown region of the infected plant becomes dark in colour. Shredding of root bark and disintegration of root system.

Pathogen

- Fungus produces large number of round and black **sclerotia**. Sclerotia are black and globular to irregular in shape.
- **Pycnidia** appear on the stalks.
- **Pycndiospores** are colourless, oval and single celled and borne in black flask shaped pycnidia.

Epidemiology of charcoal rot The diseases caused by the pathogen are more prevalent under the condition of high soil temperature 30 - 42°C, low soil moisture and low soil pH (5.4 - 6.0) or when plants are under water stress

Mode of spread and survival

- Primary spread – Sclerotia in infected crop debris.
- Secondary spread – Wind-borne pycnidiospores.

Integrated PFSR disease management

Moisture control

Moisture stress condition is favor by the *F. verticillioides* whereas, water stress for 30 days before harvest increased late wilt.

Temperature

Temperature has a marked influence on infection and development of stalk rot. Dry and hot weather during and after flowering favors development of disease Rot is favored by soil temperature of 30-45°C and low soil moisture.

Soil solarization

Soil solarization before sowing of crop cause deterrent effect on pathogen. Plant maturity Maize does not generally become susceptibility to stalk until silking time. Its infection increased with plant age from 5 to 95 days.

Plant population

A large plant population per hectare generally makes plants more prone to stalk rot. First, the stalks of plants grown under crowded conditions are smaller in diameter and, therefore, less rot is required to weaken the stalks to the breaking point.

Organic manure Organic amendment stimulates the population of beneficial soil bacteria and actinomycetes resulting in a lower incidence of post flowering stalk rot in maize. Soil fertility In general, stalk rot incidence and severity increases with increased fertility. There is evidence that potassium fertilizers reduces the severity of stalk rot and that nitrogen fertilizers, especially if in excess compared with potash, increases the severity of stalk rot except in case of late wilt. Potassium plays a vital role in reducing stalk lodging in corn. Hence, maintaining a sufficient supply of potassium to prevent lodging needs more attention in maize hybrids Biological control *Pseudomonas cepacia* is a potential suppressor of maize soil borne diseases. The seed treatments with *T. harzianum* (4 g kg⁻¹ seed) along with castor and neem cake gave an effective control of post flowering stalk rot diseases and gave better cost: benefit ratio.

Genetic resistance: Resistance to Fusarium stalk rot is controlled by two genes and is dominant in expression. These two genes are located in the short arm of chromosome 7 and long arm of 10. Resistance to Fusarium stalk rot in inbred 61 C is also attributed to two genes. Resistant variety Source of resistance against Fusarium stalk rot of maize identified are CM 103, CM 119, CM 125, CI 21 E, CML 31,77,79,85,90, and CML 3 81.

Chemical control

Diseases intensity of Fusarium stalk rot can be reduced by seed treatment with *T. viride* + bavistin along with two additional irrigation at tasselling and silking stage reduced the disease intensity from 70.08 to 13.24%

Stewart's Bacterial Leaf Blight

Stewart's Bacterial Leaf Blight is caused by the bacterium *Pantoea stewartii*. This disease is transmitted to corn by corn flea beetles. Flea beetles carry the bacterium and introduce it into the corn when feeding.



Symptoms

Young plants when infected most often wilt and die due to systemic infection. Leaf blight associated with this disease occurs later in the growing season usually after tasseling. Leaf blight lesions are long and linear with wavy margins (> 1 inch). These lesions will start in the upper part of the plant unlike most foliar diseases that start at the bottom of the plant. Flea beetle feeding may be evident in the lesion

Banded leaf and Sheath blight (BLSB) is one of them caused by most widespread, destructive and versatile pathogen *Rhizoctonia solani* f. sp *Sasakii* (teleomorph: *Corticium sasakii*, syn *Thanatephorus cucumeris*) which claims significant yield loss (Saxena, 2002). It was first reported by Bertus (1927) in Sri Lanka under the name Sclerotial disease. Since, it develops on leaf and sheath; the symptoms appear in concentric spots that cover large area of infected leaf and husk. The pathogen spreads from the basal sheath to the developing ear under favorable environmental conditions. The main damage reported in the humid tropics is a brownish rotting of ear, which shows conspicuous, light brown, cottony mycelium with small, round and black sclerotia.



Propaconazole, 0.1%, and Carbendazim, 0.05%), by applying as foliar sprays at 30, 40 and 50th day of planting, alone or in combinations.

Aspergillus rot / Aflatoxin contamination rot:

- *Aspergillus flavus* and *A. parasiticus*. *Aspergillus* ear rot is one of the most important diseases in maize crop. The fungus produces a mycotoxin known as aflatoxin (B1, B2, G1 and G2) - inside the diseased maize kernels. The presence of aflatoxin will affect grain quality and marketability, as well as livestock health if the grain is consumed.

Favourable Condition:

- *Aspergillus* ear rot is commonly observed during hot, dry years on stressed plants (such as those exhibiting symptoms of nutrient deficiency or drought stress). Feeding damage from ear-invading insects also contributes to disease development and aflatoxin contamination.

Aspergillus ear rot is one of the most important diseases of maize. It is caused primarily by the fungus *Aspergillus flavus* and *A. parasiticus*. *Aspergillus* ear rot appears as an



olive-green mould on the kernels that may begin at the tip of the ear or be associated with injury from insects, birds, or hail. The fungal spores appear powdery and may disperse like dust when you pull back the husk. These signs are most commonly observed at the tip of the ear but can be scattered throughout the ear and all the way to the base of the ear.

Aspergillus species produce a mycotoxin called aflatoxin. Aflatoxin affects grain quality and marketability and is primarily a threat to livestock health. Aflatoxin is extremely carcinogenic and most countries have regulations (20 ppb) in place to prevent aflatoxin from entering the human food and livestock feed supply. The aflatoxin, which will accumulate as the fungus spreads in subsequent hot and dry

weather. The fungus can infect the ear and produce more aflatoxin after physiological maturity, particularly during periods when rainfall delays harvest. It's important to note that kernels with no visible injury or mould may still contain aflatoxin.

Management

Maize must be dried to below 15 percent moisture to prevent further fungal growth and mycotoxin production. An important factor in preventing *Aspergillus* ear rot is to reduce stress on the corn plant. Hybrids that tolerate water stress and/or irrigation can reduce drought stress on the plant. Also, provide adequate nitrogen fertilizer and maintain appropriate fertility within a field

Gibberella ear rot is caused by the fungus **Gibberella zeae** (also known as *Fusarium graminearum*), the same pathogen that causes stalk rot of corn and head scab of wheat. The fungus typically infects via the silk channel, causing a pinkish-white mold to develop at the tip of the ear (Fig. 1). Cool, wet weather (rainfall or high relative humidity) during and after silking (R1 growth stage) provides optimal conditions for the development of ear rot. During infection and colonization of the ear, the fungus produces several mycotoxins, including deoxynivalenol (DON), also called vomitoxin. As a result, high levels of Gibberella ear rot severity and moldy grain are usually accompanied by high levels of vomitoxin. Mycotoxins are harmful to both humans and animals. Vomitoxin is water soluble, heat stable, and the most commonly encountered mycotoxin in food and feed. The pathogen that causes Gib ear rot can produce two mycotoxins in the infected kernels: deoxynivalenol and zearalenone. These mycotoxins can affect the health of many monogastric animals, but swine are especially sensitive.



Sorghum Diseases

Anthracnose: *Colletotrichum sublineolum*

For Maize anthracnose : *Colletotrichum graminicola*

An important disease damages leaves, stalk, peduncles, and panicles, and causes substantial losses in grain, forage and stover yields. Lesions begin as small purple to reddish dots and expand to lesions with straw-colored centers and wide red, orange, or purple margins. As the disease progresses, the lesions can grow together, or coalesce, and expand to cover most of the leaf surface. After prolonged disease development, the leaf can die. In the tissue that has been killed (mostly within the lesions), small reproductive structures called acervuli can be seen. The midrib infections will have similar lesions and may occur independently of foliar symptoms.



The panicle and grain phase may or may not be correlated in severity with the foliar or stalk rot phase of anthracnose, but foliar anthracnose likely contributes to it. As foliar lesions sporulate, conidia are moved behind the leaf sheath into the area adjacent or onto the panicle by wind or water, where they germinate and infect. Lesions first appear on the panicle as small, oval-shaped, and dark water-soaked pockets. They become purple with age. If the panicle is split internally, tissue may appear dark and somewhat marbled. This is considered the stalk rot phase. The panicles from severely diseased plants are typically smaller, and the grain ripens prematurely. Dark streaks appear on grain when it is infected. Acervuli can be found on many parts of the panicle and head during this phase. The panicle and grain phase of disease can cause serious losses on moderately susceptible to susceptible hybrids by decreasing both grain quantity and quality.

Target leaf spot

Representative spot-causing fungal disease in the warm regions. The disease produces red purple and oval lesions of 0.5-2cm in length and 0.3-1cm in width abundantly on leaves from the end of summer to autumn. The lesions are produced by the accumulation of anthocyan responding to the infection of the pathogen. The fungus can infect the plant in all growth stages. It typically survives in soil or residue from the previous year but can also be present on grassy weed hosts. Symptoms begin as small purple spots and progress to reddish-purple oval or elliptical shaped lesions. The lesions can have a tan center with a purple border but this is rare. The lesions range in size from 1 to 10 cm and coalesce when conditions favor continued disease development. Unlike gray leaf spot, lesions are not confined by the leaf veins. Profuse sporulation can occur within the lesions and spores can be spread by wind or water splashing to neighboring plants in the field. Leaves with numerous lesions eventually become blighted, brown, and die.



Sorghum Ergot : *Sphacelia sorghi* (*Claviceps sorghi*)

Introduction

Ergot or sugary disease is a serious limiting factor in production of hybrid seed, particularly if seedset in male sterile line is delayed due to lack of viable pollen. It happens if there is a lack of synchrony in flowering between the male and female parents. Moreover, environmental conditions favorable for disease development are not congenial for rapid seed-set, thus making spikelets more vulnerable to ergot attack. Dihydroergosine is the major alkaloid in sorghum ergot sclerotia and honeydew, but four clavine alkaloids are also produced. Initially the disease was observed in Kenya (in 1915) and India (in 1917).

Symptoms

The earliest symptom of the disease may be seen on the ovary if the glumes are opened 3-5 days after infection. The infected ovary appears dull green and slightly



shriveled, in contrast to dark green and round appearance of a healthy, fertilized ovary. Within 2 days, superficially visible white mycelial



stromata appear in the base of the ovary and gradually extend upwards. The ovary is converted into fungal stromata with shallow folds.

The first external symptoms, clear to pinkish drops exuding from infected ovaries, appear 5-10 days after inoculation. The name, 'sugary' disease, for sorghum ergot originates from this sticky sweet fluid. It is also called honeydew and contains numerous conidia.

The earliest symptom is honeydew oozing from infected florets. Honeydew is a thin or viscous, sweet, sticky fluid containing the sphacelial conidia. Newly formed honeydew droplets are colourless and transparent but become progressively opaque. With time, honeydew may become uniformly yellow-brown to pink or superficially white. It may remain as intact droplets or may be so plentiful that it drips onto uninfected florets, seeds, leaves, and the ground below the panicle.

Under humid conditions, a saprophyte *Cerebella volvens* (Syn. *C. sorghi-vulgaris*) grows on honeydew and converts it into a matted, black mass. However, warm and dry conditions after the formation of honeydew will dry it, forming an easily removable, hard, white crust on the panicle. Finally, the fungal stromata are transformed into the hard, resting structure (sclerotia) that may or may not be concealed by the glumes.

Conidia

The pathogens produce three types of singlecelled, hyaline spores: oblong to oval macroconidia, spherical microconidia, and pear-shaped secondary conidia.

Mode of spread

Within 5-12 days after infection in sorghum, the pathogen produces millions of conidia, in honeydew, to infect spikelets that flower subsequently in the same panicle or in different panicles. The pathogen spreads rapidly, probably carried by flies, bees, and other insects (Futrell and Webster 1966) and rain splashes.

Management

Quarantine has been effective in excluding the pathogen the utility of early sowing to avoid ergot sowing pathogen-free seed by steeping seeds in 5% salt solution to remove sclerotia Removal of collateral hosts in and around fields and rouging of infected plants are other cultural methods of control Sowing of resistant cultivars is probably the most practical, economical, and effective method to control ergot.

Diseases of Sorghum

Smuts

Smuts are one of the most important diseases of sorghum especially where untreated seed is planted. Damage is confined almost entirely to the head or panicle, reducing both the grain yield and forage value.

The Smut Fungi

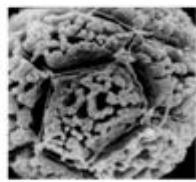
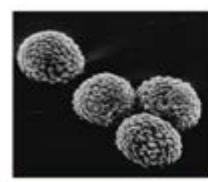
- "Smut" term comes from the dark masses of teliospores formed by many members of the group



Teliospores

- Formed singly or in spore balls
- Mostly globose, **pigmented**, with thick, ornamented walls
- Size ranges from 3.5 to 60 µm diam
- **Resistant structures**, in some species can survive up to 10 years in soil, and 25 years or more under optimal conditions

Teliospores



Three sorghum smuts are common in India

1. Covered kernel smut
2. Loose kernel smut
3. Head smut

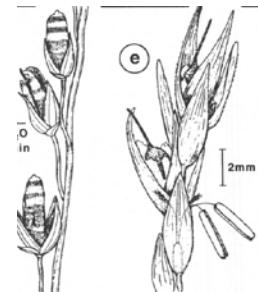
All caused by a different species of the fungus *Sporisorium*.

Covered Kernel Smut

Sporisorium sorghi (synonym *Sphacelotheca sorghi*)

It attacks all groups of sorghums, including Johnson grass. Usually, all of the kernels in a smutted head are destroyed and replaced by dark brown, powdery masses of smut spores (teliospores or chlamydospores) covered with a tough, greyish white or brown membrane.

The glumes appear normal in colour. Most sori are conical or oval, and resemble an elongated sorghum seed. They are whitish to grey or brown, and may have grey and brown stripes. Sometimes nearly all of the grains are affected, but



frequently heads are only partially smutted. Smut sori may be localized at the top, bottom or side of the infected head. The soral membrane (peridium) may remain intact until threshing or it may rupture easily, which ruptures irregularly to expose the dark brown powdery spore mass and the central columella composed of host tissues permeated by hyphae; panicles congested and stunted or not. The infected kernels (smut sori) break, and the microscopic spores adhere to the surface of healthy seeds where they overwinter.

Only seed borne spores cause infection. Smut sori are generally smooth; oval, conical or cylindrical; and vary in size from those small enough to be concealed by the glumes to those over one cm long. They may be white, gray, or brown. When a smut-infested kernel is planted, the teliospores germinate along with the seed forming a 4-celled promycelium (epibasidium) bearing lateral sporidia. The sporidia germinate and infect the developing sorghum seedling. Sometimes the teliospores germinate directly by producing germ tubes. Once inside the seedling, the fungus grows systemically, apparently without damaging the plant until heading.

Loose kernel smut : *Sporisorium cruentum*

(synonym *Sphacelotheca cruenta*)

Plants affected by loose kernel smut were initially mostly stunted, had thin stems, and panicles which emerged earlier than those of healthy plants. Later, all kernels of infected panicles were replaced by smut sori. The sori were surrounded by a thin grey membrane that often ruptured soon after the panicle emerged from the stem. Following rupture of the membrane, a powdery and black smut



Sorus (pl. sori)

- Teliospores are formed in sori
- Composed of host and fungal tissues
- Formed in host ovaries, stems, leaves, or roots depending on the smut taxon
- Characters of taxonomic importance include:
 - Thread-like structures (fungal)
 - Sterile cells
 - Columella (host)
 - Peridium (host or fungus)
 - Persistent = covered smut
 - Thin, breaking down to expose spores = loose smut

mass (teliospores) was dispersed leaving a clearly visible central and curved columella inside the sorus.

The powdery, dark brown to black spores (teliospores) are soonblown away, leaving a long, black, pointed, conical, often curved structure (columella) in the centre. Some smut spores adhere to the surface of healthy kernels on neighbouring plants in the same field or ones nearby before and during harvest. Unlike covered kernel smut, plants affected with loose kernel smut are stunted, have thin stalks, and heads emerge earlier than healthy plants.

Head smut caused by *Sporisorium holci-sorghhi* (synonyms *S. reilianum* and *Sphacelotheca reiliana*)

Stunted growth due to a lack of elongation of the peduncle and rupture of the thick, white peridium membrane. **Sori** in inflorescences that are usually completely destroyed and transformed into blackish brown granular-powdery spore masses; sometimes only single flowers are attacked and, rarely, the panicle rachis and leaves are infected. Sori initially covered by a white to light brown peridium of fungal origin which ruptures irregularly and flakes away to expose the mass of spore balls mixed with groups of sterile cells and numerous columellae (remnants of vascular bundles and fungal elements). Columellae long, sinuous or stout, thread-like.



Long smut – *Sporisorium ehrenbergii* (*Tolyposporium ehrenbergii*)

This disease is normally restricted to a relatively small proportion of the florets which are scattered on a head. The sori are long, more or less cylindrical, elongated, slightly curved with a relatively thick creamy-brown covering membrane (peridium). The peridium splits at the apex to release black mass of spores (spore in groups of balls) among which are found several dark brown filaments which represent the vascular bundles of the infected ovary.



Management of smuts:

1. Covered and loose kernel smuts are easily and effectively controlled by treating the seed with a protectant fungicide. Seed treatment prevents introducing the head smut fungus into uninfested fields. Fungicide seed treatment also improves and stabilizes

the stand when soil insects are not a problem. In addition, it provides protection against seedling blight fungi in the soil.

2. There are a number of physiologic races of the three sorghum smut fungi, which can also hybridize with one another; it is extremely difficult to develop highly resistant or immune hybrids, varieties or cultivars of sorghum. Those varieties, hybrids, and types of sorghum that are resistant to races of covered kernel smut usually are resistant to races of loose kernel smut. Most sweet sorghum varieties are highly resistant.

3. Promptly remove and burn head smut galls before the spores are scattered.

4. Since the head smut fungus may live in the soil for several years grow sorghum in the same field only once in 4 years. Such a crop rotation also helps to control other diseases that attack the leaves, heads, stalks, and roots.

Fusarium stalk rot : *Fusarium thapsinum*

Symptoms: The most obvious and important of symptoms is crop lodging. However discolouration of the pith tissue inside the stalks, often centred on the nodes, also occurs. Lodging is often the first obvious sign of Fusarium stalk rot in sorghum plants, but the diagnostic symptoms of the disease are usually not evident until the plants are stressed. When a stalk infected by Fusarium is split lengthwise, a pink-red discolouration is evident from ground level up the stem. Stalks can be infected by Fusarium but not lodge; this depends on the strength of the stalk, and on the speed at which Fusarium invades the stalk. The latter is influenced by the severity of the stress and perhaps by the tolerance of the hybrid. Fusarium or other stalk-rotting pathogens may not be the sole cause of crop lodging; physiological (non-biotic) stress factors can often be the cause.

Favourable conditions: Fusarium stalk rot is favoured by a period of physiological stress. This may be moisture stress as a result of seasonal conditions or stress caused by the application of a desiccant.

Management:

Practices that minimise moisture stress on the crop should be encouraged. However, rotation with non-host crops is also recommended.

Sorghum Downy Mildew

The fungus causes systemic downy mildew of sorghum. It invades the growing points of young plants, either through [oospore](#) or conidial infection. It is characterized by leaf chlorosis (which invariably includes the leaf base), which usually seems in two weeks or more after sowing (symptom expression being dependent on the timing of infection). The intersection between the diseased and healthy tissue is sharply defined (resulting in the 'half-leaf symptom'). Progressively greater proportions of the lamina on subsequently emerged leaves show chlorosis, until most or all of the lamina is chlorotic. The first leaf is free from infection; this may be caused by the first leaf outgrowing the pathogen, which requires time to invade the root and stem tissue, or the existence of a passive defence mechanism in the first leaf, which prevents entry of the pathogen.



Favourable Conditions

In cool, humid weather the asexual structures (conidiophores and conidia) of the pathogen appear on the surface of the diseased leaves, giving a white, down-like appearance. Maximum sporulation takes place at 100 per cent relative humidity. Optimum temperature for sporulation is 21-23°C during night. Light drizzling accompanied by cool weather is highly favourable.

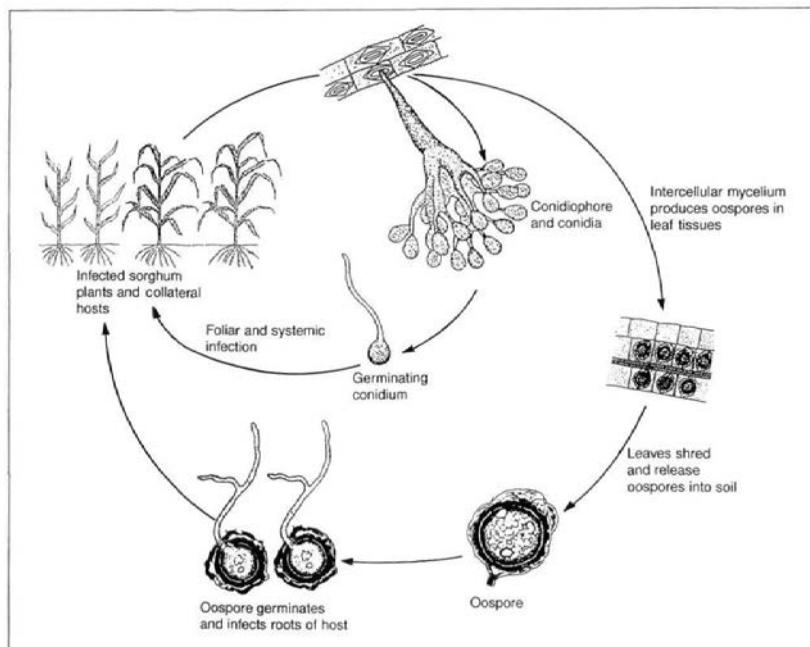
'Leaf-shredding' symptom:

On sorghum, whitish streaks develop from the base of the younger leaves, which turn brown as the oospores produced in rows in the fibrovascular bundles mature; the lamina of these leaves begins to tear length-wise, causing the characteristic symptom of 'leaf-shredding'. This process releases oospores from within the sorghum leaf. Leaf-shredding does not often occur in maize,



Pathogen

Oogonia of *P. sorghi* are spherical and are embedded among mesophyll cells between fibrovascular bundles. Oospores are hyaline, spherical, with light yellow walls, and germinate by germ tube. Conidiophores are erect, fragile, usually dichotomously branched 2–4 times and emerge through stomata on the lower sides of leaves. Conidia are hyaline, oval to spherical, and borne on elongated, tapered sterigmata. They germinate by germ tube under high humidity.



Management

As same as maize downy mildew

Charcoal rot: *Macrophomina phaseolina*

Charcoal rot in sorghum is caused by the soil borne fungus *Macrophomina phaseolina* and is a major stalk rotting disease in sorghum which can lead to plant lodging. The causal agent, *M. phaseolina* can infect via the roots of sorghum plants at almost any stage of plant growth, but develops more rapidly in plants closer to maturity.

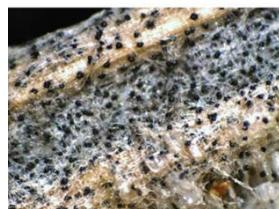


Symptoms: The pathogen is easily identifiable when stems are split longitudinally. The characteristic appearance of black microsclerotia (resting bodies) in the vascular tissue and inside the rind of the stalk results in a ‘peppered’ look in conjunction with shredded internal vascular tissue which is grey/charcoal in colour.

Favourable conditions: Extensive colonisation of stem tissue generally occurs post flowering when plants are placed under a stress, such as unfavourable environmental conditions, particularly hot, dry conditions

Management: strategies for Fusarium stalk rot and charcoal rot are closely related. There are no effective foliar fungicides for either disease.

Soil moisture—planting into adequate soil moisture and ensure row spacing and plant populations are suitable for the field and seasonal situation, to minimise possible post flowering moisture stress.



Adequate nutrition—application of adequate fertilisers should be exercised to maintain plant health and vigour reducing nutrient related stress. More specifically, excessive N and low levels of K should be avoided.

Crop rotations—rotating out of susceptible crop hosts can be effective in reducing the build-up of *Fusarium* and/or *M. phaseolina* which may have occurred in mono-cropping systems

Use of lodging resistant, drought tolerant, non-senescent varieties.

Rust : *Puccinia purpurea*

Symptoms: Early symptoms on leaves are small purple-red or tan spots. These enlarge to produce elongated raised pustules that break open to release brown, powdery masses of spores. Sorghum rust is more serious in late-sown crops or susceptible hybrids in humid areas. If the disease is serious, leaves are destroyed and pinching of the grain results. Select hybrids with resistance for late planting.



Favourable conditions: The spores germinate on wet leaves, penetrating the leaf and will then take 10–14 days for the pustules to appear. The spores are primarily dispersed by wind.

Management : spray Mancozeb at 1kg/ha. Repeat fungicidal application after 10 days

Sorghum Leaf Blight : *Excerohilum turcicum*

The disease develops on [sorghum](#) leaves particularly under humid conditions by producing reddish-purple or tan spots that coalesce to form large lesions. It attacks seedlings as well as older plants.

Leaf blight is caused by the fungus *Excerohilum turcicum*. This fungus also causes Northern corn leaf blight. The disease is most readily identified by large cigar-shaped lesions on the leaf with reddish or purple margins. Symptoms of the disease include small reddish or tan spots that can enlarge to long elliptical reddish purple or tan lesions. These lesions can be 12 mm wide and 2.5 to 15 cm long.

Within the lesion, there are often noticeable black conidia formed by sporulation of the fungus giving the lesion an ashy gray to dark olive appearance. Like many foliar pathogens, the conidia are blown or splashed to neighbouring leaves or other plants and will infect when free moisture is available on the leaf. Some hybrids are resistant to the fungus. Resistant reactions have lesions that are smaller, often no larger than a purple fleck, with little to no sporulation. The fungus survives as mycelia on plant residue, buried in the soil, or on the surface.



Favourable conditions

Disease development is favoured by moderate temperatures (18° to 27°C) and heavy dews or rain during the growing season. The disease can make its appearance early in the season and continue to develop throughout the growing season unless retarded by dry weather.

Disease cycle

The pathogen is found to persist in the infected plant debris. Seed borne conidia are responsible for seedling infection. Secondary spread is through wind-borne conidia.

Management

- Use disease free seeds.
- The disease is controlled by the use of resistant cultivars
- Crop rotation and tillage may also offer some control, but the proximity of maize may provide enough inoculum blowing from the corn field to the sorghum field to cause disease.
- Treat the seeds with [Captan](#) or [Thiram](#) at 4 g/kg.
- Spray [Mancozeb](#) 1.25 kg or [Captafol](#) 1 kg/ha.

Anthracnose

Anthracnose is caused by the fungus *Colletotrichum sublineolum*

Anthracnose has multiple phases

- The foliar phase,
- The panicle and grain phase
- The stalk rot phase

The foliar phase of anthracnose describes disease of the leaf and leaf midrib. Lesions begin as small purple to reddish dots and expand to lesions with straw-colored centers and wide red, orange, or purple margins.

As the disease progresses, the lesions can grow together, or coalesce, and expand to cover most of the leaf surface. After prolonged disease development, the leaf can die. The midrib infections will have similar lesions.



Lesions first appear on the panicle as small, oval-shaped, and dark water-soaked pockets. They become purple with age. If the panicle is split internally, tissue may appear dark and somewhat marbled. This is considered the stalk rot phase. The panicles from severely diseased plants are typically smaller, and the grain ripens prematurely. Dark streaks appear on grain when it is infected.

Favourable Conditions

It is most often observed when the weather is hot and humid and on susceptible hybrids can be severe and cause tremendous yield losses.

Pathogen

Small reproductive structures called acervuli can be seen with the aid of a 10x hand lens. The acervuli are dark colored, mostly black, and have spiny structures protruding called setae. These acervuli are diagnostic for foliar anthracnose.

Disease cycle

The disease spread by means of seed-borne and air-borne conidia and through the infected plant debris.

Management

- Treat the seeds with Captan or Thiram at 4 g/kg.
- Spray the crop with Mancozeb 2 kg/ha.

Minor Diseases

Bacterial leaf spot is caused by *Pseudomonas syringae* pv. *syringae*.

Symptoms of the disease consist of an initial water soaked lesion on the lower leaves. As lesions grow and mature, they become elliptical to circular and often develop red or brown margins. As lesions dry, the centres become light coloured. At this stage, leaf spot can resemble pesticide injury, physiological spotting and one or more fungal diseases.

Bacterial leaf streak is caused by *Xanthomonas campestris* var *holcicola*. The typical symptoms of the disease include small inter veinal water soaking areas that increase in size and that become several centimetres in length and purple in colour. The purple lesions often remain between the leaf veins and may appear as stripes. In very susceptible varieties, the stripes coalesce to become blotches and leaf shredding and death may occur at this stage.

Bacterial top and stalk rot is caused by the bacterium *Erwinia chrysanthemi*. The most recognizable symptom of the disease is the upper four to five leaves dead in the whorl. When the plant is removed from the soil and split longitudinally, the interior of the stalk is reddish, water-soaked, and a putrid odour emanates from the diseased tissue.



Head Blight, Grain Moulds and Head Molds

Head blight and molds are caused by a variety of fungal pathogens. Head molds generally refer to fungi that mold the grains as they mature on the seed head. *Fusarium moniliforme*, *Fusarium semitectum*, *Curvularia lunata*, *Phoma sorghina*, *Helminthosporium* spp. and *Alternaria* spp. are generally considered to be head molds.

The most obvious symptoms of head molds are the pink, orange or white seeds found on heads infected by *Fusarium* and by the presence of black seeds on heads infected by *Curvularia*, *Alternaria* or *Helminthosporium*. The presence of small black dots may indicate *Phoma* pycnidia or the acervuli of *Colletotrichum*.



Head blight is usually reference to the infection of panicle or rachis branches that result in premature death of all or parts of a panicle. Head blights can be caused by *Fusarium* but also can be caused by *Colletotrichum*. Head blights and molds can be partially avoided by adjusting planting dates so that plants mature during a period without frequent rains.

Target spot is caused by *Bipolaris sorghicola*

The symptoms of the disease first appear as reddish or grayish spots which later develop into elliptical, oval or more commonly cylindrical shapes. The lesions vary in

size from 1 to 10 cm in length. On rare occasions the purple lesions may have a tan-coloured centre. Under wet conditions, numerous spores can be produced on lesions. Spores are brownish to grey in colour.

Sooty stripe

Sooty stripe is caused by the fungus *Ramulispora sorghi*. The disease can occur at any time during the growing season and be severe on susceptible hybrids. Sooty stripe first appears as non-descript lesions on the lower leaves of the plant. During warm, humid weather, conidia form in the lesions and spread to healthy tissue. As lesions mature, they become elongated, with a yellow or reddish border and sclerotia form in the insides of the lesions. The sclerotia are dark coloured and easily wipe away having a sooty appearance.

Crazy top is caused by the microorganism *Scleropthora macrospora*. It is an oomycete, which is different from a fungus. In young plants, the disease produces mottled yellow plant tissue similar to that of a mosaic symptom caused by some viruses. As the plant matures, the heads often do not emerge or are malformed. Leaves of the plant are thick, with many leaves twisted and appearing to emerge from a similar location on the stem and the plant appears to have “too many” leaves.

3. Etiology, symptoms, mode of spread, survival, epidemiology and integrated management of Diseases of pearl millet, finger millet and small millets

Diseases of pearl millet

1. Downy mildew -*Sclerospora graminicola*

Symptoms

Infection is mainly systemic and symptoms appear on leaves (downy mildew) and inflorescence (green ear). The initial symptoms appear in seedlings at three to four leaf stages. The affected leaves show profuse downy white growth consisting of sporangiophores and sporangia of the pathogen occurs predominantly on the under surface of the leaves. Corresponding to the upper surface of the leaf, yellowing or patches of light green to light yellow colour discolouration occur. The yellow discolouration often turns to streaks along veins. Each florets of the inflorescence of infected plants gets completely or partially malformed into green beard leafy like structures, giving the typical symptom of green ear.



Downy mildew



Green ear symptom

Pathogen

It is a biotroph and cause systemic infection. The mycelium is filamentous intercellular, hyaline, non-septate and branched. It produces haustorium. The sporangiophores emerge through stomata singly or in clusters, they are swollen, short, upright branches bearing sporangia. Sporangia of *Sclerospora* show indirect germination that is zoospores are produced inside the sporangia and then infect the crop. Sexual spores are oospores and are thick walled, brown in color and round shaped

Favourable conditions

Cool (20 -23°C) and moist weather (>90 % RH) favours the disease development

Mode of spread and Survival

Primary spread is by Oospores in soil (viable for 3-4 years) and seed-borne inoculums, while the secondary spread is through air-borne sporangia. Pathogen survives as oospores in soil and crop debris

Management

Grow downy mildew resistant varieties CO7, WCC 75, CO(Cu)9, TNAU-Cumbu Hybrid-CO9. Transplanting reduces disease incidence. At the time of planting, infected seedlings should be removed. In the direct sown crop, infected plants should be removed up to 45 days of sowing as and when the symptoms are noticed. Spray any one of the fungicides Metalaxyl + Mancozeb @500 g or Mancozeb 1000g/ha.

2. Blast - *Magnaporthe grisea*

Symptoms

The disease appears as grayish, water-soaked lesions on foliage that enlarge and become necrotic, resulting in extensive chlorosis and premature drying of young leaves. The lesion size varies from small, roundish, elliptical, diamond shaped to elongated, measuring 1-2 mm to 20 mm. Lesions are often surrounded by a chlorotic halo, which turns necrotic, giving the appearance of concentric rings. The lesions are usually confined to interveinal spaces on the foliage. Lesions grow and coalesce to cover large surface areas and cause necrosis of tissues. Severely infected plants produce no grain or few shriveled grains in blasted florets.



Pathogen

The mycelium is dark brown to olivaceous green and septate. Asexual conidia are pyriform, hyaline, mostly three-celled with a small appendage on the basal cell. Conidia measure approximately 17-31 x 6-9 µm and germinate by producing appresorium. Through sexual reproduction four celled ascospores are formed in peritheciun.

Favourable conditions

Prevalence of high humidity (>90% RH) and moderate temperature (25- 30°C) favors infection and disease development. The disease becomes more severe during humid weather conditions especially with dense plant stands.

Mode of spread and survival

The pathogen sporulates profusely in the lesions on foliage and the conidia can be easily dispersed by the wind and splashing rain. These spores can overwinter in stubble and can infect the next crop the following year. Conidia generated in the diseased plant can further spread the infection.

Management

Remove collateral weed hosts from bunds and channels, use only disease free seedlings and avoid excess nitrogen. Seed Treatment with Carbendazim @ 2g/ kg or Metalaxyl + Mancozeb 35 SD @ 6g/kg seed and spray any one of the following fungicide after observing initial infection Carbendazim 250g/ha or Mancozeb- 1.0 kg or Metalaxyl + Mancozeb @ 2g/litre.

3. Rust - *Puccinia penniseti/ Puccinia substriata*

Symptoms

Symptoms first appear mostly on the distal half of the lamina. Raised reddish rusty pustule (urediosori) appear both upper and under surface but appear more on the upper surface. The pustules may be formed on leaf sheath, stem and on peduncles also. Later, black colored telial formation takes place on leaf blade, leaf sheath and stem. While brownish uredia are exposed at maturity, the black telia remain covered by the epidermis for a longer duration.



Pathogen

The pathogen is biotrophic, heterocous, macrocyclic in nature. Uredinial and telial, stages occur on pearl millet. The spermagonial and aecial stages are seen on *brinjal* (alternate host). Uredospores are oval, elliptic, sparsely echinulated and pedicellate. Teliospores are dark brown in colour, two celled, cylindrical to club shaped, apex flattened, broad at top and tapering towards base.

Favourable Conditions

Closer spacing and rainy weather favours the disease development. Presence of abundant brinjal plants and other species of *Solanum* viz., *S.torvum*, *S. xanthocarpum* and *S. pubescens* may result in severe disease spread.

Mode of Spread and Survival

Air-borne uredospores are the primary sources. Presence of alternate host helps in perpetuation of the fungus.

Management

Sowing during December - May results in less incidence. Adopt control measures when there is rust incidence in the early stages as spread of infection to top leaves results in poor grain filling. Spray Wettable sulphur 2500g / ha Mancozeb 1000g/ha when the initial symptoms of the diseases are noticed and repeat application 10 days after if necessary.

4. Ergot or Sugary disease - *Claviceps fusiformis*

Symptoms

Honey dew stage: The symptom is seen by exudation of small droplets of cream to pink mucilaginous droplets of "honeydew" from the infected spikelets. Under severe infection many such spikelets exude plenty of honey dew which trickle along the earhead. This attracts several insects.

Ergot stage: In the later stages, the infected ovary turns into small dark brown to black sclerotium which projects out of the spikelet. Sclerotia are larger than seed and irregularly shaped, and generally get mixed with the grain during threshing.



Honey dew secretion



Ergot –Sclerotia

Pathogen

The pathogen is having high organ specificity that is it infects only the ovary of the cereals and entire ovary is converted into sclerotia in place of normal seed development from the ovary. The pathogen infects the florets, grows through the stylar tube to the base of the ovary where it ramifies the entire ovary tissue. Honey dew secretion contains conidial suspension and survives for a long period as sclerotia

Favorable conditions

Conditions favoring the disease are relative humidity greater than 80%, and 20 to 30°C temperatures during flowering. Cool night temperature and cloudy weather aggravate the disease.

Mode of spread and survival

The primary source of infection is through the sclerotia. The secondary spread takes place through air and insect-borne conidia. Rain splashes also help in spreading the disease. 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*.

Management

Adjust the sowing date so that the crop does not flower during October 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. Remove collateral hosts and Spray Carbendazim 500g or Mancozeb 1000g /ha when 5 - 10% flowers have opened and again at 50% flowering stage

5. Smut - *Moesziomyces penicillariae* (Syn: *Tolyposporium penicillariae*)

Symptoms

The pathogen infects few florets and transforms them into plump sori containing smut spores. The smutted grains are initially bright green later color changes to dirty black in colour containing smut spores. The smutted grains are two to three times bigger than the normal grain. Matured sori ruptures to release dark-brown to black spore balls of numerous teleutospores. Relatively small proportion of the florets is infected.



Pathogen

The fungus produces teleutospores and sporidia. Teleutospores occur in compact, ball-like masses called spore balls in the infected florets. Spore balls vary in shape and size, and the number of teleutospores aggregated in a ball varies from 200 to 1400. Teleutospores germinate to produce four-celled promycelium on which sporidia are borne in chains. These sporidia germinate to cause infection. Two sporidia of compatible mating types are needed to form a dikaryotic infection hypha, which penetrates through young emerging stigma of a pearl millet floret.

Favourable conditions

Smut infection and spread is most favored by the prevalence of high relative humidity (80-95%) and optimal temperature (25-35°C) at the flowering stage of the crop. As with ergot, rapid pollination is known to reduce or even prevent smut infection in pearl millet lines with shorter protogyny that facilitates self-pollination.

Mode of spread and survival

It survives as spore balls in the soil that serves as primary source of inoculum. Secondary spread is by air-borne smut spores.

Management

Soil solarization to increase the soil temperature to kill the soil-borne teliospores . Collect smutted earheads in cloth bags and destruct by dipping in boiling water. Treat the seed with Carboxin@ 2g/kg or Captan/Thiram 4g/kg of seed. Spray any fungicides viz., carboxin or carbendazime or captafol during boot stage

6. Head Mold: Various fungi

Symptom

Appearance of pink, white, brown or grey fungal growth will be seen on grain. Apparently asymptomatic seed may be contaminated. Many pathogens cause grain molds. Grain molds on pearl millet tend to be more severe with humid conditions during grain fill and if grain harvest is delayed. Several fungi cause grain molds, and these differ by the region of cultivation, crop management, environmental conditions prior to harvest, and storage conditions.



Head mould

Management

Spray mancozeb 1kg/ha or Captan 1kg +Aureofungisol 100g/ha if intermittent rainfall occurs during earhead emergence, a week later and during milky stage.

7. Bipolaris Leaf Spot: *Bipolaris setariae*

Symptom

Foliar symptoms vary, as brown flecks, fine linear streaks, small oval spots, large irregular oval, oblong, or almost rectangular spots measuring 1-10 x 0.5-3 mm. Large fusiform lesions are sometimes produced. Lesions may expand and coalesce. Lesions may be solid dark brown but usually become tan or greyish brown with a more or less distinct dark brown border.



Management

Spray mancozeb 1kg/ha.

Diseases of small millets

I. Diseases of finger millet

1. Blast - *Pyricularia grisea*

Symptoms

Symptoms of ragi blast are the same as described in rice blast. The pathogen attacks the crop from seedling stage to the time of grain formation. The lesions are spindle shaped with grayish green centre and brown margin. Under humid conditions, an olive grey overgrowth of fungus is seen on the centre of the spot. Later the centre become whitish grey and disintegrates. Nodal infection causes blackening of the nodal region. Neck infection shows black discoloration at the neck region. Infection may also occur at the basal portions of the panicle branches including the fingers. The affected portions turn brown and ears become chaffy and only few shriveled grains are formed.



Leaf blast



Nodal blast



Finger blast

Pathogen

The pathogen is hemibiotroph dark brown to olivaceous green and septate mycelium. Conidia are produced in clusters on long septate, olivaceous conidiophores. Conidia are pale olive green, three celled, pyriform, attached to the conidiophore at the broader base by a hilum. Through sexual reproduction four celled ascospores are formed in peritheciun.

Favourable conditions

Application of excessive doses of nitrogenous fertilizers, intermittent drizzles, cloudy weather, high relative humidity (> 85%), long dew periods with dew deposition on leaves, low night temperature (below 15 - 25°C) and availability of collateral hosts

Mode of spread and survival

Mycelium and conidia in the infected straw and seeds are important sources of primary inoculum. The fungus also survives on collateral hosts and secondary spread is through air-borne condia.

Management

Use of blast resistant varieties like GPU-28, GPU-26, GPU-48, and CO15 coupled with carbendazim seed treatment at 2g/kg increases yield anywhere between 50-100 per cent. Seed treatment with *Pseudomonas fluorescens* @ 6 g/kg coupled with two sprays of *Pseudomonas fluorescens* at 0.3% first at the time of flowering followed by second spray 10 days later can control leaf, neck and finger blasts very effectively. In the absence of varieties with inbuilt resistance, sprays of fungicides are advisable to minimise the disease. Two sprays of Carbendazim+Mancozeb (0.2%) or carbendazim 0.05% or tricyclazole 0.05% with first spray at 50 per cent flowering followed by the second 10 days after were also effective.

2. Seedling blight or Leaf blight - *Drechslera(Helminthosporium) nodulosum*

(Sexual stage : *Cochliobolus nodulosum*)

Symptoms

The pathogen affects both seedling and the adult plants. Minute, oval, light brown lesions on the young leave and become dark brown. Several such lesions coalesce to form large patches of infection on the leaf blade. The affected blades wither prematurely and the seedlings may be killed. Linear oblong and dark brown spots appear on the leaves of grown up plants. The leaves give blighted appearance. The pathogen also attacks the nodal region causing black lesion. While nursery infection causes heavy damage due to the seedling blight, neck infection causes heavy chaffiness and severe loss in grain yield.

Pathogen

Hypha of the fungus is brow and septate. Conidiophores are long, septate, dark brown in colour, often branched and geniculate. Conidia are straight ovoid, pale to dark

golden brown, 5-7 pseudoseptate. *C. nodulosum* produces spherical perithecia and ascii contain 1 to 8 ascospores.

Favourable conditions

Optimum temperature for infection is 30-32°C and 80-90% relative humidity, rains during ear head emergence.

Mode of spread and survival

Primary spread is through seed-borne inoculum and the secondary spread by air-borne conidia.

Management

Treat the seeds with Captan or Thiram @4g/Kg. Spray Mancozeb @ 1.25Kg/ha or Spray 1% Bordeaux mixture or Copper oxy chloride or Dithane Z-78 (2g/lit. water)

3. Wilt -*Sclerotium rolfsii* (Sexual stage : *Corticium rolfsii*)

Symptoms

The infected plants become pale, chlorotic and stunted. The fungus attacks basal stem portion and later the leaf sheath and culm. The infected portion becomes soft and dark brown in colour. A whitish mycelial mass can be seen on the basal stem and on the nodal portions. On the surface of the lesions, small spherical, dark coloured sclerotia are formed.

Pathogen

The mycelium of the fungus is septate and white to tan coloured. Sclerotia are minute, mustard seed like structures, regular spherical in shape and black in colour. Sclerotia contain viable hyphae and serve as primary inoculum for disease development.

Favourable conditions

High soil moisture and high temperature (more than 30°C)

Mode of spread and survival

The fungus survives in the soil as sclerotia and spreads through irrigation water and implements.

Management

Keep the plants healthy and robust, and providing good drainage and other optimum soil conditions to avoid the disease. Plough deeply before sowing and proper crop sequences involving non-poaceous crops to reduce disease intensity. Spot drench with Copper oxychloride at 0.25 per cent for prevent spread of disease. Crop rotation with non host plants.

4. Mosaic/Mottle streak/ streak

Ragi severe mosaic

Symptoms

The virus induces mosaic symptoms, which are more clear and pronounced on young leaves. Infected plants remain stunted and the ears of severely affected plants malformed. Such plants produce few seeds of smaller size, which reduces the yield

considerably. In addition, the affected plants appear pale yellow due to severe chlorosis and in severe cases become brownish-white. Thus, the entire field appears yellow and can be readily distinguished from non-infected stands from a distance. Stunted plants do not recover, develop roots at nodes, generally do not produce ears and if produced are mostly sterile.

Pathogen

Caused by *Sugarcane mosaic virus* and transmitted by *Longiunguis sacchari* and *R. maydis*. Particles were flexuous rods with an average length of 667 ± 8 nm and an approximate diameter of 12-14 nm. The virus, thus, was identified as a strain of sugarcane mosaic virus. Virus is neither seed borne nor soil borne.

Ragi Mottle Streak

Symptoms

The infected plants exhibit regular dark-green areas all along the leaf veins when the plants are 4-6 weeks old. Other symptoms on leaf include chlorosis and streak. In some cases occasional yellowing to almost albino symptoms are also observed. However, in the lower leaves, the symptoms are of mottle type in the form of white specks and the affected plants are generally stunted bearing small ears.

Pathogen

Caused by *Ragi mottle streak virus* which are rhabdoviruses transmitted by transmitted by two species of jassids viz., *Cicadulina bipunctella* and *C. chinai*. *C. bipunctella*. Virus particles are short rod like, bacilliform particles. The particles measure 80 nm in cross section and 285 nm lengthwise.

Ragi Streak

Symptoms

Symptoms appear on unfolding young leaves as pale specks or stripes of different size. The specks coalesce involving larger areas resulting in chlorotic bands running almost the entire length of the leaf parallel to the midrib. These bands are occasionally interrupted by dark green areas. The new emerging leaves of both the main shoot and the tillers show number of well defined chlorotic streaks having almost uniform width running parallel to the midrib throughout the length of the leaf lamina. The infected plants in the field produce comparatively more number of tillers and bear yellowish sickly ears, often bearing few shrivelled seeds. The plants infected very early in the crop growth stage die before they bloom.

Pathogen

Eleusine strain of *maize streak virus* and transmitted by leaf hopper *Cicadulina chinai*

Management

Rogue out the infected plants. Spray Monocrotophos or Methyl demeton 500 ml/ha. Spray first on noticing symptoms and repeat twice at 20 days interval.

II. Diseases of Tenai

1. Blast : *Pyricularia setariae*

Symptoms

The spots are seen on the leaf blade. They are circular with light centre and are surrounded by a dark brown margin. The spots are small and scattered. When the disease appears in severe form the leaves wither and dry up. Neck infection is very rare.



Pathogen

The conidiophores emerge through epidermal cells or through stomata. Several conidia are formed one after another from each conidiophore. They are sub-hyaline, three celled and obpyriform. Thickwalled, olivebrown and globose chlamydospores are also developed at the tips of the germ tube.

Favourable Conditions

The optimum temperature is 30OC. High relative humidity (90 per cent), low night temperature and cloudy weather.

Mode of Spread and Survival

The primary source of infection is through seed-borne conidia and to some extent soil-borne. The secondary spread is through air-borne conidia which are produced on ragi, bajra, wheat and *Dectyotacnium aegyptium*.

Management

Treat the seeds with Captan or Thiram 4 g Carbendazim 2 g/kg. Spray the crop with Iprobenphos (IPB) or Edifenphos 500 ml/ha.

2. Leaf spot or Leaf blotch: *Helminthosporium setariae*

(Sexual stage : *Cochliobolus setariae*)

Symptoms

Leaf spots are brown in colour and small. Sometimes lesions also appear as blotches and the rotting of the secondary roots may also occur.

Pathogen

The conidiophores are simple, erect, cylindrical, brown, slightly swollen at the base and geniculate at the apex. Conidia are ellipsoid, straight or slightly curved and pale to moderately dark brown.

Favourable Conditions

Optimum temperature for growth and sporulation is 30OC

Mode of Spread and Survival

Externally seed borne.

Management

Treat the seeds with Captan or Thiram at 4 g/kg.

3. Smut: *Ustilago crameri*

Symptoms

The fungus grows systemically inside the host and express the symptom at the time of flowering. The sori are seen in the flowers and the basal parts of the palea. The fungus affects most of the grains in an ear but sometimes the terminal portion of ear may escape. The sori are pale grey in colour and measures 2 to 4 mm in diameter. When the crop matures the sori rupture and liberate dark powdery mass of spores.

Pathogen

The chlamydospores are dark brown in mass but lighter singly, irregular or angular in shape and smooth walled. The chlamydospores are inter calary in hyphal strands.

Mode of Spread and Survival

The fungus is externally seed-borne and secondary spread by air-borne chlamydospores.

Management

Treat the seeds with Captan or Thiram at 4 g/kg.

4. Rust: *Uromyces setariae italicae*

Symptoms

Numerous minute, brown uredosori appear on both surface of the leaf and are covered by the epidermis for very long time. The pustules are small, oblong and cinnamon brown in colour. The telia are smaller but covered by epidermis for quite a longer period and are grayish black in colour. Severe incidence of disease reduces the yield.

Pathogen

The uredospores are round, spiny, yellowish brown with 3 or 4 germ pores. The teliospores are one celled, smooth, oblong globose and thick walled especially at the apex.

Mode of Spread and Survival

The fungus can also attack other species of *Setaria* viz., *S. glauca*, *S. viridis* and *S. verticillata*. The air-borne uredospores cause primary infection.

Management

No control measure is generally taken against this disease.



III. Diseases of Kuthiraivali

1. Leaf blight: *Helminthosporium crusgalli*

Symptoms

The pathogen attacks all the parts of the plants including roots, base of the plants, culms, leaf sheath, leaf blade, neck of the panicle and the fingers. Both pre-and post- emergence rot may be seen. On young leaves the disease appears as minute, light brown oval spots. The affected leaves wither prematurely and seedlings may be killed. The fungus affects the base of the plants and cause root rot and foot rot. In grown up plants, spots are oblong and dark brown. The spots on the leaf sheath and culms are irregular and are generally found on the junction of blade and sheath. Infection on the neck causes discoloration and sooty growth in the inflorescences.

Pathogen

Hypha of the fungus is light brown coloured and septate. Conidiophores are long, septate, dark brown in colour, often branched and geniculate. Conidia are straight ovoid, pale to dark golden brown, 5-7 pseudoseptate. Pathogen produces spherical perithecia and ascii contain 1 to 8 ascospores.

Favourable Conditions

Optimum temperature for infection is 30-32OC and 80-90% relative humidity. Rains during earhead emergence are favourable.

Mode of Spread and Survival

The pathogen readily infects *Setaria italica*, *Eleusine indica*, *Echinochlora* sp, *Panicum miliaceum*, *Pennisetum typhoides*, *Sorghum bicolor* and *Zea mays*. Primary spread is through seed-borne inoculum and the secondary spread by air-borne conidia.

Management

Treat the seeds with Captan or Thiram at 4 g/kg. Spray with Mancozeb at 1.25 kg/ha.

2. Smut : *Ustilago crus-galli*

Symptoms

The infected ear heads are completely destroyed. The fungus also produces gall-like swellings on the stem, the nodes of young shoots and in the axils of older leaves. The gall-like swellings are covered by a hairy tough membrane of host tissue.



Pathogen

The smut spores are mikado-brown, spherical and echinulated.

Mode of Spread

Externally seed-borne.

Management

Treat the seeds with Captan or Thiram at 4 g/kg.

IV. Diseases of Varagu

1. Head smut: *Sorosporium paspali-thunbergii*

Symptoms

The entire panicle is transformed into a long sorus and cream coloured thin membrane covers the sorus. In some cases it is enclosed in the flag leaf and may not emerge fully. The membrane bursts open and exposes the black mass of spores.

Pathogen

Spores are globose to angular and dark brown with a thick smooth epispor.

Mode of Spread and Survival

Mainly seed-borne. The spores stick to surface of the grains and infect the next crop.

Management

Treat the seeds with Thiram or Captan at 4 g/kg.



4. Etiology, symptoms, mode of spread, survival, epidemiology and integrated management of Diseases of pigeonpea, urdbean, mungbean, soybean and cowpea

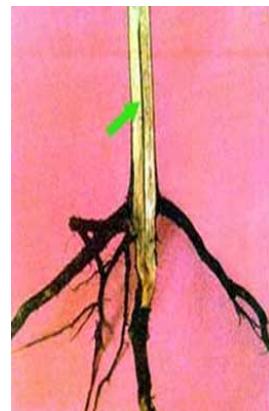
Diseases of pigeonpea or Redgram

1) Wilt : *Fusarium udum* Butler (Perfect stage: *Gibberella indica*)

Introduction: *Fusarium* wilt of pigeon pea is common in Bangladesh, India, Kenya, Thailand and Uganda. It was first reported from Bihar in India during 1931. Wilt of pigeon pea is common disease throughout India. It is very destructive in parts of Bihar, Madhya Pradesh, Maharashtra, and Uttar Pradesh. This disease is responsible for 15 to 25 % mortality rate of plants and this may be more than 50% in epidemic years.

Symptoms

- ❖ It usually occurs near or at the reproductive stages (flowering to pod-filling) of crop growth.
- ❖ Symptoms include the drooping and wilting of the uppermost leaflets and discolouration of the vascular system. Plants become completely yellow and die.
- ❖ When the plants are affected during the mid- to late-pod filling stages, seeds are often shriveled.



Pathogenic characters

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

Mode of spread

- ❖ The fungus is soil borne and survive in the soils. Fungus spreads about 3 m through the soil in one season, apparently along roots.

- The fungus was found to survive in infected plant stubble for 2.5 years in Vertisols and 3 years in Alfisols

Favorable conditions

- Low soil temperature and increasing plant maturity favours wilt.
- Fungal population is highest at 30% soil water-holding capacity and at the soil temperatures between 20 and 30° C.

Management

Grow disease resistant varieties such as AL 1, BDN 2, Birsa Arhar 1, DL 82, H 76-11 , H 76-44, H 76-51 , H 76-65, ICP 8863 (Maruti), ICP 9145, ICPL 267, Mukta, Prabhat, Sharda, TT 5 and TT 6.

Cultural practices

Select a field with no previous record of wilt for at least 3 years. Select seed from disease-free fields. Grow pigeon pea intercropped or mixed with cereal crops, e.g., sorghum. Rotate pigeon pea with sorghum, tobacco, or castor every 3 years. Uproot wilted plants and use them for fuelwood. Solarize the field in summer to help reduce inoculum.

Chemical control

Seed dressing with Benlate T ® (benlate 50 % + thiram 5 0 % mix) @ 3 g kg-1 seed.

2. Root Rot : (Sclerotial stage :*Rhizoctonia bataticola*) (Pycnidial stage :*Macrophomina phaseolina*)

Symptom

- The disease occurs both in young seedlings and grown up plants.
- The lower leaves show yellowing, drooping and premature defoliation.
- The discolored area later turns black and death of plants occur.
- The infected plants can be easily pulled out due to the rotting of the roots.
- Minute dark sclerotia are seen in the shredded bark (collar region and root).
- Large number of brown dots on the stem portion represent the pycnidial stage.
- Prolong dry weather or drought followed by irrigation or rain favours this disease build up.



Pathogen: *Macrophomina phaseolina*

- The mycelium was initially hyaline and later became grey in colour.
- Sclerotia were minute, black, round to oblong or irregular in shape with mycelial attachment

Mode of spread and survival:

The disease is seed borne. The pathogen survives as sclerotia in plant debris in the air. Secondary spread is through soil-borne sclerotia carried by irrigation water and implements. Air borne pycnidiospores help to cause infection in the field.

Management

- Seed treatment with talc formulation of *T. viride* @ 4g or *P. fluorescens* @ 1g/kg seed (or) Carbendazim or Thiram @ 2 g/kg , Neem cake @ 150 Kg/ha
- Soil application of *P. fluorescens* or *T. viride*@ 2.5 Kg / ha + 50 Kg of well decomposed FYM or sand at 30 days after sowing.Spot drenching with Carbendazim @ 1 gm/ litre

3. Alternaria Leaf Spot: *Alternaria alternate*

Symptom

- Initially small necrotic spots appear on the leaves, and these gradually increase in and form characteristic lesions with dark and light brown concentric ring with a wavy purple margin.
- As infection progresses, the lesions coalesce and cause blighting of leaves
- The disease is mostly confined to older leaves in adult plants, but may infect new leaves of young plants, particularly in the post rainy-season.

Pathogen: *Alternaria alternate*

- Hyphae septate, branched, dark coloured
- Conidiophores are short and dark in colour,
- Conidia are beaked, obclavate, formed singly, olive brown, muriform with both horizontal and vertical septation.
- Primary spread is through dormant mycelium in plant debris and Tuber, while secondary spread is through conidia

Mode of spread and survival:

- The disease spreads by airborne conidia in the field. Seed infection has only been reported from Puerto Rico

Epidemiology:

It is not a serious problem in rainy season crops sown at the normal time, but it causes problems in crops sown late September or in the post rainy season on the plains of north eastern India.



Management

- Use disease free seeds. Remove and destroy infected plant debris
- Spray Mancozeb 0.2% or chlorothalonil 0.2%

4) Powdery mildew: (*Oidiopsis taurica* (Lev.) Salmon (Teleomorph: *Leveillula taurica* [Lev.] Amaud)

Symptoms

- It is an Oidiopsis type of powdery mildew in which the mycelium is endophytic.
- The affected leaf shows powdery patches on the lower surface corresponding with yellowing on the upper surface.
- Usually older leaves show symptom first.
- There will be premature defoliation of affected leaves.
- The disease is air borne
- Field symptom

lower

upper surface



Epidemiology

This is a polycyclic disease, i.e., there is an initial infection and secondary spread. Infection is directly proportional to the quantity of inoculum available as conidia. Indian varieties with thin, succulent leaves that are easily colonized by the fungus are more susceptible than those from Kenya that have thicker leaves. The disease develops at temperatures ranging from 20 to 35°C, but 25°C is the optimum. A cool, humid climate is congenial to fungal infection and colonization, but a warm humid climate is good for sporulation and spore dispersal. Sporulation is more frequent on young leaves than on older ones. Plants attacked by sterility mosaic or phyllody support abundant sporulation. Since sterility mosaic and phyllody-infected plants remain green in the field for long periods they provide a continuous source of inoculum. The fungus survives on perennial pigeon peas and volunteer plants growing in the shade, and on the ratoon growth of harvested stubbles. It also survives as dormant mycelium on infected plant parts, e.g., axillary buds. In India early sowing and irrigation encourage disease establishment.

Pathogenic characters

- The mycelium is endophytic bears conidiophore and conidia (oidiopsis type)
- Formation of conidia singly (Pseudoidium type) on long lengthy conidiophore
- The size and shape of conidia mainly ellipsoid–ovoid
- Sexual spores are ascospores from chasmothecium

Management

- ❖ Grow disease resistant varieties such as ICP 9150, ICP 9177.
- ❖ **Cultural practices:** Select fields away from perennial pigeon peas affected with the disease which are a source of inoculum. Sowing late (after July) in India, to reduce disease incidence.
- ❖ **Chemical control:** Spray wettable sulfur @ 1 g L⁻¹ or triadimefon (Bayletan ® 25 % EC) @ 0.03%.

5) Leaf spot : *Cercospora cajani* Hennings, *Cercospora cansescens* (most prevalent)

Economic importance

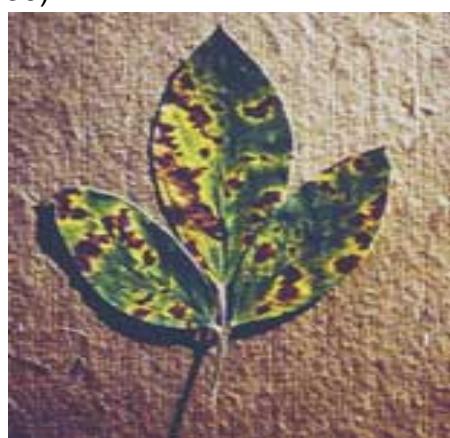
The disease is a problem in humid regions. Yield losses up to 85% have been reported from eastern Africa, and losses are severe when defoliation occurs before flowering and podding.

Epidemiology

Cool temperatures (25°C) and humid weather favor the disease, which normally appears when plants are flowering and podding. Cyclonic rains in southern and north-eastern peninsular India result in sudden outbreaks of the disease in certain years. The disease is more common in the long-duration and perennial pigeon peas grown in eastern Africa.

Symptoms

- ❖ First appear as small circular to irregular necrotic spots or lesions usually on older leaves. These lesions coalesce causing leaf blight and defoliation.
- ❖ During epidemics lesions appear on young branches and cause their tips to dry and die back.
- ❖ The Indian isolates of the pathogen produce a fluffy mycelial growth on their lesions, while the African isolates produce concentric zonations on their lesions (Fig. 30).



Pathogenic characters

The fungus produces large number of whip-like, hyaline, 7-9 septate conidia in groups on the conidiophores which are light to dark brown in colour.

Management

Grow disease resistant varieties such as UC 796/1, UC 2113/1, UC 2515/2, and UC 2568/1.

Cultural control: Select fields away from perennial pigeon peas which are a source of inoculum. Select seed from healthy crops.

Chemical control: Spray maneb (Indofil M 45 ®) @ 3 g L-1 water.

6) Phytophthora Blight: *Phytophthora drechsleri* Tucker f. sp. *Cajani*

Symptoms

- Phytophthora blight resembles damping off disease in that it causes seedlings to die suddenly.
- water-soaked lesions on their leaves and brown to black, slightly sunken lesions on their stems and petioles.
- leaves lose turgidity, and become desiccated.
- Lesions girdle the affected main stems or branches which break at this point and the foliage above the lesion dries up.
- It 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: *Phytophthora Cajani*

- Hyaline coenocytic mycelium.
- Sporangiophores are slender sympodially branched which bears elliptical non-papillate sporangia with slight apical thickening with kidney shaped, biflagellate zoospores.

Sexual spore is oospore

Epidemiology

Cloudy weather and drizzling rain with temperatures around 25°C favor infection that requires continuous leaf wetness for 8 hours to occur. Pigeonpeas gradually develop tolerance to the disease as they grow older and are not infected after they are 60 days old. The disease is more common on Alfisols than on Vertisols, and appears first in

low-lying areas of fields where water temporarily stagnates. The thick canopy of short-duration types occasioned by close spacing (30 x 10 cm), and their higher genetic susceptibility, seem to encourage blight build-up. Warm and humid weather following infection results in rapid disease development and plant death. Wind and rain help to disseminate zoospores and by damaging the plant, facilitate infection. *Cajanus scarabaeoides* var. *scarabaeoides*, a wild relative of pigeonpea is also a host of the blight pathogen.

Mode of spread and survival

The disease is soilborne. The fungus survives as chlamydospores, oospores, and dormant mycelium in soil and on infected plant debris

Management

Grow resistant varieties such as Hy 4, ICPL 150, ICPL 288, ICPL 304, KPBR 80-1-4, and KPBR 80-2-1 (Field resistant)

Cultural practices

Select fields with no previous record of blight. Avoid sowing pigeonpea in fields with low-lying patches that are prone to waterlogging. Prepare raised seedbeds and provide good drainage. Use wide interrow spacing.

Chemical control

Seed dressing with Ridomil M Z ® @ 3 g kg-1 seed. Two foliar sprays of Ridomil M Z ® at 15-day intervals starting from 15 days after germination

7) Pigeon pea sterility mosaic virus

Distribution: Bangladesh, India, Myanmar (Burma), Nepal, and Sri Lanka.

Economic importance: A serious problem in Nepal and India where it is estimated to cause annual pigeon pea grain losses worth US \$ 282 million.

Symptoms:

- ❖ In the field, the disease can be easily identified from a distance as patches of bushy, pale green plants without flowers or pods.
- ❖ The leaves of these plants are small and show a light and dark green mosaic pattern. The mosaic symptoms initially appear as vein-clearing on young leaves.
- ❖ When infection occurs at 45 days after emergence or later, only some parts of the plant may show disease symptoms, while the remaining parts appear normal.
- ❖ Strains of sterility mosaic cause severe internodal shortening of the branches and clustering of leaves. Sometimes these leaves become filiform.
- ❖ Mild chlorosis typical chlorotic ring complete sterility before flowering



Etiology: Unknown Vector.

Eriophyid mite: *Aceria cajani* Channabasavanna

❖ **Epidemiology:** A single eriophyid mite vector is sufficient to transmit the disease. The mites are very small and can be seen easily under a stereobinocular (40x) microscope, they can be wind borne up to 2 km from the source of inoculum. Both pathogen and the mite vector are specific to *Cajanus cajan* and its wild relative *C. scarabaeoides var. scarabaeoides* that is commonly found on wastelands and field bunds. Perennial, volunteer pigeonpeas, and the ratooned growth of harvested plants provide reservoirs of the mite vector and the pathogen. Disease incidence is high when pigeon peas are inter- or mixed cropped with sorghum or millets. Symptoms are suppressed during the hot summer months but with monsoon rains they reappear on the new growth. Shade and humidity encourage mite multiplication, especially in hot summer weather.

Management

Grow disease resistant varieties such as Bageshwari, Bahar, DA 11, DA 13, ICPL 86, ICPL 146, ICPL 87051, MA 165, MA 166, PDA 2, PDA 10, and Rampur Rahar.

Cultural practices: Select a field well away from perennial or ratoon pigeon peas. Destroy sources of sterility mosaic inoculum, i.e., perennial or ratooned pigeon peas. Uproot infected plants at an early stage of disease development and destroy them. Rotate crops to reduce sources of inoculum and mite vectors.

Chemical control: Seed dressing with 25 % Furadan 3 G® or 10% aldicarb @ 3 g kg- 1 seed. Spraying acaricide or insecticides like Kelthane ® , Morestan ® , metasystox @ 0.1 % to control the mite vector in the early stages of plant growth.

BLACK GRAM and GREEN GRAM

1. Powdery mildew – *Erysiphe polygoni*

Symptoms

Small, irregular powdery spots appear on the upper surface of the leaves, sometimes on both the surfaces. The disease becomes severe during flowering and pod development stage. The white powdery spots completely cover the leaves, petioles, stem and even the pods. The plant assumes grayish white appearance, leaves turn black and finally shed. Often pods are malformed and small with few ill-filled seeds.

Pathogen

This is oidium type of powdery mildew. 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, chasmothecia appear as minute, black, globose structures with myceloid appendages. Each chasmothecium contains 4-8 ascii and each ascus contains 3-8 ascospores which are elliptical, hyaline and single celled.

Favourable Conditions

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

Mode of Spread and Survival

The fungus is an obligate parasite and survives as chasmothecia in the infected plant debris. Primary infection is usually from ascospores from perennating chasmothecia. The secondary spread is carried out by the air-borne conidia. Rain splash also helps in the spread of the disease.

Management

Remove and destroy infected debris. Spray Carbendazim 500g or wettable sulphur 1.5g or Tridemorph 500ml/ha at the initiation of disease and repeat 15 days later.

2. **Anthracnose - *Colletotrichum lindemuthianum* (Sexual stage: *Glomerella lindemuthianum*)**

Symptoms

The symptom can be observed in all aerial parts of the plants and at any stage of crop growth. The fungus produces dark brown t black sunken lesions on the hypocotyls area and cause death of the seedlings. Small angular brown lesions appear on leaves, mostly adjacent to veins, which later become grayish white centre with dark brown or reddish margin. The lesions may be seen on the petioles and stem. The prominent symptom is seen on the pods. Minute water soaked lesion appears on the pods initially and becomes brown and enlarges to form circular, depressed spot with dark centre with bright red or yellow margin. Several spots join to cause necrotic areas with black dots (Acervuli). The infected pods have discolored seeds.

Pathogen

The fungus mycelium is septate, hyaline and branched. Conidia are produced in acervuli, arise from the stroma beneath the epidermis and later rupture to become erumpent. A few dark coloured, septate setae are seen in the acervulus. The conidiophores are hyaline and short and bear oblong or cylindrical, hyaline, thin walled, single celled conidia with oil globules. The perfect stage of the fungus produces perithecia with limited number of ascii, which contain typically 8 ascospores which are one or two celled with a central oil globule.

Favourable Conditions

High relative humidity (Above 90 per Cent), Low temperature (15-20° C) and cool rainy days.

Mode of Spread and Survival

The fungus is seed-borne and cause primary infection. It also lives in the infected plant tissues in soil. The secondary spread by air borne conidia produced on infected plant parts. Rain splash also helps in dissemination.

Management

Remove and destroy infected plant debris in soil. Treat the seeds with Carbendazim at 2 g/kg. Spray Carbendazim 500g or Mancozeb 2kg/ha soon after the appearance of disease and repeat after 15 days

3. Leaf spot - *Cercospora canescens*

Symptoms

Small, circular spots develop on the leaves with grey centre and brown margin. Several spots coalesce to form brown irregular lesions. In severe cases defoliation occurs. The brown lesions may be seen on petioles and stem 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.

Favourable Conditions

Humid weather and dense plant population.

Mode of Spread and Survival

The fungus survives on diseased plant debris and on seeds. The secondary spread is by air-borne conidia.

Management

Remove and burn infected plant debris. Spray Mancozeb at 1 kg/ha or Carbendazim at 250g/ha.

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

Symptoms

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 an 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.

Favourable Conditions

Cloudy humid weather, temperature of 21-26° C and Nights with heavy dews

Mode of Spread and Survival

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

Primary infection is by the sporidia developed from teliospores. Secondary spread is by windborne uredospores. The fungus also survives on other legume hosts.

Management

Remove the infected plant debris and destroy. Spray Mancozeb 1 kg or Carbendazim 500 g or Propiconazole 1L/ha, immediately on the set of disease and repeat after 15 days.

5. Dry root rot- *Rhizoctonia bataticola* (Pycnidial stage: *Macrophomina phaseolina*)

Symptoms

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

Pathogen

The fungus produces dark brown, septate mycelium with constrictions at hyphal branches. Minute, dark, round sclerotia of 110-130 are produced in abundance. The fungus also produces dark brown, globose ostiolated pycnidia on the host tissues. The pycnidiospores (Conidia) are thin walled, hyaline, single celled and elliptical.

Favourable conditions

Day temperature of 30°C and Prolonged dry season followed by irrigation.

Mode of Spread and Survival

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 pycnidiospores which are air-borne.

Management

Treat the seeds with carbendazim or thiram at 4g/kg or pellet the seeds with *Trichoderma viride* at 4g/kg of seed.

6. Yellow mosaic disease

Symptoms

Initially small yellow patches or spots appear on green lamina of young leaves. Soon it develops into a characteristics bright yellow mosaic or golden yellow mosaic symptom. Yellow discoloration slowly increases and leaves turn completely yellow. Infected plants mature later and bear few flowers and pods. The pods are small and distorted. Early infection causes death of the plant before seed set.

Favourable Conditions

Summer sown crops are highly susceptible. The presence Weed hosts viz., *Croton sparsiflorus*

Acalypha indica, *Eclipta alba* and *Cosmos Pinnatus* and other legume hosts.

Mode of Spread and Survival

The Virus survives in the weed hosts and other legume crops The disease spreads through wind-borne viruliferous white fly, *Bemisia tabaci*.

Management

Rogue out the diseased plants up to 25 days after sowing. Remove the weed hosts periodically.

Increase the seed rate (25 kg/ha).

7. Leaf crinkle disease - *Urdbean leaf crinkle virus (ULCV)*

Symptoms

The symptom apperars 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 wellas internodes are shortened. The infected plant gives a stunted and bushy appearance Flowering is delayed, inflorescence, if formed are malformed with small size flower buds and fails to open. The age of the plant is prolonged with dark green leaves till harvest

Mode of Spread and survival

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 sap transmissible.

Management

Use increased seed rate (25 kg/ha). Rogue out the diseased plants at weekly interval up to 45 days after sowing. Cultivate seed crop during rabi season. Remove weed hosts periodically. Spray methyl demeton on 30 and 40 days after sowing at 500 ml/ha.

8. Leaf curl / Necrosis - *Groundnut bud necrosis virus (GBNV)*

Symptoms

The infection starts as chlorosis of lateral veins near the leaf margins and margins slowly curl downwards. The infected leaves are brittle and sometimes show vein necrosis on the under surface of the leaves, extending to the petiole. Plants affected in the early stages of growth develop top necrosis and die. Plant may produce a few small and malformed pods.

Favourable Conditions

Rainy days during kharif season show high incidence of disease. the presence of the weed host viz ,*Acanthospermum hispidum*, *Ageratum conyzoides*, *Amaranthus viridis* , *Calotropis gigantean* , *lagasca millis*, *Trianthema portulacastrum*, *cassia tora*, *cleome gynandra*, *solanum nigrum* and *Datura metal* and other legume crops.

Mode of spread and survival

The virus is transmitted by thrips viz., *Frankliniella schultzii*, *Thrips tabaci* and *Scirtothrips dorsalis*. The virus survives in weed hosts, tomato, petunia and Chilli.

Management

Rogue out infected plants up to 30 days after sowing. Remove the weed hosts which harbour virus and thrips. Spray imidachlor at 500 ml/ha on 30 and 45 days after sowing.

Diseases of Soyabean

1. Disease Name: Charcoal rot / Dry root rot

Causal organism: Pycnidial stage: *Macrophomina phaseolina*

Sclerotial stage: *Rhizoctonia bataticola*

Symptoms

- ✓ Disease occurs in patches
- ✓ Symptoms first appear as yellowing of leaves.
- ✓ Within a day or two leaves droop and finally they may drop-off. The plants wilt suddenly within a week.
- ✓ Dark lesions may be seen on the bark at the ground level leading to bark shredding.
- ✓ The basal stem and the main roots may show dry rot symptoms.
- ✓ Dark brown sclerotia seen on roots giving charcoal like appearance and black minute pycnidia on stem near the soil level.
- ✓ Infected plants are easily pulled out.
- ✓

Pathogen character

- ✓ Thallus filamentous, branched, septate, colored (black) mycelium.
- ✓ Parasitism/Life style - necrotroph.
- ✓ Asexual reproduction - Asexual fruiting body -Pycnidia is dark brown, globose with an ostiole. Inner wall of the pycnidium is lined with pycnidiphore bearing pycnidiospore. Pycnidiospores are hyaline, thin, one celled, rod or oval shaped
- ✓ Sexual reproduction – No sexual stage.
- ✓ Resting bodies -irregular, black colored sclerotia which are microscopic
- ✓ Host plants – major food crops (maize, sorghum, pulse crops (common bean, green gram, blackgram), fiber crops (jute, cotton), and oil seed crops (soybean, sunflower, sesame).

Epidemiology

- ✓ Post flowering and grain filling stage
- ✓ Warm temp 36 – 40°C and low moisture

Management

- ✓ Maintaining soil moisture through irrigation, if possible, during the post-flowering period can minimize charcoal rot infestation.
- ✓ Collection and destruction of infected crop residues
- ✓ Treat the seed with *Trichoderma* @4 g/kg and Spot drenching with 0.1 % carbendazim.

2. Disease Name : Wilt

Causal organism: *Fusarium oxysporum f. sp. tracheiphilum*

Symptoms

- ✓ Premature yellowing of leaves
- ✓ Partial wilting of the plant due to infection of lateral roots
- ✓ Patches of dead plants at flowering or podding
- ✓ Visible browning or blackening of the xylem when the main stem split open (**vascular browning**).
- ✓ Die-back symptoms of branches
- ✓ Mode of spread and survival
- ✓ Seed and Soil borne.
- ✓ Survives on infected plant debris in the soil.



Favorable conditions

- ✓ More severe on black soils than on red soils.
- ✓ Ratooning pre-disposes the plant to wilt.
- ✓ Long and medium duration types suffer more than short-duration types.

Pathogen condition

- ✓ Macroconidia – linear, curved, pointed at both ends, thin walled, 3-4 septate
- ✓ Microconidia – small, elliptical, thin walled, single or two celled
- ✓ Chlamydospores – oval or spherical, single or in chains, terminal or intercalary

Management

- ✓ Select fields with no previous record of wilt for at least 3 years.
- ✓ Use disease-free seed.
- ✓ Grow pigeonpea intercropped or mixed with sorghum.
- ✓ Crop rotation with sorghum, tobacco, cumbu or castor
- ✓ Remove wilted plants
- ✓ Solarize the field in summer to help reduce inoculum.

- ✓ Seed treatment with *Trichoderma viride* or *P. fluorescens*
- ✓ Collection and burning of plant residues after harvesting.
- ✓ Green manuring / Compost application @ 12.5 t / ha
- ✓ Spot drenching with Carbendazim @ 1 gm/ litre

3. Disease Name: Soybean Rust

Causal Name: *Phakopsora pachyrhizi* and *P. meibomiae*

Symptoms:

- ✓ Dark brown raised pustules full of uredia on the lower leaf surfaces
- ✓ Infected leaves desiccate and drop off.
- ✓ Extensive defoliation occurs in severe infections.



Pathogen Character:

- ✓ Parasitism - obligate parasite and autoecious and macrocyclic .
- ✓ Uredospores are unicellular, globoid or ellipsoid, yellowish brown.
- ✓ Teliospores are globose or elliptical, unicellular, chestnut brown
- ✓ Aecia are cup shaped; orange coloured and aeciospores are unicellular and elliptical.

Favorable condition:

- ✓ Cloudy humid weather, temperature of 21-26 C and nights with heavy dews.

Mode of Spread and Survival:

- ✓ The pathogen survives in the soil as teliospores and as uredospores in crop debris.
- ✓ Primary infection is by the sporidia developed from teliospores. Secondary spread is by wind-borne uredospores. The fungus also survives on other legume hosts.

Management

- ✓ Remove the infected plant debris and destroy. Spray Mancozeb 1 kg or Wettable sulfur 1 kg/ha
- ✓ Avoid sowing pigeonpea close to bean fields.
- ✓ Rotate crops to reduce the chance of pathogen survival.
- ✓ Spraying Mancozeb 2 g / l of water is effective.

4. Disease Name : Leaf spot

Causal organism: *Cercospora sojana*

Symptoms:

- ✓ Light to dark gray or brown areas varying from specks to large blotches appear on seeds.
- ✓ The disease primarily affects foliage, but, stems, pods and seeds may also be infected.
- ✓ Leaf lesions are circular or angular, at first brown then light brown to ash grey with dark margins.
- ✓ The leaf spot may coalesce to form larger spots. When lesions are numerous the leaves wither and drop prematurely. Lesions on pods are circular to elongate, light sunken and reddish brown.



Pathogen character

- ✓ **Cercospora (anamorph)**
- ✓ **Mycosphaerella (Teleomorph)**
- ✓ Thallus – filamentous, septate, colored (dark grey) mycelium
- ✓ Life style/parasitism – Hemibiotroph (older version Facultative parasite).
- ✓ Asexual reproduction – it produces sporodochium type of asexual fruiting body and inside of it conidiophores are borne that bear septate, long and slender needle shaped condia (multi-celled) with 5-10 transverse septa

Favourable conditions

- ✓ Fungus survives in infected seeds and in debris.
- ✓ Warm, humid weather favor disease incidence

Management

- ✓ Use resistant varieties.
- ✓ Use healthy or certified seeds.
- ✓ Rotate soybean with cereals.
- ✓ Completely remove plant residue by clean ploughing the field soon after harvest.
- ✓ Destroy last years infected stubble.
- ✓ Seed treatment with Thiram + Carbendazium (1:1) @ 2g/kg seed.

- ✓ Spray Mancozeb @ 2g/L or Carbenzadum (500 mg/L).

✓

5. Disease Name: Powdery mildew

Causal organism: *Microsphaera diffusa*

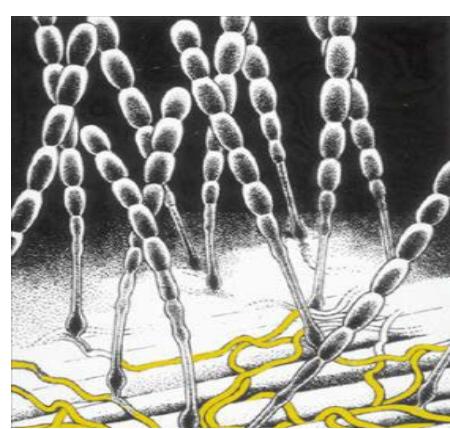
Symptom:

Symptoms

- ✓ White talcum powder like powdery growth occurs on the leaves spreading to cover the stem and other plants parts.
- ✓ The symptoms are severe at flowering stage.
- ✓ In severe cases the entire plant dries up.

Pathogen Character

- ✓ Thallus – filamentous, hyaline, septate mycelium. mycelia are epiphytic (ectoparasites) on the surface of the leaves and pathogens absorbs the nutrients by producing haustoria from the superficial mycelium into the epidermal cells.
- ✓ Parasitism – biotrophs.
- ✓ Asexual reproduction – There is no asexual fruiting body. Asexual spore is conidium. Conidiophore: short, Club shaped , unbranched, non-septate, hyaline
- ✓ Conidia: Barrel shaped , single celled, hyaline , in chain
- ✓ Sexual reproduction - sexual fruiting body called cleistothecia (syn: chasmothecium). Inside the chasmothecium, ascus and ascospores (sexual spores) are produced.



Favourable condition

- ✓ Warm humid weather
- ✓ The disease severe during late Kharif and rabi seasons

Mode of spread and Survival

- ✓ 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 air borne conidia
- ✓ Rain splash also helps in the spread of the disease

Management

- ✓ Remove and destroy infected plant debris
- ✓ Spray Wettable sulphur 1.5 kg or Tridemorph 500 ml / ha

6. Anthracnose – *Colletotrichum lindemuthianum*

Symptoms

- ✓ All aerial parts of the plants are affected.
- ✓ Water-soaked ulcer like sunken lesions on pods - usually with dark centre and bright – red, yellow or orange margins.
- ✓ Small angular brown lesions with greyish white centre and dark brown margin on leaves, petiole and stem.
- ✓ The affected parts may wither off.



Pathogen character

- ✓ Fungus *Colletotrichum lindemuthianum* (anamorph) - *Glomerella cingulata* (Teleomorph)
- ✓ Mycelium is septate, hyaline and branched.
- ✓ Life style/parasitism- hemi - biotroph
- ✓ Asexual stage – asexual fruiting body –acervulus and asexual spore – conidia- are single celled, hyaline, mostly sickle shaped and cylindrical shaped and contains oil globules

Favourable Conditions

- ✓ High RH (>90 per cent), low temp (15-20 C) and cool rainy days.

Mode of Spread and Survival

- ✓ The fungus is seed-borne and cause primary infection. The secondary spread by air borne conidia

Management

- ✓ Spray Carbendazim 500g or Mancozeb 1 kg/ha

7. Disease Name: Downy mildew

Causal organism: -*Peronospora manshurica*

Symptom:

- ✓ Downy mildew appears on the upper surface of young leaves as pale green to light yellow spots.

- ✓ As the spots enlarge, they become pale yellow and of irregular size and shape.
- ✓ During periods of high humidity, a white to tan colored tuft of moldy growth often develops on the undersides of the spots. The spots eventually turn tan in color as they die.
- ✓ Rarely does downy mildew progress to levels high enough to cause defoliation.



Pathogen character

- ✓ Asexual spore – Conidia
- ✓ Sexual spore - Oospore

Favourable condition:

- ✓ The development of this disease is favored by high humidity and cool temperatures. The increased resistance of older leaves and high temperatures slow the development of downy mildew during mid-season, and extensive disease development rarely occurs.

Mode of spread and survival

- ✓ The primary infection is by means of Oospore present in the soil which germinate and initiate the systemic infection
- ✓ The secondary spread is by means of conidia

Management

- ✓ Rouge the affected plant
- ✓ Spray Mancozeb 1 kg/ ha

8. Disease Name : Sclerotinia Stem Rot (White Mold)

Causal organism: *Sclerotinia sclerotiorum*

Symptom:

- ✓ The plants normally rot at the collar region or at any point on the branch.
- ✓ A web of white mycelial strands appears at the collar region (white rot fungus)
- ✓ Whitish or brownish mycelial strands on branches or inside the stem .

Pathogen character:

- ✓ Life style -polyphagous, necrotroph
- ✓ Asexual – never produce conidia and conidiophores.

- ✓ Sexual – homothallic sexual fruiting body- apothecium, ascospores –single celled, hyaline colored
- ✓ Sclerotia – is the most clear sign of the pathogen and the important survival structure. They are macroscopic, black and irregular. They are either mycelogenous or carpogenous type.



Favourable condition:

- ✓ Cool, wet weather favours the disease and mists, dews and fogs provide enough moisture for infection.

Mode of spread and survival:

- ✓ Sclerotinia survives as sclerotia (hard, dark resting bodies) in the soil for many years, and infects many broadleaf crops and weeds.
- ✓ Spread through airborne ascospore

Management:

- ✓ Crop rotation can have some effect, although sclerotia survive for significant periods. Cereals are non-hosts and provide the most effective disease break.

9. Disease Name: Bacterial blight

Causal organism: *Pseudomonas syringae* pv. *Glycinea*

Symptom:

- ✓ Foliar symptoms begin as small, water-soaked spots that turn yellow and then dark brown to black with a yellow border.
- ✓ The spots often coalesce to form irregular brown patches.
- ✓ Portions of the infected area may fall out, giving the leaves a ragged appearance.



Favourable condition:

- ✓ Bacterial blight is favored by cool rainy weather, and is generally more prevalent early in the growing season.
- ✓ Dry and hot weather slows the development of this disease.

Mode of spread and survival:

- ✓ Initial infections may occur during seedling emergence, with secondary disease outbreaks often following windy rainstorms or crop cultivation while the foliage is wet.

Management:

- ✓ Control is obtained by planting disease-free seed, rotating crops, and avoiding cultivation during times when the soybean foliage is wet

10. Disease Name: Soyabean Mosaic Disease

Causal organism: Soyabean Mosaic Virus (SMV)

Symptoms:

- ✓ Diseased plants are usually stunted with distorted (puckered, crinkled, ruffled, narrow) leaves.
- ✓ Pods become fewer and smaller seeds. Infected seeds get mottled and deformed.
- ✓ Infected seeds fail to germinate or they produce diseased seedlings.



Pathogen character

- ✓ It is caused by *Soybean mosaic virus* - a potyvirus. Flexuous particles 750 - 900nm long, ss RNA genome

Mode of spread and survival

- ✓ Soybean mosaic virus is seed borne. The SMV can be transmitted through sap, 32 aphid species are involved in transmission.

Favorable conditions

- ✓ Temperature around 18o C
- ✓ Humid weather

Management

- ✓ Deep summer ploughing.
- ✓ Use resistant or tolerant varieties.
- ✓ Use healthy/certified seeds.
- ✓ Keep the field free from weeds.
- ✓ Rogue out infected plants and burn them
- ✓ Pre-sowing soil application of Phorate @ 10 kg/ha.
- ✓ Two foliar sprays of Thiamethoxam 25 WG @ 100 g/ha or Methyl demeton 800 ml/ha at 30 and 45 days after sowing.

DISEASE OF COWPEA

1) Anthracnose : *Colletotrichum lindmuthianum*

Symptoms

- ❖ Typical symptoms are deep, shrunken lesions containing flesh-colored spores on bean pods
- ❖ Lesions on stems, hypocotyls and leaf veins of seedling plants
- ❖ complete girdling and eventually death of the plant.
- ❖ Infection of the bean pods results in rust-colored lesions that develop into sunken cankers with black ring borders.
- ❖ Severely infected premature pods abort and fall early, while pods that mature produce infected seed with dark cankers that make the seed unmarketable to consumers



Pathogenic characters

- Mycelium is septate.
- Conidiophore bears the Conidia were hyaline, aseptate, falcate, fusiform, tapered gradually to each end.
- Appressoria were dark brown to black, mostly lobed, rarely circular to clavate. Acervuli and setae present.

Favourable Conditions

- ❖ High relative humidity (Above 90 per cent)
- ❖ Low temperature (15-20° C)
- ❖ Cool rainy days.

Mode of spread

The pathogen overwinters in seed and crop residues (primary source of infection) and infects all aerial parts of the bean plant.

Management

Primary inoculum may come from seed (40 percent seed transmission) or from diseased plant debris. Secondary spread is rapid during cool, wet weather. The disease may be controlled by using clean seed, application of benomyl or mancozeb (0.2 percent a.i.) or by growing resistant varieties. Pathogenic variants occur.

2) Dry root rot - *Macrophomina phaseolina* (Sclerotial stage: *Rhizoctonia*)

***bataticola*)**

Symptoms

- ❖ Disease occurs both in young seedlings and grown up plants.
- ❖ Infected seedlings can show reddish brown discoloration at collar region.
- ❖ lower leaves show yellowing, drooping and premature defoliation.
- ❖ Discolored area later turns to black and sudden death of the plants occurs in patches.
- ❖ The bark near the collar region shows shredding.
- ❖ Plant can be easily pulled off leaving dark rotten root in the ground.
- ❖ Minute dark sclerotia are seen in the shredded bark and root tissues.
- ❖ Large number of brown dots seen on the stem portion represents the pycnidial stage of the fungus



Pathogen characteristics

- The mycelium was initially hyaline and later became grey in colour.
- Sclerotia were minute, black, round to oblong or irregular in shape with mycelial attachment

Epidemiology (Favourable Conditions)

- ❖ Prolonged drought followed by irrigation.
- ❖ High temperature of 28-35°C.

Mode of survival and spread

The primary spread of the disease is by seed and soil. Secondary spread is by air-borne conidia. The pathogen survives as sclerotia in the soil as facultative parasite and in dead host debris.

Management

- ❖ Treat the seeds with carbendazim or thiram at 2g/kg or pellet the seeds with *Trichoderma viride* at 4 g/kg (10^6 cfu/g).
- ❖ Apply heavy doses of farm yard manure or green leaf manure like Gliricidia maculata at 10 t/ha or apply Neemcake at 150 kg/ha.

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

Symptoms

- ❖ 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 characteristics

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

Epidemiology (favourable Conditions)

- ❖ Cloudy humid weather, temperature of 21-26° C.
- ❖ Nights with heavy dews

Mode of Spread and Survival The pathogen survives in the soil through teliospores and as uredospores in crop debris. Primary infection is by the sporidia developed from teliospores. Secondary spread is by windborne uredospores. The fungus also survives on other legume hosts.

Management

- ❖ Remove the infected plant debris and destroy.
- ❖ Spray Mancozeb 2 kg or Carbendazim 500 g or Propiconazole 1L/ha, immediately on the set of disease and repeat after 15 days

4) Leaf spot : *Cercospora cruenta*

Symptoms:

- ❖ Spots appear on both sides of leaf
- ❖ On upper surface the spots appear brown with distinct dark border.
- ❖ On lower surface lesions have less distinct margin with grey centre due to the production of condioophores and conidia.
- ❖ The lesions become angular as they are limited by the veins. Leaves dry and drop.
- ❖ die and fall off.



Pathogen: *Cercospora cruenta*

- Sexual stage – *Mycosphaerella cruenta*
- Geniculate conidiophores bears pale olivaceous both ends blunt 2 to 8 septate conidia.
- Black perithecia bears ascospores with 8 ascospores.
- Primary spread is through soil borne ascospores and secondary spread is through wind borne conidia

]Mode of survival and spread

The fungus survives between crops on seed and in crop debris. It may also survive on legume weeds. Transmission is by air-borne spores produced on the underside of the leaf. Both windborne ascospores and conidia are thought to be spreading agents.

Management

Cultural approaches:

- ❖ VRB-10 is completely resistant to Cercospora leaf spot while VRB7 is highly susceptible, so care should be taken in choosing varieties to plant.
- ❖ Manual seed cleaning, to remove plant debris, will prevent carry-over of the fungus and should be encouraged, given the many farmers who save seeds for the next season.
- ❖ Intercropping by planting alternate rows of cowpea and another suitable non-legume crop, such as maize or sorghum, will limit spread of the disease within a field but not eliminate it.
- ❖ Burying or destroying the remains of a cowpea harvest will reduce the amount of fungus able to infect new crops, as will removing alternative hosts, but these are costly and time-consuming measures which may not appeal to, or be feasible for, all farmers.

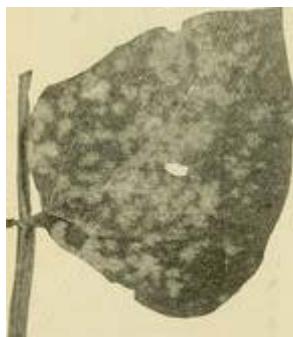
Chemical control: Mancozeb should be applied after the crop has flowered and pods are starting to develop, with a maximum of 2-3 applications per planting season

5) Powdery mildew : *Erysiphe polygoni*

Symptoms

- ❖ Small, irregular powdery spots appear on the upper surface of the leaves, sometimes on both the surfaces.
- ❖ The disease becomes severe during flowering and pod development stage.
- ❖ The white powdery spots completely cover the leaves, petioles, stem and even the pods.

- ❖ The plant assumes greyish white appearance; leaves turn yellow and finally shed. Often pods are malformed and small with few ill-filled 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 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 ascii and each ascus contains 3-8 ascospores which are elliptical, hyaline and single celled.

Favourable Conditions

- ❖ Warm humid weather.
- ❖ The disease is severe generally during late kharif and rabi seasons.

Disease cycle

The Pathogen 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.

Management

- ❖ Remove and destroy infected plant debris.
- ❖ Spray Carbendazim 500g or Wettable sulphur 2kg or Tridemorph 500 ml/ha at the initiation of disease and repeat 15 days later.

6) Bacterial blight : *Xanthomonas phaseoli*

Symptoms

- ❖ Initial symptoms are tiny, water-soaked dots under the leaf. These vary from pinpoint size to more than 1.25cm in diameter, with a yellow halo. They often expand, join up and develop into large necrotic lesions.
- ❖ The pathogen also invades the stem, causing cracking with brown stripes, and the pods, where they manifest as dark green, water-soaked areas.
- ❖ Infected seeds are discoloured and shrivelled. In a severe infestation, pod development is poor and most of the seeds are shrivelled and unable to germinate.



Pathogenic characters

- Bacteria is gram negative, rod shaped bacteria. It enters into the host through wounds or natural opening. It spreads through water, propagating materials, pruning tool etc.

Epidemiology (Favourable conditions)

The bacteria can remain viable for nearly 400 days in infected seed and debris at temperatures of 5°C to 10°C, and for 250 days at temperatures between 10°C and 40°C. The pathogen can survive in the soil for 260 days at 10°C and 100 days at 40°C. Farmers who plant their own seed from the previous season could suffer a 100% crop loss if the seed is already infected and cool, wet weather conditions prevail.

Mode of survival and spread

This aggressive pathogen is carried on the seeds and can survive in soil, crop residue, and seed, as well as on alternative hosts such as lablab bean, common bean and sun hemp. Seed and crop residue are the main source of infection, but it is also spread by insects and wind-driven rain.

Management

- ❖ The disease can be managed in several ways: cultural practices, intercropping cowpea with maize or cassava, planting disease-free seed, and timely application of registered chemicals.
- ❖ A combination of extracts of pawpaw, neem and red acalypha reduced disease incidence by 73.68% and improved yield by 1.58 tons/ha (a 73.49% increase) compared with untreated control
- ❖ *Erwinia herbicola* and *Pseudomonas oxalicum* are showing antagonistic relationship against this pathogen.
- ❖ Streptomycin sulfate of 0.2% concentration was applied as a positive control and sterile distilled water as a negative control.

7) Cowpea Mosaic Virus

Symptoms

- ❖ Mosaics, mottling, interveinal chlorosis and vein-banding.
- ❖ Seed borne leaves shows vein-clearing, vein-yellowing, diffused chlorotic spots or patches
- ❖ vein-yellowing, or variable degrees of yellow mosaic with or without dark-green, irregular vein-banding and blistering.
- ❖ Mosaic mottling with dark green vein – banding, leaf distortion, blistering, stunting and reduced leaf lamina.



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Vectors: Aphid - *Aphis craccivora*, *A. gossypii*, *A. fabae* and *Myzus persicae*

Pathogenic characters

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.

Epidemiology

The seed transmissibility of CABMV reflects its wide geographical distribution, and probably also virus survival in the off-crop season. The role of weeds and wild legumes as reservoirs of CABMV infection has yet to be determined; however, there is evidence that irrigation and perennially damp areas provide reservoirs of CABMV in the semi-arid savannah of West Africa. Infection from infected seeds plays an important role in initiation of the disease, whereas aphids are important in the secondary spread of the disease under field conditions. Cultivation of virus-susceptible cowpea cultivars in a large area is another factor which favours disease spread.

Management

- ❖ Early sowing and intercropping of cowpea with cereals,
- ❖ The use of virus-free seed
- ❖ Pyrethroid cypermethrin restricts the acquisition and inoculation of the virus, and protects against its transmission; however, the initial virus introduction was not prevented by these synthetic pyrethroids.

5. Etiology, symptoms, mode of spread, survival, epidemiology and integrated management of Diseases of groundnut, sesame and castor

GROUNDNUT or Peanut (*Arachis hypogaea*)

List of diseases:

| S.No. | Name of the disease | Causal organisms |
|--------------|--------------------------------------|---|
| 1. | Groundnut Early leaf spot | <i>Cercospora arachidicola</i> (<i>Mycosphaerella arachidicola</i>) |
| 2. | Groundnut Late Leaf Spot | <i>Phaeoisariopsis personata</i> (Syn. <i>Cercospora personata</i>) |
| 3. | Groundnut Rust | <i>Puccinia arachidis</i> |
| 4. | Stem and Pod rot | <i>Sclerotium rolfsii</i> |
| 5. | Dry root rot | <i>Macrophomina phaseolina</i> |
| 6. | Crown rot / Black mould | <i>Aspergillus niger</i> |
| 7. | Yellow mould / Aflaroot | <i>Aspergillus flavus</i> |
| 8. | Bud Necrosis or Stem Necrosis | Thrips |
| 9. | Groundnut rosette virus | Aphids |
| 10. | Kalahasti Malady (Pod Scab nematode) | <i>Tylenchorhynchus brevilineatus</i> |

1. Groundnut Early leaf spot

Introduction:

Groundnut (*Arachis hypogaea*) is one of the important oil seeds and food crops of the world as it provides an inexpensive source of high quality dietary protein and edible oil. Early and late leaf spots, commonly called as tikka disease in groundnut - economically important foliar fungal diseases. Infection causes early death of the leaves and dramatic yield loss. This is estimated to range from 10 % to 80 % and varies according to the environment and availability of control methods. Leaf spot causes significant yield loss, and can be found wherever Groundnut is grown. This includes areas such as USA, Australia, Fiji, Solomon Islands and Tonga, worldwide and in India states such as Gujarat, Andhra Pradesh, Karnataka, Tamil Nadu and Maharashtra.

Causal Organism:

Anamorph stage: *Cercospora arachidicola*

Teleomorph stage: *Mycosphaerella arachidicola*

Symptoms:

- The disease occurs on all above ground parts of the plant, more severely on the leaves.
- Occurs early in crop season – 3 to 4 weeks after sowing.

- Sub-circular to irregular dark brown spots are produced on the leaf
- Yellow halo is seen around the brown spots.

Advanced stage:

- Oval to elongate spots are also seen on stems, petioles, and pegs.
- Severe disease attack leads to shedding of leaflets resulting in premature ageing of the crop.

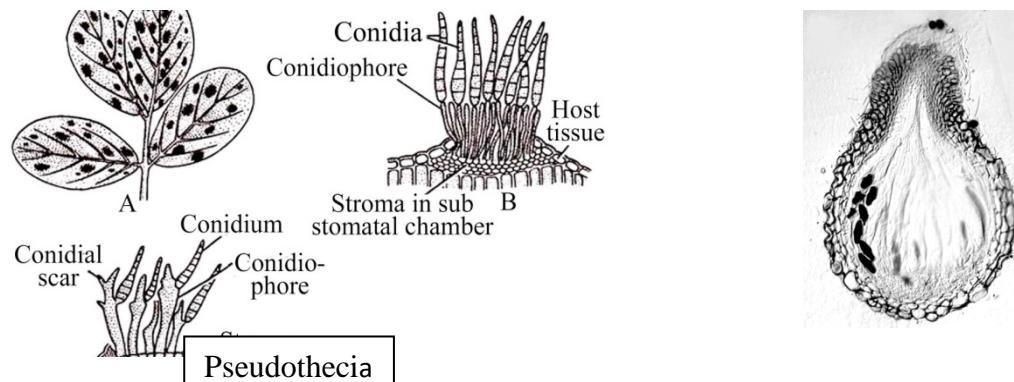


Pathogen character:

Fungus is both intercellular and intracellular. Abundant sporulation on the upper surface of the leaves.

Conidiophores are olivaceous or yellowish brown in colour, short, 1-2 septate, unbranched and geniculate and arise in clusters. Conidia are sub hyaline or pale yellow, obclavate, often curved, 3-12 septate. $35 - 110 \times 2.5 - 5.4 \mu\text{m}$ in size with rounded to distinctly truncate base and sub-acute tip.

In 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 2 celled. Apical cell larger than the lower cell.



Favourable conditions/Epidemiology:

In anamorph stage, it survives as stroma or mycelium in crop residue.

Primary infection usually occurs after a period of rain, where the leaf is continually wet, and the pathogen thrives in areas of high relative humidity and moderate temperature (25-30 °C).

Mode of spread and survival:

The pathogen survives for a long period in the infected plant debris through conidia, dormant mycelium and perithecia in soil. The volunteer groundnut plants also harbour the pathogen. The primary infection is by ascospores or conidia from infected plant debris or infected seeds. The secondary spread is by wind blown conidia. Rain splash also helps in the spread of conidia.

Management:

- Remove and destroy the infected plant debris.
- Eradicate the volunteer groundnut plants.
- Keep weeds under control.
- Follow cereal-cereal-groundnut crop rotation.
- Grow moderately resistant cultivars like ALR 1, 3, VRI (Gn) 5
- Spray 10 % Calotropis leaf extract
- Treat the seeds with Carbendazim or Thiram at 2g/kg.
- Spray carbendazim at 1 g/lit or chlorothalonil at 2 g/litor tebuconazole at 1 g/lit.
at 10 – 15 days interval starting from disease occurrence.

2. Late Leaf Spot

Introduction:

Late leaf spot (LLS) is a major foliar disease that reduces the pod yield and severely affects the fodder and seed quality in groundnut. Late leaf spot (LLS) and rust are two major foliar diseases of peanut that often occur together leading to 50–70% yield loss in the crop.

Causal Organism:

Phaeoisariopsis personata (Syn. *Cercospora personata*)

Anamorph stage: *Phaeoisariopsis personata* (Syn. *Cercospora personata*)

Teleomorph stage: *Mycosphaerella berkeleyii*

Symptom:

- Late leaf spots are nearly circular and darker than early leaf spots
- Late leaf spots are darker with no or light yellow halo.
- Lower surface lesions are carbon black in colour.
- Spots appear 5 to 7 weeks after sowing.

Advanced stage:

- Severe disease attack leads to shedding of leaflets prematurely.

- Oval to elongate spots similar to early leaf spot are also formed on stems and pegs.
- Late leaf spot attack is usually seen along with rust disease.

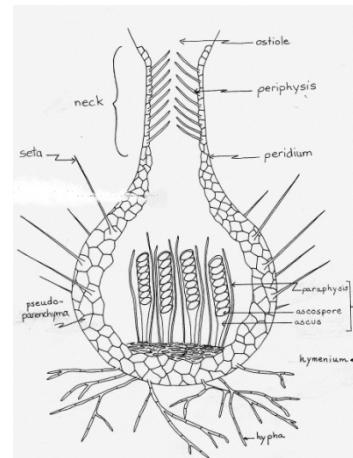


Pathogen character:

Fungus produces intercellular and intracellular mycelium. The conidiophores are long, continuous, 1 – 2 septate, geniculate, arise in clusters on lower surface and are olive brown in colour.

Conidia are cylindrical or obclavate, short, 18 - 60 x 6 – 10 µm, 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, contains 8 ascospores. Ascospores are 2 celled and constricted at septum and hyaline.



Favourable conditions/Epidemiology:

Temperatures in the range of 25 to 30°C and high relative humidity favor infection and disease development.

Mode of spread and survival:

The primary infection is by ascospores or conidia from infected plant debris or infected seeds.

The secondary spread is by wind blown conidia. Rain splash also helps in the spread of conidia.

The pathogen survives for a long period in the infected plant debris through conidia, dormant mycelium in soil.

Management:

- Intercropping pearl millet or sorghum with groundnut (1 : 3) is useful in reducing the intensity of late leaf spot.

- Crop rotation with non-host crops preferably cereals.
- Deep burying of crop residues in the soil, removal of volunteer groundnut plants are important measures in reducing the primary source of infection.
- Spray Carbendazim 0.1% or Mancozeb 0.2% or Chlorothalonil 0.2%.
- Spay hexaconazole (0.1%) or difenoconazole (0.1%) at three times on 30, 45 and 60 days old plant to manage leaf spots and rust of groundnut.

Difference between Early Leaf Spot and Late Leaf Spot:

| Character | Early Leaf Spot | Late Leaf Spot |
|--|--|--|
| Stages of occurrence | Early infection | Late infection |
| Shape of spot | Irregular | Circular |
| Colour of the spot | Brown with yellow halo | Black without yellow halo |
| Perfect stage | <i>Mycosphaerella arachidicola</i> | <i>Mycosphaerella berkeleyii</i> |
| Mycelium | Intercellular without haustoria | Intercellular with haustoria |
| Conidia | Sub-hyaline, Slightly olivaceous, often curved, 3-12 septate, lengthier one. | Hyaline, olive brown, short, straight, 3-4 septate |
| Leaf surface on which most spores are produced and their arrangement | Upper surface, random arrangement | Lower surface, in concentric rings |

3. Rust

Introduction:

Rust causes serious damage to groundnut crops in many parts of the world with pod yield losses of up to 70% being reported (Harrison, 1973; Subrahmanyam and McDonald, 1987). It is a destructive disease in several South and Central American countries. In the Caribbean region, rust limits commercial groundnut production.

Causal organism:

Puccinia arachidis



Symptoms:

- Infection seen on 6 weeks old crop.
- Brick red pustules on lower surface of leaf.
- Upper surface necrotic brown spots.
- Pustules seen on leaf and stem.
- Dark coloured teliosori appear which produces teliospores.

- Leaves dry, drop prematurely, seeds small and shriveled.

Pathogen Character:

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 alternate host.

Favourable conditions/Epidemiology:

High relative humidity (above 85 per cent), heavy rainfall and low temperature (20-25 C).

Mode of spread and survival:

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

Management:

- Removal of volunteer groundnut plants from the field
- Adopt cereal-cereal-groundnut crop rotation.
- Adjust the sowing time to avoid the most conducive environmental condition for rust development
- Grow resistant cultivars like ALR 1, 3 and VRI (Gn) 5
- Spraying of tridemorph at 1.0 ml/lit or chlorothalonil at 2 g/lit or mancozeb 2 g/lit or wettable sulphur 5 g/lit of water controls rust.

4. Stem and Pod rot

Introduction:

Stem and pod rot, also called southern blight, is a fungal disease that occurs wherever groundnuts are grown. It causes up to 10-25% reduction in pod yields worldwide. Losses in Africa are not well recorded but, as it is present in more than 45 countries, they are likely to be high.

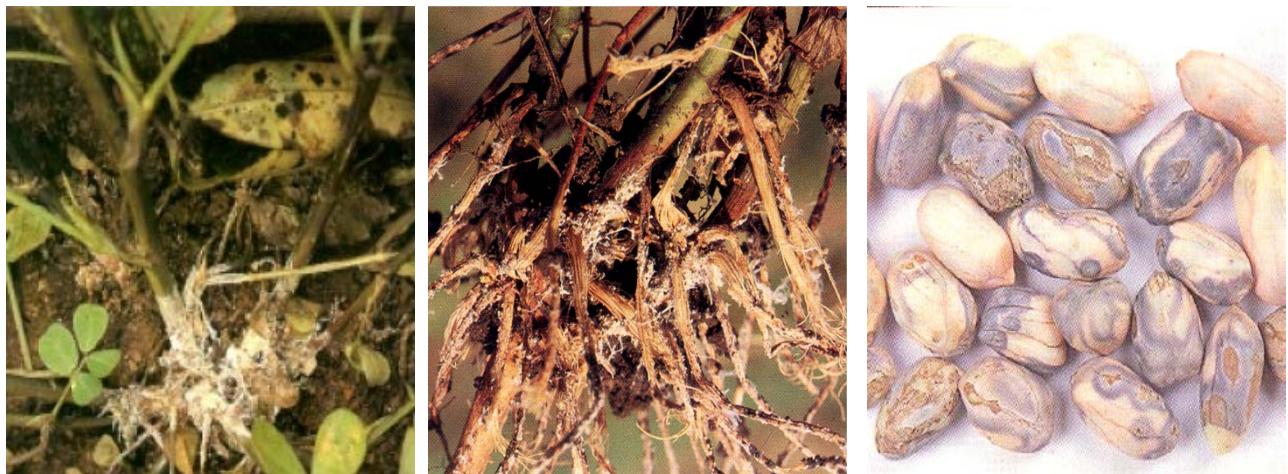
Causal Organism:

Asexual stage: *Sclerotium rolfsii*

Sexual stage: *Athelia rolfsii*

Symptoms:

- Yellowing and wilting of branches near the base of the plant is the first symptom.
- White fungus growth develops at or near the soil around the affected stem.
- Mustard seed like, spherical, brown colored sclerotia are formed as a sign of the disease at the collar region.
- Infection of pegs, pods, and roots occurs either independently or together with stem infection.
- Infected developing pegs may retard pod development.
- Dark brown colored sclerotia are formed on the stem.
- Severely infected pods are completely covered with a white fungal growth, and eventually decay.
- In some cases the seeds from the diseased pods show a characteristic bluish-grey discolouration known as 'blue damage' due to production of oxalic acid.



Pathogen Character:

Sclerotium rolfsii is a necrotrophic, soilborne fungal plant pathogen that produces abundant white mycelium on infected plants and in culture. Advancing mycelium and colonies often grow in a distinctive fan-shaped pattern and the coarse hyphal strands may have a somewhat rosy appearance.

Favourable conditions/Epidemiology:

Alternate wet and dry periods favours the disease.

Prolonged rainy season at seedling stage.

Low lying areas and acidic soil.

Mode of spread and survival:

It survives as sclerotia in soil and as mycelium in crop debris. Thrives in highly aerobic environments and thus survives best near the soil surface.

Management:

- Cultural practices like deep ploughing, sanitation and control of leaf spots to prevent leaf drop helps in controlling stem rot.
- Control of moisture and soil solarization helps in controlling stem rot.
- Crop rotation with cotton, wheat, onion and garlic is an effective means of control.
- Seed treatment with Captan at 4 g/kg seed reduce stem rot incidence. Tebuconazole and propiconazole soil drenching.
- Biological control with *Trichoderma* spp

5. Dry root rot

Introduction:

In India groundnut plantation has suffered a 55 to 85 percent root rot disease caused by multiple pathogen complex mainly *Aspergillus niger*, *Aspergillus flavus*, *Sclerotium rolfsii*, *Thielaviopsis basicola*, *Rhizoctonia solani* and *Pythium aphanidermatum* perennating in soil and seed.

Causal organism:*Macrophomina phaseolina*

Symptoms:

- Brown water soaked lesion on the stem just above the soil level.
- The lesion darkens, affected collar region is girdled and plants wilts.
- In the dead tissue, many sclerotia are formed.
- Bark shreds and studded with numerous sclerotia.
- Blackening of shells.



Pathogen Character:

- The fungus invades the host both inter and intra-cellularly. It grows rather fast, covering large areas of the host tissues and eventually killing them in a short time.
- It produces numerous sclerotial bodies on the host tissue, which measure about 110-130 μ in diameter.
- Often the pycnidial stage is produced on the host. The pycnidia are dark brown, ostiolate and of varying size. The pycnidiospores are elliptical, thin walled, single celled, hyaline and measure 10-42x6-10 μ .

Favourable conditions/Epidemiology:

High C:N ratio in the soil and low bulk density as well as low soil moisture content favours the disease.

Mode of spread and survival:

It survives in the soil mainly as microsclerotia that germinate repeatedly during the crop-growing season. The microsclerotia overwinter in the soil and crop residue and

are the primary source of inoculum in the spring. They have been shown to survive in the soil for up to three years.

Management:

- Good Agricultural Practices (GAP) such as balanced fertilization, timely irrigation and pest management encourage good crop growth which may help in reducing the disease.
- Seed treatment with *Trichoderma viride*.
- Seed treatment with Carbendazim 2g/kg or Captan 3g/kg or Thiram @ 4g/kg.
- Spot drench with Carbendazim at 0.5 g/lit.
- PCNB (Brassicol & 75% WP) 0.5% can also be applied or in the form of soil dust 25 kg/ha in two split applications, 12.5 kg/ha before sowing and the rest 12.5 kg/ha at 15 days after first application.

6. Crown rot / Black mould

Introduction:

Collar rot is one of the economic important disease. Collar rot damaged regularly due to its seeds and soil borne nature. This disease has prevalent in almost all groundnut growing states. This disease is extensive in rainy season than summer season.

Causal organism:*Aspergillus niger*

Symptom:

The fungus causes pre emergence rot post emergence rot and crown rot symptoms

Pre-emergence rot – rotting of seeds, germination affected.

Post-emergence rot - Circular brown spots occurs on the cotyledons of seedling.

- Brown spots occurs on collar region that become soft and rots. Collapse of seedling and stem shredding are seen.
- The infected areas of seedlings are covered with black fungal spores.
- Mature plants are also attacked.

Crown rot

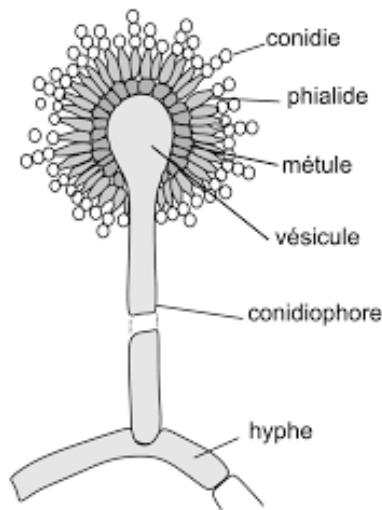
- Large lesions develop on the stem below the soil and spread upwards along the branches causing drooping of leaves and wilting of plant.
- Infected pods reveal patches of black sooty spores



Pathogen Character:

The mycelium of the fungus is hyaline to sub-hyaline. Conidiophores arise directly from the substrate and are septate, thick walled, hyaline or olive brown in colour. The vesicles are mostly globosed and have two rows of hyaline phialides viz., primary and secondary phialides. The conidial heads are dark brown to black. The conidia are globose, dark brown in colour and produced in long chains.

Favourable



conditions/Epidemiology:

High soil temperature 37° C and light textured soil, deep sowing and spreading type of varieties.

Mode of spread and survival:

Pathogen is seed-borne and spores are present on the seeds.

Pathogen perpetuate through the contaminated soil.

Management:

- Deep sowing of seed should be avoided.
- Avoiding mechanical damage, destroying plant debris and deep ploughing.
- Crop rotation with gram and wheat is useful in reducing the collar rot disease incidence.
- Seed treatment with *Trichoderma viride*/*T. harzianum* @ 4g/kg seed and soil application of *Trichoderma* @2.5kg/ha along with well decomposed FYM @50 kg/ha.
- Seed treatment with Thiram or Captan @ 2g/kg seed.

7. Yellow mould / Aflaroot

Introduction:

This disease does not reduce the yield, but the quality of the produce is very poor. In the early 1960s, aflatoxin, a toxic metabolite of *Aspergillus flavus* was found in peanut meal. Feed prepared with this meal caused the death of 100 000 turkeys in Great Britain. A very small amount (10-20 ppb) can produce fatal liver cancer in young animals. Yellow mould is more severe in the tropics, with symptoms appearing both

early in the growth of the peanut seedlings, and near harvest time on pods and seeds in the soil.

Causal organism: *Aspergillus flavus*

Symptom:

Seeds are covered with yellowish green spore masses.

Seeds disintegrate in 4-8 days (pre-emergence seed rot)

In the emerging seedlings, cotyledons are attacked and covered by yellowish-green spores, leading to death.



Pathogen Character:

Fungus produces both inter and intra cellular mycelium. It produces numerous sclerotial bodies on the host tissue, which measure about 110 to 130 µm in diameter. Often the pycnidial stage is produced on the host. Pycnidia are dark brown, ostiolate and of varying size. Pycniospores are elliptical, thin walled, single celled, hyaline and measures 10 - 42 x 6 – 10 µm.

A. flavus produces aflatoxin (mycotoxin)

Mode of spread and survival:

Seed -borne and soil-borne

Management:

- Since the fungus is a weak parasite, agronomic practices which favour rapid germination and vigorous growth of seedling will reduce the chance of *A. flavus* infection.
- Seed treatment with carbendazim or captan or thiram at 2g/kg seed.

8. Bud Necrosis or Stem Necrosis

Introduction:

This is a virus disease causing more damage to groundnut crop during kharif and summer. Virus is mainly transmitted through thrips.

Causal organism:

Tomato spotted wilt virus (TSWV). RNA virus, multipartite virus. Persistent virus - It is transmitted by thrips like *Thrips tabaci*, *Frankliniella schultzei* and *Scirtothrips dorsalis*

Symptom:

- First symptoms are visible 2-6 weeks after infection as ring spots on leaves.
- Young 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 exhibit necrotic streaks. Later the plant become stunted with short internodes and short auxiliary shoots.
- 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 testas.



Pathogen Character:

Virus particles are spherical, 30 nm in diameter, enveloped ssRNA with multipartite genome.

Mode of spread and survival:

Primary spread – Thrips. (*Thrips palmi*, *T. tabaci* and *Frankliniellasp.*)

Secondary spread – Infected plants within the same field.

Spread of this virus in groundnut, is mostly Monocyclic and disease incidence in this host depends on infection by thrips that acquire the virus from other crops or weed hosts (*Tagetes minuta* and *Trifolium subterraneum*)

Management:

- Use resistant/tolerant cultivars
- Rogue out bud necrosis affected plants up to 6 weeks after sowing.
- Early sowing (15 June to 15 July) and maintain high plant density.
- Adopt 10 x 40 cm spacing (70 X 15 cm higher incidence) and remove infected plants up to 6 weeks after sowing
- Intercropping with cereals like pearl millet will restrict spread of the virus.
- Avoid groundnut cultivation adjacent to the crops that are susceptible to bud necrosis, such as green gram or black gram.
- Spray with AVP (anti viral principles)
- Spray methyl demeton 500 ml/ha 30 DAS either alone or in combination with AVP – sorghum or coconut leaf extract 10 % on 10 and 20 DAS to manage the insect vector.

9. Groundnut rosette virus

Introduction:

Groundnut rosette virus was first described in Africa in 1907 and causes serious damage to groundnut crops on that continent. In 1939 it was reported to infect 80 to 90% of plants in the Belgian Congo causing major losses in yield.

Causal organism:

Mixture of viruses like, Groundnut Rosette Assistor Virus (GRAV), Groundnut Rosette Virus and Groundnut Rosette Satellites.

Transmitted by *Aphis craccivora*



Symptom:

Chlorotic rosette:

Faint mottling of young leaflets, newly formed leaves

are smaller chlorotic, curled and distorted.

Internode become shortened. Flowering is decreased.

Green rosette:

Plant are severely stunted with extreme proliferation and rosetting of secondary shoots.

Pathogen Character:

Isometric, not enveloped and 28 nm diameter (Reported from India)

It gives no overt symptoms in groundnut.

Groundnut Rosette Virus is with ssRNA genome, which becomes packaged in GRAV virus and thus depends on it for aphid transmission, but produces no overt symptoms in groundnut.

The Groundnut Rosette Satellites are satellite RNA that control the symptoms and cause the different types of rosette (chlorotic, green and mosaic).

Mode of spread and survival:

The primary source of spread by aphid vector, *Aphis craccivora* and *A. gossipii* in a persistent manner, retained by vector but not transmitted congenitally. The virus is not transmitted by any other means like mechanical or seed or pollen. The virus can survive on the volunteer plants of groundnut and other weed hosts.

Management:

- Practice clean cultivation.
- Use heavy seed rate and rogue out the infected plants periodically.
- Spray methyl demeton at 500 ml/ha.

10. Kalahasti Malady (Pod Scab nematode)

Introduction:

A severe nematode disease of groundnut, popularly called 'Kalahasti malady', caused by the nematode *Tylenchorhynchus brevilineatus* has been prevalent since 1976 in certain parts of Andhra Pradesh, India. One of the resistant genotypes is a high-yielding breeding line (TCG 1518) and this is being released for use in disease-affected areas of Andhra Pradesh State.

Causal organism: *Tylenchorhynchus brevilineatus*

Symptom:

- Small 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.



Management:

- Grow resistant varieties
- Crop rotation with rice
- Irrigate the field.
- Apply carbofuran 3G @ 1.0 kg a.i/ha 25 -30 days after sowing along with irrigation water.
- Application of gypsum @ 200 kg/ha at the time of earthing up.

Reference:

1. <https://www.ikisan.com/up-groundnut-disease-management.html>
2. <http://agropedia.iitk.ac.in/content/integrated-disease-management-groundnut#:~:text=Dry%20root%20rot%2Fdry%20wilt%3A,borne%20and%20seed%20borne%20sclerotia.>
3. https://www.biologydistribution.com/plants/plant-diseases/diseases-of-groundnut-plant-diseases/43123#Disease_10_Groundnut_Rosette

Seasame or Gingelly diseases

Disease: Root rot/ Stem rot/ Charcoal rot

Causal organism: *Rhizoctonia bataticola* (Pycnidial stage: *Macrophomina phaseolina*)

Symptoms:

- Dark brown lesions at the collar region
- Yellowing, drooping and defoliation of leaves
- Shredding of bark. Rotten roots and stem tissues harbours sclerotia
- Plant can be easily pulled out
- Pods infected and open prematurely
- Pycnidia seen on infected capsules and seeds



Pathogen: The fungus produces a large number of black, round to irregular shaped sclerotia. The pycnidia are dark brown to black with an ostiole and contain numerous single celled, thin walled, hyaline and elliptical pycnidiospores.

Favourable conditions: The disease is favoured by moisture stress and high soil temperatures. Fungus is soil borne and survives in the form of sclerotia in the soil.

Management

- Frequent irrigations to avoid moisture stress and early season planting and selection of short duration cultivars
- Spot drenching with carbendazim @ 1 g/ litre
- Soil application of *Pseudomonas fluorescens* or *Trichoderma viride* –2.5 Kg / ha + 500 Kg of well decomposed FYM or vermicompost or sand at 30 days after sowing

Disease: Leaf blight

Causal organism: *Alternaria sesami*

Symptoms

- Small,circular reddish brown spots with concentric rings on leaves
- Dark brown lesions on petioles,stem and capsules
- Blighting of leaves,defoliation ,splitting, of capsules and shriveled seeds

Pathogen: The pathogen produces cylindrical conidiophores, which are pale grey-yellow coloured, straight or curved, geniculate, simple or branched, septate and bear chain of conidia. Conidia are pale grey-yellow to pale brown, pyriform with beak.



Favourable conditions/Epidemiology: High humidity and moderate to warm temperatures favours the disease. The fungus can survive as mycelium in refuse from diseased plants at least for one season and possibly two years in dry conditions. Conidia are air borne.

Mode of spread and survival: Primary inoculum comes from fungus survives on infected plant debris and secondary spread is through wind born conidia.

Management:

- Selection of healthy seeds for sowing; Early sowing during kharif season; Crop rotation has to be followed and Removal and burning of the infected plant residues;

- Seed treatment with mancozeb 63% + carbendazim 12% @ 2g per kg of seed and foliar application of propiconazole 25% EC @ 0.1% for two times at 30 and 45 days after sowing.

Powdery mildew : *Erysiphe cichoracearum*

- White powdery growth on upper surface later turn grayish forming cleistothecia
- Flowers and young capsules affected and premature shedding

Pathogen Character: The fungus produces ectophytic, branched, septate mycelium. During asexual reproduction, it produces hyaline, cylindrical, thin walled, single celled conidia in chain on a short club shaped conidiophores



Favourable conditions/Epidemiology: Moderate temperatures (60° to 80°F) and shady conditions generally are the most favorable for powdery mildew development.

Mode of spread and survival: Cleistothecium developed on left over crop in isolated areas serve as primary inoculum. Wild weed plant harbour the conidial stage of the fungus and release conidia for primary infection to ensuing crop. Conidia are spread by wind.

Management

- Avoiding high humidity area and having full sunlight most of the day and providing morning irrigation and maintaining optimum population; following crop rotation.
- Foliar application of wettable sulphur 80 WP @ 3g or difenconazole @ 0.05% for two times.

Phyllody : Phytoplasma disease

Vector Jassid – *Orosius albicinctus*

- Floral parts transformed into green leafy structure – Phylloid structure
- Small leaves and malformed flowers cluster at the top. Flower sterile
- Stamens leaf like, anthers green and ovary transformed into shoot like structure
- Plants with reduced internodes – gives bushy appearance



Pathogen: This are Mollicutes bound by a triple-layered membrane, pleiomorphic and is less than 1 µm in diameter. Sensitive to tetracycline antibiotic.

Management

- Intercrop sesame with pigeon pea (6:1)
- Remove and burn infected plants

- Seed treatment with imidacloprid protects the crop from all sucking pests including leaf hoppers for about a month
- Spray dimethoate 30 EC @ 500 ml/ ha at 30, 40 and 60 days after sowing to control the leaf hopper

Disease : Wilt

Pathogen: *Fusarium oxysporum f.sp sesami*

Symptoms: Symptoms are visible initially as drooping of the leaves followed by wilting of stems and complete drying and sudden death of the plants. Anatomically dark brown or black discolouration will be observed in the vascular tissue. Roots infected with this pathogen harboured whitish mycelia.

Favourable conditions/Epidemiology: Around 30°C favours the disease development. High soil moisture due to frequent irrigation increases infection



Pathogen: Micro conidia are oval to cylindrical, hyaline, single celled, normally arise on short conidiophores. Macro conidia which borne on branched conidiophores, are thin walled, 3 to 5septate, fusoid and pointed at both ends. Chlamydospores act as resting spore are rough walled or smooth, terminal or intercalary, may be formed singly or in chains

Management

- Seed treatment with carbendazim 2g/kg of seeds and soil drenching with 0.1% carbendazim
- Crop rotation with other crops can be followed

CASTOR (*Ricinus communis L.*)

List of Diseases:

1. Seedling blight : *Phytophthora parasitica*
2. Wilt : *Fusarium oxysporum*
3. Rust : *Melampsora ricini*
4. Powdery Mildew : *Leveillula taurica*.
5. Leaf blight : *Alternaria ricini*
6. Brown / Cercospora leaf spot : *Cercospora ricinella*
7. Charcoal Rot / Root rot / dieback : *Macrophomina phaseolina*
8. Grey Mould : *Botrytis ricini*
9. Bacterial Leaf Spot : *Xanthomonas campestris pv. ricini*
10. Bacterial wilt : *Pseudomonas solanacearum*

1. Seedling blight:

Introduction:

Castor (*Ricinus communis L.*) is one of the important non edible oilseed crops and considered as the ancient nonedible oilseed crop. It is indigenous to eastern Africa and most probably originated in Ethiopia . This crop iswidely distributed throughout the tropics and sub-tropics and is well adapted to the temperate regions of the world.

- The disease seedling blight is known to exist in India since 1909 when it was reported from Pusa, Bihar.
- It generally appears during rainy season, i.e., about the end of June and continues up to September.
- It destroys nearly 30-40 per cent seedlings particularly those which are 6-8 inches high.
- The disease has also been reported from Uttar Pradesh in1948 and from Hyderabad in 1947

Causal organism:

Phytophthoraparasitica

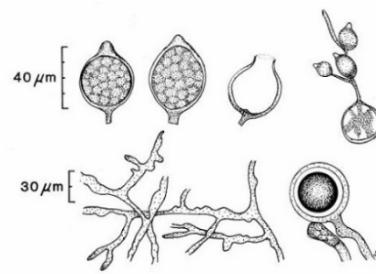
Symptoms:

- The disease appears circular, dull green patch on both the surface of the cotyledonary leaves.
- It later spreads and causes rotting.
- The infection moves to stem causing withering and death of seedling.
- In mature plants, circular, dull green patch appears on the young leaves
- Spreads to petiole and stem causing black discoloration
- In advanced stage severe defoliation occurs.
-



Pathogen Character:

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.



Favourable conditions/Epidemiology:

Continuous rainy weather, low temperature (20-25°C) low lying and ill drained soils favours the disease.

Mode of spread and survival:

- The fungus remains in the soil as chlamydospores and oospores, which act as primary source of infection. The fungus also survives on other hosts like potato, tomato, brinjal, sesame etc.
- The secondary spread takes place through wind borne sporangia.

Management:

- Remove and destroy infected plant residues.
- Avoid low lying and ill drained fields for sowing.
- Treat the seeds with Thiram or Captan at 4g/Kg.

2. Wilt: *Fusarium oxysporum*

Introduction:

Castor wilt (*Fusarium oxysporum* f. sp. *ricini*) is the most important disease of castor at present in India

Causal organism: *Fusarium oxysporum*

Symptoms:

- When seedlings are attacked cotyledonary leaves turn to dull green colour, wither and die subsequently.
- Leaves are droop and drop off leaving behind only top leaves.
- Diseased plants are sickly in appearance.
- Wilting of plants, root degeneration, collar rot, drooping of leaves and necrosis of affected tissue and finally leading to death of plants.
- Necrosis of leaves starts from margins spreading to interveinal areas and finally to the whole leaf.



Wilt

- Spilt open stem shows brownish discolouration and white cottony growth of mycelia much prominently in the pith of the stem.

Pathogen Character:

- Macro and microconidia are produced.
- Microconidia are hyaline, single celled and oval.
- Macroconidia are slightly sickle shaped, and two to five celled.

Management:

Cultural

- Selection of disease free seed.
- Grow tolerant and resistant varieties like Jyothi, Jwala, GCH-4, DCH-30 and SHB 145.
- Avoid water logging
- Burning of crop debris
- Green manuring and intercropping with Red gram

Chemical

- Treat the seed with Thiram @ 3g/ kg or carbendiazim @ 2g/ kg seed.
- Seed treatment with 4g *Trichoderma viride* formulation.
- Multiplication of 2kg *T.viride* formulation by mixing in 50kg F.Y.M, Sprinkling water and covering with polythene sheet for 15 days and then applying between rows of the crops is helpful in reducing the incidence.

3. Rust:

Introduction:

- The disease occurs in Bombay, Deccan districts, Coimbatore and Nagpur.
- It usually appears in Bombay between November and February on castor sown in June.
- The damage caused by this disease was very severe in moist localities and at places where the disease appeared quite early.
- In Hyderabad the disease appears only in December when the capsule formation has already started so that little damage is done to the crop.

Causal organism: *Melampsora ricini*

Symptoms:

- Minute, orange yellow coloured raised pustules appears
- 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, leads to drying of leaves.



Pathogen Character:

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.

Mode of spread and survival:

The fungus survives on the self sown castor crops in the off season.

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 at 1kg/ha or dust sulphur at 25 kg/ha or wettable sulphur at 1kg/ha

4. Powdery Mildew:

Introduction:

The disease in India is reported to be prevalent during November to March at Coimbatore.

Causal organism: *Leveillula taurica*.

Symptoms:

- It is characterised by typical mildew growth which is generally confined to the under-surface of the leaf.
- When the infection is severe the upper-surface is also covered by the whitish growth of the fungus.
- Light green patches, corresponding to the diseased areas on the under surface, are visible on the upper side especially when the leaves are held against light.

Pathogen Character:

Mycelium - Intercellular

Haustoria - Absorbs nutrition

Conidiophores - Which arise through stomata, are hyaline, long, non septate, slender and rarely branched and bear single conidium at the tip.

Conidia - Hyaline, single celled and elliptical or clavate.

Cleistothecia - black, globose with simple myceloid appendages.

They contain 9-20 cylindrical ascii. Each ascus contains 3-5 ascospores which are also hyaline and unicellular.

Favourable conditions/Epidemiology:

Warm sultry weather.

Mode of spread and survival:

Air borne conidia and spores in the debris.

Management:

- When weather is comparatively dry spray twice Wettable Sulphur 3g/lit at 15 days interval , starting from 3 months after sowing.
- Spray 1ml Hexaconazole or 2ml Dinocap /litre of water at fortnight intervals. The variety jwala is resistant to this disease.

5. Leaf blight:

Introduction:

- The disease has been reported from different parts of our country from time to time and is assuming serious proportions.
- In some other countries also, Alternaria leaf spot is considered to be one of the serious diseases of castor.

Causal organism: *Alternaria ricini*

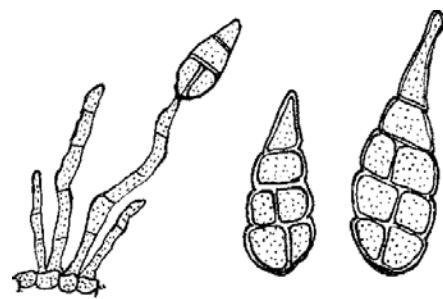
Symptoms:

- Irregular brown spots with concentric rings form on the leaves.
- When the spots coalesce to form big patches premature defoliation occurs.
- The stem, inflorescence and capsules are also show dark brown lesions with concentric rings.
- Brown sunken spots initially appear, enlarge rapidly and cover the whole capsules.
- The capsules crack and seeds also get infected.



Pathogen Character:

The pathogen produces erect or slightly curved, light grey to brown conidiophores, which are occasionally in groups. Conidia are produced in long chains. Conidia are obclavate, light olive in colour with 5-16 cells having transverse and longitudinal septa with a beak at the tip.



Favourable conditions/Epidemiology:

High atmospheric humidity (85-90 %) and low temperature (16-20 C)

Mode of spread and survival:

The fungus also survives on hosts like Jatropha pandurifolia and Bridelia hamiltoniana. The pathogen is extremely and internally is seed borne and causes primary infection. The secondary infection is through air-borne conidia.

Management:

- Treat the seeds with Captan or Thiram at 4g/kg.
- Remove the reservoir hosts periodically.

- Spray Mancozeb at 1kg/ha.

6. Brown/ *Cercospora* leaf spot:

Introduction:

The disease has been reported from Bihar, Uttar Pradesh and Hyderabad and is probably present in many other parts of the country.

Causal organism:

Cercosporaricinella

Symptoms:

- Minute specks surrounded by a pale green halo.
- These spots are visible on both the surfaces of the leaf.
- The spots enlarge to grayish white centre portion with deep brown margin.
- Spots enlarge coalesce to form brown patches but restricted by veins.
- Infected tissues often drop off leaving shot hole symptoms.
- In severe infections the older leaves may be blighted and withered.



Pathogen Character:

The fungal hyphae collect beneath the epidermis and form a hymenial layer. Clusters of conidiophores emerge through stomata or epidermis. They are septate and unbranched with deep brown base and light brown tip. The conidia are elongated, colourless, straight or slightly curved, truncate at the base and narrow at the tip with 2-7 septa.

Mode of spread and survival:

- The fungus remains as dormant mycelium in the plant debris.
- The fungus mainly spreads through wind borne conidia.

Management:

- Remove the infected plant debris,
- Spray Mancozeb at 1Kg/ha.
- Treat the seed with thiram or captan 3gm/kg seed.

7. Charcoal Rot / Root Rot / Die Back:

Introduction:

Charcoal rot caused by *Macrophomina phaseolina* is one of the most important diseases of castor. The fungus causing charcoal rot infects more than 500 plant species, including some of the world's most important crops such as soybean, cotton and corn. Root-rot and charcoal rot of castor is considered to be a major devastating disease in India also.

Causal organism: *Macrophomina phaseolina*

Symptoms:

- Small brown depressed lesions on and around nodes.
- Increase in size on both directions causing 2 to 20 cm necrotic area
- Lesions often coalesce and girdle the stem causing leaf drop.
- Entire branch and top of the plant withers.
- Drying and death starts from apex and progress.
- Infected capsules discoloured and drop easily.
- Sudden wilting of plants in patches under high moisture stress coupled with high soil temperature.
- Plant exhibit symptoms of drought and drooping of leaves.
- At ground level black lesions are formed on the stem.
- Young leaves curl inwards with black margins and drop off later, such branches Die-back.

Pathogen Character:

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

Management:

Cultural

- Grow tolerant and resistant varieties like Jyothi, Jwala, GCH-4, DCH-30 and SHB-145.
- Avoid water logging.
- Destruction of crop debris.
- Selection of healthy seed.
- Providing irrigation at critical stages of the crop.

Chemical

- Treat the seed with Thiram @ 3g / Kg or carbendazim @ 2g/ kg seed.
- Seed treatment with *Trichoderma viride* formulation at 4g/kg of seed .
- Soil drenching with carbandazim (1g/1 litre of water) 2-3 times at 15 days interval.

8. Grey Mould/Rot:

Causal organism:

Teleomorph: *Botrytis ricini*

Anamorph: *Amphobotrys ricini*

Symptoms:

- Initially symptoms are small blackish spots on inflorescence from which drops of yellow liquid may exude.
- Fungal threads which grow from there spots, spread the infection and produce characteristic appearance of affected receme.
- The disease is problematic when rains occur during capsule formation and during prolonged we weather.
- Total plant parts like leaves, stem, flower and capsules weathered.
- The effected flowers are rot and are covered by gray coloured fungus.
- The disease spreads upwards infecting all flowers and capsules.
- Blue spots are appear on the branches and laterals of the spike.
- Affected parts are break off.
- Infection at the time of flowering results in flower rot and affects seed filling.
- Infected capsules are rotted and shed off.



Pathogen Character:

Sclerotia produced and germinate in moist weather and produce conidia

Favourable conditions/Epidemiology:

A succession of several (5 days) continuously wet days with high relative humidity (>90%) and cool temperature (25-28°C) during flowering and capsule formation is essential for disease development.

Mode of spread and survival:

The fungus survives in soil through mycelia/sclerotia in crop debris. It can survive in infected crop debris on the soil surface for 6 months. Spores formed on the infected spikes get readily disseminated by wind and rain, which facilitate the secondary spread of infection. The pathogen also survives in seeds especially in the caruncle and beneath the seed coat.

Management:

Cultural

- Use of non spiny varieties (48-1)
- Avoid excess irrigation
- Avoid close spacing
- Destruction of crop debris.

- Selection of variety with Non-spiny capsules and less compact inflorescence like JWALA.

Chemical

- Spray Carbendazim (0.05%) or Thiophanate methyl (0.05%) at 15 days interval.
- Seed treatment with carbendazim at 3g per kg and spraying with carbandazim at 1g/lit depending on weather forecast atleast 6-8 hours before rain.
- Application of 20kg area and 10kg of Muriate of Potash after removal of diseased panicles.

9. Bacterial Leaf Spot:

Introduction:

The disease has been reported from Bombay.

Causal organism:

Xanthomonas campestris pv. ricini

Symptoms:

- The pathogen attacks cotyledons, leaves and veins and produces few to numerous small, round, water-soaked spots which later become angular and dark brown to jet black in colour.
- The spots are generally aggregated towards the tip. At a later stage the spots become irregular in shape particularly when they coalesce and areas around such spots turn pale-brown and brittle.
- Bacterial ooze is observed on both the sides of the leaf which is in the form of small shining beads or fine scales.



Pathogen Character:

- It is an aerobic, Gram-negative rod having single polar flagellum
- The optimum temperature for the growth of the bacterium and its thermal death point are 31°C and 51°C respectively.

Management:

Cultural

1. Field sanitation help in minimizing the yield loss as pathogen survives on seed and plant debris.
2. Hot water treatment of seed at 58°C to 60°C for ten minutes.
3. Grow tolerant varieties.

Chemical

- Spray Copper oxy Chloride(0.3%) or (500 PPM) Streptocycline 1g in 10 litres of water or Paushamycin (0.025%).

10. Bacterial wilt:

Causal organism: *Pseudomonas solanacearum*

Symptoms:

Yellowing of plants, stunted, downward curling of leaflets

Xylem blackening

Pathogen Character:

It is an aerobic non-spore-forming, Gram-negative, soil-borne and motile with a polar flagellum

Mode of spread and survival:

It can spread through contaminated soil and debris and also through weed

Management:

- Field sanitation help in minimizing the yield loss as pathogen survives on seed and plant debris.
- Hot water treatment of seed at 58°C to 60°C for ten minutes.
- Grow tolerant varieties.
- Spray Copper oxychloride 2kg/ha or Streptomycin 100g/ha or Paushamycin 250g/ha.

Reference:

1. <https://vikaspedia.in/agriculture/crop-production/integrated-pest-management/ipm-for-oilseeds/ipm-strategies-for-castor/diseases-and-symptoms>
2. <https://ikisan.com/ap-castor-disease-management.html>
3. <http://www.icar-iior.org.in/ckmp/page.php?p=botryotinia-gray-mold>

6. Etiology, symptoms, mode of spread, survival, epidemiology and integrated management of Diseases of tobacco, jute and mulberry

Diseases of Tobacco

1. Disease Name: Damping Off

Causal Organism: *Pythium spp*

Symptoms:

- In the pre-emergence 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.



Pre-emergence and post-emergence damping off

Pathogen Character:

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, light to deep yellow or yellowish brown coloured, measuring 17 -19 µm in diameter.

Favourable conditions/Epidemiology:

Overcrowding of seedling, ill drained nursery beds, heavy shade in nursery, high atmospheric humidity (90-100 percent), high soil moisture, low temperature (below 24 °C) and low soil temperature of about 20 °C

Mode of spread and Survival:

The fungus survives in the soil as Oospores and Chlamydospores. The primary infection is from the soil borne fungal spores and secondary spread through sporangia and zoospores transmitted by wind and Irrigation water.

Management:

- Prepare raised bed nursery with adequate drainage facility
- Burn the seed beds with paddy husk before sowing
- Drench the seed bed with 1 per cent Bordeaux mixture or 0.2 per cent Copper oxychloride, two days before sowing
- Avoid overcrowding of seedlings

- Avoid excess watering of the seedlings
- Spray the nursery beds two weeks after sowing with 1 per cent Mancozeb and repeat subsequently at 4 days interval under dry weather and 2 days interval under wet cloudy weather or spray 0.2 per cent Metalaxyl compound at 10 days interval commencing from 20 days after germination

2. Disease Name: Tobacco Black shank

Causal Organism: *Phytophthora parasitica* var *nicotinane*

Symptoms:

- The most common symptom of the disease is a root and crown rot
- The root system will be black and rotting.
- The lower portion of the pith of the stem will be blackened and classically the pith area will be divided into disks of tissue.
- Black shank is more severe in wetter or poorly drained portions of the field
- Due to root rot, the plants show temporary wilt that progresses into chlorosis of leaves and permanent



Blackening of root

Wilt



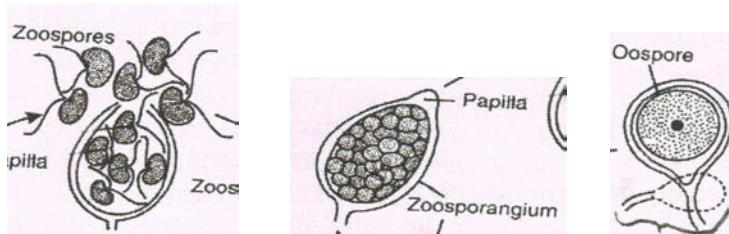
→ Necrotic spots also occur on leaf



→ Hyphae often are readily observed in the necrotic pith tissues upon splitting of the stem

Pathogen Character:

Phytophthora sp. produces pear/lemon shaped sporangium which is either papillate/non papillate. Sporangia germinate by producing zoospores. Zoospores, asexual spores, are produced inside the sporangium. They are reniform/kidney shaped, hyaline, biflagellate, laterally positioned; heterokont-i.e. zoospores have tinsel and whiplash flagella. Tinsel flagellum is directed anteriorly and whiplash flagellum is directly posteriorly. *Phytophthora* sp. produces Oogonium and antheridium (amphigynous). Sexual spore is Oospore



Favourable conditions/Epidemiology:

- High moisture and temp between 16 and 30°C
- Heavy soil and incidence of root knot nematode, *Meloidogyne incognita* and *M. javanica*

Mode of spread and Survival:

- Soil-borne and infected plant debris
- Secondary spread by wind-borne sporangia, rain and irrigation water

Management:

- Burn seed bed with paddy husk or groundnut shell @ 15-20 cm thick layer
- Provision of adequate drainage
- Drench 1.0 % Bordeaux mixture or 0.2 % copper oxychloride
- Spray the bed after two weeks with 0.2 % metalaxyl or 0.2 % copper oxychloride or 1.0 % Bordeaux mixture

3. Disease Name: Frog eye spot

Causal Organism: *Cercospora nicotianae*

Symptoms:

- Several small, round brown lesions with 2-10 mm diameter on lower and mature leaves occur.
- Typical lesion with white parchment centre surrounded by brown or tan colored margin resembling eye of frog.
- Different spots coalesce causing drying of leaves which wither prematurely.



Pathogen Character:

The mycelium is intercellular and collects beneath the epidermis and clusters of conidiophores 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.

Favourable conditions/Epidemiology:

Temperature of 20- 30 ° C, high humidity (80-90 per cent), closer spacing, frequent irrigation and excess application of nitrogenous fertilizers.

Mode of spread and survival

The fungus is seed borne. The fungus also persists on crop residues in the soil. The primary infection is from the seed and soil borne inoculum. The secondary spread is through wind borne conidia.

Management

- Remove and burn plant debris in the soil
- Avoid excess nitrogenous fertilization
- Adopt optimum spacing
- Regulate irrigation frequency
- Spray with 0.4 per cent Bordeaux mixture or Thiophenate Methyl 750 g/ha and repeat after 15 days

4. Powdery mildew - *Erysiphe cichoracearum* var. *nicotianae*

Symptoms

Initially the disease appears as small, white isolated patches on the upper surface of the leaves. Later, it spreads fast and covers the entire lamina. The disease initially appears on the lower leaves and as disease advances, the rest of the leaves are also infected and sometimes powdery growth can be seen on the stem also. The affected leaves turn to brown and wither and show scorched appearance. The severe infection leads to defoliation and reduction in quantity and quality of the curable leaves.

Pathogen

The fungus is ecotrophic and produces hyaline, septate and highly branched mycelium. Short, stout and hyaline conidiophores arise from the mycelium and bear conidia in chains. The conidia are barrel shaped or cylindrical, hyaline and thin walled. Cleistothecia are black, spherical with no ostiole, with numerous densely-woven septate, brown-coloured appendages. They contain 10-15 asci which are ovate

with a short stalk. Each ascus contains two ascospores which are oval to elliptical, thinwalled, hyaline and single celled.



**Conidia and
conidiophores**

Favourable Conditions

- Humid cloudy weather.
- Low temperature (16-23°C).
- Close planting and excess doses of nitrogenous fertilizers.

Mode of spread and survival

- The fungus remains dormant as mycelium and cleistothecia in the infected plant debris in soil. The primary infection is mainly from soil-borne inoculum. The secondary spread is aided by windblown conidia.

Management

- Apply balanced fertilizers.
- Avoid overcrowding of plants.
- Remove and destroy the affected leaves.
- Plant early in the season so that crop escapes the cool temperature at maturity phase.
- Spray dinocap at 375 ml or Carbendazim at 500g/ha.

5. Disease Name: Anthracnose

Causal Organism: *Colletotrichum tabacum*

Symptoms:

- Symptom appears as small water-soaked spots with sunken center on leaves.
- Spots become white with brown margin.
- Lesions occur also on midribs, petioles and lateral veins causing distortion and ragged.
- Lesions on stem weaken the stem.

**Pathogen Character:**

The fungus produces thin, hyaline, septate, profusely branched hyphae containing oil droplets. The fungus produces black, minute velvety acervuli. Conidiophores are closely packed inside the acervulus which contains conidia.

Favorable conditions/Epidemiology:

Rain and high humidity are favourable for the development of disease.

Mode of spread and Survival:

The primary infection by sowing infected seeds and secondary by wind.

Management:

- Spraying with Mancozeb 1 kg/ha and repeat after 15 days

6. **Disease Name:** Brown spot

Causal Organism: *Alternaria longipes*

Symptoms:

- Initially it appears on lower and older leaves as small brown, concentric circular lesions, which spread, to upper leaves, petioles, stalks and capsules even.
- In warm weather under high humidity, the leaf spots enlarge, 1-3 cm in diameter, centers are necroses and turn brown with characteristic marking giving target board appearance with a definite outline.
- In severe infection spots enlarge, coalesce and damage large areas making leaf dark-brown, ragged and worthless

**Pathogen Character:**

- Sexual spore is Ascospore and Asexual spore is conidia

Favorable conditions/Epidemiology:

- Warm weather with high humidity.

Mode of spread and Survival:

- The pathogen survives through spores (conidia) or mycelium in diseased plant debris or weed.

Management:

- Spray with Mancozeb 1 kg/ha

7. Wild fire - *Pseudomonas tabaci*

Symptoms

The leaf spots may occur at any stage of plant growth including the nursery seedlings. Dark brown to black spots with a yellow halo spreads quickly causing withering and drying of leaves. In advanced cases, lesions develop on the young stem tissues leading to withering and drying of the seedlings. In the fields, initially numerous water soaked black spots appear and later become angular when restricted by the veins and veinlets.



Symptoms

- Several spots may coalesce to cause necrotic patches on the leaves. In advanced conditions, the entire leaf is fully covered with enlarged spots with yellow haloes. The leaves slowly wither and dry. Under humid weather condition, the disease spreads very fast and covers all the leaves and the entire plant gives a blighted appearance.

Pathogen

- The bacterium is a rod, motile with a single polar flagellum, non-capsulated, non-spore forming and Gram negative.

Favourable Conditions

- Close planting.
- Humid wet weather.
- Strong winds.

Mode of spread and survival

- The bacterium survives in the infected crop residues in the soil, which is the primary source of infection. The secondary spread of the pathogen in the field is through wind splashed rain water and implements.

Management

- Remove and burn the infected crop residues in the soil.
- Avoid very close planting.

8. Disease Name: Tobacco Mosaic Disease

Causal Organism: **Tobacco Mosaic Virus, *Nicotiana virus* or *Marmor tabaci***

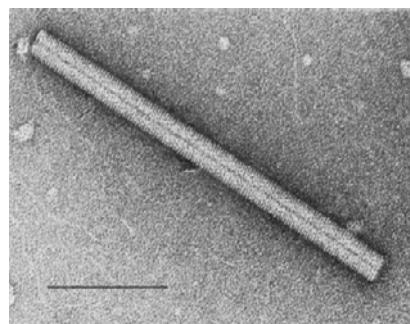
Symptoms:

- Symptoms include a general mottling of the leaves with irregular light and dark green splotches over the leaf surface.
- Expanding bud leaves may be distorted, narrow and crinkled in appearance.
- Dark green blisters and enations (leafy growth) appear on lower leaf surface
- Sometime necrotic brown spots or scorched patches appear on leaves resulting in mosaic scorch or mosaic burn under hot sunny dry periods.
- Growth is retarded. Leaves are narrowed, puckered, distorted, thin and malformed. It lowers the market value of leaves
- Partial sterility of infected plants



Pathogen Character:

- Virus is hollow, rod shaped particle, ss RNA
- Mode of spread: Sap transmissible; enters through wounds
- Mechanical means by wind, water, farm workers in the field
- Cultural practices like topping or clipping
- Not transmitted by insects



Favorable conditions/Epidemiology:

- TMV is highly contagious and transmitted by sap. It is easily transmitted by mere contact of a diseased plant with a healthy one.
- Air-dried tobacco is a common source of new infection. Workers who chew or smoke natural leaf tobacco during nursery operations may spread the virus into the seedlings.
- Old stems and leaf trash of affected plants buried in the soil are the other sources of infection and spread.
- In the nurseries, seedlings may get affected due to the presence of susceptible weed hosts.

Mode of spread and Survival:

- The virus remains viable in the plant debris in the soil. The virus has a wide host range, affecting nearly 50 plant species belonging to nine different families. 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. The implements used in the field also transmit the virus.

Management:

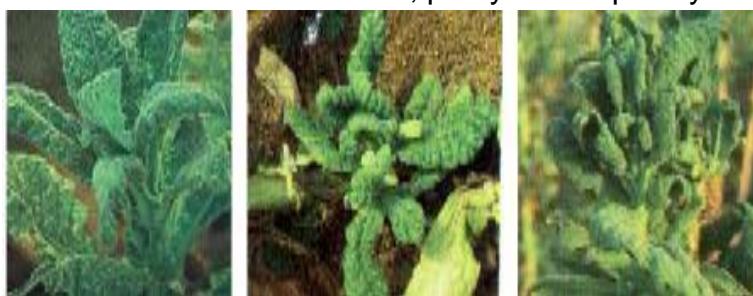
- Roguing
- Free from weeds
- Wash hands with soap water before and after field operation
- Crop rotation
- Use resistant varieties like TMV RR2, TMV RR2a, TMV RR3
- Spray *Bougainvillea* / *Basella alba* leaf extract at 1 litre in 150 litres of water, two to three times at weekly intervals.

9. Disease Name: Tobacco Leaf Curl Disease

Causal Organism: Tobacco Leaf Curl Virus, Nicotiana Virus 10 (*Ruga tabaci*)

Symptoms:

- Disease is characterized by downward curling & rolling of leaves; thickening; dark green in colour with vein clearing effect; brittle; enation (cup like or frill like outgrowth), reduction in size.
- Infected plants become stunted due to shortening of internodes and formation of more lateral branches.
- Flowers are deformed; partly or completely sterile.



Pathogen Character:

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

Favorable conditions/Epidemiology:

- White flies become more active in dry periods after monsoon showers. Leaf-curl is therefore, noticed more during this period

Mode of spread and Survival:

- The virus has wide host range of 63 crops species belonging to fourteen families. 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 Methyl demeton 0.1 to 0.2 per cent control the vectors
-

10. Disease Name: Broom rape

Causal Organism: *Orobanche cernua* var. *desertorum* and *O. indica* (Phanerogamic parasite - Total root parasite)

Symptoms:

- Young parasitic plants emerge from soil at the base of tobacco plants
- Plants attacked early show general stunting and wilting
- Plants attacked late in season do not show visible symptoms but yield and quality of leaves are reduced.
- Mode of spread: Seeds in soil and sec. spread through irrigation water, animals, human beings and implements



- Other crops: Brinjal, Tomato, Cauliflower, Turnip and other cruciferous plants

Pathogen Character:

- Very frequently 10-15 Orobanche shoots found attached to the roots of a single plant emerges in clusters, pale brown or purple, 15-45 cm tall.
- Stem solitary round and thickened at the base

- Flowers are long and curved; Fruit is a capsule contain many oval brown seeds

Favorable conditions/Epidemiology:

- Optimum temperature for germination is 15° - 20°C for at least 18 days.
- It can get fail to parasitize if the soil temperature is too high.
- Seed germinates in presence of roots of host plant (Root exudates contain growth regulators like IAA, GA₃ and kinetin stimulates germination).

Mode of spread and Survival:

- 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 are stimulated to germinate by root exudates of tobacco and attaches itself to the roots by forming haustoria. Later, it grows rapidly to produce shoots and flowers. Orobanche also attacks the crops like brinjal, tomato, cauliflower, turnip and other cruciferous crops.

Management

- Sowing clean seeds of tobacco
- Rouging (by regular weeding)
- Spray soil with 25 % Copper sulphate
- Spray drenching the emerged shoot with 0.1 % allyl alcohol (tender shoot stage) 0.2 % at later stages
- Apply few (3-4) drops of kerosene directly on shoot
- Grow trap crops like chillies, moth bean, and sorghum, cowpea in rotation to stimulate seed germination and kill the parasite
- Application of (2-3 drops of) sunflower, linseed, castor, safflower, neem oil on young shoots, which will kill the parasite

<https://vikaspedia.in/>

Diseases of Jute

1. Disease Name : Root & Stem Rot

Causal organism: *Macrophomina phaseolina*

Symptoms

Occurs at all stages of crop growth

- On young seedlings:

- ✓ Dark, thin streaks on the collar region & also on cotyledon.
- ✓ During high humid condition, lesions enlarge & spread killing the seedling.
- ✓ Spread of the disease is so rapid, often called as damping off.
- ✓
- On fairly grown up plants:
- ✓ Buff to black coloured lesion on the leaves along the margin and apex, on midribs and petioles.
- ✓ As the disease advances, the fungus attacks the stem at nodal region, causing small dark brown to black lesions, enlarges to girdle the stem.
 - ✓ Lesion spread along the stem causing bark shredding. Affected plants shows wilting and premature- defoliation.
 - ✓ Disease spreads from basal stem to root, killing the plant.
 - ✓ Pycnidia formed on the infected root & stem.
 - ✓ On inflorescence:
 - ✓ Capsules are discoloured black, seeds discoloured & small. Sclerotia seen on the infected capsules.
 - ✓ Disease is disseminated by seed, soil and air.
 - ✓ Deshi and Tossa jute are infected by this disease.



Pathogen Characters

- ✓ Sclerotial stage: *Rhizoctonia bataticola*

Favorable condition

- ✓ Alluvial and lateritic soils with low pH (5.6-6.5), high level of nitrogen, high rainfall and high humidity favour infection of *M. phaseolina*. Higher soil temperature and low soil moisture predispose the older plants.

Mode of spread and survival

- ✓ Wide host range: Potato, cotton, legumes, tobacco, sesamum, mulberry, egg plant. Survives all the year round.
- ✓ Deficiency of potassium in the soil has been found to increase the incidence of stem rot.

Management Practices

- ✓ Field sanitation and balanced fertilizer application.
- ✓ Burn the crop debris.
- ✓ Spray Dithane M-45 @ 18.56g/10 litres water.
- ✓ Spray Dithane M-45, Manner M-45 @ 2g/1litre H₂O 2-3 times at the plant base soil.

2. Disease Name: Powdery Mildew

Pathogen Name: *Oidium* sp

Symptoms

- ✓ Powdery white to ash coloured growth on the leaves, later turn brown and wither
- ✓ Diseased plants are usually weak and quality of fibre is poor
- ✓ Foggy weather is favourable for the growth

Pathogen Character

- ✓ 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

Favourable Conditions

- ✓ Warm humid weather.
- ✓ The disease is severe generally during late kharif and rabi seasons.

Mode of spread and survival

- ✓ The Pathogen 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.

Management

- ✓ Remove and destroy infected plant debris.

- ✓ Spray Carbendazim 500g or Wettable sulphur 2kg or Tridemorph 500 ml/ha at the initiation of disease and repeat 15 days later.

3. Disease Name: Anthracnose

Pathogen Name: *Colletotrichum corchorum*

Symptoms

- ✓ Yellowish brown water-soaked lesions, depressed spots, on the stem which soon develops into characteristic irregular spots.
- ✓ Spot turn dark brown and finally black.
- ✓ Several spots coalesce- forms large patches, girdling of stem.
- ✓ Depending upon the depth of infection, the plant may wilt immediately or survive to produce pods.
- ✓ Necrotic lesion are produced on pods.
- ✓ The fungus invades the vascular bundles, weakening the bast fibre bundles.
- ✓ Rapid spread and severe damage is during hot humid months of july- aug.
- ✓ With high humidity, acervuli are produced on the spots their characteristic bristles can be seen under hand lens.



Favorable condition

- ✓ Continuous rain, high relative humidity and temperature of around 35°C

Mode of spread and survival

- ✓ The pathogen survives in the soil, infected crop debris and in seeds

Management

- ✓ Seed treatment with Carbendazim 50 WP @ 2 g/ kg or Captan @ 5 g/kg and spraying of Carbendazim 50 WP @ 2 g/l or Captan @ 5 g/l or Mancozeb @ 5 g/l control the disease.
- ✓ Seeds having 15% or more infection should not be used even after treatment. Removal of affected plants and clean cultivation reduce the disease.

4. Disease Name: Stem Gall

Causal Organism: *Physoderma corchori*

Symptoms:

- ✓ Symptoms appear first when the plant is about 8 – 10 inches high, producing small greenish gall on the lower portion of the stem, above the ground level.
- ✓ Galls gradually increase in size, turn dark brown crack at maturity.
- ✓ Sometimes several galls coalesce to form a large erupted lesion.
- ✓ Galls contain resting sporangia.



Pathogen Character

- ✓ Pathogen produces rhizomycelium and intercalary swellings.
- ✓ Globose resting sporangia (smooth, dark brown exospores and thin, hyaline endospore).
- ✓ Infection sites remain restricted and the rhizomycelium penetrates to the xylem but not beyond.
- ✓ Fibre strands from infected plants are shattered at the base and discontinuous above that point.

Management

- ✓ Avoiding submerged conditions by arranging proper drainage system
- ✓ Growing resistant varieties like capsularies type

5. **Disease Name:** Die-back (Black band disease)

Causal organism: *Diplodia corchori*

Symptom:

- ✓ Discolouration of the tips of main shoots that proceeds gradual darkening and withering of branches, which ultimately resemble blackened stocks. Innumerable, erumpent pycnidia, which extrude masses of spores are produced.



Control measures:

- ✓ Two times spray of Dithane M-45 @ 18.56g/10 litres water at the interval of 2-3 days.
- ✓ Crop rotation with deshi jute instead of tossa, Spraying of dithane M-45, Manner M-45 @ 2g/l litre water 2-3 times.

6. Disease Name: Wilt

Causal Organism: *Rhizoctonia solani*

Symptom:

- ✓ Root system of affected plant becomes infested with a soil borne fungi.
- ✓ All the leaves become flaccid at a time and after few days dropping occurs.
- ✓ At the flowering stage, wilting occurs severely on jute plants.
- ✓ The *Olitorius* varieties are affected by this disease more than the *Capsularis*.
- ✓ Disease disseminated by seed and soil

Control measures:

- ✓ The crop debris will be destroyed or burned.
- ✓ Crop rotaion will be maintained with *Capsularis* varieties
- ✓ Dithane M-45, Manner M-45 @ 18.56g/10 litres water.

7. Disease Name : Bacterial Leaf Spot:

Causal Organism: *Xanthomonas campestris* pv. *Nakataecorchori*

Symptom:

- ✓ Small, dark green spots that turned brown. Spots often coalesced to form large necrotic areas in leaves, which turned yellow and dropped.
- ✓ Lesions on the stem surrounded by yellow border causes girdling and death of the plant above the infected portion
- ✓ Circular, brown, Sunken lesions developed on capsules.



8. **Disease Name** : Bacterial wilt

Causal Organism: *Pseudomonas solanacearum*

Symptom:

- ✓ Affected plants were stunted and become chlorotic before the leaves dropped. The root systems of plants with symptoms were seriously deteriorated

Pathogen:

- ✓ The causal bacterium, which produced short, Gram – negative rods with single, polar flagella, also caused wilting of several solanaceous crops

Pathogen Character

- ✓ The causal bacterium, which produced short, Gram – negative rods with single, polar flagella, also caused wilting of several solanaceous crops.

Management:

- ✓ Avoid movement of infected plants or soil from around infected plants and to prevent surface water from running to other fields from fields. Jute should not be planted in rotation with susceptible, solanaceous crop plants

9. **Disease Name:** Golden mosaic

Causal organism: Jute Yellow Mosaic Virus

Symptom:

- ✓ Symptoms remained latent for a time,
- ✓ Mildly affected plants show retarded growth, but flowering and pod production were nearly normal.
- ✓ Severely affected plants were noticeably stunted and eventually killed.



Mode of spread and Survival:

- ✓ Transmitted by grafting. No insect vector has been identified, but evidence of seed transmission of the disease, as well as transmission through pollen from infected plants, has been reported.

Management

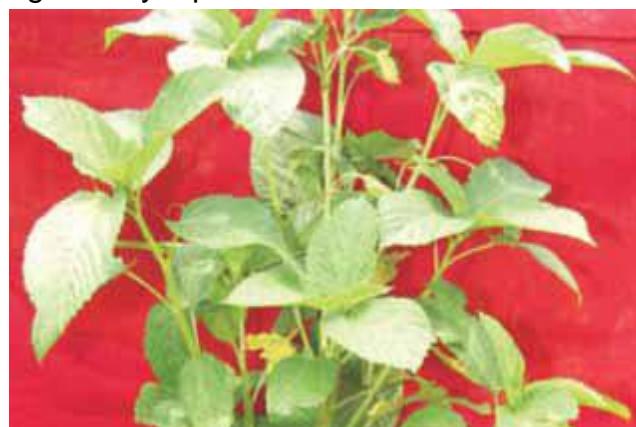
- ✓ Uprooting of infected plants
- ✓ Spraying of Heyzine/Hemithrin @ 15ml/10 litres water 2-3 times with 7 days interval

10. Disease Name: Little leaf and bunched top

Causal Organism: Phytoplasma

Symptom:

- ✓ The infected plants showed profuse lateral branching with a bushy appearance.
- ✓ Branching at apical portion developed bunched top symptom with tufts of smaller leaves.
- ✓ Infected plant showing bunched top and little leaf



Diseases of Mulberry

1. Disease name: Leaf spot

Causal organism: *Cercospora moricola*

Symptom:

- ✓ Circular light brown spots appear on both sides of the leaves.
- ✓ The adjacent spots unite together to form a larger spot
- ✓ The necrotic tissues of such spots drop out and form the characteristics - shot holes.
- ✓ Highly infected leaves defoliate prematurely



Pathogen Character:

- ✓ Conidia are hyaline, tapering at one end and 70x3 µm in size, 3-7 celled conidia

Favorable Condition:

- ✓ Temperature of 24-26 °C and 70-80 % relative humidity are most congenial for the disease development.

Mode of spread and Survival

- ✓ The disease is air borne spreading by conidia primarily through rain droplets

Management

- ✓ Spray 0.1% Bavistin (carbendazim) when disease symptoms appear 2-3 times at ten days interval.

2. Disease Name: Powdery mildew

Causal Organism: *Phyllactinia corylea*

Symptom:

- ✓ White powdery patches on the lower surface of the leaves.
- ✓ The corresponding portions on the upper surface develop chlorotic lesions.
- ✓ When severe, the white powdery patches turn to brownish-black; the leaves become yellow, coarse and lose their nutritive value.



Favourable condition

- ✓ 24 - 28° C and 75-80 %

Mode of spread and survival

- ✓ air borne conidia

Management

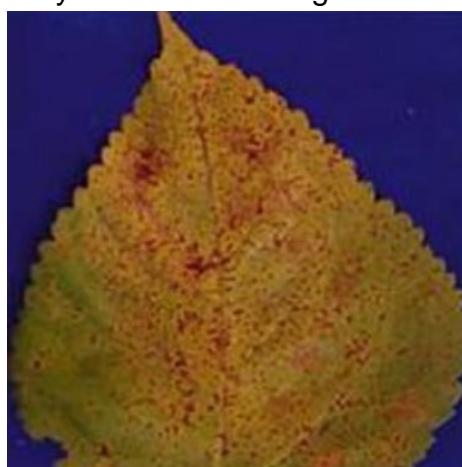
- ✓ Follow wider spacing of plantation (90 cm x 90 cm) or paired row planting system [(90 +150) x 60 cm]
- ✓ Spraying of 0.2 % Karathane (Dinocap 30% EC) / Bavistin on the lower surface of the leaves. Safe period 5 days. Or spray Sulfex (80WP) 0.2%, safe period 15 days.

3. Disease Name: Leaf Rust

Causal Organism: *Cerotelium fici*

Symptom:

- ✓ Several small pin head shaped brown postules appear on the lower surface of mature leaves
- ✓ Reddish brown spot appears on the upper surface of the infected leaves.
- ✓ Severely infected leaves turn yellowish and margin of the leaves become dry.



Favorable condition:

- ✓ Temperature 22-26°C, RH 70 %

Mode of spread and survival

- ✓ The disease is air borne dispersing by uredospores through water droplets and wind current

Management

- ✓ Follow wider spacing of plantation (90 cm x 90 cm) or paired row planting system [(90+150) x 60 cm]. Avoid delayed leaf harvest Spraying 0.2% Kavach (Chlorothalonil 75 % WP) on the leaves

4. Disease Name: Sooty mould

Causal organism: group of fungi

Symptom:

- ✓ Thick black coating develops on the upper surface of the leaves.
- ✓ The disease occurs due to the presence of white flies in the mulberry field.
- ✓ The fungi develop on the honey like substance produced by the whiteflies



Favourable condition:

- ✓ Temperature of 20-24° C and high relative humidity above 70 %

Mode of spread and survival:

- ✓ Air borne conidia

Management:

- ✓ Spray 0.2% Indofil-M45 to check growth of saprophytic fungi.
- ✓ Foliar spray of 0.02% monocrotophos on 15th and 30th day after pruning to control white fly infestation. Safe period: 15 days.

5. Disease Name: Root rot

Causal organism: *Rhizoctonia bataticola* (*Macrophomina phaseolina*), *Fusarium solani*, *F. oxysporum*, *Botryodiplodia theobromae*

Symptom:

- ✓ Above ground symptoms of root rot (yellowing/withering of leaves). Initially the above ground symptom of the disease appears sudden withering of plants and leaves fall off from the bottom of the branches and progressing upwards.
- ✓ The below ground symptoms include decaying of root cortex or skin, turn black due to fungal spores/ mycelium below the bark. The severely affected plants lose the hold in the soil and can be easily uprooted. On severity, the entire root system gets decayed and plants die. Affected plants after pruning, either fail to sprout or plant sprouted bears small and pale-yellow leaves with rough surface.



Management

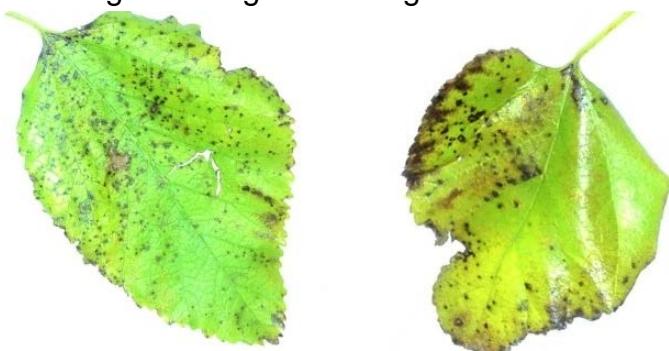
- ✓ Prune off the dried shoots above 15-30 cm from ground. Make shallow ring around stump and apply the Navinya solution made by adding 10 g of Navinya in 1 liter of water (i.e. 1 kg Navinya in 100 litre water; sufficient for 100 plants @ 1 liter/plant).
- ✓ Pour the solution over the pruned stump to drench completely. Cover with soil around the stump to prevent exposure to sunlight. Treat the surrounding mulberry plants also to prevent spreading of the disease.

6. Disease Name: Bacterial blight

Causal organism: *Pseudomonas syringae* pv. *mori* / *Xanthomonas campestris* pv. *mori*

Symptoms:

- ✓ It shows numerous blackish brown irregular water-soaked patches on the leaves resulting in curling and rotting of leaves.



Management:

- ✓ Step-up pruning (30 cm above the ground) during rainy season in high rainfall areas and spraying 0.2% Streptomycin or Dithane M45 (Mancozeb 75% WP) with safe period of 2-3 days are recommended.

7. Causal organism: Root Knot nematode (*Meloidogyne incognita*)

Symptom:

- ✓ Severely affected mulberry plants show stunted growth with low water moisture in leaves, later yellowing of leaf margins.
- ✓ Formation of knots / galls on roots is the main indicator of the disease symptom.



Nematode Character:

- ✓ Galls are spherical and vary in size; young galls are too small and yellowish-white in colour, old galls are big and pale brown

Favourable condition:

- ✓ 27-30 °C, soil moisture of less than 40 % and pH of 5 to 7

Mode of spread and survival:

- ✓ Primarily through contaminated soil, farm implements and run-off irrigation

Management

- ✓ Apply neem oil cake @ 800 kg/acre/yr in 4 split doses during intercultural operation or after pruning the plant or after leaf harvest by making the trenches of 10 –15 cm deep near the root zone of plant and cover with soil and irrigate

7. Etiology, symptoms, mode of spread, survival, epidemiology and integrated management of Diseases of banana

Fusarium wilt :

Fusarium wilt of banana, popularly known as **Panama disease**, is a lethal fungal disease caused by the soil-borne fungus *Fusarium oxysporum* f. sp. *cubense* (*Foc*). It is the first disease of bananas to have spread globally in the first half of the 20th century. Although the disease probably originated in southeast Asia, the first recording of the disease was made in 1874 in Brisbane,Australia. It was then reported from Panama in 1890.

The epidemic strated in Central America on the susceptible 'Gros Michel' banana, had a major impact on the global export trade. In the 1950s, 'Gros Michel' was replaced by Cavendish cultivars due to race 1. At the end of the 1980s, the so-called TR4 strain, to which Cavendish cultivars are susceptible, was isolated from samples from Taiwan. It has since spread through Asia and reached Africa in 2013.In India it was reported from Bihar state during 2019

Causal organism: *Fusarium oxysporum* f. sp. *cubense* (*Foc*).

Symptoms:

Fusarium wilt is a typical vascular wilt disease. Two types of symptom expression can be seen. i) Internal symptoms seen at the early stage ii) External symptoms seen at later stages of crop growth.

External symptoms: First signs of disease are usually wilting and yellowing of the older leaves around the margins. The yellow leaves may remain erect or collapse at the petiole. Sometimes, the leaves remain green, except for spots on the petiole, but still snap. The collapsed leaves hang down the pseudostem like a skirt. Eventually, all the leaves fall down and dry up. Splitting of the base of the pseudostem is another common symptom. Infected suckers do not start showing symptoms of Fusarium wilt until they are about 4 months old, a situation that has contributed to the spread of the disease through planting material.

Internal symptoms: The characteristic internal symptom of Fusarium wilt is vascular discolouration, which varies from pale yellow in the early stages to dark red or almost black in later stages. Internal symptoms first develop in the feeder roots, which are the initial infection sites. The fungus spreads to the rhizome and then the pseudostem.



Petiole breaking



Pseudostem splitting



Rhizome discolouration

Pathogen :The pathogenic isolates are classified into races based on the cultivars on which they cause disease. Race 1 infects Gros Michel,Silk and Pome group,Race 2-Monthan varieties,Race 3-Ornamental crops(*Heliconium* sp) and Race 4 –cavendish groups. Race 4 is divided into TR- 4 and STR-4. Fungus produces 3 types of spores

- Microconidia - 1 celled, hyaline
- Macroconidia – Sickle shaped, tapered, 3-4 septate hyaline
- Chlamydospores - Terminal or Intercalary

Favourable conditions/Epidemiology:

Nematodes predispose the fusarium wilt pathogen.Nematodes like *Radopholus similis*, make wounds in the root surface through which fusarium pathogen easily enters.Acidic soil,nematodes infected field will favour the disease incidence.

Mode of spread:

Primary : Infected sucker

Secondary : Water borne conidia and chlamydospores

Management:

Cultural method:

- ✓ Flooding for six months,can be done growing paddy as succeeding crop
- ✓ Application of enriched farm yard manure
- ✓ Growing sunhemp,Daincha and ploughing insitu before flowering
- ✓ Removal of infected suckers and application of calcium in the infected pit
- ✓ Burning of infected rhizomes and pseudostem
- ✓ Avoiding irrigation from infected to healthy fields

Chemical Method

- ✓ Uproot and destroy severely affected plants
- ✓ **Pairing and pralinage :** To avoid wilt disease, infected portions of the corm may be pared, dipped for 45 minutes in carbendazim 0.1% solution (1 g in 1 l of water) solution

for Rasthali, Monthan, Neyvannan, Virupakshi and other wilt susceptible varieties. Pralinage with 40 g of carbofuran 3 CG granules per sucker (dip the corm in slurry solution of 4 parts clay plus 5 parts water and sprinkle carbofuran to control nematodes).

- ✓ **Corm injection:** Remove a small portion of soil to expose the upper portion of the corm. Make an oblique hole at 45° angle to a depth of 10 cm. Immediately insert a gelatin capsule containing 60 mg of carbendazim or inject 3 ml of 2 % carbendazim solution into the hole with the help of corm injector' on 2nd, 4th and 6th months after planting.

Biological control :

- ✓ Apply press mud at 5 kg per plant to reduce the wilt incidence or apply *Pseudomonas fluorescens* (Pf1) liquid formulation @ 4 l/ha at 2nd, 4th and 6th months after planting through drip system to manage panama wilt and nematode complex.
- ✓ Application neem cake @250gms /plant, *Trichoderma viride* 25gms/plant at the time of planting, 2nd and 4th month after planting.

Yellow Sigatoka leaf spot - *Mycosphaerella musicola* (I.S: *Cercospora musae*)

Black sigatoka – *Mycosphaerella fijiensis*

- ✓ Disease was first reported in Java 1902
- ✓ Disease was named after the epidemic occurred at Sigatoka of Fiji island.
- ✓ Occurs in all most all banana growing regions of India

Symptoms

Yellow sigatoka leaf spot - *Mycosphaerella musicola*

- ✓ Disease incidence observed in older leaves and progress towards the younger one.
- ✓ Light yellow or brownish green narrow streaks along the veinlets, sometimes spindle shaped spots with grey centre.
- ✓ Central portion necrotic, surrounded by dark brown band, yellow halo. Spots coalesce and whole leaf blade dries up
- ✓ On the upper surface of the spots, fructifications of the fungus appear as black specks
- ✓ Rapid drying of leaves

Black sigatoka leaf spot - *Mycosphaerella fijiensis*

Black colour rectangular spots/ linear spots on leaf lamina without yellow halo

Rapid drying of leaves



Pathogen: Pathogen produces branched, septate hyphae, branched, septate cylindrical conidiophores on which brown colour, slender, 4 to 6 septate conidia in *M. musicola*, while brown colour, needle shaped, multi-septate conidia in *M. fijiensis*. Under perfect stage, fungus produces perithecia, asci and ascospores (1-2 celled).

Favourable condition:

High humidity, heavy dew and rainy weather with temp above 21 0C. Soils with poor drainage and low fertility favour the disease incidence. Thick planting, presence of weeds and increased number of suckers in a mat promote disease development.

Mode of spread

Primary : Ascospores from the infected plant debris

Secondary : Air borne conidia

Management:

- ✓ Remove affected leaves and burn
- ✓ Spraying carbendazim @ 1 g/l or mancozeb @ 2 g/l or copper oxychloride @ 2.5 g/l or ziram @ 2 ml/l or chlorothalonil @ 2 g/l at monthly interval from November onwards
Always add 5 ml of wetting agent like Sandovit, Triton AE, Teepol etc. per 10 l of spray fluid
- ✓ Alternatively spray propiconazole @ 1 ml/l or 0.5 ml/l along with petroleum based mineral oil @ 10 ml/l
- ✓ Spray pyraclostrobin 133 g/l + epoxiconazole e 50g/l SE @ 3 g/l or tebuconazole 50% + trifloxystrobin 25% WG @ 300 g/ha
- ✓ or *Pseudomonas fluorescens* @ 0.5% three times at 15 days interval to control sigatoka leaf spot incidence..

Anthracnose / Fruit Rot: *Gloeosporium gloeosporioides*

The most important disease problem of export bananas (Cavendish cultivars) is crown rot. This disease may involve several species, but the most commonly associated organism is *Colletotrichum musae*

In India, post-harvest losses to disease, including anthracnose, have been estimated at 30–40%. *C. musae* causes lesions on the fruit peel after ripening, as well as finger and crown rots. Anthracnose is a latent infection where fungal spores infect immature banana in the field.

Symptoms:

- ✓ Circular to irregular black sunken lesions, coalesce with pinkish or salmon coloured spore mass appear on the fruit
- ✓ Skin of the fruit turns black and rots
- ✓ Latent infection – Pathogen infects green fruits in the field without expression of symptoms and symptoms appear during ripening



Pathogen: Hyaline, septate and branched conidiophore on which cylindrical to oval single celled, tapered towards apex, thin walled conidia with large number of oil globules are produced.

Favourable condition: Atmospheric temperature, relative humidity, wounds made during harvest favours the disease development, Disease incidence is more during rainy season

Mode of spread

Primary : Air borne conidia

Secondary : Spread through the contact of infected fruits

Management:

- ✓ Foliar spray of carbendazim (0.1%) twice at 15 days interval after bunch emergence.
- ✓ Proper sanitation ,handling,proper cooling to 14°C

Cigar end rot: *Verticillium theobromae*

Symptoms: The initial infection in the perianth slowly spreads along the finger and it causes peel blemishes as black or brown sunken spots of various sizes on fruit that may bear masses of salmon-colored acervuli with their associated conidia on the fruit peel after ripening. The noted 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.



Spread: Survives in dead or decaying leaves and also on fruits. Its spores can be spread by wind, water and insects as well as by birds and rats feeding on bananas

Management:

- ✓ Removal of perianth by hand or soft brush 8-10 days after bunch formation
- ✓ Preharvest spray of copper oxychloride @ 0.25 per cent along with wetting agent.
- ✓ Proper storage and sanitation

Bacterial diseases:

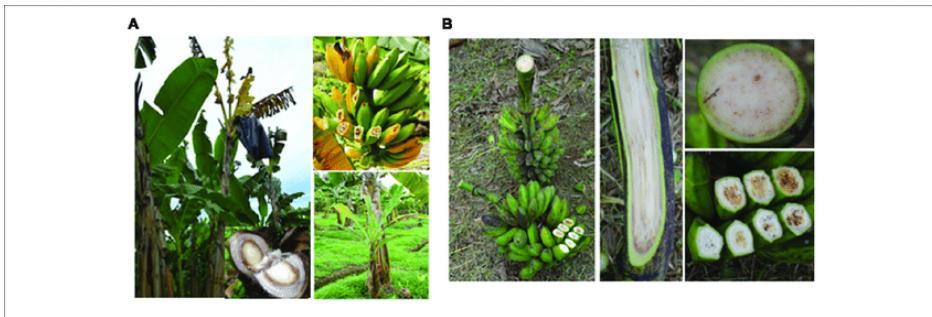
1. Moko wilt: *Ralstonia solanacearum* Race II

(Previously named as *Burkholderia solanacearum*)

First reported in Guyana in 1840. In India it was reported from West Bengal. This disease was reported in Poovan and Robusta in Tamil Nadu.

Symptoms:

- ✓ Rapid development of yellow discoloration of inner lamina close to the petiole and wilting of inner leaves.
- ✓ Necrosis of unfurled heart leaf.
- ✓ Blackening of internal fruit pulp.
- ✓ Pale yellow to dark brown strands are seen on vascular bundles and yellow slimy bacteria oozing out from the infected corm.
- ✓ Discolouration of rhizome starts from central part of rhizome and moves to the periphery.
- ✓ Production of bacterial ooze (grayish brown) in colour seen in the pseudostem of affected plant is cut transversely.



Pathogen: Moko disease is caused by race 2 of *Ralstonia solanacearum* which infects *Musa* and *Heliconia*. They are gram negative..Rod shaped gram negative bacterium with single polar flagellum (monotrichous).

Mode of spread:

Primary : Infected suckers

Secondary : Bacteria carried by irrigation water

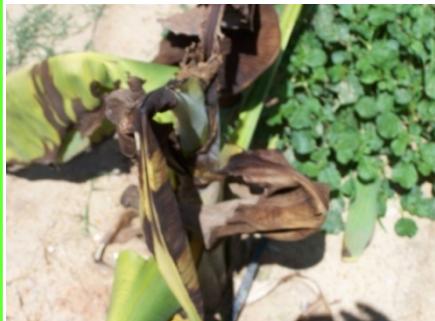
Management :

- ✓ Strict plant quarantine and phytosanitary measures
- ✓ Use of healthy planting material.
- ✓ Exposure of soil to sunlight during dry hot weather.
- ✓ Eradicate infected plants and suckers killing *in situ* by application of herbicides.
- ✓ Disinfestation of tools with formaldehyde diluted with water in 1:3 ratio
- ✓ Bacterization with *Pseudomonas fluorescens*

TOP ROT or TIP OVER or HEART ROT: *Pectobacterium (Erwinia) carotovorum sub sp. carotovorum*

Symptoms:

- ✓ Tissue culture banana and Nendran,Gro michel are highly susceptible
- ✓ The disease will be severe during summer months.
- ✓ Disease incidence is more up to 5 months.
- ✓ Infected plants can be pushed over easily and are very susceptible to wind damage.
- ✓ In severely infected soil newly planted rhizomes may rot and fail to sprout.
- ✓ When young plants are infected, a dark brown necrosis appears in the lamina of the older leaves. Later the plant becomes stunted and yellow.
- ✓ Rotting of basal portion of pseudostem at the point of attachment with suckers/corm in young plants due to production of pectinase enzyme.
- ✓ Bad odour emitted from the rotten tissues.
- ✓ Swelling of pseudostem base and rotting of collar portion.
- ✓ Toppling of pseudostem affected plants.



Pathogen: Rod shaped gram negative bacterium with flagella around the cell wall (peritrichous).

Mode of spread:

Primary : Infected suckers and soil borne bacteria

Secondary : Bacterial carried by irrigation water

Favourable conditions:

- ✓ Prevalence of high temperature during initial stages of crop
- ✓ Tissue culture plants var Grand Naine is highly susceptible

Management:

- ✓ Avoid planting during summer
- ✓ Apply bleaching powder at the rate of 6g/plant and irrigation should be given immediately,
- ✓ Sunhemp can be grown as intercrop and should be ploughed insitu
- ✓ Application of *Pseudomonas fluorescens* @25g per plant will reduce the inoculum.

viral diseases

Viral diseases are considered a major concern for banana production because of their effects on yield and quality. There are many (about 20) different viruses reported to infect banana worldwide. However, the economically most important viruses are: *Banana bunchy top virus* (BBTV), *Banana streak viruses* (BSV), *Banana bract mosaic virus* (BBrMV) and *Cucumber mosaic virus* (CMV).

Banana Bunchy Top virus Disease

Banana bunchy top virus disease (BBTD) is the most important and devastating disease first recorded in 1889 in Fiji and has since spread to a number of countries in the South Pacific, Asia, and Africa. In India, BBTV is reported to cause serious losses in many states involved in banana cultivation. This disease caused a serious havoc in hill banana cv. Virupakshi reduced the production area from 18,000 to 2000 ha.

Symptoms: Infected plants, new leaves emerge with difficulty and are narrower with wavy leaf lamina and yellow leaf margin. Leaves become progressively smaller in size with limited elongation of petiole. Leaves remain abnormally erect. When several abnormal leaves have emerged, an extreme congestion or bunching appears at the top of the plant the symptom from which the disease is named.

BBTV-affected and healthy leaves are different in texture. The petiole, midrib, and lamina of infected plants are harsh and brittle and can be easily snapped when bent or crushed in contrast to healthy ones being elastic and pliable in nature. Severely infected plants usually do not fruit, but if produced, the banana hands and fingers are likely to be distorted and twisted. Occasionally, bracts of male flower buds turn to a leafy structure and exhibit dark-green dots and streaks . BBTV symptoms are sometimes referred as “Morse code streaking” due to the presence of irregular streaks and series of dots and dashes. Rubbing the waxy white coating on petiole and midrib makes it easier to see the streaking.



Pathogen:Banana bunchy top virus (BBTV)has circular, single-stranded DNA genome.

Mode of spread :

The primary spread - infected planting materials.

Secondary spread- banana aphid(*Pentalonia nigronervosa*) which transmits the virus in a persistent and circulative manner

Banana Streak Disease

Banana streak disease is known to be the most widely distributed in banana plantations throughout the world. The disease was first observed in the Niek Valley on the Ivory Coast in 1958 The disease is now reported to occur in over 43 countries of Africa, Asia, Australia, Europe, Oceania, and tropical America .

Symptoms:The symptoms produced by most isolates are discontinuous sometimes continuous chlorotic or yellow dots or streaks that turn necrotic which run from the midrib to the leaf margin Sometimes the leaf lamina can be distorted. At later stage, the streaks darken to orange and often become brown or black. Necrosis has been observed to occur on the leaf midrib and petiole especially under low temperature and short-day conditions. Stunting, reduced bunch size, and distortion of fingers have also been reported.

Pathogen:Banana streak virus (BSV), a member of the genus Badnavirus of family Caulimoviridae, is the causal virus of the disease. The virions of BSV are bacilliform-shaped (120–150×30 nm), double-stranded circular DNA.



Mode of spread: BSV fail to transmit by mechanical inoculations. The initial or long-distance spread occurs through vegetative propagation, spread of the virus from infected to healthy banana plant occurs by mealybug *Planococcus citri* and *Saccharicoccus sacchari* in a semipersistent manner. In many tropical regions, banana is found growing in close proximity to sugarcane, and it may be possible that sugarcane may act as source of inoculum for banana

Banana Bract Mosaic Disease

Occurrence of the virus was discovered in other Asian countries including India, Samoa, Sri Lanka, Thailand, and Vietnam.

Symptoms: The virus typically caused distinctive mosaic patterns on bracts. Spindle-shaped purplish streaks on bracts, pseudostems, midribs, peduncles, and even fruits are the characteristic symptoms of the virus. The symptom color may darken through red to brown and even black. In some cultivars, such as Nendran, the leaves appear as "traveler's palm" plant. Necrotic streaks on fruits, leaves, pseudostems, and midribs have also been recorded (Selvarajan and Jeyabaskaran 2006).



Pathogen: BrMV belong to the genus Potyvirus and family Potyviridae. Flexuous filamentous RNA virus .

Mode of spread:

The primary source of infection occurs through virus-infected vegetative planting material. The BBrMV is transmitted by several aphid species (*P. nigronervosa*, *Rhopalosiphum maidis*, *Aphis gossypii*, *A. craccivora*) in a nonpersistent manner

Infectious chlorosis

Banana mosaic is also known as infectious chlorosis, heart rot, sheath rot, and cucumber mosaic. However, severe strain (heart rot) of the virus is known to cause significant economic damage.

Symptoms: The virus causes variable symptoms from mild chlorosis to severe chlorotic streaks on leaf lamina. Symptoms are more pronounced which include necrosis of emerging leaves and internal tissues of pseudostem. Fruits may show mosaic symptoms and bunches may bear malformed fruit or no fruit. Plant death may occur in very severe cases especially when plants get infected with severe strain soon after planting.



Pathogen: Cucumber mosaic virus (CMV) which is a member of Cucumovirus group . Spherical virus particles , having single-stranded positive-sense RNA as genome.

Mode of spread: The spread of the disease occurs in nature through vegetative planting material and by over 60 different species of aphid vector including *Aphis gossypii*, *A. craccivora*, *Rhopalosiphum maidis*, *R. prunifolium*, and *Myzus persicae* .

Management :

- ✓ exclusion of the disease by effective quarantine,
- ✓ use of healthy planting materials by a well-regulated certification program,
- ✓ vector control.
- ✓ destruction of diseased stool

8. Symptoms, mode of spread, survival, epidemiology and integrated management of Diseases of guava, papaya and pomegranate

Diseases of Guava and their management

1. Wilt – *Fusarium oxysporum f.sp. psidii*, *F. solani*, *Macrophomina phaseolina*, *Cephalosporium sp.*, *Gliocladium roseum* and *Verticillium alboarum*.

This disease was first reported in 1935 from Allahabad. Severe incidence of wilt was reported that seven thousand acres of land in A.P under guava cultivation was reduced to half the land value by the presence of the disease.

Symptoms

- ✓ The affected plants show yellow colouration with slight leaf curling at the terminal branches, becoming reddish at the later stage and subsequently premature shedding of leaves takes place.
- ✓ Twigs become bare and fail to bring forth new leaves or flowers and eventually dry up. Fruits of all the affected branches remain underdeveloped, become hard, black and stony
- ✓ The entire plant becomes defoliated and dies. A few plants also show partial wilting, which is very common symptom of wilt in guava.
- ✓ The finer roots show black streaks which become prominent on removing the bark.
- ✓ The roots also show rotting at the basal region and the bark is easily detachable from the cortex.
- ✓ The cortical regions of the stem and root show distinct discolouration and damage.
- ✓ Light brown discoloration is noticed in vascular bundles. Bark splitting can be seen in wilted plants in later stages.
- ✓ The disease can be categorized into slow wilt and sudden wilt. In slow wilt, plant takes several months or even a year, to wilt after the appearance of initial symptoms and in sudden wilt, infected plant wilts in 15 days to one month.



Pathogen: Fungus produces 3 types of spores

- ✓ Microconidia-Ovate / elongate / 1 celled, hyaline
- ✓ Macroconidia – Spindle shaped, tapered, 3-4 septate hyaline
- ✓ Chlamydospores - Terminal or Intercalary

Mode of spread:

Primary : Soil borne chlamydospores

Secondary : Water borne conidia

Survives as saprophytes and chlamydospores which remain dormant for several years.

Favourable conditions:

- ✓ **pH 6.0** is optimum for disease development. Both pH 4.0 and 8.0 reduces the disease.
- ✓ Disease is more in clay loam and sandy loam compared to heavy soil.
- ✓ Higher disease incidence in monsoon period.
- ✓ Disease appears from august and increases sharply during September-October.
- ✓ The presence of nematode, ***Helicotylenchus dihystera***

Management

Cultural:

- ✓ Proper sanitation of orchard.
- ✓ Wilted plants should be uprooted, burnt and a trench of 1.0-1.5m should be dug around the tree trunk. Treat the pits with formalin and cover the pit for three days and then transplant the seedlings after two weeks.
- ✓ While transplanting seedlings avoid damage to the roots.
- ✓ Maintain proper tree vigour by timely and adequate manuring, inter-culture and irrigation.
- ✓ Growing marigold around the basin and irrigation channels.
- ✓ Soil solarization with transparent polythene sheet during summer months.
- ✓ Application of oil cakes like neem cake, mahua cake, kusum cake supplemented with urea. Apply 6kg neem cake + 2kg gypsum per plant.
- ✓ Judicious amendments of N and Zn.

Chemical:

Biological:

- ✓ ***Aspergillus niger* strain AN 17**, ***Trichoderma viride***, ***Trichoderma harzianum*** and ***Penicillium citrinum*** can be used as biocontrol agents

Stem Canker : *Physalospora psidi*

Symptom

Causes cracks in branches wilting of branches due interference of translocation of water and nutrients.

Mode of spread

Primary spread through conidia and secondary spread through ascospores (perithecia)

Survival

- ✓ As dormant mycelium below the bark

Management

- ✓ Removal of dead stem paint the cut end with bordeaux paste

Red rust : *Cephaeleros virescens*

Symptom

Favourable conditions

Bright sunlight

Management

Spray copper oxy chloride @ 2.5 g/l or 0.5 % Bordeaux mixture or wettable sulphur @ 2 g/l

Red Rust: *Cephaleuros virescens*

In India it has been observed in Bihar, Karnataka and U.P. The disease appeared in an epidemic form in orchards of Tarai in 1956. Reduction in photosynthetic activity and defoliation as a result of algal attack lower vitality of the host plant.

Symptoms:

- ✓ It is an algal parasite. Affect leaves and fruits
- ✓ On fruits the lesions are small, dark green to brown or black some times. On the leaves small brown specks are seen in patches.
- ✓ Produces small red orange raised velvety mycelial growth on the upper surface of the leaf. In severe cases, symptoms seen on the fruits.



Pathogen:

The alga produces sporangia on sporangiophore. Sporangia produce numerous zoospores. 5-8 sporangia are found on each vesicle. Zoospores are involved

in the disease spread.

Management:

- ✓ Avoid close plantations
- ✓ Spray Bordeaux mixture 1-2% or Fytolan / Blitox 0.5% or lime sulphur 0.1%

Anthracnose: *Gloeosporium psidii*

Symptoms:

- ✓ Die back of young twigs and branches
- ✓ Circular to irregular black sunken lesions with pinkish sporulation appear on the fruit
- ✓ Skin of the fruit turns black and rots
- ✓ Latent infection – the infection originate from the green fruits in the field and expressed during ripening



Pathogen: Hyaline, septate and branched conidiophore on which cylindrical, single celled, hyaline and thin walled conidia with large number of oil globules are produced.

Mode of spread

Primary : Air borne conidia

Secondary : Spread through the contact of infected fruits

Management:

- ✓ Cowdung paste and actionmycetes isolated from cow dung paste has also shown positive response in control of dieback, anthracnose in guava.
- ✓ Foliar spray of mancozeb 1gm/lit

SOOTY MOULD :*Capnodium sp*

Symptoms:

- ✓ Black encrustation formed on flowers, leaves ,stem and fruit
- ✓ Mycelium superficial and lives on the sugary secretion of the sucking pests like hoppers, jassids, aphids and mealy bugs.
- ✓ Photosynthetic activity is reduced which results in reduced fruit set and fruit fall

Pathogen: Fungus produces 5 types of conidia such as 1. Torula 2. Trichothecium 3. Coniothecium 4. Brachysporium 5. Ascospores from Pseudothecia



Mode of spread : Primary and Secondary spread are through wind borne conidia

Management:

Spray Maida 5% (1 kg Maida or starch) boiled with 1 l of water and diluted to 20 litres. Avoid during cloudy weather

Diseases of Pomegranate

Anthracnose: *Gloeosporium gloeosporioides*

Symptoms:

- ✓ Spots seen on leaves and fruits
- ✓ Leaf : Necrotic spots on leaf
- ✓ Fruit : Circular, slightly sunken spots .Latex oozes out



Pathogen: Hemi biotroph produces hyaline septate branched mycelia. Fungus produces acervuli without black setae. Conidia are single celled, hyaline, thin walled with full of oil globules.

Mode of spread : Primary spread and Secondary spread through wind borne conidia

Favourable conditions

- ✓ Severe during August-September
- ✓ High humidity and temp ranging from 20 to 27o C

Management:

Spray carbendazim@0.1% or Thiophanate-methyl @0.1% or mancozeb@0.2% at fortnightly intervals

Cercospora leaf spot: *Cercospora punicae*

Symptoms:

- ✓ Light brown zonate spots appear on leaves and fruits
- ✓ Black elliptical spots appear on the twigs
- ✓ Affected areas in the twigs become flattened and depressed with raised edge.

Pathogen: Conidiophore are olivaceous brown, short fasciculate, sparingly septate. Conidia are olivaceous, cylindrical and multi-septate.

Mode of spread : Primary spread through ascospores from fallen leaves and Secondary spread is through wind borne conidia

Management:

- ✓ Pruning and destruction of diseased twigs.
- ✓ Application of thiophanate- methyl 0.1% or chlorothalonil 0.2% or mancozeb 0.2%

Bacterial leaf spot : *Xanthomonas campestris* pv. *punicae*

Symptom

- ✓ Dark coloured irregular spots 2-5 mm in diameter
- ✓ The leaves often distorted and malformed
- ✓ Premature dropping of leaves. The normal growth of the plant is affected
- ✓ Raised spots are seen on the fruits which are irregular.
- ✓ Fruit splitting noticed in advanced infection stages



Pathogen: Gram negative, rod, motile with single polar flagella

Mode of spread & Survival : The bacteria infect through wounds and stomatal openings. Survive in the soil.

Management:

Foliar spray of **Streptomycin + Tefracycline (Agrimycin 100 ppm)**

DISEASES OF PAPAYA

1. Foot or stem rot : *Pythium aphanidermatum*

Symptom

- ✓ This pathogen also cause damping off in nursery
- ✓ Water soaked patches on stem at ground level
- ✓ Girdling of the stem
- ✓ Terminal leaves turn yellow, droop and wilt
- ✓ Due to disintegration of parenchymatous tissues at the base of the stem, the internal tissues of the bark give a **honey comb appearance**
- ✓ Roots deteriorate and entire plant topples and dies





Pathogen : Coenocytic mycelium ,produces sporangiospore bearing irregular shaped sporangia which bears the vesicle .Inside the vesicle the zoospores are produced.

Mode of spread

Primary : Soil borne Oospores

Secondary : Water borne zoospores

Survival and spread

- ✓ Pathogen survive in the soil Spread through irrigation water

Control

- ✓ Remove affected plants and burn Avoid water logging condition
- ✓ Drench the soil with 1% Bordeaux mixture.

POWDERY MILDEW: *Oidium caricae*

Symptoms:

- ✓ Powdery growth on undersurface of the leaves.
- ✓ Some times on upper surface. Flower stalks and fruits are also affected and exhibit white powdery patches



Pathogen : Obligate parasite produces short conidiophore and also produces haustoria. Barrel shaped conidia, mycelium hyaline septate conidiophore short hyaline and produces conidia in chains.

Mode of spread

Primary : Soil borne ascospores

Secondary : Wind borne conidia

Management:

- ✓ Spray wettable Sulphur@0.3% at 10 days interval
- ✓ Spray systemic fungicides like carbendazim@0.1% at monthly intervals

Leaf spot : *Myrothecium roridum*/

Symptom

- ✓ Small dirty yellow water soaked spots on the leaves Having brown to violet periphery with chlorotic halo Spots coalesced and leaf dries.

Survival

- ✓ Survive in infected leaf and fruit
- ✓ Spread through wind borne conidia

Control

Spray 0.2% mancozeb or 0.25% copper oxychloride.

Leaf spot : *Phyllosticta sulata cercospora leaf spot/Asperisporium caricae*

Symptom

- ✓ Round irregular oval or elongated spots on the leaves White in centre bounded by yellowish or brownish margin Centre of the spot thin and papery.

Survival

- ✓ Survive in plant debris Spread through wind

Control

Three spraying with 1% Bordeaux mixture

Dry root rot : *Macrophomina phaseolina*

Symptom

- ✓ Yellowing and dropping of leaves
- ✓ Red to black wet rot developed just before death
- ✓ Disintegration of tissue in advanced stage especially near soil level.

Survival

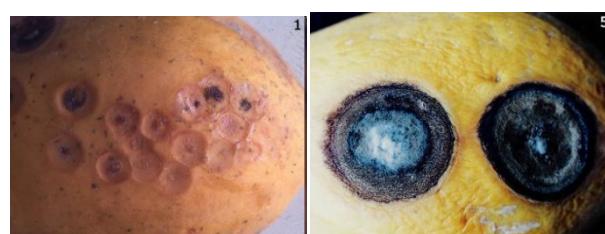
- ✓ Soil borne and in debris
- ✓ High temperature induce the disease development.

Control

- ✓ Removal of affected plants
- ✓ Drench with 1% Bordeaux mixture or Carbendazion 0.1%

Papaya anthracnose:- *Colletotrichum gloeosporioides*

- ✓ Circular, sunken, brown discolouration on fruits
- ✓ Necrotic spots with acervuli on leaves and stems
- ✓ Latent infection in field



Mode of spread: Conidia spread by wind & rain splashes

Management: Foliar spray of Carbendazim 0.1% / mancozeb 0.2% / Chlorothalanil 0.2%.

Papaya ringspot virus

Papaya ringspot virus was first reported in western India in 1958 . Since then it has spread to different geographical locations of India irrespective of the agro-climatic conditions, and causes crop losses of up to 85–90% . In papaya it causes mottling and distortion of leaves, ringspots on fruit and water-soaking streaks on stems and

petioles. It stunts the plant and drastically reduce the size of the fruit. Filiformity observed in the young seedlings.

PRSV is a member of *Potyvirus*, PRSV contains a single-stranded positive sense RNA

The virus is transmitted in a non-persistent manner,(by three aphid vectors viz., *Aphis gossypii*, *A. craccivora*, and *Myzus persicae*) meaning that the virus does not multiply within the aphid but is instead carried on its mouthparts and is transmitted from plant to plant while feeding.. It is unlikely that PRSV can be transmitted by mechanical means such as by using the same garden tools on both infected and non-infected papaya trees. The main mode of transmission is through the feeding of aphids.



Management:

Raising papaya seedlings in insect proof net house. Spray with a systemic insecticide 3 days before transplanting. Grow two rows of border crop with maize at one month before transplanting of seedlings. Apply FYM @10 kg/pit. For vector management, spray dimethoate @ 1.5 ml / l or neem oil @ 3% or acephate 1.5 g/lit or imidacloprid 0.075% (7 ml per 10 litres of water) at monthly intervals up to 5 months after planting followed by zinc sulphate @ 0.5% + boron @0.1% at 4th and 7th month after planting .Installation of yellow sticky traps (12 nos./ha) swabbed with grease or castor oil to attract the aphids is advisable.

Papaya leaf curl virus disease:

Leaf curl disease of papaya caused by Papaya leaf curl virus (PaLCV) is a devastating disease prevailing in south India. This disease is characterized by curling, crinkling and distortion of leaves, Severity is more on young leaves, accompanied by vein clearing inward rolling and thickening of leaves. Leaves become leathery, brittle

and distorted. Plants stunted affected plants do not produce flowers and fruits. This disease is transmitted by whitefly *Bemisia tabaci*.



Management

- Removal and destruction of the affected plants
- Vector management – Foliar spraying of systemic insecticides.
- Avoid tomato tobacco near papaya field.

Papaya Mosaic : Virus

Symptom

- ✓ Mottling, puckering, chlorotic and malformed appearance of leaf Increase in number of leaves
- ✓ Leaves are reduced in size
- ✓ Old leaves get defoliated leaving tuft of small one at the top
- ✓ New leaves formed after infection showing yellow mosaic symptom.

Spread

Through *Aphis gossypi* and also by *myzus persicae*

Control

Removal of affected plant.

Check the insects by spraying systemic insecticide.

9. Symptoms, mode of spread, survival, epidemiology and integrated management of Diseases of tomato

Crop : Tomato

Disease name: Damping off

Introduction

The disease is common in many parts of India. Tomato seedlings are highly susceptible to Damping off. The fungus attacks the seedlings in the greenhouse and nursery leading to heavy loss in germination and establishment.

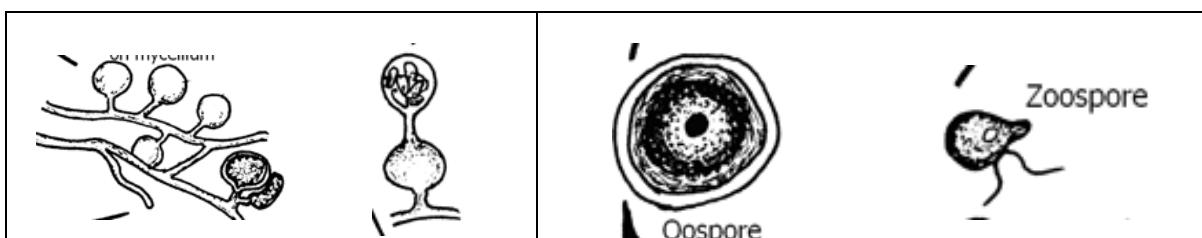
Causal organisms: *Pythium aphanidermatum*, *Phytophthora nicotianae* var. *parasitica*, *Rhizoctonia solani*

Symptoms

- Pre-emergence damping off: The disease may attack the seedlings before the emergence of seedlings from the soil. The young seedlings are killed before emergence which leads to poor seed germination and gappiness in the nursery.
- Post-emergence damping off: After the emergence of seedlings, water soaked, soft, brown lesions appear on the stem at or below the ground level. The stem is constricted at the base and subsequently collapse (toppling over of seedlings).



Pathogen character : *Pythium* produces coenocytic mycelium with sporangiophore bearing lobed sporangia. The sporangia contains vesicle which bears the zoospores. Sexual spore is oospore.



Favourable conditions/ Epidemiology : Excess soil moisture due to water stagnation, high humidity, overcrowding of seedlings and high seed rate.

Mode of survival: As oospores or sclerotia in soil or plant debris.

Primary spread: From soil-borne oospores or sclerotia.

Secondary spread: Through irrigation water.

Management

- Provide proper drainage and use of raised nursery bed
- Cover the nursery with polythene sheet for 7-10 days
- Treat the seeds with thiram or captan @4g/kg or *Trichoderma viride* @4g/kg or *Pseudomonas fluorescens* @10g/kg.
- Soil application of *Pseudomonas fluorescens* or *Trichoderma viride* @10g /sq.m along with FYM.
- Soil application of Pf1 @ 2.5 kg mixed with 50 kg FYM /ha at 30 days of transplanting.
- Soil drench with Bordeaux mixture @1.0% or copper oxychloride @0.25%

Crop : Tomato

Disease name : Early blight

Introduction

Fungi belonging to the genus *Alternaria* are commonly found on leaf spots and dead tissue. Large number of strains which attack different host are available. The fungus also causes blemishes and storage rots.

Causal organisms : *Alternaria solani*

Symptoms

- On leaves circular to irregular, dark brown to black spots with concentric rings appear.
- The spots coalesce and cause drying and defoliation of leaves.
- Dark spots at the base of the stem near the ground level gradually girdle the stem.
- Dark brown sunken spots appear on fruit. Immature fruits are shed.



Pathogen character : It is a polycyclic necrotrophic pathogen, produces dark colored, multinucleate muriform and beaked conidia which arise singly in simple conidiophore.



Favourable conditions: June-July sowing and weak and older plants.

Mode of survival: In plant debris and seeds

Primary spread: From infected plant debris and seeds.

Secondary spread: Air-borne conidia.

Management

- Keep the field clean.
- Remove infected plants and debris.
- Follow crop rotation with non-solanaceous crops.
- Use disease free seeds. Collect healthy seeds from disease-free fruits.
- Treat the seeds with thiram or captan @4g/ kg.
- Provide optimum spacing to avoid overcrowding.
- Avoid heavy N and dense planting to minimize relative humidity.
- Spray zineb (or) chlorothalonil (or) mancozeb (or) difolatan @0.2 %.

Crop : Tomato

Disease name : *Fusarium* wilt

Introduction

Fusarium wilt is one of the most prevalent and damaging diseases of tomato which is most destructive in warm climates and warm, sandy soils of temperate regions. Massee first described *Fusarium* wilt disease of tomato in 1895. Three races of the pathogen have been identified based on the ability to cause disease on tomato with different forms of disease resistance. Race 1 infects varieties with no genetic resistance to *Fusarium* wilt. Race 2 was first identified in 1945 and Race 3 was first identified in Australia in 1978.

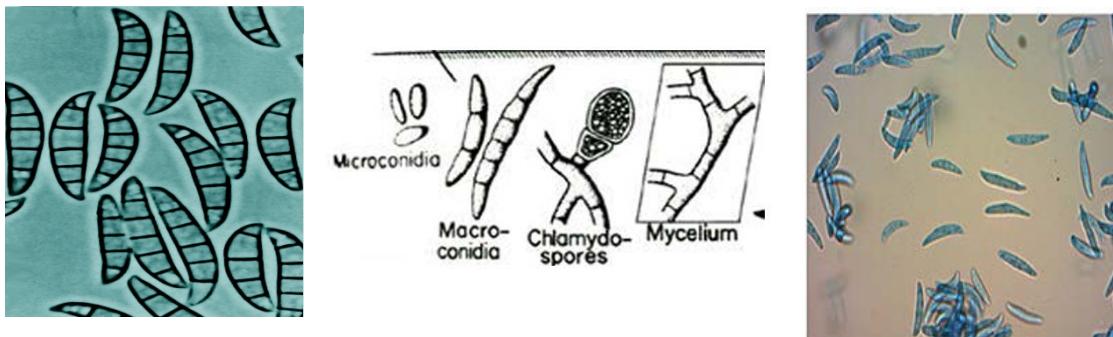
Causal organisms : *Fusarium oxysporum* f. sp. *lycopersici*

Symptoms

- Affected plants exhibit yellowing and drooping of lower leaves
- Internal stem portion exhibits vascular browning.
- Plants wilt and die in due course.
- Though the disease appears in all stages, young plants are more vulnerable for infection.



Pathogen character : Mycelium is septate. The fungus produces microconidia, macroconidia and chlamydospores. Microconidia are hyaline, oval, single celled or one septate. Macroconidia are sickle-shaped, 3-5 celled and hyaline. Chlamydospores are thick walled, round resting spores, produced in terminal or intercalary.



Favourable conditions: Root-knot nematode predisposes the plant to disease incidence.

Mode of survival: In crop debris and soil as chlamydospores.

Primary spread : From soil and diseased seedlings.

Secondary spread : Through irrigation water

Management

- Follow deep summer ploughing.
- Follow crop rotation with non-solanaceous crops (cereals).
- Remove infected plants and plant debris.
- Treat the seeds with carbendazim @ 2g/kg or *Trichoderma viride* @4g/kg or *Pseudomonas fluorescens* @10g/kg.
- Nursery application of *Pseudomonas fluorescens* @ 20g/m² and seedling root dip with *Pseudomonas fluorescens* @10g/lit of water and soil application of *Pseudomonas fluorescens* @ 2.5 kg/ha + 50 kg of FYM.
- Spot drenching with carbendazim @ 0.1%.

Crop : Tomato

Disease name : *Septoria* leaf spot / Defoliation disease

Introduction

Septoria leaf spot is one of the most common foliar diseases of tomato. It was first reported in Argentina in 1882 and later in the US in 1896. Depending on the weather conditions, the fungus cause epidemic in the field and has been known to cause complete crop failure.

Causal organisms : *Septoria lycopersici*

Symptoms

- Numerous, circular spots with grey centre and dark brown margins are seen on the leaves, stem and calyx.
- Centre of the spots shows minute black fructifications.
- Severe infection causes defoliation during rainy season.



Pathogen character : The mycelium is hyaline, thin walled and sparingly septate. Pycnidia are subglobose, composed of 2-3 layers of brown cells. Pycnidiospores are filiform, slightly curved, hyaline and septate with pointed or rounded ends.

Favourable conditions: Rainy season

Mode of survival: In infected plant parts

Primary spread: From infected plant parts

Secondary spread: Air-borne pycnidiospores

Management

- Follow crop rotation (avoid potato and brinjal)
- Treat the seeds with thiram @4 g/kg
- Spray zineb or mancozeb @ 0.2 % or copper oxychloride @0.25 % or Bordeaux mixture @1.0%

Crop : Tomato

Disease name : Late blight

Introduction

Phytophthora infestans, commonly known as the Irish potato famine pathogen cause late blight of tomato. The disease cause yield loss of 100 % during the favorable conditions. Two mating types viz., A1 and A2 have been recorded. Recently, severe outbreaks of late blight on tomato in South India have been reported.

Causal organisms : *Phytophthora infestans*

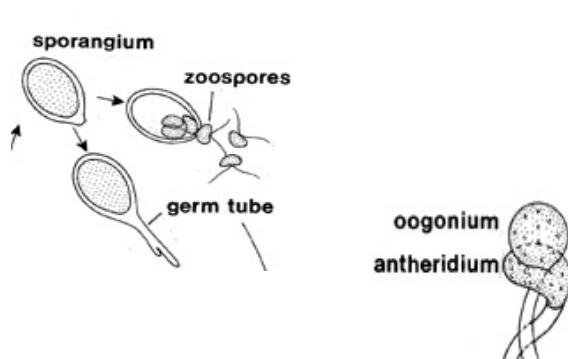
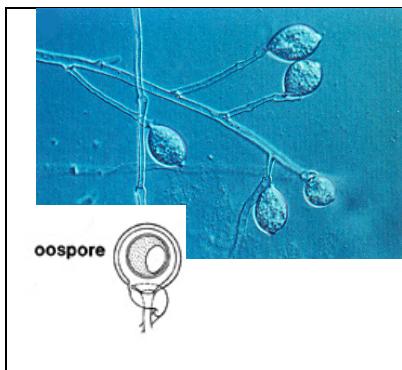
Symptoms

- Infection occurs on all the above ground plant parts.
- Water-soaked lesions with faded green patches appear on the leaves.
- Infections spread fast to entire leaf and petiole. Dead areas appear in leaf tip and margins.
- Whitish mildew like growth appears on leaves under humid conditions. The leaves gets blighted and dry.
- Dark olivaceous greasy spots with cracking and appearance of white fungal growth on the fruits are seen resulting in soft rot.

- Internal tissues become spongy with bad odour.
- In severe cases the whole plant dies.



Pathogen character: Mycelium is hyaline and coenocytic, endophytic, intercellular with haustoria. Sporangia are hyaline, pear-shaped or lemon-shaped with papilla at the tip. Each sporangium releases 3 to 8 zoospores. Zoospores are reniform and biflagellate. Oospores produced late in the season and they are the thick walled resting spores which help in the survival of the pathogen.



Favourable conditions: High humidity and low temperature, cloudy weather and rainfall with splashing rains.

Mode of survival: In infected plant debris and soil as oospores.

Primary spread: Oospores in soil.

Secondary spread: Air-blown sporangia and rain splashes.

Management

- Follow crop rotation with cereals.
- Provide good drainage.
- Follow summer ploughing and apply green manures/ biocontrol agents
- Avoid dense planting and maintain proper aeration in the collar region.
- Follow staking of plants or mulching with straw to avoid the contact of fruits in soil.
- Remove and destroy infected plants and drench the soil with copper oxychloride 0.25%.

- Spray mancozeb or difolatan @ 0.2% or metalaxyl + mancozeb @0.2%.

Crop : Tomato

Disease name : Bacterial wilt

Introduction

Bacterial wilt is a widespread devastating disease in tropical, sub-tropical and temperate regions that mainly affects the Solanaceous crops. In India, bacterial wilt of tomato was first reported in Solan area of Himachal Pradesh by Gupta *et al* 1998. Generally Race 1, Biovar 3 induces the wilt. The first report of bacterial wilt caused by *Ralstonia solanacearum* Race 1, Biovar 2 on tomato is from Egypt. Recently, *R. solanacearum* species complex is classified into three species such as *R. pseudosolanacearum* (Phylotypes I and III strains); *R. solanacearum* (Phylotype II strains), and *R. syzygii* (Phylotype IV strains) based on the significant variations in whole genome (Prior *et al.* 2016).

Causal organisms : *Ralstonia solanacearum*

Symptoms

- Stunting, yellowing and wilting of foliage leading to collapse of the entire plant.
- Lower leaves droop before wilting.
- Cross section of the stem near the base show vascular browning.
- When the affected stem is cut bacterial ooze can be seen.
- Adventitious roots are formed from the stem.
- The affected plants collapse and die.





Pathogen character: It is Gram-negative and rod-shaped, occurring in pairs with 1-4 polar flagella.

Favourable conditions: Humid weather and high temperature.

Mode of survival: Survives for several years in soil.

Primary spread: Soil and seed infestation.

Secondary spread: Through irrigation water.

Management

- Follow crop rotation with cereal crops or cruciferous vegetables avoiding solanaceous crops
- Incorporate bleaching powder in soil @ 15 kg/ha before transplanting.
- Seedling root dip in Streptocycline 25 ppm (2.5g/100 lit of water) for 30 min.
- Soil drench with Bordeaux mixture @1%.

Crop : Tomato

Disease name : Bacterial leaf spot

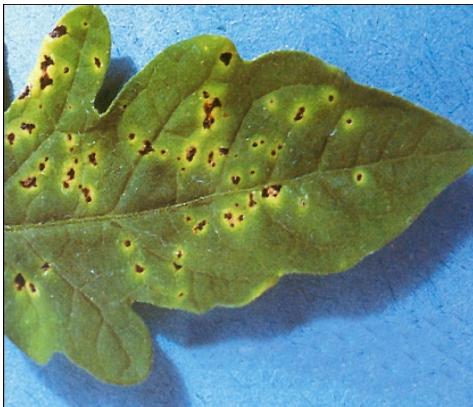
Introduction

Bacterial spot was first discovered on tomato in South Africa in 1914, and named *Bacterium vesicatorium* by Doidge (1920). The disease was later identified in Indiana by Gardner and Kendrick (1921) who later discovered that *B. vesicatorium* also caused leaf spot of pepper (*Capsicum annuum*), but Higgins (1922) first described the disease. *B. vesicatorium* was thought to be the only causal agent of bacterial spot and reclassified several times to *Pseudomonas vesicatoria*, *Phytomonas vesicatoria* and *Xanthomonas campestris* pv. *vesicatoria*.

Causal organisms : *Xanthomonas vesicatoria*

Symptoms

- Small translucent water-soaked spots on the leaves enlarge to black greasy or dark-brown spots surrounded by yellow halo.
- Several spots cause chlorosis in the leaves and defoliation occurs.
- Black cankerous spots occur on stem and petiole.
- Water-soaked lesions on unripe green fruits become corky resembling small scabs with irregular margins.



Pathogen character : It is Gram-negative, rod-shaped motile with single polar flagellum.

Favorable conditions: Temperature of 25-30°C, relative humidity of more than 90% and humid weather.

Mode of survival: In plant debris and seed (seed-borne).

Primary spread: From infected seeds.

Secondary spread: Air-blown rain splashes.

Management

- Follow crop rotation.
- Follow summer ploughing.
- Collect seeds from disease-free plants.
- Dip the seeds in streptocycline 100ppm or treat seeds in hot water @ 50 °C for 25 min.
- Follow soil solarization in nursery bed to avoid seedling infection.
- Spray copper oxychloride @ 0.25%.

Crop : Tomato

Disease name : Bacterial canker

Introduction

Bacterial canker was one of the first bacterial diseases reported on plants. Over a hundred years ago, Erwin F. Smith was the first to describe this disease in 1909 in Michigan, USA. It is currently being reported in tomato production areas worldwide. During the favorable conditions, disease outbreaks to almost 100 % occur resulting in heavy losses.

Causal organisms : *Clavibacter michiganensis* sub sp. *michiganensis*

Symptoms

- Leaves shows wilting symptoms.
- Stem shows streaks and canker
- Small brown scabby lesions surrounded by white halo appear on the fruits which resembles the birds eye.



Pathogen character : It is Gram-positive, aerobic, coryneform, rod-shaped motile with single polar flagellum.

Favourable conditions: More severe during wet weather.

Mode of survival: In plant debris and seed (seed-borne).

Primary spread: From infected seeds.

Secondary spread: Air-blown rain splashes.

Management

- Follow crop rotation.
- Follow summer ploughing.
- Collect seeds from disease-free plants.
- Dip the seeds in streptocycline @100ppm or treat seeds in hot water @ 50°C for 25 min.
- Follow soil solarization in nursery bed to avoid seedling infection. Spray copper oxychloride @0.25%.

Crop : Tomato

Disease name : Mosaic

Introduction

Tomato Mosaic was reported in 1909 in US (Connecticut) which is a distinct viral species, transmitted by contact. It is more frequently observed in tomato and pepper in field and under protected conditions.

Causal organisms : *Tomato mosaic virus* (ToMV).

Symptoms

- Typical mosaic pattern with dark green and light green areas appear on leaves.
- Leaflets are distorted, puckered and are small.
- Leaflets exhibit, fern- leaf symptoms.
- Necrotic sunken lesions are seen on fruits.
- Infection on matured fruits shows internal necrosis.
- The affected plants are stunted and pale green.



Pathogen character : rod shaped RNA virus

Favourable conditions: Injuries to plant.

Mode of survival: Solanaceous hosts and on the seeds (externally seed borne).

Primary spread: From infected seeds and from other host plants.

Secondary spread: The virus is transmitted by contact (sap transmissible), hands of workers, plant debris, implements and on the surface of seeds.

Management

- Follow crop rotation with non-solanaceous crops.
- Select seeds from disease free plants.
- Rogue out infected seedlings in the nursery.
- Raise protected nursery (in net house or green house).
- Raise barrier crops like sorghum or maize or pearl millet 5-6 rows around the field before planting tomato.
- Remove weed hosts, volunteer plants and infected plants.

Crop : Tomato

Disease name : Leaf curl

Introduction

Leaf curl is one of the major diseases of tomato found in tropical and subtropical regions causing severe economic losses. The virus is transmitted by Whitefly (*Bemisia tabaci* Genn.) under natural conditions. Several crop as well as weed species are known to harbor ToLCVs in India. Maximum temperature and rainfall play an important role for the spread of the disease in southern India.

Causal organisms : *Tomato leaf curl virus* (ToLCV)

Symptoms

- Typical downward curling, crinkling and chlorosis of leaves.
- Infected plants are stunted with shortened internodes giving bushy appearance.
- No flowering and fruiting at later stage of infection.
- Small leaf-like growth called enations on the midrib on the lower surface of leaf.



Pathogen character: single circular single-stranded (ss) DNA molecule (2787 nt in size) which is a common distinction among viruses in the family *Geminiviridae*. The ssDNA genome encodes for six open reading frames (ORF).

Favourable conditions: Abundance of insect vector (White fly)

Mode of survival: In susceptible host plants.

Primary spread: From susceptible crops and weed hosts.

Secondary spread: Insect vector, whitefly (*Bemisia tabaci*).

Management

- Removal of weed hosts and infected plants
- Raise protected nursery (in net house or green house) to prevent whitefly infection.
- Treat the nursery bed with carbofuran @1.0 kg ai/ha and another dose at one week after transplanting @ 1.5 kg ai/ha followed by foliar spraying with dimethoate or methyl demeton @ 2ml/lit on 15, 25, 45 DAT to control vector.
- Dip seedling roots in Imidacloprid solution @ 4-5 ml/lit for one h before transplanting.
- Adopt mulching with straw to repel whiteflies.
- Install yellow sticky traps to manage insect vector.
- Raise barrier crops of sorghum or maize (5-6 rows) around the field before planting tomato.

Crop : Tomato

Disease name : Tomato spotted wilt / Bronzy wilt

Introduction

Tomato spotted wilt is one of the most economically devastating diseases of tomato around the world. Brittlebank, (1919) published the first description of this disease on tomatoes in 1915 in the state of Victoria (Australia) and he called it as "spotted wilt virus of tomato". Losses of 75–100 % from tomato spotted wilt have been reported in Hawaii. The first characterization of this virus as the causal agent of the disease was reported by Samuel *et al.* (1930), who gave it its current name "*Tomato spotted wilt virus*". TSWV ranks second in the top ten most detrimental viruses worldwide (Scholthof *et al.*, 2011).

Causal organisms : *Peanut bud necrosis virus* (PBNV)

Symptoms

- Necrotic irregular spots are seen on the leaf surface.
- Leaves are reduced in size and exhibit thickened, bronzy veins.
- The place where the branch arises from the main stem becomes weak and necrotic leading to bending of branches from the main stem.
- On fruits, numerous pale yellow or yellowish concentric circular rings are seen.
- Younger plants wilt and die but older plants survive with spotted fruits that may not ripe properly.
- Poor seed recovery and reduced lycopene content.



Pathogen character : Tospoviruses - negative single stranded RNA viruses belonging to family *Bunyaviridae*. They are quasi spherical particles with 80-120 nm diameter containing three single-stranded nucleic acid segments, denoted as L, M and S RNAs. Favourable conditions: Abundance of thrips population.

Mode of survival: In susceptible crop hosts and weeds.

Primary spread: From susceptible crops and weeds.

Secondary spread: By insect vector, thrips (*Frankliniella schultzei*, *Scirtothrips dorsalis*, and *Thrips tabaci*).

Management

- Remove weed hosts and keep the field clean.
- Raise barrier crops like sorghum, maize or bajra 5-6 rows around the field before planting tomato.
- Select virus free seedlings for planting.
- Use aluminium surfaced plastic mulch to repel vector.

- Rogue out infected plants upto 45 DAT.
- Spray AVPs (leaf extracts of sorghum or coconut 10%) on 10, 17 and 24 DAP.
- Apply carbofuran @ 33 kg/ha in nursery at sowing followed by second application on 10 DAP in the main field and spray phosalone @1.5 ml/lit on 25, 40 and 55 DAP.

10. Etiology, symptoms, mode of spread, survival, epidemiology and integrated management of Diseases of brinjal and okra

Crop : Brinjal

Disease name : Damping off

Introduction

Damping- off disease causes about 60% losses in brinjal at seedling stage in the nursery and field conditions. The disease will lead to poor germination and death of brinjal seedlings which cause economical loss to the farmers. The disease is known by various name viz., collar rot, necrose du collet, water rot, stem rot of seedling which causes mortality upto 90% (pre emergence and post emergence seedling mortality) if infected early.

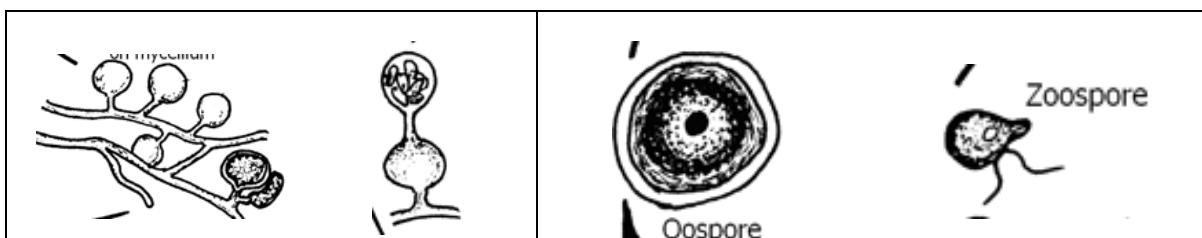
Causal organisms : *Pythium aphanidermatum*

Symptoms

- Pre-emergence damping off : Seed and seedling rot before emergence from soil.
- Post-emergence of damping off : Characterized by water-soaked, brown discolouration at collar region followed by softening of tissues and collapse at soil level.



Pathogen character : It produces hyaline, aseptate mycelium, oospores may form a germ tube directly to infect the plant or they may form sporangia. The sporangia produce zoospores, which are the motile form of the fungus.



Favourable conditions: High soil moisture and moderate temperature along with high relative humidity especially in the rainy season.

Mode of survival: As oospores in infected plant parts in soil.

Primary spread: From oospores in the soil.

Secondary spread: Through irrigation water.

Management

- Sow healthy seeds.
- Treat seeds with thiram or captan @4g/kg or *Trichoderma viride* @ 4g/kg or *Pseudomonas fluorescens* @10g/kg.
- Avoid raising nursery continuously in the same area.
- Drench nursery soil with copper oxychloride @0.25% or Bordeaux mixture @ 1%. Solarize the soil by spreading 25µm transparent polythene sheet over the seed bed for 40-45 days during summer.
- Application of *Trichoderma viride* or *Pseudomonas fluorescens* @ 40 g/ sq.m along with FYM.

Crop : Brinjal

Disease name : *Fusarium* wilt

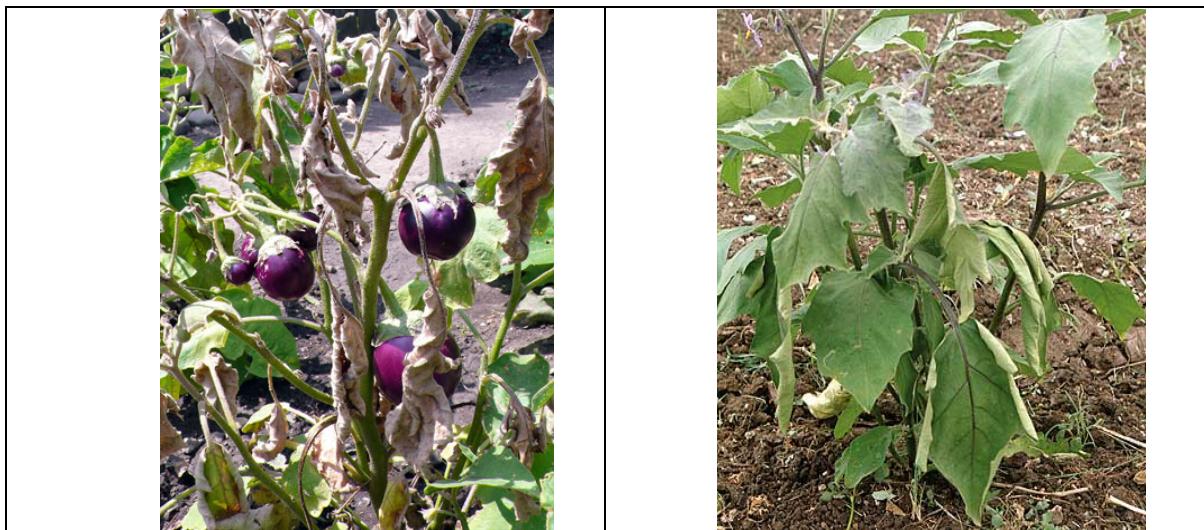
Introduction

It is an important vascular disease in India which causes distinct loss to the crop under favourable conditions. The disease has been first reported in Iran followed by Japan, Korea, and Turkey. In India it is first reported from pune in 1984. It causes yield loss upto 75-81% in India during summer. It is highly prevalent in Tamil Nadu and Karnataka. It is also responsible for fruit rot in brinjal.

Causal organisms : *Fusarium solani*

Symptoms

- It attacks seedlings as well as matured plants.
- Leaves become chlorotic, flaccid and hang down.
- Vascular brown streaks are seen in the longitudinal section of the stem.
- Roots exhibit rotting.
- Whitish fungal growth seen on the basal stem and roots.



Pathogen character : Macroconidia, Microconidia, Chlamydospores

Favourable conditions: Root-knot nematode predisposes the plant to disease incidence.

Mode of survival: In crop debris and soil as chlamydospores.

Primary spread: Soil and diseased seedlings.

Secondary spread: Irrigation water

Management

- Treat the seeds with *Trichoderma viride* @ 4g/kg or *Pseudomonas fluorescens* @ 10g/kg or carbendazim @ 2g/kg.
- Soil application of *Trichoderma viride* or *Pseudomonas fluorescens* @ 2.5 kg + 50 kg FYM/ ha on 30 DAP in the main field.
- Remove and destroy infected plants and spot drench with carbendazim @ 0.1%.

Crop : Brinjal

Disease name : *Verticillium* wilt

Introduction

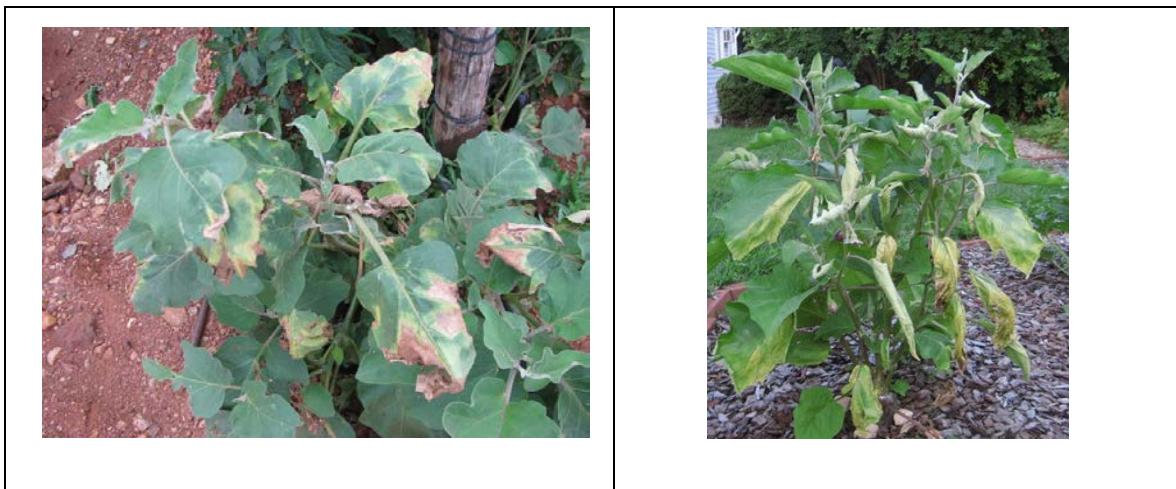
Verticillium wilt of brinjal causes economic losses in crops in temperate region of the world. The disease was first reported in North America in the year 1940. The disease cause yield loss of upto 10 %. The strains of this pathogen have been classified into several vegetative compatibility group (VCG) based on their genetic characters.

Causal organisms : *Verticillium dahliae*

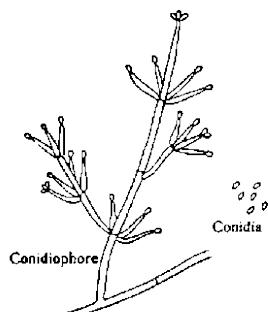
Symptoms

- The disease attacks the young plants as well as mature plants.

- The leaves show presence of irregularly scattered necrotic pale yellow spots which later coalesce resulting in complete wilting of leaves.
- Drying of branches followed by wilting and collapse of plant.
- The longitudinal section of the root shows a characteristic dark brown discoloration on the xylem vessels.



Pathogen character : The mycelium is hyaline, septate, and multinucleate. Conidia are ovoid or ellipsoid and usually single-celled. They are borne on phialides, which are specialized hyphae produced in a whorl around each conidiophore. *Verticillium* is named for this verticillate (=whorled) arrangement of the phialides on the conidiophore.



Favourable conditions: Temperature of 19-21°C and other crop hosts.

Mode of survival: Microsclerotia in soil.

Primary spread: Soil-borne inoculum.

Secondary spread: Irrigation water

Management

- Follow crop rotation avoiding bhendi, tomato, cotton and potato.
- Drench the soil in root region of plants with carbendazim or benomyl @0.1%.

- Treat the seeds with *Trichoderma viride* @4g/kg or *Pseudomonas fluorescens* @ 10g/kg or carbendazim @ 2g/kg.
- Follow soil application of *Trichoderma viride* or *Pseudomonas fluorescens* @ 2.5 kg + FYM 50 kg of /ha.

Crop : Brinjal

Disease name : *Alternaria* leaf spot

Introduction

It is one of the important diseases of brinjal which infects the leaf. The disease was first observed in Guntur district during 2000. The disease causes an yield loss of upto 25%. The disease transmission was more during rabi than kharif.

Causal organisms : *Alternaria solani*

Symptoms

- Irregular, brown and necrotic spots with concentric rings appear on leaves.
- Leaves dry due to bigger necrotic patches and then fall down prematurely.
- Spots on fruits are dark brown, sunken, turn yellow and cause fruit to drop.



www.apsnet.org

Pathogen character : It is a polycyclic necrotrophic pathogen, produces dark colored, multinucleate muriform and beaked conidia which arise singly in simple conidiophore.

Favourable conditions: Temperature of 19-21°C and other crop hosts.

Mode of survival: In seed and soil (Seed-borne and soil-borne).

Primary spread: From infected seeds and plant debris.

Secondary spread: Air-borne conidia

Favourable conditions:

Management

- Use disease free seeds.
- Remove and destroy infected plant debris.
- Spray mancozeb @0.2 %.

Crop : Brinjal

Disease name : *Cercospora* leaf spot

Introduction

One of the most common fungal disease of egg plant in home and community gardens. Major problem of large scale grower and backyard gardeners. In India it is reported by Chupp in 1953. The pathogen attack the leaves alone causing an yield loss upto 10-20 %. This disease weakens plants and reduces yield by causing premature defoliation of infected leaves.

Causal organisms : *Cercospora melongenae*

Symptoms

- Large irregular or circular, brown spots with grey centre are seen on leaves.
- Spots are also seen on fruits.

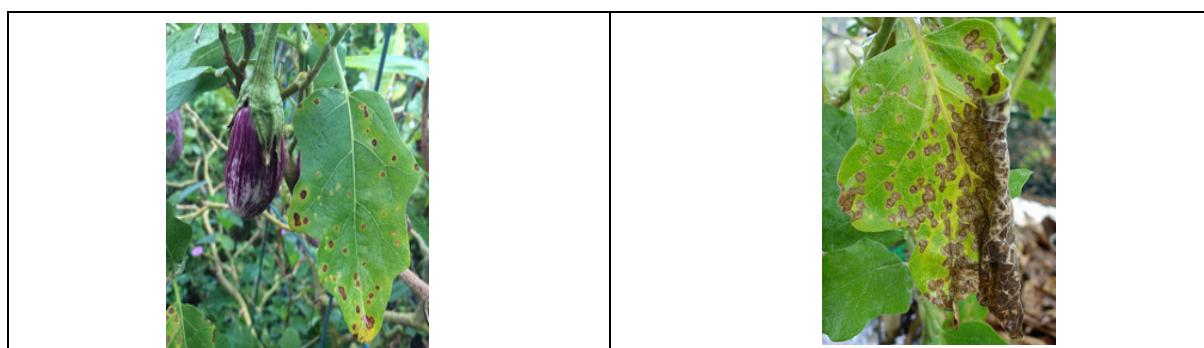
Pathogen character : Conidiophores appear in fascicles (clusters) of 3 to 12. They are pale brown, occasionally septate, and unbranched, and they bear hyaline, mildly curved conidia.

Favourable conditions: Leaf wetness and high relative humidity.

Mode of survival: In infected plant debris in soil upto one year and seed.

Primary spread: From infected plant debris and seed.

Secondary spread: Air-borne conidia



Management

- Use disease free seeds
- Remove and destroy affected leaves
- Spray mancozeb or difolatan @ 0.2%.

Crop : Brinjal

Disease name : Rust

Introduction

In India the disease was first reported from Gujarat in 1914. After then it has been reported from almost all the states wherever brinjal is grown. It causes around 30-35% yield losses in severe condition. The crop is an alternate host for cumbu rust.

Causal organisms : *Puccinia substriata* var. *penicillariae*

Symptoms

- Pycnial and aelial stages of pearl millet rust are formed on leaves of brinjal (alternate host).
- Upper surface of leaf becomes depressed and the corresponding lower surface is convex.
- Pycnia are formed as orange yellow pustules on the upper leaf surface.
- Aelial cups are formed in groups on the lower surface of leaves.
- Severe infection of rust causes drying of leaves.



(<https://blog.plantwise.org/>)

Pathogen character : It is a macrocyclic heterocious with uredial and telial stage on *Pennisetum* spp. and spermogonial and aelial stages on *Solanum* spp.

Favourable conditions: Availability of pearl millet crop in nearby fields.

Mode of survival and spread: Primary host (pearl millet)

Management

- Spray mancozeb or difolatan 0.2% or tridemorph 0.1%.

Crop : Brinjal

Disease name : *Phomopsis* blight and fruit rot

Introduction

The disease is severe in tropical and sub tropical area of the world. The pathogen attacks foliage and fruits, but the latter phase is more destructive. The disease was first described by halsted in 1892. In India it was first reported in Gujarat in 1914. It causes yield loss 30-35%. The disease occurred almost every year in the month of March and October.

Causal organisms : Sexual stage : *Diaporthe vexans* (Sacc. & Syd.) Gratz.

Asexual stage : *Phomopsis vexans* (Sacc. & Syd.).

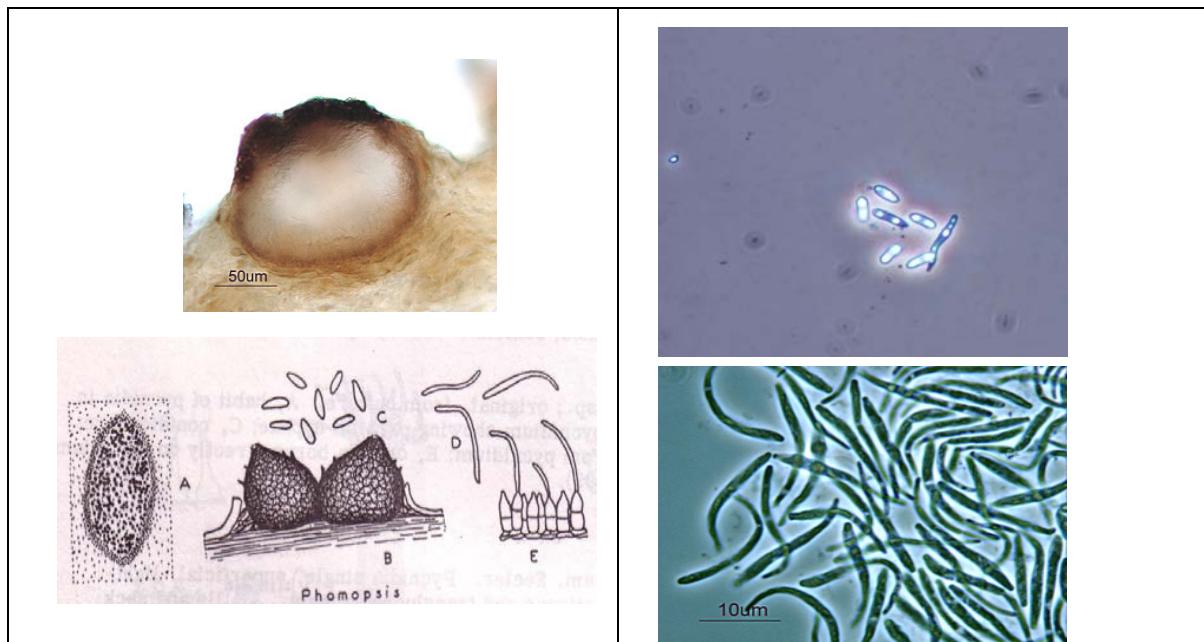
Symptoms

- The pathogen infects the crop from seedling to harvest.
- On young seedlings, brown or dark sunken lesions appear on stem slightly above the soil surface which results in collapse and death (seedling infection).
- Circular or irregular, grey to brown spots with light coloured centre embedded with pycnidia appear on leaves.
- Affected leaves turn yellow and drop prematurely (leaf infection).
- Stem lesions appear at basal part near nodal region as grey dry rot with constriction.
- The infected branch dries giving a partial wilting symptom (stem infection).
- The fruit is attacked while in the plant.
- Pale, sunken, oval spots on fruits enlarge and cover the entire fruit at the end. Internal portion of fruits rots.
- Diseased fruits are unmarketable.



Pathogen character : The mycelium was hyaline and septate; the conidiophores (phialides) within the pycnidium were hyaline, simple, or septate. Two types of conidia (alpha and beta) were observed. Alpha conidia were hyaline, single celled, biguttulate

and subcylindrical ($4.1\text{--}6.5 \times 1.2\text{--}1.9 \mu\text{m}$). Beta conidia were filiform, curved, hyaline and septate ($6.2\text{--}7.6 \times 0.5\text{--}0.8 \mu\text{m}$).



Favourable conditions: Temperature of $21\text{--}32^\circ\text{C}$ and hot and wet weather.

Mode of survival: In plant debris, soil and seed.

Primary spread: Plant debris and seed.

Secondary spread: Rain splashes, insects, contaminated equipment and air.

Management

- Follow crop rotation with cereals.
- Use disease free seeds/ tolerant varieties.
- Treat seeds with carbendazim @ 2g/kg or thiram @ 4g/kg or hot water @ 50°C for 30 min.
- Plant disease-free seedlings
- Collect and burn infected crop residue and fallen mummified fruits.
- Spray with mancozeb @ 0.2% in the nursery.
- Spray difolatan @ 0.2% or carbendazim @ 0.1%

Disease name : Sclerotium collar rot

Introduction

Collar rot caused by *Sclerotium rolfsii* Sacc is becoming one of the major threats both under nursery and field cultivated brinjal crop. The pathogen has been reported to inflict the fruit yield losses upto 90-100%. The fungus can overwinter as mycelium in infected tissues or plant debris or as sclerotia near the soil surface or buried in soil.

Sclerotia disseminate by cultural practices with infected soil and contaminated tools, infected seedlings, water, wind and possibly as concomitant contaminants along with seeds. The disease was first reported in India by Rao (1969) in Vijayawada district on Andhra Pradesh.

Causal organisms : *Sclerotium rolfsii* Sacc. [Sexual stage: *Athelia rolfsii* (Curzi)].

Symptoms

- Necrosis of stem tissues is seen near the soil line.
- White, cottony and silvery mycelial growth is visible on the affected portion.
- Fungal growth below the soil surface is also common.
- White to light brown mustard like sclerotia is observed on the infected collar region and soil.
- Progressive drooping and wilting of the entire plant is observed.



Pathogen character : It is a soil living saprophytic pathogen, produces ribbon like hyphae with clamp connections. Basidia are club-shaped, bearing four smooth, ellipsoid basidiospores. Small, brownish sclerotia (hyphal propagules) are also formed.

Favourable conditions: High soil moisture, ill drained soil, dense canopy and several cultivated crop hosts and weeds.

Mode of survival: In plant debris as sclerotia.

Primary spread: From soil-borne sclerotia.

Secondary spread: Irrigation water.

Management

- Follow crop rotation with cereals.
- Removal of weed hosts.
- Deep summer ploughing

- Use disease free seeds/tolerant varieties.
- Apply green manure followed by *Trichoderma viride* or *Pseudomonas fluorescens* @ 2.5 kg/ha in soil.
- Avoid closer spacing and maintain proper aeration and sunlight near the collar region.
- Spot drench carbendazim @ 0.1% or copper oxychloride @ 0.25%.

Disease name : Bacterial wilt

Introduction

A soil bacterium formerly known as *Pseudomonas solanacearum*. First report of bacterial wilt of brinjal is from Egypt in 2008. The severely infected fields had 80-100% yield losses. The pathogen has five different races, each infecting different plant species. It leads to yield loss upto 30-100%.

Causal organisms : *Ralstonia solanacearum* (Yubucchi et al.)

[Syn. *Pseudomonas solanacearum* (Smith), *Burkholderia solanacearum* (Smith)].

Symptoms

- Wilting of foliage and sudden death of plant.
- The vascular browning of stem followed by rotting of roots and rootlets.



Pathogen character: The bacterium is rod-shaped, gram negative, lophotrichus bacteria (more than one polar flagellum), multiplication occurs by bacterial fission.

Favourable conditions: High soil moisture, acidic pH, intercultural wounds and nematode injury.

Mode of survival: In soil

Primary spread: Soil

Secondary spread: Through irrigation water

Management

- Follow deep summer ploughing.
- Grow disease resistant varieties.
- Follow crop rotation avoiding bhendi, tomato and potato.
- Soak seeds in a solution of streptocycline @ 25 ppm (2.5g/100 litres of water) for 30 min before sowing.
- Avoid water stagnation in the field.
- Remove and destroy affected plant parts.

Disease name : Little leaf

Introduction

It is one of the destructive diseases in Brinjal. In India it is reported from Coimbatore in 1838. Loss of upto 100% is noticed in diseased plants. *Candidatus phytoplasma asteris* (16SrI group) associated with little leaf brinjal was first reported in India by Thomas and Krishnaswami.

Causal organisms : *Candidatus Phytoplasma*

Symptoms

- The infected plants exhibit general chlorosis and reduction in plant height.
- Leaf size is abnormally reduced.
- Axillary buds are induced to produce many, small narrow and thin leaves.
- Reduction in internodal length leads to clustering and overcrowding of leaves giving bushy appearance.
- Flowers are phylloid and fruits very rare.
- Early infected plants are completely sterile.



Pathogen character : *Candidatus Phytoplasma trifolii*

Favourable conditions: Susceptible varieties, presence of other crop hosts (Potato, Tomato, chillies, *Datura*) and weeds and abundance of vector.

Mode of survival: Survives in weed hosts (*Argemone mexicana*, *Datura metel*, *Physalis minima*, *Solanum indicum*, *Solanum trilobatum*, *Catharanthus roseus* and *Withania somnifera*).

Primary spread: Weed hosts.

Secondary spread: Insect vector, leafhopper (*Hishimonas phycitis*). Acquisition feeding period 1 hr, Latent period in the vector 16 days, Inoculation feeding period <1 hr, No transovarial transmission.

Management

- Eradicate solanaceous and other weed hosts.
- Rogue out infected plants
- Spray dimethoate or methyl demeton @ 2ml/lit to control insect vector, leafhoppers.
- Spray tetracycline @ 100 ppm to suppress multiplication of phytoplasma.
- Grow moderately resistant varieties viz., Arka Sheel, Banara Giant, Pusa Purple Cluster.

Disease name : Mosaic

Causal organisms : Potato virus Y (PVY) Tobacco mosaic virus (TMV)

Symptoms

- Mosaic mottling of leaves and stunting of plants
- 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.
- Severely infected leaves become small and misshapen.
- Plants infected early remain stunted.



TNAU Agriportal



Pathogen character : ssRNA virus *Potato virus Y (PVY)* *Tobacco mosaic virus (TMV)*

Favourable conditions: Susceptible varieties, Mode of survival: Survives in weed hosts (*Solanum nigrum* and *S. Xanthocarpum*), presence of other crop hosts (cucurbits, legumes, pepper, tobacco, tomato), Presence of vector.

Primary spread: Weed hosts.

Secondary spread: Insect vector, PVY – Aphids (*Aphis gossypii*, *Myzus persicae*)

Management

- Field sanitation. Nursery application of neem cake @ 1.0 kg/sq.m
- Growing of maize as border crop
- Eradicate solanaceous and other weed hosts.
- Foliar spraying of neem oil formulation @ 3 ml/lit
- Installation of yellow sticky traps @ 12/ha
- Rogue out infected plants. Spray dimethoate or methyl demeton @2ml/lit to control insect vector

Crop : Bhendi

Disease name : Powdery mildew

Introduction

Bhendi powdery mildew is an important disease which affects the quality and marketability of Bhendi. It has been identified in Australia, Brazil, Greece, India, Israel, Japan, Sudan, and Taiwan (Farr and Rossman 2018). The first report of powdery mildew caused by *P. xanthii* on okra was from Korea.

Causal organisms : *Golovinomyces cichoracearum*

Symptoms

- The disease is found mainly on the older leaves and stems of plants.
- White powdery patches appear on the upper surface of leaves covering the entire leaf.
- Fungal growth changes brown and the affected leaves dry and fall.
- Infection on stem and fruit is also common.



Pathogen character : Ectophytic mycelium producing chain of single celled, hyaline barrel shaped conidia arranged in a basipetal succession (catenate). Sexual fruiting body is chasmothecium which bears ascospores.



Favourable conditions:

Mode of survival: As

Primary spread: From

Secondary spread: Air-borne conidia

Management

- Spray wettable sulphur or dinocap @ 0.2% tridemorph or carbendazim @ 0.1%.

Cool and humid weather.

cleistothecia in plant debris
infected plant debris

Disease name : *Cercospora* leaf spot

Introduction

This disease was recorded first time in 1893 in Jamaica. *Cercospora* leaf spot (CLS), are caused by different *Cercospora* spp. viz., *Cercospora abelmoschi*, *Cercospora malayensis*, and *Cercospora hibisci*. This disease is considered to be the one of the important diseases that can appear at any growth stage of the plant. In India, two species of *Cercospora*, produce leaf spots on bhendi which cause defoliation during humid seasons. The disease incited by *C. abelmoschi* becomes more severe in Southern transition zone of Karnataka.

Causal organisms : *Cercospora abelmoschi*, *Cercospora malayensis*, *Cercospora hibisci*

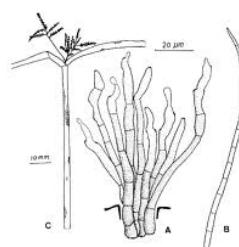
Symptoms

- Irregular, brown spots with grey centre and dark coloured margins appear on leaves.
- Severely affected leaves fall off prematurely.



Pathogen character : Conidiophores - Brown, multiseptate, branched and irregular.

Conidia - Cylindric, brown, straight to curved.



Favourable conditions : Temperature - 25-29°C.

Mode of survival : In crop refuse.

Primary spread : From infected crop debris.

Secondary spread : Air-borne conidia.

Management

- Remove and destroy the infected plant parts.
- Spray copper oxychloride @ 0.25 % or Bordeaux mixture @ 1.0 % or zineb or captan or difolatan @ 0.2 % or carbendazim @ 0.1 %.

Disease name : Wilt

Introduction

Fusarium wilt is one of the most important diseases which is being widely distributed throughout the tropical and subtropical areas of the world. The first report of *Fusarium oxysporum f.sp. vasinfectum* in Brazil occurred in 1935. The genus of fusarium includes saprophytic species, as well as plant pathogens. The disease was responsible for significant yield losses in many areas where the crop is grown.

Causal organisms : *Fusarium oxysporum f.sp. vasinfectum*

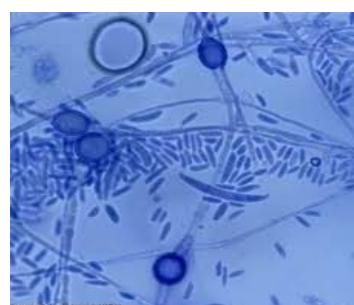
Symptoms

- Yellowing, and stunting of plants
- Wilting and rolling of leaves
- Dark brown vascular discolouration

Pathogen:
microconidia,
and
chlamydospores



Produces
macroconidia



Favourable conditions: Susceptible varieties, weed hosts and abundance of insect vector.

Mode of survival : In weed hosts.

Primary spread : From weed hosts and volunteer plants.

Secondary spread : By insect vector, whitefly (*Bemisia tabaci*)

Management

- Follow deep summer ploughing.
- Follow crop rotation with non-solanaceous crops (cereals).
- Remove infected plants and plant debris.
- Treat the seeds with carbendazim @ 2g/kg or *Trichoderma viride* @ 4g/kg or *Pseudomonas fluorescens* @ 10g/kg.
- Soil application of *Pseudomonas fluorescens* @ 2.5 kg/ha + 50 kg of FYM.
- Spot drenching with carbendazim @ 0.1%.

Disease name : Yellow vein mosaic / Vein clearing

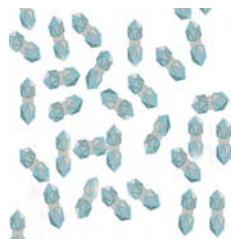
Introduction

Bhendi yellow vein mosaic was first reported in okra plants in 1924 in India and Sri Lanka. This is the most important, serious and destructive viral disease in bhendi. The disease infects all the stages of crop growth which severely reduces growth and yield.

Causal organisms : *Bhendi yellow vein mosaic virus*

Symptoms

- The green colour of main and lateral veins is bleached.
- The veins and veinlets exhibit yellow network (vein clearing).
- Veins are thickened.
- The interveinal portions exhibit small portions of green colour.
- As the disease develops, these green portions turn chlorotic and finally the entire leaves become yellow and papery
- The affected plant is severely stunted with smaller leaves and shortened internodes.
- Fruits are smaller, malformed, thick, fibrous, hard and yellow and unfit for consumption.



Pathogen character : Spherical bipartite particles with ssDNA as genome
Favourable conditions: Susceptible varieties, weed hosts and abundance of insect vector.

Causal agent: *Bhendi yellow vein mosaic virus* (BYVMV).

Mode of survival: In weed hosts.

Primary spread: From weed hosts and volunteer plants.

Secondary spread: By insect vector, whitefly (*Bemisia tabaci*)

Management

- Grow resistant cultivars (TNAU Bhendi hybrid CO4, Arka Abhay)
- Rogue out affected plants and weed hosts
- Raise barrier crops of sorghum, pearl millet, maize with 5-6 rows before raising bhendi.
- Treat the seeds with imidacloprid @ 5 ml/kg
- Install yellow sticky traps
- Spray dimethoate or methyl demeton @ 2ml/lit or Thiamethoxam 25%WG @ 10g/lit to control insect vector.

Disease name : Enation leaf curl

Introduction

Bhendi enation is one of destructive viral disease in bhendi in recent years which cause complete failure of the crop. The disease infects all the stages of crop growth which severely reduces growth and yield. The OELCV incidence has reached serious proportions in recent years both in Northern India (Sanwal *et al.*, 2014) and Southern India (Sayed *et al.*, 2014).

Causal organisms : *Bhendi enation leaf curl virus*

Symptoms

- Small pin-head enations on the under surface of leaves.
- Warty and rough texture of leaves.
- Leaves curl upwards.
- Plants show twisting of the stem and lateral branches
- Leaves becoming thick and leathery
- Fruits are unfit for consumption.



Pathogen character : Single stranded DNA virus with geminate particles

Favourable conditions: Susceptible varieties, weed hosts and abundance of insect vector.

Mode of survival: In weed hosts.

Primary spread: From weed hosts and volunteer plants.

Secondary spread: By insect vector, whitefly (*Bemisia tabaci*)

Management

- Grow resistant cultivars
- Rogue out affected plants and weed hosts
- Raise barrier crops of sorghum, pearl millet, maize with 5-6 rows before raising bhendi.
- Spray dimethoate or methyl demeton @ 2ml/lit or Thiamethoxam 25%WG @ 10g/lit to control insect vector.

11. Etiology, symptoms, mode of spread, survival, epidemiology and integrated management of Diseases of crucifers

1. CLUB ROOT OF CABBAGE/ FINGER AND TOE DISEASE: *Plasmodiophora brassicae*

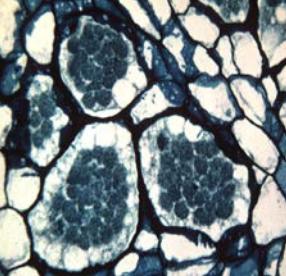
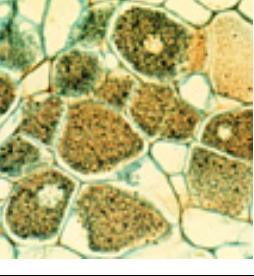
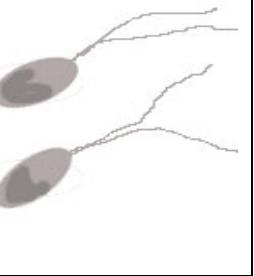
Symptoms:

- Leaves pale green to yellowish
- Plants stunted
- Fail to produce marketable heads
- Small or large spindle like, spherical, knobby or club shaped swellings on the roots and root lets
- Diseased plants later die

| | |
|--|---|
|  |  |
| Club root – Hypertrophy and Hyperplasia | Club root initial stage |

Pathogen: It is a Protozoa. Plasmodium (naked protoplasm) produces primary and secondary zoospores.

zoospores are biflagellate. Sexual spores are resting spores

| | | |
|---|--|---|
|  |  |  |
| Plasmodium | | Zoospore |

Mode of spread:

Primary: Resting spores; Through farming implements, Surface flood water and from infected seedlings.

Manure from cattle fed on diseased root crops.

Secondary: Zoospores

Favourable conditions

Low lying areas, ill drained soils with a soil temperature of 15-25°C.

Acid soils are highly favourable for the pathogen.

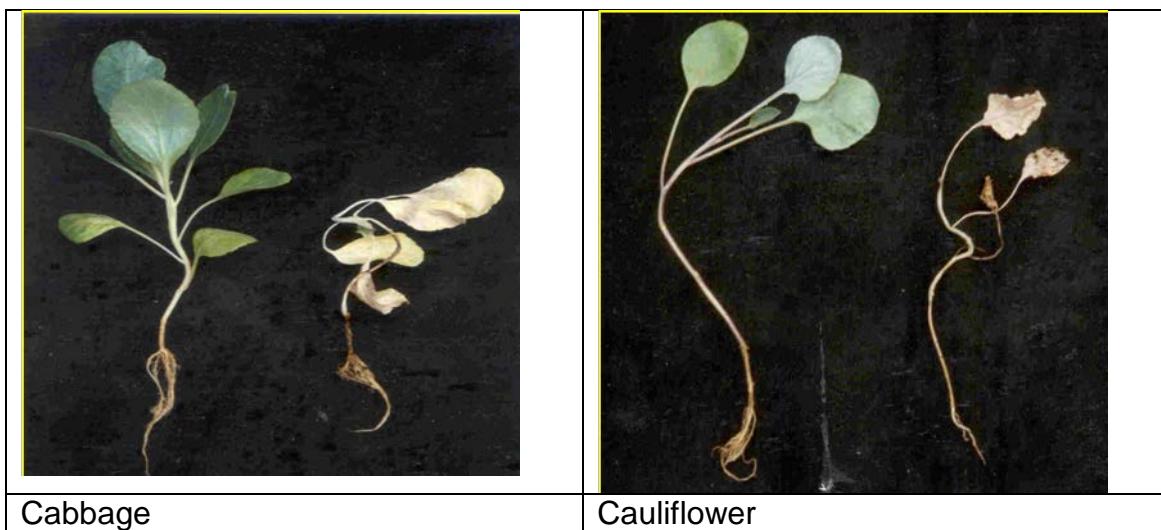
Management

- Maintain a soil pH of 7.2 by liming (Field with pH 5.0 needs approximately 2.5 t of lime/ha). Finally ground lime alters the pH more quickly than coarse granules
- Follow crop rotation with non cruciferous crops for 7 years
- Apply formalin 2 % at 10 lit/ sq.m of seed bed
- Treat the seeds with *Pseudomonas fluorescens* @10g/kg followed by seedling dip of *Pseudomonas fluorescens* at 5 g/ lit and soil application of *Pseudomonas fluorescens* at 2.5 kg/ha + 50 kg FYM before planting or carbendazim 2g/kg followed by seedling dip in carbendazim solution at 2 g/lit for 20 min
- Improve drainage facilities and disease free seedlings
- Avoid moving infected transplants and/or infested soil on farm equipment to clean fields
- Remove brassica weeds
- Clean and disinfect all machinery before moving it from infested to non infested land
- Spot drench with carbendazim at 0.1% or copper oxychloride 0.25%

2. DAMPING OFF/WIRE STEM OF CABBAGE: *Thanetophorus (Rhizoctonia) solani*

Symptoms:

- Serious in nursery of cabbage
- Causes wire stem in seedlings
- Tissues water soaked and toppling of seedlings
- Damping off in seedling, wire stem in seedlings (plants bend or twisted without breaking in cabbage)
- Bottom rot, head rot and root rot in older plants





Head rot of Cabbage

Pathogen: Septate mycelium. Sclerotia are irregular brown to black. Produces terminal and intercalary chlamydospores. Basidia with basidiospores are produced

Favourable condition: cool, cloudy weather, high humidity, wet and compact soil and overcrowding.

Mode of spread: Primary: Sclerotia and Basidiospores; Secondary: Water borne sclerotia and chlamydospores

Management :

Adopt raised seedbed method. Treat the seeds with *Trichoderma asperellum* 4g/kg

Avoid excessive irrigation to reduce humidity around the plants

Discard the diseased seedlings before transplanting

Soil drench with carbendazim 0.1 % or copper oxychloride 0.25%

3. BLACK LEG OF CABBAGE: *Phoma lingam*

Symptoms:

- Fungus attacks the crop at seedling or any stage of crop growth
- Oral, depressed, height brown canker appear near the base of stem
- Canker enlarges and girdles the stem
- Attacked roots show dark brown cankers
- Elliptical lesions appear on seed stalk and pods



Stem and leaf symptom



Symptom

Pathogen: Septate mycelium, produces pycnidia. Ascocarps bear asci with ascospores

Favourale condition: continued drizzling and wet weather

Mode of spread: Primary: Ascospores; Secondary: Water borne conidia

Management

Follow deep summer ploughing to incorporate crop debris promptly after harvest to hasten decay

Practice four year rotation in seedbeds and fileds

Use certified/disease free seeds

Treat seeds with hot water as it spreads mainly through seeds

Dress the seeds with imidacloprid+metalaxyl+carbendazim based products

Rouge out and destroy diseased plants from seedbeds

Improve soil drainage and air circulation. Avoid working in the fields when wet

4. CABBAGE YELLOWS/FUSARIUM WILT OF CABBAGE: *Fusarium oxysporum f.sp. conglutinans*

Symptoms:

- Plants show uniform yellowing
- Stem and leaves curl laterally
- Later leaves turn brown, brittle and die
- Browning of vascular system seen

Pathogen: Produces microconidia, macroconidia, and chlamydospores

Favourable condition: soil moisture, acidic pH, root wounds and the temperature about 27 °C

Mode of spread:

Primary : Chlamydospores

Secondary : Water borne conidia

Management

- Use only certified, disease free seeds/transplants
- Growing seedlings in seed bed disinfested by steam or soil fumigant
- Grow resistant varieties. Early sowing of cabbage
- Follow crop rotation with crops like lettuce, chilli, peas, tomato, sweetpotato or cotton
- Remove and destroy infected plant debris.

5. WHITE BLISTERS OR RUST OF RADISH: *Albugo candida*

Host: Cabbage, mustard, radish, turnip

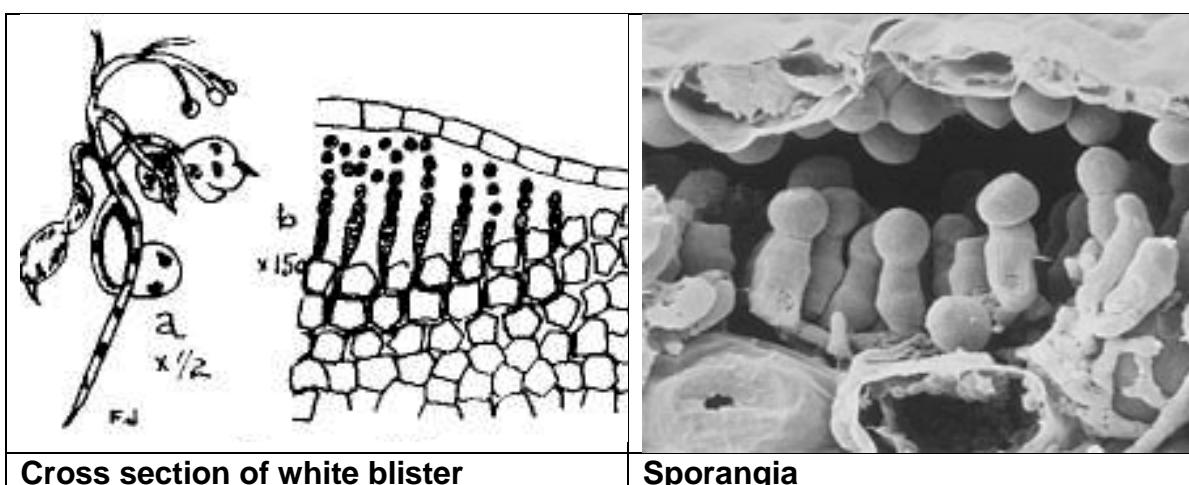
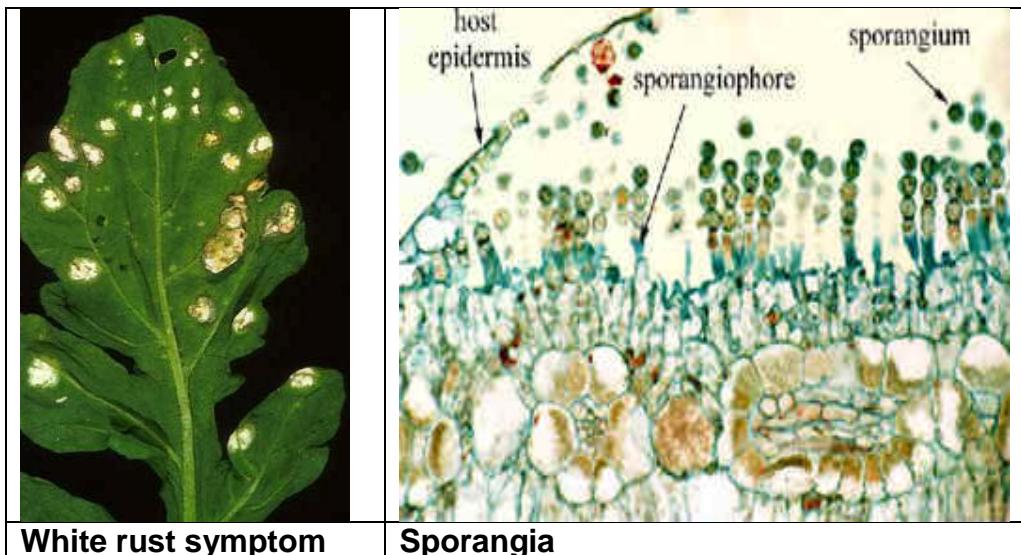
Two types of infection: Local and systemic

Symptoms:

- **Local infection:** isolated pustules or sori develop in leaves and stems. Pustules merge to form larger patches. Host epidermis rupture after maturity of pustules
- **Systemic infection:** When young stems and flowering parts are infected it becomes systemic. Stimulates hypertrophy and hyperplasia
- Results in enlarged and variously distorted organ mostly flower parts sepals become enlarged to several times than the normal sepals. Petals enlarge and become

green pistils and anthers are distorted.

- Seed development is arrested.



Pathogen: Pathogen produces sporangia. Sporangia are spherical / hyaline in nature contains 4-8 zoospores

Sexual spore is tuberculate oospore

Favourable condition: Moist cool weather and a thin film of water on the leaves

Mode of spread:

Primary : Oospore

Secondary : Sporangia and Zoospores

Management

Reove and destroy infected plant debris

Treat the seeds in hot water at 52 C fr 20 min or metalaxly 4-6g/kg

Remove cruciferous weeds in the filed

Spray Bordeaux mixtre 1.0% or mancozeb or metalaxyl+mancozeb0.2%

6. DOWNY MILDEW: *Peronospora parasitica*

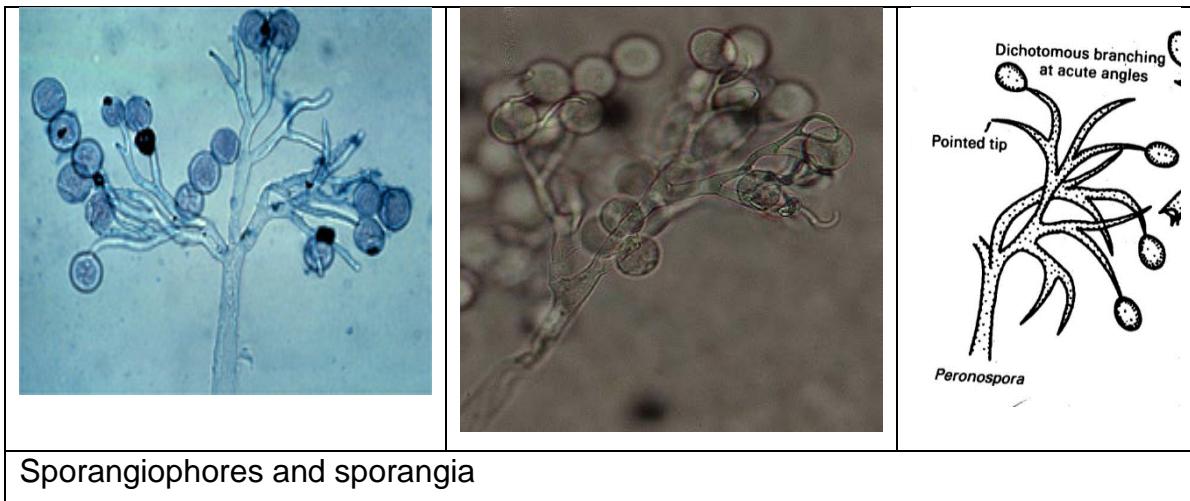
Symptoms:

- It affects seedlings and mature plants.
- **On seedlings:** The entire plant becomes covered in a whitish coating of the pathogen and dies rapidly
- **On larger plants:** Yellowish patches of discolouration on the upper surfaces of leaves, often angular and limited by veins. On the corresponding lower surface is a fuzzy whitish outgrowth of the pathogen. Eventually, the affected tissues die, shrivel and may drop out
- **On cauliflower curds and radishes:** Leaf damage may be accompanied by internal browning of the cauliflower curds and radish flesh.
- These infections in turn release spores and spread to other brassicas by wind and rain.

| | | |
|--|---|--|
|  |  | |
| Downy mildew symptom on lower surface of leaf | Downy mildew symptom on upper surface of leaf | |

| | | |
|---|--|--|
|  | | |
| Cabbage head symptoms | | |

Pathogen: The sporangiophores are dichotomously branched at acute angles and taper into gracefully curved pointed tips on which sporangia are borne.



Sporangiophores and sporangia

Favourable condition

Cool moist conditions, high humidity, fog, drizzling rains and heavy dew.
Optimum temperature for the conidia is 8 to 10°C
For conidial germination 8 to 12°C and for host penetration 16°C, rapid development 15 to 20°C.

Mode of spread:

Primary : Oospore

Secondary : Sporangia and Zoospores

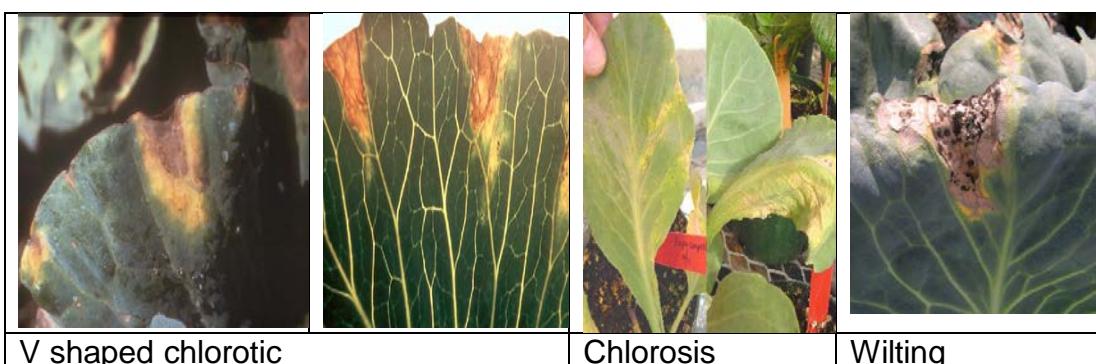
Management

- Follow crop rotation with non cruciferous crops
- Grow tolerant varieties. Treat seeds with metalaxyl 6g/kg
- Remove cruciferous weeds in the field
- Spray metalaxyl+mancozeb or metalaxyl 0.4% or mancozeb0.2%

7. BLACK ROT: *Xanthomonas campestris* pv. *campestris*

Symptoms:

- Infected young plants killed. Plants stunted
- Cotyledons turn yellow to black, hang down and drop off prematurely
- Leaves yellow with blackened veins and vein lets .Become necrotic and brittle
- V shaped chlorotic to yellow lesions develop from the leaf margin
- Disease spreads onto the roots
- Vascular bundles become black and the fleshy tissue break down
- Cabbage heads and cauliflower curds are infected and discoloured



V shaped chlorotic

Chlorosis

Wilting

Pathogen: Gram negative, rod shaped bacterium with single polar flagellum

Mode of spread: Wind driven rain splashes carry bacteria from infected to healthy leaves, Twigs and fruits

Favourable condition

Optimum temperature 26.5 to 30°C and frequent rain

Management

Grow tolerant varieties. Follow atleast four year rotation with cruciferous crops

Treat the seeds in hot water at 50 C for 30 min or with streptocycline 100 p[pm for 30 min kills bothinternally and externally seed borne bacterium

Grow seedlings on raised beds in non infested area or frequently change th nursery site and get disease free seedlings

Avoid low, wet soils with poor soil drainage. Keep the field fallow for 2 years

Raise intercrops like mungbean or urdbean as mulches to reduce the spread of pathogen by rain splashes

Remove and destroy diseased plants and weed hosts

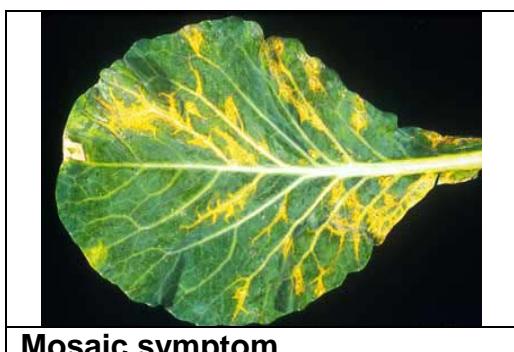
Drench the nursery soil with formalin 0.5% ro apply bleaching powder 12.5 kg/ha

Spray copper oxychloride 2.5 g/lt +streptocycline at 100 ppm or Kasugamycin 0.2%

8. CAULIFLOWER MOSAIC: Cauliflower mosaic virus, Double strand DNA virus

Symptoms:

- Leaves are mottled with a pattern of light and dark green
- Plants are stunted, central leaves smaller
- Mild infection leads to production of a small and poor quality heads
- Infected plants die



Mosaic symptom

Transmission: Aphids - *Brevicoryne brassicae*

Other disease

- Whiptail disease: Caused by molybdenum deficiency

Management

Use resistant /tolerant varieties. Establish seed beds away from susceoptile crops

Grow seedlings in insect proof condition

Discard seedlings with mosaic symptoms before planting

Early rouging and destruction of diseases plants. Use mild strains of CaMV

Control of aphid vectors through insecticides like Methyl demeton 0.1% at an appropriate stage.

Reference Books or links

Arjunan, G. Dinakaran, D and Parthasarathy,S. 2018. Diseases of Horticultural corps.

12. Etiology, symptoms, mode of spread, survival, epidemiology and integrated management of Diseases of sweet potato and beans

SWEET POTATO

Crop : Sweet potato

Disease name : *Cercospora* leaf spot

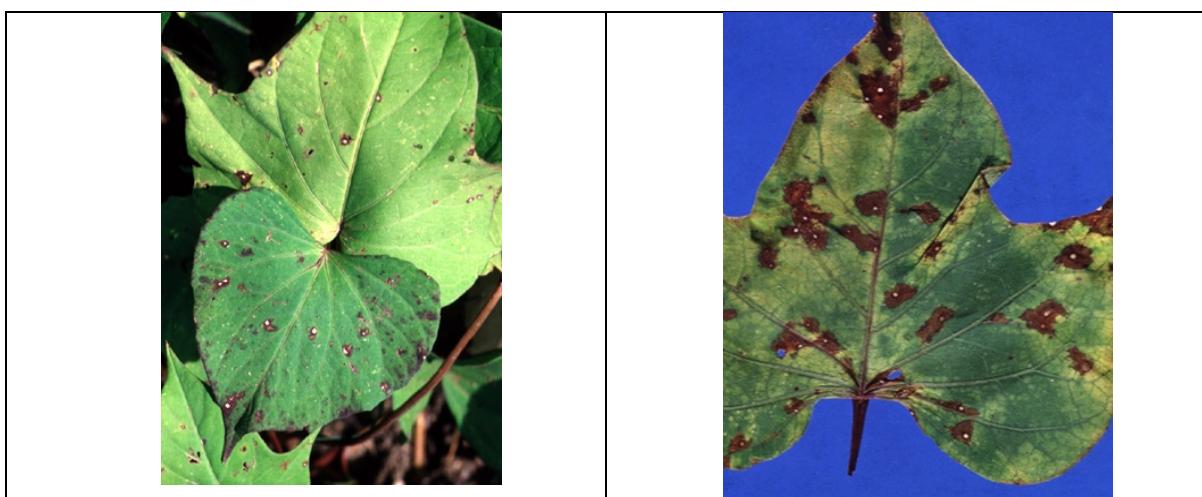
Introduction

The primary host of *C. bataticola* is *Ipomoea batatas* (sweetpotato). It is most prevalent in the hot and humid tropics and is seldom observed during the dry season. The disease is commonly found throughout the tropics, mainly in the Caribbean and South and Central America. It has also been noted in India, Japan, Italia, Netherlands, Antilles and USA.

Causal organisms : *Cercospora ipomoeae* G. Winter; *Pseudocercospora timorensis* (Cooke) Deighton.

Symptoms

- Light green spots initially appear on both surfaces of the leaf.
- They gradually increase in size and turn brown with yellow green border.
- Later the centre becomes pale brown to whitish grey and the border is brown to purple black. The veins limit the lesions.
- The lesions increase in size, coalesce and blight the leaf.
- Severely blighted leaves turn yellow leading to defoliation.
- More spots are found on older leaves.
- Fruiting structures of the fungus are seen at the centre of the spots, especially after rainfall.



Pathogen character : Conidiophores are septate, dark brown, geniculate. Conidia are acicular to obclavate, multiseptate, hyaline, straight curved.

Mode of survival: In weed hosts and infected plant debris.

Primary spread: From infected crop debris and weeds.

Secondary spread: Air-borne conidia and by splashing rains.

Favourable conditions: Hot humid climate with relative humidity of 90-95% and intermittent rain.

Management

- Remove and destroy severely diseased and fallen leaves.
- Spray zineb or mancozeb @0.2%

Disease name : Black rot

Introduction

Black rot of sweetpotato is caused by the ascomycete fungus *Ceratocystis fimbriata* which cause significant losses in storage. The black rot fungus and other secondary fungi induce the sweetpotato to produce toxins such as ipomeamarone and ipomeanol that have caused serious poisoning and death of cattle. Black rot can develop over a wide range of temperatures, and soil moisture.

Causal organisms : *Certatocystis fimbriata* Ellis & Halst.

Symptoms

- This is mainly a storage disease.
- On underground roots, the first symptom appears as small circular dark brown, slightly sunken spots.
- These spots enlarge upto 5 cm in dia during transit, storage and marketing.
- Characteristic small, black fungal structures (perithecia) with long neck protrude in the spotted area and they appear to the naked eye as dark bristles.
- Spots enlarge and appear greenish black to black when wet and greyish black when dry and coalesce to cover the entire root.
- If secondary fungi or bacteria invade the tissue, the flesh beneath the spot turns black and go deep and this blackened area extend to the centre of the root.
- Tissue near the discoloured area has bitter taste but fruity odour and the entire root rots. (The bitter taste is due to ipomeamarone (phytoalexin) produced by the storage roots in response to *C. fimbriata* infection while the fruity odour is from the volatile compound, amyl acetate produced after infection)
- The disease is serious on young sprouts and adult plants also.
- Infected sprouts develop black, sunken, necrotic lesions or cankers on the stem under the soil.
- In the field, plants are stunted and chlorotic due to the cankers present in the underground stem. Chlorotic leaves drop.



(NC



State Extension Publications)

Mode of survival: As chlamydospores and perithecia in plant debris, in soil and tubers.

Primary spread: From infected roots and shoots.

Secondary spread: Through sweetpotato weevil and rodents in store houses.

Favourable conditions: Temperature of 25°C, high soil moisture and injuries on storage roots during storage, transit and marketing.

Management

- Propagate plants from disease free planting material
- Cultivate sweetpotato once in 2-3 years
- Dip tubers in thiabendazole @ 0.1 %.
- Do not manure the field with cattle fed with diseased tubers.
- Store clean sound and disease-free tubers in store house
- Cure roots immediately after harvest at 85-95°F and 85-90% relative humidity for 5-10 days.
- Do not wash and package the roots showing symptoms of black rot.
- Decontaminate the equipment that comes into contact with an infected crop.
- Fumigate the crates/containers and store room.
- Control the insects and rodents in store houses.

Disease name : Surface rot / Stem rot /*Fusarium* wilt

Introduction

Surface rot is likely to be widely distributed and is caused by a non- specialized cortical rotting *Fusarium* found common in soils of most sweetpotato growing areas. It is morphologically undistinguishable from *F. oxysporum* f.sp *batatas*. These fungi are soil-borne and can persist in the soil for many years. Infection is usually through wounds obtained during and after harvesting. Both diseases develop during storage but do not spread to other roots unless new wounds occur.

Causal organism: *Fusarium oxysporum* f.sp. *batatas* (Wollenw.) W.C. Snyder & H.N.

Hansen

Symptoms

- Fleshly roots are infected usually in storage through wound made during harvest. Roots damaged by splitting, insect, or nematode feeding at the time of harvest also show symptoms.
- Circular, light to dark brown, dry and firm lesions are seen on the storage roots.
- The infection may start at the end attached to the mother root or at wounds and slowly extends to the cortical tissues.
- The surface of the root shrinks and the flesh underneath dries out.
- A white mycelial growth is observed on it and then the root becomes hard and mummified.



Favourable conditions: Temperature of 32-37°C and low soil moisture.

Mode of survival: As chlamydospores in soil for many years.

Primary spread: From infected tubers.

Secondary spread: Through irrigation water

Management

- Grow resistant cultivars.
- Adopt crop rotation once in 3-4 years.
- Raise the soil pH to 6.5 or 7.0 and use nitrate nitrogen rather than ammoniacal nitrogen to retard disease development.
- Use certified seed-roots for planting. Obtain cuttings from sprouts 5 cm above the soil line. Shoots pulled from mother roots should not be used.
- Dip seed material in 0.1% carbendazim for 20 min.
- Harvest the roots when soil is neither too wet nor too dry to avoid wounding during harvest.
- Follow proper curing and handling of storage roots after harvest to avoid surface rot during storage.

Disease name : Soft rot / *Rhizopus* rot

Introduction

Rhizopus soft rot is caused by the necrotrophic, Zygomycete fungus *Rhizopus stolonifer* which is a ubiquitous fungus that causes postharvest soft rot. *Rhizopus* soft rot typically appears during postharvest handling and transport and is rarely observed in the field. *R. stolonifer* (Ehrenb. ex Fr.) (syn *R. nigricans*) was first described in 1818 and first recognized as a pathogen on sweetpotato in 1890.

Symptoms

- Infection and decay commonly occur at one or both ends of the roots.
- Under humid conditions the affected sweet potatoes become soft and watery and the entire root rots within few days.
- If the humidity is high, the sweet potatoes show greyish black fungal growth. This feature distinguishes *Rhizopus* soft rot from other storage rots.
- The colour of the root is not changed, but an odour produced by it attracts fruit flies.



(Charles Averre, North Carolina State University)

Pathogenic characters : : Coloured, coenocytic mycelium with rhizoids and tuft of sporangiophore with sporangia. Aplanospores are sticky in nature.

Causal organism: *Rhizopus stolonifer*
(Enrenb. ex. Fr.) Lind.

Mode of survival: As zygospores in crop refuse.

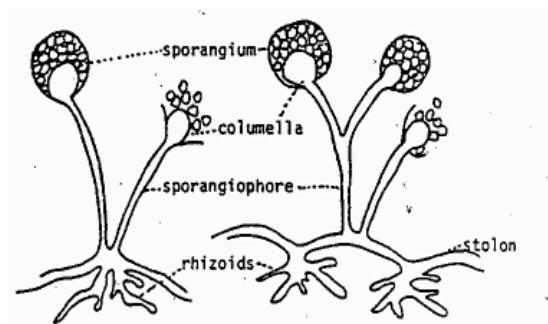
Primary spread: From infected tuber and in many crop hosts and through contaminated equipments.

Secondary spread: Air-borne spores or infested soil.

Favourable conditions: Temperature of 23-28°C, relative humidity of 75-85% during storage or transport, longer roots, chilling and heat damage when sweetpotatoes are washed, packed, or shipped to market during cold weather. Long stored sweetpotato tuber decay occurs at 15-23°C.

Management

- Avoid planting sweet potatoes in poorly drained, heavy soils and avoid very high rainfall areas.
- Intercrop with less susceptible crops or with cover crops to reduce fungal inoculum levels in the fields.
- Manage weevil population to reduce holes in the sweetpotatoes and to avoid pathogen entry.
- Clean up crop debris in fields after harvest.
- Dry the tubers for 1-2 h before storage
- Wash and clean storage house with copper sulphate 2.5% soultion.
- Follow crop rotation
- Carefully handle sweet potatoes before and after harvest and during transport or storage to prevent unnecessary wounding.
- Cure properly the roots immediately after harvest.
- Do not allow sweet potatoes to be exposed to sunlight for extended periods (to prevent heat damage) or to be chilled in the field.
- Avoid lengthy storage period before sale in store houses or in markets
- Avoid marketing diseased sweet potatoes in plastic bags without aeration.



Disease name : White rust/blister

Symptoms

- Presence of chlorotic or yellowish blotches, initially roundish to angular where they are limited by veins, on the upper surface of leaves.
- On the lower surface, white creamy pustules produced
- Corresponding upper surface show yellow discoloration
- Complete yellowing and pre mature shedding of leaves

Pathogenic characters: The mycelium is intracellular with typical knob-like haustoria. The sporangiophores are hyaline, club shaped, unequally curved at the base. The sporangia are produced in chains. They are short, cylindrical, with more rounded terminal, hyaline, and smooth; The oospores are light yellowish brown with papillate episporic and curved ridges.



Mode of survival: Soil borne Oospores.

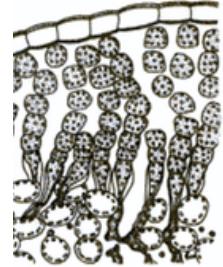
Primary spread: Oospores

Secondary spread: Zoospores, Air –borne sporangia

Favourable conditions: Rainfall

Management

- Clean up crop debris in fields after harvest.
- Soil application of *Pseudomonas fluorescens* or *Trichoderma viride* @10g /sq.m along with FYM.
- Soil application of Pf1 @ 2.5 kg mixed with 50 kg FYM /ha at 30 days of transplanting.
- Soil drench with Bordeaux mixture @ 1.0% or copper oxychloride @ 0.25%
- Spray mancozeb or difolatan @ 0.2% or metalaxyl + mancozeb @0.2%.



BEANS

Disease name : Anthracnose

Introduction

Bean anthracnose, caused by a fungus, is a major disease of common bean, *Phaseolus vulgaris* in both temperate and tropical countries worldwide. Anthracnose was first described in Germany in 1875. The disease is most common and severe on dry and snap beans, runner bean, mung bean, cowpea, and broad bean. The disease cause an yield loss of \$1.5 million of eastern USA.

Causal organisms : *Colletotrichum lindemuthianum* (Sacc. & Magnus)

[Sexual stage: *Glomerella lindemuthianum* Shear]

Symptoms

- The pathogen can cause infection on all above-ground parts
- Dark brown-black, slender lesions that follow the leaf vein on the underside of the leaf.
- Older lesions are sunken and occur on both sides of the leaves.
- Brown to black, slender to oval-shaped lesion appears on petioles and stem. The margins of the lesions are slightly raised, well-defined black ring surrounded by a red-brown halo. The centre of the lesions appear light to dark brown.
- Lesions on pods are circular, sunken with reddish-brown margins and reddish centres. The fruiting bodies on the lesions are with orange-pink spores.
- Infected seeds are shriveled and discoloured with very poor germination and the seedlings from them die at an early stage.



Pathogen character : It is a hemibiotrophic pathogen, it spends part of its infection cycle as a biotroph, and the other part as a necrotroph, and upon infection light pink color conidia were produced Conidia are 1-celled, hyaline, oblong, cylindrical with rounded ends. Asexual fruiting body is acervuli.

Favourable conditions: Temperature of 17°C, relative humidity of 92%, presence of free moisture on plant surface and disease intensity increases with age of the plant.

Mode of survival: In seeds (upto 5 years) and in the crop refuse (upto 2 years).

Primary spread: From infected seeds and plant refuses.

Secondary spread: Air-borne conidia and air-driven rain splashes.

Management

- Follow deep summer ploughing to incorporate the infected debris.
- Use resistant varieties and adopt crop rotation.
- Collect and use seeds from disease free pods / certified seeds for sowing.

- Collect and destroy infected plants.
- Treat the seeds with carbendazim @2g/kg
- Spray carbendazim @0.1% or mancozeb or Chlorothalonil @0.2%.

Disease name : Rust

Introduction

Bean rust, is one of the most prevalent diseases of beans which cause economical loss to the farmers. Several pathogenic races of the bean rust fungus are known to exist. Although rust is widespread, its severity is usually low to moderate. The disease occurs sporadically and is favoured by prolonged periods of warm, and moist weather. In India, it is found in the Manipur state. It is more severe in humid areas.

Causal organisms : *Uromyces phaseoli-typica*

Symptoms

- Reddish brown pustules are mostly seen on the lower surface of the leaves.
- The corresponding upper surface of pustules becomes chlorotic.
- Black telia are formed later.
- The severely infected leaves turn brown, dry and fall off prematurely.

(Howard
Schwartz,
Colorado State University)



F.



Pathogen character : It is a macrocyclic rust, produces five spores viz., spermatiospores, aeciospores, urediospores, teliospores and basidiospores.

Favourable conditions: Free moisture on leaves, temperature of 17-27 °C, relative humidity of more than 95 % and cloudy and humid weather with night dew.

Mode of survival: Teliospores in plant debris and volunteer plants.

Primary spread: Teliospores from infected plant debris.

Secondary spread: Air-borne basidiospores and uredospores.

Management

- Use resistant varieties
- Adopt crop rotation with non-host crops.
- Collect and destroy severely diseased leaves and volunteer bean plants.
- Spray mancozeb or chlorothalonil @0.2%.

Disease name : Powdery mildew

Introduction

Common bean powdery mildew caused by *Erysiphe poligoni* DC. (Ferreira et al. 1999) is one of the important diseases of bean which occurs in humid conditions. Recent studies suggest that it is closer to *Erysiphe diffusa* (Cooke & Peck) U. Braun & S. Takam, formerly *Microsphaera diffusa* Cke. & Pk. (Almeida et al. 2008). High relative humidity at night and low relative humidity during day favoured the occurrence of powdery mildew disease.

Causal organisms : *Erysiphe poligoni*

Symptoms

- Small and white talc-like spots on the upper surface of leaves. These spots increase in size and form a whitish, powdery growth spreading to cover a large area of the leaves.
- Infected leaves gradually curl downward, pale yellow or brown, die and fall off.



Pathogen character : It produces amphigenous white septate mycelium, lobed single or paired appressoria, oidium type conidiophore, solitary cylindrical conidia, brown, septate myceloid Cleistothecia with 3-10 ascii, containing 3-4 spores.

Favourable conditions: Shady conditions, high N fertilization, high relative humidity and moderate temperature.

Mode of survival: Cleistothecia in the plant debris in soil

Primary spread: Infected plant debris.

Secondary spread: Air-borne conidia.

Management

- Grow resistant varieties.
- Remove and destroy infected plant materials.
- Spray wettable sulphur 0.3%.



Disease name : Dry root rot

Introduction

Aphanomyces root rot (ARR) of beans caused by *Aphanomyces euteiches* f.sp. *phaseoli* is a soil borne fungal disease causing browning of roots and stems of green

beans (*Phaseolus vulgaris*). The fungus causing the disease is favoured by wet conditions.

Causal organisms : *Macrophomina phaseolina* (Tassi) Goid. (Pycnidial stage)
Rhizoctonia bataticola (Taub.) Butler (Sclerotial stage)

Symptoms

- It attacks seedlings and grown up plants.
- Black sunken lesions appear near the base of the cotyledons and it spreads to the stem and first pair of unfolded leaves.
- Infection on stem of grown up plants shows sunken and reddish brown lesions which spread both upward and downward causing death of plants.
- Affected tissues show dry rot and large number of fruiting bodies (sclerotia on root barks or pycnidia on stem).



Pathogen character : It is a soil borne monocyclic fungus possesses two asexual phases, a saprophytic phase (*R. bataticola*) that forms microsclerotia and mycelia and a pathogenic phase (*M. phaseolina*) present in host tissues that forms microsclerotia, mycelia and pycnidia.

Favourable conditions: Dry weather, high temperature and low soil moisture.

Mode of survival: As sclerotia in infected plant refuse in soil.

Primary spread: From soil-borne sclerotia.

Secondary spread: Through irrigation water and implements.

Management

- Adopt long crop rotation.
- Rogue out and destroy infected plant debris.
- Treat the seeds with carbendazim @ 2g/kg or *Trichoderma viride* @4g/kg or *Pseudomonas fluorescens* @10g/kg.
- Spot drench with carbendazim @ 0.1%.

Disease name : Bacterial blight

Introduction

Common bacterial blight (CBB) is a significant seed borne disease of common bean, caused by the gram-negative bacterial pathogen *Xanthomonas axonopodis* pv. *phaseoli* (Xap). CBB is ranked among the most important constraints to common bean production. CBB can cause significant losses in common beans in tropical and subtropical climates. It also attacks different legume crops as a secondary host and make a reasonable losses.

Causal organisms : *Xanthomonas axonopodis* pv. *phaseoli*

Symptoms

- Small, angular, light green, water-soaked or translucent spots appear on the leaves.
- The spots rapidly enlarge and merge.
- As they develop, the centre of the spot becomes brown and surrounded by a distinct yellow zone.
- In susceptible varieties, the lesions expand and coalesce. Such leaves appear scorched, wither and fall off.
- Pod lesions are round and water-soaked that merges and form sunken, irregular reddish brown blotches.
- Infected pods are shriveled and dry.
- The seeds in severely affected pods do not develop properly but get shriveled.



Pathogen character: Rod shaped Gram – negative bacterium

Favourable conditions: Optimum temperature of 26- 32°C, wounds by air-blown soil particles and insects.

Mode of survival: In seed (more than two years) and in the diseased plant debris.

Primary spread: From infected seeds.

Secondary spread: Air-blown rain and irrigation water

Management

- Follow crop rotation.
- Grow resistant / tolerant varieties.
- Sow only disease-free healthy seeds
- Soak the seeds in streptocycline @ 100 ppm before sowing.
- Avoid water stagnation and maintain proper drainage
- Rogue out and burn severely infected plants and crop debris.
- Spray copper oxychloride @0.25%.

Disease name : Bean common mosaic

Introduction

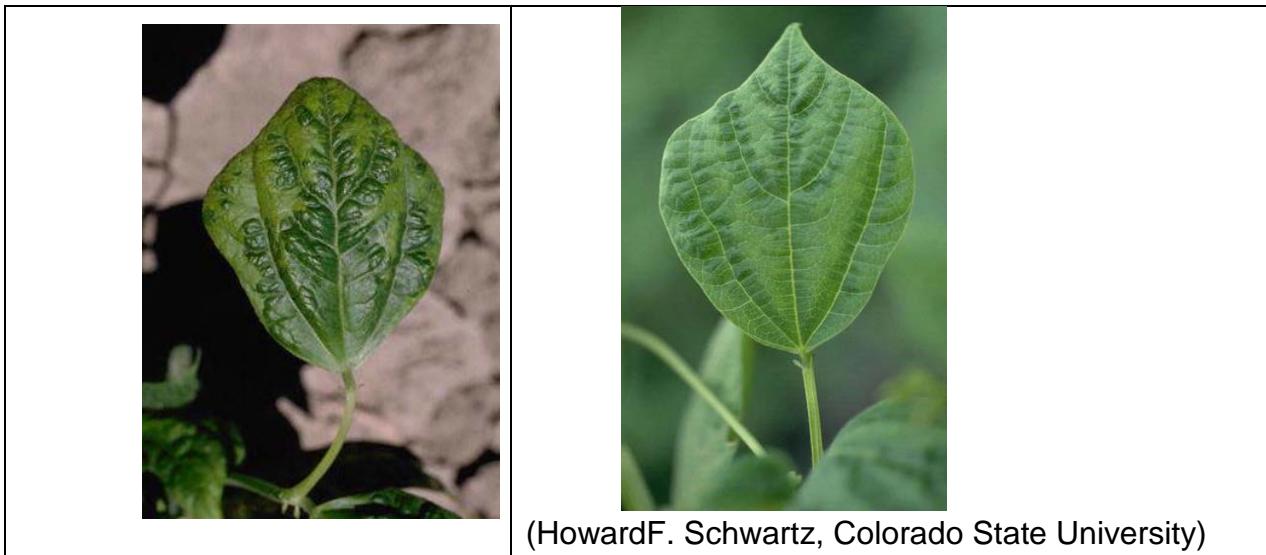
Bean yellow mosaic virus is a plant pathogenic virus in the genus Potyvirus and the virus family Potyviridae. A mosaic disease, believed to be bean yellow mosaic virus, was first reported in the early 1900s infecting garden peas (*Pisum sativum*) in the North eastern United States. The virus is currently believed to be distributed worldwide.

Causal organisms : *Bean common mosaic virus*

Symptoms

- Leaves show irregular-shaped, light-yellow and dark-green areas in a mosaic-like pattern.
- Infected leaves show puckering, stunting, malformation and downward curling.
- Infected leaves are narrower and longer than normal ones.
- Early infected plants are yellowish and dwarfed.

- Systemic infection causes necrosis roots, stems, leaf veins and pods.
- Infected pods are chlorotic, undersized with fewer seeds.



Pathogen character : Single-stranded positive sense RNA virus

Favourable conditions: Temperature of 20-25°C, monocropping, abundance of aphid vector and weed hosts.

Mode of survival: In seed (seed-borne) and weed hosts.

Primary spread: From infected seeds.

Secondary spread: By insect vector, aphids (*Myzus persicae*).

Management

- Grow tolerant varieties.
- Collect seeds from healthy plants.
- Remove and destroy infected plants.
- Control insect vectors by spraying methyl demeton @2ml/lit or other systemic insecticides.

13. Etiology, symptoms, mode of spread, survival, epidemiology and integrated management of Diseases of Coconut and arecanut

1. 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 breaking 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. Sporangium releases reniform, biflagellate zoospores upon germination. The fungus also produces thick walled, spherical oospores.



Favorable Conditions

High rainfall, high atmospheric humidity (above 90 per cent), low temperature (18-20°C) and wounds caused by tapper 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 disease 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 Bordeaux paste. Give prophylactic spray with 1% Bordeaux mixture or copper oxy chloride (0.25%) to all the healthy plams in the vicinity of diseases one and also before onset of monsoon rains.

2. Leaf blight : *Lasiodiplodia (Botryodiplodia) theobromae*

Symptoms

The pathogen causes damage in leaflets, fronds and nuts. Generally the disease occurred in the leaflets of matured fronds in lower whorls. Heavily infected coconut palms its delayed flowering than healthy palms and the incidence is severe in older leaves and the younger leaves completely free from disease. The affected leaflets start drying from the tip downwards. The drying leaflets margins are dark grey to brown colour with wavy to undulated margins and spread the lesion throughout the leaflets then the fronds exhibits a charred or burnt appearance. The fungus entered in to the kernel through mesocarp, resulting in a decay of the endosperm. The affected nuts were desiccated, shrunk, deformed and dropped prematurely. In severe cases of the disease. the nut yield loss extends up to 10 to 25 per cent.

Leaf blight



Necrotic patches on petiole



Nut rot



Pathogen

Colonies are grey to black, fluffy with abundant aerial mycelium; reverse fuscous to black. It produced pycnidia, simple or compound, stromatic, ostiolate, frequently setose in nature. Conidiophores are simple, hyaline, sometimes single septate, rarely branched cylindrical, arising from the inner layers of cells lining the pycnidial cavity. Conidia are initially unicellular, hyaline, granulose, sub-ovoid to ellipsoid-oblong, thick-walled, base truncate; mature conidia is one-septate, cinnamon to fawn and often longitudinally striate.

Favourable Conditions

The incidence was noticed throughout the year. Maximum incidence was observed during summer months and low in cooling/ winter months. High temperature and humidity favoured the disease development. Spores and the resting structures on the affected portion of the leaves served as inoculums for further spread through wind.

Management

Remove and burn the severely affected leaves to avoid further spread. Spray bordeaux mixture (1%) or Copper oxy chloride (0.25%) along with sticking agent (1m/lit) for two times at 15 days interval during summer months. Give root feeding of Carbendazim 2 g or Hexaconazole 2 ml in 100 ml water for 3 times at 3 months interval. Apply *Pseudomonas fluorescens* @ 200g along with 50 kgs of FYM / 5 kgs of Neem cake /palm /yr. Apply an additional quantity of 1.5 kgs of Murite of Potash .

3. Grey leaf spot: *Pestalotia palmarum*

Symptoms

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

4. 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 some time and later turn yellowish brown. The new fronds produced become successively smaller and yellowish in colour which does 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.



Yellowing, drying and drooping of leaves



Oozing of reddish brown liquid



Bracket formation

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 basidiospores. 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 Hexaconazole 2ml/100 ml of water through root feeding at 3 months intervals for 3 times in an year.

5. 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 exudate 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 outright but become uneconomical to maintain. In extreme cases, the trees may become barren and die.



Pathogen

The fungus produces two types of conidia. Macroconidia are produced on conidiophores singly or in chains. They are spherical and dark green in colour. Microconidia are produced endogenously inside the long cells ruptures when mature and release the microconidia 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. Ascii 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 survives in the infected plant debris and soil as perithecia. 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.

6. 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 matured nuts was 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 shriveling 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.



Flaccidity



Marginal Necrosis



Necrosis of inflorescence

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 typicus*.

Mode of Spread and Survival

The severely infected plants serve as primary sources of inoculum. The MLO is transmitted by the plant hopper (*Protista moesta*) and lace wing bug *Stephanitis typicus* from diseased to healthy palms.

Management

Cut and remove disease advanced and uneconomic palms yielding less than 10 nuts/palm/year as well as juvenile palms. Grow disease resistant/tolerant varieties viz., Kalphasree and Kalpha Sankara. Apply balanced dose of chemical fertilizers (Urea – 1.3 kg; superphosphate – 2.0 kg; Muriate of Potash – 3.5 kg/palm/year), 1 kg magnesium sulphate + organic manure – FYM @ 50 kg + *Pseudomonas fluorescens* 100 g + neem cake 2 kg/palm/year. Mulch the basin with coconut leaves. Grow green manure crops - cowpea, sunhemp (*Crotalaria juncea*), *Calopogonium mucanoides*, *Pueraria phaseoloides* etc. may be sown in coconut basins during April-May and incorporated during September-October. Irrigate properly in summer (250 litres/day) and also provide proper drainage. Grow suitable inter and mixed crops (banana, pepper, cocoa, vanila, turmeric, ginger, pineapple, coffee, nutmeg or tapioca etc.). To manage the vectors (Green lace wing bug and Plant hopper), apply 20 g Phorate 10G mixed with 200 g fine sand around the base of the spindle leaf or spray Dimethoate or Monocrotophos 1.5 ml + 1ml sticking agent dissolved in 1 litre of water for two times at monthly interval. For manage the leaf rot disease, cut and remove the rotten portions of the spindle and the adjacent two leaves and dissolve 2 ml of Contaf 5 EC or 3 g of Dithane M45 in 300 ml of water and pour around the spindle leaf. Apply talc based formulation of *Pseudomonas fluorescens/Bacillus subtilis* @ 50 g in 500 ml of water by dispensing around the base of the spindle leaf.



Diseases of Arecanut

1. Anabe roga or basal rot or foot rot: *Ganoderma lucidum*

Symptoms

The leaflets in the outer whorls of leaves become yellow and spreads to the whole leaf and the leaves drooping down covering the stem. Later, the inner whorl leaves also become yellow. Subsequently all the leaves droop, dry up and fall off, leaving the stem alone. The stem will become brittle and easily broken by heavy wind. The base of the stem shows brown discolouration and oozing of dark fluid. Bracket shaped fructifications of the fungus called 'anabe' appears at the base of the trunk. Roots become discoloured, brittle and dried.

Yellowing, drying and drooping of leaves



Bracket formation



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 basidiospores. 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 Hexaconazole 2ml/100 ml of water through root feeding at 3 months intervals for 3 times in a year.

2. Mahali disease: *Phytophthora arecae*

Symptoms

Water soaked spots initially develop at the base of the nut. Fruit stalks and rachis of inflorescence are also affected. Rotting and excessive shedding of immature nuts from the trees occur. Nuts show large vacuoles and dark brown radial strands. Very often the top of the affected trees dries up resulting in withering of leaves and bunches. Affected nuts fall off and show the white mycelial growth of the fungus.

Bud rot

The first symptom is the change of spindle leaf colour from green to yellow and then brown. The leaves rot and the growing bud rots causing death of the palm. The affected young leaf whorl can be easily pulled off. The outer leaves also become yellow and droop off one by one leaving a bare stem.



Withering of leaves

Mycelial growth on nuts

Bud rot

Pathogen

The fungus produces intercellular, non septate, hyaline mycelium. Sporangiophores are hyaline and simple or branched occasionally. The sporangiophores are hyaline, thin walled, pear shaped with a prominent papillae. Sporangium releases reniform, biflagellate zoospores upon germination. The fungus also produces thick walled, spherical oospores.

Favorable Conditions

High rainfall, high atmospheric humidity (above 90 per cent), low temperature (18-20°C) and wounds caused by tappers 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 disease 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.

Management

Remove and burn badly affected trees which are beyond recovery. If disease 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 Bordeaux

paste. Give prophylactic spray with 1% Bordeaux mixture or copper oxy chloride (0.25%) to all the healthy plams in the vicinity of diseases one and also before onset of monsoon rains.

3. Yellow leaf disease: Phytoplasma

Symptoms

Yellowing of tips of leaflets in 2 or 3 leaves of outermost whorl is the common symptom. Brown necrotic streaks run parallel to veins in unfolded leaves. The yellowing extends to the middle of the lamina. Tips of the chlorotic leaves may dry up. In advanced stage all the leaves become yellow. Yellowing of leaves is conspicuous during October to December. Finally the crown leaves fall off leaving a bare trunk. Root tips turn black and gradually rot.

Survival and spread

Caused by Phytoplasma Like Organism and transmitted by plant hopper (*Proutista moesta*)

Management

Apply balanced nutrients with additional quantity of superphosphate. Apply 1 kg of lime/tree/year. Apply organic manures @ 12 kg/ tree/year.

14. Etiology, symptoms, mode of spread, survival, epidemiology and integrated management of Diseases of tea

Diseases of Tea

A. MAJOR DISEASES

1. Blister blight -*Exobasidium vexans*
2. Grey blight -*Pestalotia theae*
3. Black rot - *Corticium invasum*, *C.theae*
4. Red rust - *Cephaleuros mycoidea*
5. Root rot diseases

Brown root disease : *Fomes lamoensis* (*basidiomycota*)

Black root disease : *Rosellinia arcuata* (*ascomycota*)

Red root rot : *Poria hypolateritia* (*basidiomycota*)

Armillaria root rot : *Armillaria mellea* (*basidiomycota*)

Inter root disease : *Botryodiplodia theobromae* (*ascomycota*)

B. Minor diseases

Pink disease : *Pellicularia salmonicolor*

Sooty mould : *Capnodium* sp.

Leaf spot : *Cercospora theae*

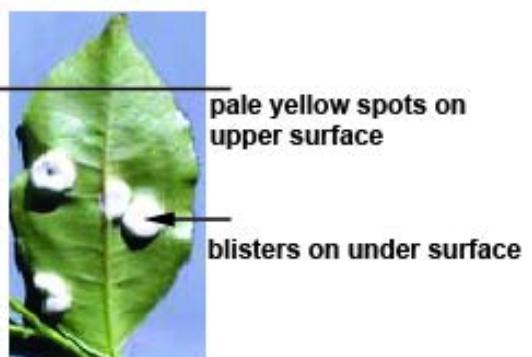
Thread blight : *Pellicularia koleroga*

1. Blister blight: *Exobasidium vexans*

Symptoms:

- Only infect on young succulent leaves and green shoots/stem (these are the economic parts)
- Attacks first flush of 2-3 young leaves and kills the young shoots and buds.
- On the upper surface of the leaf, small, circular, pale yellow or pinkish, translucent spots occur initially and later they are depressed into a shallow cavity.
- Correspondingly the under surface of the leaf bulges as a trough-like depression forming a classic blister – like swelling.
- The lower bulged surface is covered with white fungal growth comprising basidia.
- Leaves become curled and distorted.
- Later, the blisters turn to dark brown and shrink to flattened patch.

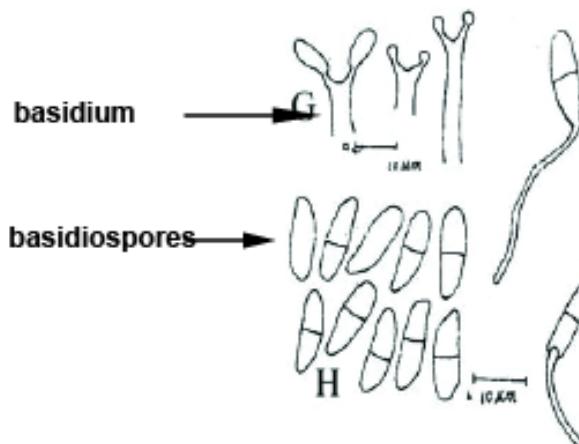
Blister blight of tea



-

Fungal characters: *Exobasidium vexans*

- Mycelium – Septate intercellular - collect in bundles below lower epidermis to produce erumpent hymenial layer from which vertical hyphae are projected by rupturing the epidermis on the surface of the spots. homothallic fungus
- Parasitism – obligate parasite
- Sexual reproduction: Basidia are intermingled with sterile hyphae. Basidia are long club shaped (clavate) generally bearing two sterigmata and two basidiospores in each basidia
- Basidiospores are ovate to oblong, ellipsoid, hyaline initially unicellular, becomes bicelled on maturity (1-septate at maturity). Non- teleosporic basidiomycetes fungus,



Pathogenesis

- Basidiospore germinates and forms germtube followed by primary hyphae. Later by somatogamy forms homothallic secondary hyphae. Germ tube and mycelium enters through the stomata, grows intercellularly and colonize the epidermal and spongy cells.
- Later, hyphae ramify the tissue, ruptures the lower epidermis exposing several basidia arranged in a layer externally on the surface of the lower epidermis (that is why the genus name exobasidium)
- Basidiospores are formed on basidia and carried by wind and causes secondary infection.

Favorable conditions

- High RH and temperature between 17 to 22°C.
- Temp > 24°C is fatal to the disease development
- If RH is < 80 %, infection is delayed

Mode of spread and survival

- It is a typical polycyclic disease. Since the crop is grown throughout the years, inoculum mainly basidiospore and less importantly mycelium always available for infection in the pre-existing infected bushes.
- Secondary spread is by wind borne basidiospore.

Management:

- Removal of affected leaves and shoots by pruning and destruction
- Protective fungicide – Copper fungicides, Spraying Bordeaux mixture or Copper fungicides – COC, copper oxides and copper hydroxide or

- (A mixture of 210 g of copper oxychloride + 210 g of nickel chloride per ha at 5 days interval from June – September and 11 days interval in October – November gives economic control)
- Spraying with 420 g of COC + 27 g of Agrimycin 100 per also gives better control
- Chlorothalonil gives both protective and therapeutic effects.
- Systemic fungicides like Tridemorph (calixin), Triadimefon (Bayleton), hexaconazole (Contaf 5E) and propiconazole (Tilt 25EC) are recommended for blister blight control in both pruning and plucking fields.

2. Grey blight: *Pestalotiopsis theae*

Symptoms:

- The disease generally attack older leaves.
- The disease initiates as minute round brown spots that enlarge and turn grey center with dark brown margin.
- Fructification appears as minute black dots in the form of concentric rings.
- The fungus attacks plucking points causing die back.



Fungal characters:

- Mycelium – filamentous, colored, branched mycelium.
- Parasitism - necrotroph
- Asexual reproduction : asexual fruiting body is acervulus. Conidia are spindle shaped with five cells (having 4 septa). The central three cells are dark and terminal cells are hyaline. The upper hyaline cell bears sterile appendages called setae.



Favorable conditions

wet conditions on foliage, insect pucture, sun scorch and postash deficiency aggravates the disease

Mode of spread and survival

- Air- borne conidia

Management

Spraying Bordeaux mixture @1% or copper oxychloride 0.25% or mancozeb 0.25%

3. Red Rust: *Cephaleuros mycoidea* and *Cephaleuros parasiticus*

Symptoms:

- On leaves, the alga occurs sometime as parasite or some time as epiphytic.
- The alga occurs as orange yellow, reddish pustules or circular patches on the upper surface of the leaves.
- The patches may be few or numerous, crowded or scattered and may occupy most of the leaf.
- The host cells in contact get killed and become brown and dried up.
- On stem, the alga is normally parasitic producing cankers and killing the tissues. It occurs as red hairy patches.



Pathogen characters

- *Cephaleuros* species consist of branched filaments that comprise a thallus.
- This thallus is pigmented (orange to red-brown) consists of a vegetative filaments and sporangiophores.
- The sporangiophores bear one or more head cells with Sterigmata. Each sterigmata bears single gametangium
- Sporangia produce numerous zoospores

Mode of spread and survival:

Primary and secondary spread by sporangia and zoospores and rain water helps in the spread of sporangia and zoospores.

Epidemiology

The infection increases during rainy season.

Max. temp of 30°C, Min temp of 25°C with high RH Management:

Management

- Removal and destruction of infected parts
- Improve the vigour of the plants by adequate irrigation and fertilizer application
- Spraying Bordeaux mixture 1% or copper fungicides 0.3%

4. Brown root disease: *Fomes noxius*

Symptoms:

- The roots are encrusted with a mass of earth and small stones cemented to the root by the mycelium
- Between the bark and wood, there is a thin layer of white or brown mycelium
- Wood turns soft and spongy and honey- comb like reticulations on the wood
- Fructification seen on stumps- bracket shaped, irregular and hard

Black root disease: *Rosellinia arcuata*

- The fungus originates from the heaps of dead leaves.
- The fungal attack at collar regions.
- Black woolly strands of mycelium closely adhere to the roots and collar as loose cob webby mass.
- These enter the bark and spread out into star – like sheets of white mycelium.
- Girdling and canker seen at collar region
- The mycelium then ramifies between the bark and wood.

Red root disease: *Poria hypolateritia*

- Fast spreading and slow killing pathogen
- Fungus is confined to underground parts.
- When the bark is peeled off, characteristic, flat, black rhizomorphs are seen.
- These strands form branched markings on the surface of the wood.

Armelliella* root rot: *Armillaria mellea

- Usually the disease becomes apparent after it has severely damaged the root system
- The foliages become chlorotic, wilted and dropped off.
- Death of whole plants then follows.
- Sheets of creamy mycelium are seen beneath the bark along with flattened brown rhizomorphs.
- In advanced stages sporophores are produced at the collar region.
- The sporophores occur in clumps and are pale brown and mushroom – shaped.

Inter root disease: *Botryodiplodia theobromae*

- The roots of a dead bush do not bear any external mycelium.
- In some cases the bark becomes rough and abnormally thickened.
- The mycelium runs within the tissues of the plant. The fructifications of the fungus are minute, black, spherical bodies embedded in the bark.
- Fructifications are not visible externally. When bark is slightly scrapped, the spheres are cut across and black circles with white centre may be seen.
- The fungus enters through the fine rootlets and attacks the tap root also.

Minor diseases

Pink disease: *Pellicularia salmonicolor*

- The branches become irregularly swollen.
- The fungus forms pink fructification over affected f stem.
- Young branches lose the leaves and die back.
- Bark killed in patches.
- Basidiospores are wind borne.
- Application of potash promotes recovery.

Cercospora* leaf spot / Birds eye spot: *Cercospora theae

- Small spots with brown centre and reddish brown margin are formed on leaves.

- Several spots coalesce to form irregular patches with shot holes.
- Severe infection causes pre mature leaf fall.

Thread blight: *Pellicularia koleroga*

- Sterile white threads or strands pass along the branches.
- Spread into a fine web like film on the under surface of the leaves.
- This causes browning and death of leaf cells.

Sooty mould: *Capnodium* spp.

- The leaves and shoots are covered with black sooty growth.

15. Etiology, symptoms, mode of spread, survival, epidemiology and integrated management of Diseases of coffee

Coffee Leaf rust / Oriental leaf disease - *Hemileia vastatrix*

Collar rot - *Rhizoctonia solani*

Anthracnose - *Colletotrichum coffeaeum*

Cercospora leaf spot / Brown eye spot / Berry Blotch / Fruit spot - *Cercospora coffeicola*

Black rot / thread blight- *Corticium koleroga*

American leaf spot- *Mycena citricolor*

Sooty mould - *Capnodium brasiliense*

Pink Diseases - *Corticium salmonicolor*

Rosellina root rot/ Maya disease / Black root disease - *Rosellina arcuata*, *R. bunders*, *R. necatrix* and *R. pepo*

Brown root disease / Stump rot - *Fomes noxius*

Red Root rot disease - *Poria hypolateritia*

Santavery root disease - *Fusarium oxysporum f. sp. Coffeae*

1. Coffee Leaf rust / Oriental leaf disease/*Hemileia vastatrix*

Introduction:

Coffee originates from high altitude regions of Ethiopia, Sudan and Kenya and the rust pathogen is believed to have originated from the same mountains. Introduced in Ceylon from Ethiopia

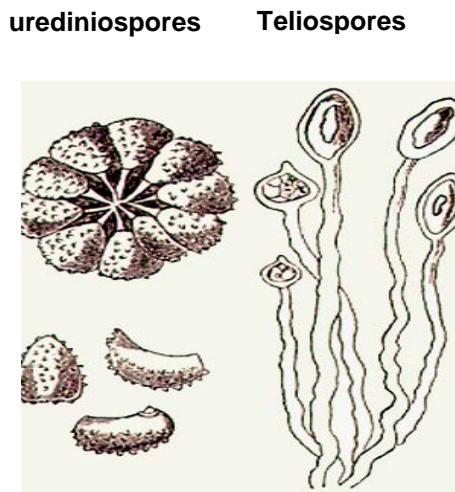


Rust was first reported in the major coffee growing regions of Sri Lanka in 1867 and the causal fungus was first fully described by the English mycologist Michael Joseph Berkeley and Christopher Edmund Broome. They named the fungus *Hemileia vastatrix*, *Hemileia* referring to the half smooth characteristic of the spores and *vastatrix* for the devastating nature of the disease.

Symptoms

- The disease is mainly restricted to leaves but sometimes seen on berries and on tender shoots.
- Young leaves are highly susceptible than matured ones.
- The disease initiates as small yellow or blotchy, orange powdery pustules (uredosori), or lesions on the under surface of the leaves.
- Corresponding to the pustules chlorotic patches appear on the upper side
- Soon the spots enlarge on the under surface of the leaves.
- The colour turns orange brown with powdery masses of spores called uredosori.
- Severely affected leaves shed prematurely.
- Vegetative growth is reduced.
- Die-back of the branches also occur.

- Fungal characters**
- Spermogonia and aecia unknown.
 - Uredinia hypophyllous, densely scattered and giving a powdery appearance on yellowish-orange rounded blotches. Urediniospores are packed together like orange segments, looking kidney-like shaped, warted on the convex face, smooth on the straight or concave face,
 - Telia as the uredinia, hypophyllous; Teliospores are rarely found. teliospores often produced in uredinia, sometimes in teliosori borne in cluster on short pedicels, 1-celled, more or less spherical to limoniform (turnip shaped) and smooth with a terminal papilla,
 - Although heteroecious, but no alternate host is known.



Mode of spread and survival:

- The fungus survives and spread by urediniospores through wind (air-borne) and rain splash
- Teliospores do not infect coffee.

Epidemiology

- Rainy weather, mist or dew conditions and moderate temperature favour the development of the disease.
- The showers and sunny weather with occasional mist prevailing during NE monsoon period is ideal for severe infection.

Management

- Use of resistant varieties like SIn. 5B, SIn. 8 SIn. 9
- Wider spacing; Shade management
- Pruning of dead and dying twigs after harvest
- Apply balanced nutrients to maintain plant vigour
- Diseased fallen leaves should be collected and destroyed
- Spraying Bordeaux mixture 0.5% during pre blossom (May- June) and post blossom (September – October)
- Spraying triadimephon 0.05% a.i. or bayleton 160 g or hexaconazole (contaf) 400 ml or propiconazole (tilt) 160 g during August – September
- Biological control with *Verticillium hemileiae* during winter

2. Collar rot: *Rhizoctonia solani*

The disease occurs on 1-3 months old seedlings in nursery.

Symptoms

Causes both pre – and post - emergence damping off

Pre emergence Damping off

- The fungus invades embryo and endosperm before germination
 - It causes decay of germinating seeds
 - Young radical and plumule undergo complete rotting
 - Seedlings are infected and died before the emergence from the soil
 - Causes poor and uneven stand of seedlings in nursery beds
- Post emergence Damping off
- Occurs after the emergence of seedlings from the soil
 - Watery, soft rot develops at the collar region of the stem
 - Affected portion shows shrinking and brown discolouration due to rotting
 - The affected seedling become collapsed and toppled down
 - Mortality of seedling is very conspicuous

Mode of spread and survival

- soil-borne sclerotia

Epidemiology

- Excessive soil moisture in the nursery bed
- Thick overhead pandal shade
- Hot and humid condition
- Overcrowding of seedlings

Management

- Expose the nursery soil to the sun for 2-3 months
- Preparation of raised – bed nursery
- Application of decomposed FYM
- Good drainage.
- Excess watering should be avoided.
- Overcrowding of seedlings should be avoided.
- Use of filtered overhead shade using green leaves coir mats / nylon mats
- *Trichoderma viride* or *Pseudomonas fluorescens* – soil application
- Seed treatment with carbendazim (1g/kg) carboxin (0.7g/kg)
- Soil drenching with carbendazim 0.05% or Mancozeb 0.05% or Captan 0.05%

3. Anthracnose: *Colletotrichum coffeatum*

- The disease also known as die back or brown blight or black berry or Nilgiri twig disease

Symptoms:

- The fungus causes spots on leaves and berries.
- On leaves circular to irregular grayish spots are produced.
- On berries small dark sunken spots are formed. The infection spreads to internal tissues and affects the bean turning black hence it is called black berry.
- Infection on the twigs causes wilting of young leaves exhibiting die back symptoms.



Favourable conditions

- Low temperature, mist or dew during night & early hours; inadequate over-head shade, prolonged drought & soil moisture stress are the pre disposing factors for the disease development.

Management

- Pruning of badly affected plants during Feb-Mar.
- Maintaining adequate overhead shade to protect the bushes from sun scalding & mulching of leaf litter around the plants for conserving soil moisture during dry weather.
- Application of balanced nutrients to maintain vigour of the plants.
- Protect plants by spraying 0.5% Bordeaux mixture during Feb- Mar(Pre-blossom), Apr-May (Pre-monsoon) & Sep-Oct (Post-monsoon).

4. Cercospora leaf spot / Brown eye spot / Berry Blotch / Fruit spot: *Cercospora coffeicola*

Symptoms:

- Lesions begin as small, circular chlorotic spots on the upper leaf surface
- Spots expand to become necrotic with dark brown margin and gray, or white centre
- The central portion turns light grey due to sporulation and collapses leaving shot hole.
- Lesions are sometimes surrounded by a bright yellowish "halo,"
- On green berries, spots are initially brown, oval slightly sunken and necrotic with ashy centre appear.
- The spots enlarge in size and become dark brown, necrotic and cover major area of the berries.
- Around the spots purplish halo is seen
- The tissues turn brown to black.



Mode of spread and survival

- Fallen leaves constitute a primary source of infection,
- Pathogen dispersal is by spores (conidia) that are air-borne
- Also seed borne

Epidemiology

- The pathogen sporulates readily under more humid conditions and temperatures between 20- 27°C is optimum.
- plants already under stress predispose the plants

Management

- Choose the planting location to avoid very high elevations and rainy locations
- Orient rows so that they are perpendicular to prevailing winds, so plant canopies and leaves become dry more quickly after rainfall.
- Optimum planting density (number of plants per acre).
- Strive to minimize plant stresses such as drought, under-nutrition, planting on impermeable rock outcroppings,
- Avoid overhead irrigation
- Avoid working with coffee plants and moving through fields and nurseries when diseased plants are wet (this minimizes potential dispersal of fungal conidia within and among moist plants).
- Prune coffee trees to increase air circulation in the canopy.
- Spraying of Bordeaux mixture 1% or Captan or Mancozeb 0.5% or Carbendazim 0.01% foltaf 0.4%.

5. Black rot / thread blight : *Corticium koleroga*

Symptoms:

- The striking feature of the disease is the presence of dark brown or black decaying leaves twigs and berries. Therefore it is called as Black rot.
- Affected leaves get detached from branches and hang down by means of slimy fungal strands.
- The fungus develops over the slimy film.
- Mycelia threads will also be seen running along the twigs.
- Numerous minute clumps of mycelium and sclerotia scattered all over the dark patches.
- On green berries, blackening is seen as a narrow band.



Mode of spread and survival

- The pathogen spreads by contact form leaf to leaf through vegetative mycelium.
- Pathogen survives as sclerotia in infected plant debris.

Epidemiology

- Heavy rains, high humidity

Management

- Uniform and medium intensity overhead shade should be maintained.
- Pruning of bushes for free passage of air and sun light.
- Removal and destroying of diseased portion by pruning.

- Removal and destruction of infected leaves.
- Provide proper drainage
- Spraying of Bordeaux mixture 1% before and during monsoon will control the disease.
- Spray Bordeaux mixture 1% or carbendazim 0.03 % a.i. (120 g / 200 l water) during break in monsoon

6. Fusarium bark disease / Storey's bark disease: *Fusarium stilboides*

Symptoms:

- The pathogen produces three types of symptom namely, 'Storey's bark disease', 'scaly bark', and 'collar rot'
- The pathogen infects the collar region of the stem causing collar rot.
- Produces bark scaling and canker in scaly bark' type .
- Canker is then produced which girdle the trunk and kills the tree.
- Young suckers also affected.
- The sucker have constricted bottle neck appearance at the base.
- Under storeys bark type disease, cinnamon to brown sunken lesion with a water-soaked margin develops near the stem base of green stems
- It expands to eventually girdle the stem.
- The lesion may bear pink spore masses in moist conditions.

Epidemiology

Insect damage; poor soil management, irregular pruning & drought predispose the plant

Mode of spread & survival

Survives on and spread through coffee plant debris.

Management

Good soil management with adequate & timely mulching to conserve moisture in the top layers of the soil, proper pruning practices, improving soil fertility, etc. reduce the infection.

The protection of stem bases with Captan or captafol (0.4%) sprays on the trunk bases after pruning and when young suckers are maturing, also recommended.

Pruning cuts or other wounds should be protected with a fungicidal paint. Badly diseased trees should be destroyed.

Sooty mould: *Capnodium brasiliense*

- The leaves and shoots are covered with black sooty fungal growth.
- Heavy attack of the aphids and scale insects predispose for infection.
- Spraying fish oil resin soap 1kg + starch 1kg + 200m; water.

Pink Diseases: *Corticium salmonicolor*

- The fungus attacks the twigs and fruits.
- The branches wither and the fruit turn black.
- This occurs in the form of whitish or pinkish crusts on the twigs.
- Removal of affected branches by pruning.
- Application of Bordeaux paste /Spraying BM 1% as preventure method

Root diseases

Rosellina root rot/ Maya disease / Black root disease: *Rosellina arcuata*, *R. bundes*, *R. necatrix* and *R. pepo*

The disease is called as Maya Disease in Latin America.

Symptoms:

- Gradual yellowing of leaves, defoliation followed by death of the bush
- Accumulation of black superficial fungal growth (rhizomorph) or black wooly mycelium on the infected roots are also seen
- On stem near the ground level, fan- shaped fungal mats with pellet like fructifications are also seen.
- The cambium tissues at the base of the stem at soil level are affected.
- Internal discolouration of roots as thread like black line or dots. The infection spreads along main root and base of the stem also.

Brown root disease / Stump rot: *Fomes noxius*

- Brown root disease also known as 'Stump Rot,'
- Mostly associated with rotting stumps of shade trees in the plantation.
- Affected plants show gradual yellowing of leaves .
- Then the leaves wither and the bushes die rather suddenly.
- Stem near the ground becomes spongy.
- The roots system shows externally thick brown encrustation with adhered gravel pitches.
- Fungal mycelium appears as brown wooly hyphae or in brown crust on root surface.
- Black charcoal – like powdery patches are seen on the encrustation.

Red Root rot disease: *Poria hypolateritia*

- Aerial symptoms are similar to brown root disease.
- Root system shows red encrustation covered with soil and gravel adhering to it.
- The red encrustation is the fungal rhizomorph.
- The affected roots are washed it is deep red in colour.
- Stumps of felled trees serve as source of infection.
- Soil application of T.V 150g+15kg FYM – 10 days before and after monsoon. Application of lime at 1.2 kg bush Whenever a shade tree is felled, uproot the stump with root system to avoid disease spread in future.

Santavery root disease: *Fusarium oxysporum* f. sp. *coffeae*

- Sudden wilting yellowing of leaves defoliation and death of aerial parts
- Infected roots shows brown to pinkish discolouration .
- Scrapping of the bark of the stem near ground level also shows internal discolouration.
- Favourable conditions - Low or high soil temperature, poor physical conditions of soil, moisture stress, inadequate shade & wounds on the roots are the predisposing factors for disease development. Disease is soil borne.
- Soil drenching carbendazim 0.8% or Carboxin 0.4% @ 3-5 l / plant in the initial stage of infection.

16. Etiology, symptoms, mode of spread, survival, epidemiology and integrated management of Diseases of rubber and cocoa

Diseases of Rubber

| | | |
|-------------------------|---|----------------------------------|
| Powdery mildew | - | <i>Odium heveae</i> |
| Abnormal leaf fall | - | <i>Phytophthora</i> spp. |
| Secondary leaf fall | - | <i>Glomerella cingulata</i> |
| Bird's eye spot | | <i>Drechslera heveae</i> |
| Pink disease | - | <i>Corticium salmonicolor</i> |
| Root diseases | | |
| White root disease | - | <i>Fomes lignosus</i> |
| Brown root disease | - | <i>Fomes noxius</i> |
| Red root disease | | - <i>Ganoderma pseudoferarum</i> |
| Dry rot | - | <i>Ustulina zonata</i> |
| Shrinking root disease- | | <i>Sphaerostilbe repens</i> |

1. Powdery Mildew : *Odium heveae*.

Predominantly noticed on newly formed tender flush during the refoliation period of January to March.

Symptoms

- The symptoms are more predominant on tender leaves and shoots.
- The young brown as well as light green leaves are covered with white powdery masses.
- Diseased leaflets shrivel, curl, crinkle and their edges roll inwards.
- Soon the infected leaflets fall off leaving the petioles attached to the twigs giving a broom stick appearance.
- Young twigs are also infected causing die-back.
- The inflorescence and young fruits are also covered with white powdery masses



Causal organism: *Oidium heveae*

Thallus – filamentous, hyaline, septate mycelium. mycelia are epiphytic (ectoparasites) on the surface of the leaves and pathogens absorb the nutrients by producing haustoria from the superficial mycelium into the epidermal cells.

Parasitism - Biotroph

Asexual reproduction – There is no asexual fruiting body. Asexual spore is conidium.

Conidia are borne on club shaped conidiophore and conidia contains large amount of water (70%), able to grow in dry weather (free water is inhibitory).

Sexual reproduction - sexual fruiting body called cleistothecia (syn: chasmothecium) in which ascus and ascospores (sexual spores) are produced.

Mode of spread and survival

- The fungus survives as dormant mycelium inside the bark.
- Secondary spread by wind borne conidia

Epidemiology

- warm weather and low humidity favors
- Under shaded condition in high elevations the disease persists throughout the year.

Management:

- Clones PB 86, GT 1, GL1, PR 107, PB 5/139, RRIM 703, RRII 208 and PB 310 show some tolerance.
- Dusting during the refoliation period @ 11 to 14 kg 325-mesh fine sulphur dust per round per hectare.
- Wettable sulphur (2.5 g /l) is also effective in nurseries and for young plants as a spray.
- Carbendazim (1g in 1 litre water) is more effective than sulphur for nurseries and young rubber. Fogging with fungicide tridemorph such as Calixin 75 EC at 0.5% a.i/ha For efficiency, dusting may be carried out in the early morning hours when the leaves are moist and the atmosphere is calm.

2. Abnormal Leaf fall: *Phytophthora palmivora* *P. meadii* *P. nicotianae* var *parasitica* and *P. botryose*

It induces the shedding of leaves during June – August while general leaf fall occurs in December.

Symptoms

On Leaves

- Initially the affected leaves show circular water soaked dull grey lesions with fine droplets of coagulated latex at the base or the apex of the leaf
- In course of time the lesions enlarge and coalesce to form large, irregular necrotic areas with various shades of black discolouration
- The infection eventually spreads to petiole. Black lesion with a drop of white coagulated latex in the centre may develop on the petiole.
- The affected leaves extensively shed prematurely either green or after turning coppery red.

On Fruits

- Small lesions at the basal end of the pod. They enlarge into brown water-soaked areas and correspondingly the globules of latex become bigger and more apparent.
- The fungus produces a downy white growth on the surface of the green fruit pod.

On tender shoots

- When there is severe infection, the tender shoots rot.

- The infected twigs show dark brown lesions and soon exhibit die back of green shoots.

On stem

- The infection occurs at the tapping panel or anywhere on the stem including the collar region
- The infection causes the bark to swell and burst. An amber coloured liquid oozes out from the infected tissues. The bark rots, and a coagulated rubber pad, emanating a foul smell. This infection is called patch canker or bark canker.
- In the renewed bark region, small, sunken, vertically parallel depressions are formed. When infected bark is removed, characteristic, distinct vertical black lines are seen on the wood corresponding to the external depressions. This is called bark stripes or black stripes or Black thread disease.

Fungal characters:

1. Thallus - Mycelium coenocytic, hyaline, branched, inter and intracellular
2. Parasitism - hemibiotroph
3. Asexual reproduction - Sporangiophores branch dichotomously. Sporangia are thin walled, hyaline, spherical, oval, multinucleate lemon shaped papillate at the nodes.
4. Sexual reproduction - is oogamous reproduction with amphigynous anthredia
5. Chlamydospores - abundantly produced

Epidemiology:

- High atmospheric humidity, low temperature 15 – 20°C , high rainfall, and crowded canopy are predisposing factors

Management:

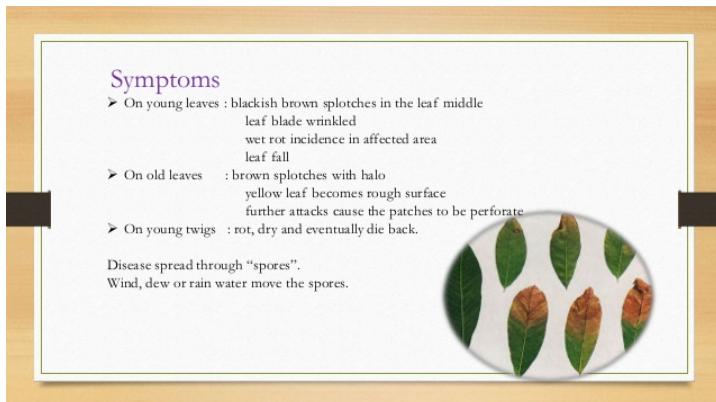
- Collection and destruction of infected fallen leaves and fruits.
- Prophylactic spray with Bordeaux mixture 1% or COC 0.3%. First spray is given prior to SW monsoon and second during the break between the monsoons to protect new flushes. Removal of bark canker affected tissues and dressing with organomercurial fungicides or copper fungicides (Bordeaux paste).
- Resistant varieties BD 10 and GL1
- curative fungicide - metalaxyl

Colletotrichum leaf fall or Secondary leaf fall: Colletotrichum gloeosporioides;

Colletotrichum acutatum (Sexual stage: Glomerella cingulata)

Symptoms

- On tender leaves, initially numerous minute circular brown spots are produced. The spots then develop a thick brown margin surrounded by yellow halo and are erumpent. The central tissues turn white. Centre of the spots dry and fall off leaving a shot hole.
- Lesions are observed on the green portions of stem also causing shoot die back.
- The fungus attacks green pods and cause rotting.



Fungal character

- Mycelium – filamentous, septate, grey colored mycelium.
- Parasitism - Hemibiotroph
- Symptoms - anthracnose disease – Anthracnose disease is characterized by sunken, necrotic and ulcer like lesions on leaves, stem, fruits/pods. Production of acervulus is the major sign on the lesions
- Asexual stage – asexual fruiting body –acervulus and asexual spore – conidia- are single celled, hyaline, mostly cylindrical shaped
- Sexual stage – sexual fruiting body – perithecia and sexual spores –ascospores- are single celled, hyaline

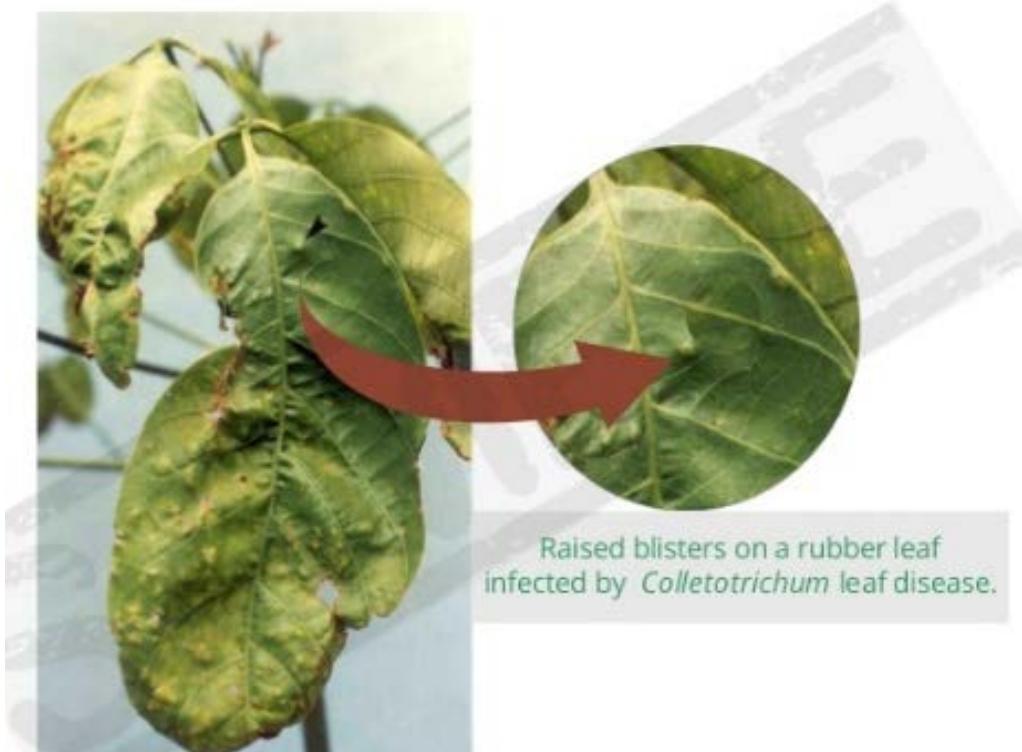
Mode of spread and survival:

- Primary inoculums and spread - ascospores from the infected plant parts
- Secondary inoculums and spread is by means of air-borne conidia.

Survival – perithecia and ascospores in the from the infected plant parts

Epidemiology

- Humid and misty condition, temperature of 24-32°C at the time of shoots, flowers and fruits development is most favourable for infection.
- Continuous wet weather during flowering causes serious blossom blight.



- RH of >95% for 12hr is essential for fruit infection.
- Excessive application of Nitrogen favourable for infection

Mode of spreads and survival:

- The fungus survives in infected plant derbies.
- Water disperses the conidia.

Management:

- Spraying with Bordeaux mixture 1%, copper oxychloride 0.25%, mancozeb 0.2% or carbendazim 0.1% at 10 - 15 day interval is also effective.

Birds eye spot – *Drechslera heveae*

Symptoms

- On tender brown leaves, disease appears as dark brown spots with yellow halo.
- This look similar to eyes of birds based on which the disease was named.
- Numerous spots are often observed on each leaflet.
- Elongated brown stripe like lesions occur on infected mid ribs, petioles and green shoots.
- The underside of lesions develop a chocolate brown colour due to sporulation of the fungus.

Pink disease – *Corticium salmonicolor*

The symptom shows fungal salmon pink incrustations on the fork region of the tree or branches where moisture is easily trapped. The white silky threads (mycelia) of the fungus appear and, under favourable conditions, spread around the branch giving a thick cobweb effect which is more visible during rainy season.

Management

Affected plant parts should be pruned and burnt

Prophylactic: Wound dressing with Bordeaux paste. Two rounds of spraying with 1% Bordeaux mixture (during May and August) on to the fork.

Curative: tridemorph 2% (Calixin 25 ml/l) in 1% ammoniated field latex or propiconazole 0.1% (Tilt 4 ml/l) in pidivyl (sticky gum), china clay and water (1:2:4 by volume) is also effective as rainwash is prevented.

Brown root disease: *Phellinus noxius*

Symptoms

- The symptoms appear as loss of shiny appearance of leaves, drooping, yellowing and buckling (curving) of leaflets
- The leaves later turn to reddish yellow and dry up.
- On the infected roots, the surrounding soil and stones form a hard and thick coating.
- brown patches of fungal rhizomorphs are visible on the surface of roots.
- Cortex appears as brown, mottled with white patches
- Wood shows brownish discolourations and brown lines.
- Large, hard, brownish purple brackets of fungus develop on old infected stumps.

Management:

- Completely killed and dried roots may be traced, pruned off and burnt along with any rotting stump
- Partially affected and healthy roots washed with tridemorph 0.5% (Calixin 6.25 ml/l) or propiconazole 0.13% (Tilt 5 ml/l) solution.

White root disease: *Fomes lignosus*

Symptoms

- The disease externally shows reduced growth rate of new shoots.
- Chlorosis of leaves followed by leaf shedding and die back.
- The fungus attacks tap root first and spreads over the root surface as white rhizomorphs with a fan like leading edge.
- These rhizomorphs are seen at the base of the trunk and roots.
- A succession of annual fruiting bodies are formed on old stumps and exposed roots or after the trees died.

Diseases of Cocoa

Major disease

- 1) White thread blight :*Marasmius scandens*
- 2) Seedling die – back :*Phytophthora palmivora*
- 3) Black pod disease :*Phytophthora palmivora*
- 4) Charcoal pod rot :*Botryodiplodia theobromae*
- 5) Witches broom :*Crinipellis pernicios*
- 6) Cherelle Rot / *Colletotrichum* Pod Rot: *Colletotrichum gloeosporioides*

II. Minor diseases

- a. Pink disease:*Pelicularia salmonicolor*
- b. Stem canker:*Phytophthora palmivora*
- d. Vascular streak:*Oncobasidium theobromae*.
- e. Cacao swollen shoot virus. (CSSV)

White thread blight: *Marasmius scandens*

White thread blight and horse hair blight are the two main types of thread blight in cocoa

Symptoms:

- White thread blight kills the leaves by spreading a network of white mycelial threads over leaves, petioles and branches.
- On the leaves it spreads as branched fine thread and also invades cortical tissue. The affected leaves turn dark brown to black.
- The dead leaves detached from stem but hung by mycelial thread in a row.
- Extensive death of young branches and suspended leaves in rows. Thick strands of mycelia are often seen on branches

Epidemiology

High humidity, less aeration and sunlight due to thick shade are the pre disposing factors **Mode of spread & survival**

The disease spread from plant to plant and different branches of the same plant through the mycelium. The dead leaves with the mycelial mat can be easily carried by wind on to the leaves and stems of the healthy plants & initiate the disease.

Management

- Removing dead materials and pruning of affected parts.
- Shade regulation and structural pruning of some branches
- Paste with copper oxychloride at cut ends.
- Bio control agent *Trichoderma viridae* application in the soil

Seedling dieback/ Black pod rot /Chupon Blight and Twig Dieback / Trunk or Stem Canker: *Phytophthora palmivora*, *P. megakarya*, *P. citrophthora* and *P. capsici*

Symptom

Seedling blight/Seedling die back:

- very common in nurseries during rainy season
- The infection starts from the tip of the stem or from cotyledonal stalk or from the collar region as dark brown to black water soaked linear lesions.
- The lesions extend to petiole and leaves resulting in wilting and subsequent defoliation of seedlings.

Black pod rot Symptoms:

- Pods of all ages are susceptible and are infected at any place on the surface, but is most often initiated at the tip.

- One or more small, brown and translucent spots appear anywhere on the pod /cherelles (immature pods) surface. Rapidly spreads in all direction with a line of demarcation of diseased and healthy tissues.
- Spots soon turn to chocolate brown colour, then darken and expand rapidly with a slightly irregular margin
- As the lesion advances, a whitish growth of the fungus consisting of mycelia and sporangia is produced over the dark brown pod surface
- The whole pod and beans are invaded by the fungus and turns black in colour, which subsequently rots and shed in large numbers
- Diseased pods eventually become black and mummified.

Chupon Blight and Twig Dieback

- Water soaked lesions appear on the apex or margin of the leaves
- They enlarge and turn dark brown to black and coalesce forming large blighted areas leading to defoliation and dieback
- Infection spreads from the leaf blade to petiole and extends backwards into the twigs or chupons
- When the lesions girdle the stem, the portion above the point of infection wilts showing twig dieback or chupon blight



Mode of spread and survival

- The fungus survives as oospores, chlamydospores and mycelium in soil, on fallen fruits, etc.
- The spread of the disease is by wind and windblown rain zoospores.

Epidemiology:

- Continuous heavy rainfall with intermittent bright sunshine, low temperature of 22-25°C, constant RH > 90%.
- Closer spacing and damp locality favour the spread of the disease.

Management:

- Collect and destroy all fallen and completely infected
- Give proper spacing between plants.
- Prune the epiphytes and chupons frequently so as to regulate shade
- Provide adequate drainage in gardens
- Prophylactic spray of Bordeaux mixture 1% or copper oxychloride 0.3 %
- Spray with metalaxyl or fentin acetate or Aluminium ethyl phosphonate.
- **Charcoal pod rot: *Lasiodiplodia theobromae* (formerly *Botryodiplodia theobromae*)**

Symptoms:

- Pods of all stages are susceptible to infection, especially during summer
- Pale yellow spots appear on the pods, mostly at the tip or stalk end.

- The spots enlarge into larger lesions and cover the entire pod giving a chocolate brown colour.
- Severely infected pods become black in color and exhibit a sooty covering all over, consisting of spores of the fungus
- Young pods become mummified and shriveled

Management

- Collect and destroy all fallen/completely dried pods. Regularly harvest ripened pods
- Give prophylactic spray of Bordeaux mixture 1% or copper oxychloride 0.3-0.4 % or Mancozeb 0.3-0.4 % at 40-45 days interval, especially on pods and flower cushions during summer months
- Curative Spray-the pod bunches twice with propiconazole or hexaconazole or carbendazim 0.1 % at 2-3 weeks interval after harvesting all mature pods. Subsequently harvest only after 45 days of spraying
-

Cherelle Rot / *Colletotrichum* Pod Rot: *Colletotrichum gloeosporioides*

- The symptom mostly starts from the stalk end and proceeds towards the tip of the pod as dark brown sunken lesion with a diffused yellow halo
- The infection also extends to the stalk and reaches the cushion
- As the infection progresses, the internal tissue of the pod becomes discoloured
- Such lesions coalesce and form bigger lesions with salmon / dark brown coloured fruiting bodies of the fungus
- Ultimately, the pod turns dark brown to black and remains mummified on the tree

Management

- Same as charcoal pod rot

Witches broom: *Moniliophthora perniciosa* (formerly *Crinipellis perniciosa*)

- It causes abnormal growths and lesions on the shoots, branches, floral cushions and fruits.
- The colonized tissues undergo several physiological and hormonal changes leading to swelling and formation of Excess of axillary bud, numerous succulent vegetative branches, known as brooms.

Mode of spread & survival

Basidiospores which cause infection are spread through wind.

Management

Young blooms should be removed before the production of sporophore. Varieties like Scavina-6 and its hybrids are resistant to the disease. Avoid inter cropping with areca, Proper spacing should be given

Swollen shoot – *Theobroma* Virus / Cocoa Swollen Shoot Virus

Symptoms

- Swelling develops at nodes, internodes and tips of the quick growing shoots.
- At early stage red vein banding appears on the leaves.
- The pigment is restricted to the mid rib, lateral veins and finer veins producing red feathering symptom.
- When the leaves turn green and harden, green vein banding seen with chlorotic or yellow translucent lesions along the major veins forming fern like pattern.
- Young unripe pods develop light and dark green mottling.
- Small abnormally shaped spherical pods

Transmitted by mealy bugs – *Planococcus* spp



Swelling of shoots



Red vein



Green vein banding