PAT 301 Diseases of Field and Horticultural crops and their management-I (1+1)

Theory

Etiology, symptoms, mode of spread, survival, epidemiology and integrated management of important diseases

Unit-I Diseases of cereals and Millets

Cereals: rice and maize; **Millets:** sorghum, bajra, finger millet and small millets

Unit- II Diseases of Pulses and Oilseeds

Pulses: pigeon pea, urd bean, mung bean, soyabean, cowpea; **Oilseeds:** ground nut, castor and Sesame

Unit- III Diseases of Cash crops: tobacco, jute and mulberry

Unit- IV Diseases of Fruits and vegetables crops

Fruits: banana, guava, papaya, pomegranate; **Vegetables:** tomato, brinjal, okra , cruciferous vegetables, beans, colacasia and sweet potato

Unit- V Diseases of Plantation crops

Plantation: coconut, arecanut, tea, coffee, rubber and cocoa

Practical

Study of symptoms and host parasite relationship of rice, maize, sorghum, bajra, finger millet ,small millets, pigeon pea, urd bean, mung bean, soyabean, cowpea, ground nut, castor Sesame, tobacco, jute , mulberry, banana, guava, papaya, pomegranate, tomato, brinjal, okra , cruciferous vegetables, beans, colacasia , sweet potato, coconut, arecanut, tea, coffee, rubber and cocoa

Theory

Etiology, symptoms, mode of spread, survival, epidemiology and integrated management of

1. Diseases of rice
2. Diseases of maize and sorghum
3. Diseases of pearl millet, finger millet and small millets,
4. Diseases of pigeonpea, urdbean, mungbean, soybean and cowpea
5. Diseases of groundnu , sesame and castor
6. Diseases of tobacco, jute and mulberry
7. Diseases of banana
8. Diseases of guava, papaya and pomegranate
9. Mid semester examination
10. Diseases of tomato
11. Diseases of brinjal and okra
12. Diseases of crucifers
13. Diseases of sweet potato and beans
14. Diseases of coconut and arecanut
15. Diseases of tea
16. Diseases of coffee
17. Diseases of rubber and cocoa

Practical

Study of symptoms and host-parasite relationship of:

1. Diseases of rice
2. Diseases of maize and sorghum
3. Diseases of pearl millet, finger millet and small millets,
4. Diseases of pigeonpea, urdbean, mungbean, soybean and cowpea

5. Diseases of groundnut , sesame and castor
6. Diseases of tobacco, jute and mulberry
7. Diseases of banana
8. Diseases of guava, papaya and pomegranate
9. Diseases of crucifers
10. Field visit/ exposure visit to hilly fruits , vegetables and plantation crops
11. Diseases of tomato, brinjal and okra
12. Diseases of sweet potato and beans
13. Diseases of coconut and arecanut
14. Diseases of tea
15. Diseases of coffee
16. Diseases of rubber and cocoa
17. Final practical examination

Reference Books

1. Arjunan.G. Karthikeyan, G, Dinakaran ,D. Raguchander,T. 1999 Diseases of Horticultural Crops, AE Publications, Coimbatore.

2. Rangasawmi ,G and Mahadevan, A. 1998. Diseases of crop Plants in India, Prentice Hall of India Pvt. Ltd., New Delhi

3. Prakasam, V., Valluvaparidasan, V., Raguchander, T. and K.Prabakar. 1997. Field crop diseases, AE Publication, Coimbatore.

E- Books

1. Agrios, G.N. 2008. Plant Pathology, Academic Press, New York.

2. Rangaswami, G. 2005. Diseases of Crop plants in India. Prentice Hall of India Pvt. Ltd., New Delhi.

Thakur, B.R. 2006. Diseases of field crops and their management

DISEASES OF RICE

RICE *(Oryza sativa)*

1. Blast

Pyricularia oryzae (Syn : P. grisea)

(Sexual stage : Magnaporthe grisea)

Symptoms

The fungus attacks the crop at all stages from seedlings in nursery to heading in main field. The typical symptoms appear on leaves, leaf sheath, rachis, nodes and even the glumes are also attacked. On the leaves, the lesions start as small water soaked blusih green flecks, soon enlarge and form characteristic **spindle shaped spots with grey centre and dark brown margin (Leaf blast).** The spots join together as the disease progresses and large areas of the leaves dry up and wither. Similar spots are also formed on the sheath. Severely infected nursery and field show a burnt appearance. In infected nodes, irregular black areas encircle the nodes can be noticed. The affected nodes may break up and all the plant parts above the infected nodes may die **(Node blast).** At the flower emergence, the fungus attacks the peduncle which is engirdled, and the lesion turns to brownish-black. This stage of infection is commonly referred to as rotten neck/neck rot/neck blast/panicle blast. In early neck infection, grain filling does not occur and the panicle remains erect like a dead heart caused by a stem borer. In the late infection, partial grain filling occurs. Small brown to black spots also may be observed on glumes of the heavily infected panicles. The pathogen caused by yield losses ranged from 30-61 per cent depending upon the stages of infection.

Pathogen

Mycelium of the fungus, is hyaline to olivaceous, septate and highly branched. Conidia are produced in clusters on **long septate, olivaceous slender conidiophores**. Conidia are **pyriform to 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.* It produces perithecia. The ascospores are hyaline, fusiform, 4 celled and slightly curved.

Favourable Conditions

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

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 important sources of inoculum. Irrigation water may carry the conidia to different fields. The

fungus also survives on collateral hosts *viz., Panicum repens, Digitaria magrginata, Brachiaria mutica, Leersia hexandra, Dinebra retroflea, Echinochloa crusgalli* and *Stenotaphrum secondatum.*

Fore casting

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

Management

Grow resistant varieties like **Ponmani, ADT40, Co25 in Samba and Co37, Co43, IR20, Co44, ADT36 and ADT39 in Thaladi seasons**. The varieties viz. Co45, ADT37, Vikas, Jaya, IR62, IR64 and ASD 18 also showed moderate resistance to blast. Avoid cultivation of highly susceptible varieties *viz.,* IR50 and TKM6 and susceptible varieties *viz.,* Ponni, White Ponni and IR36 in disease favourable season. Remove and destory the weed hosts in the field bunds and channels. Treat the seeds with Captan or Thiram or Carbendazim or Carboxin or **Tricyclazole** at 2 g/kg. Seed treatment with biocontrol agent *Trichoderma viride*@ 4g/kg or *Pseudomonas fluorescens* @ 10g/kg of seed. Avoid close spacing of seedlings in the main field. Spray the nursery with Carbendazim 25 g or Edifenphos 25 ml for 8 cent nursery. Spray the main field with Edifenphos 250 ml or Iprobenphos 500 ml or Carbendazim 250 g or Tricyclazole 400 g or Thiophanate Methyl 500 g or Pyroquilon 500 g/ha.

2. <u>Brown Spot or Sesame leaf spot</u>

Helminthosporium oryzae (Syn : *Drechslera oryzae*)

(Sexual stage : *Cochliobolus miyabeanus)*

Symptoms

The fungus attacks the crop from seedling in nursery to milk stage in main field. Symptoms appear as lesions (spots) on the coleoptile, leaf blade, leaf sheath, and glume, being most prominent on the leaf blade and glumes. The disease appears first as **minute brown dots, later becoming cylindrical or oval to circular**. The several spots coalesce and the leaf dries up. The seedlings die and affected nurseries can be often recognised from a distance by their brownish scorched appearance. Dark brown or black spots also appear

on glumes which contain large number of conidiophores and conidia of the fungus. It causes failure of seed germination, seedling mortality and reduces the grain quality and weight.

Pathogen

H. oryzae produces greyish-brown to dark brown septate mycelium. Conidiophores may arise singly or in small groups. They are straight, sometime geniculate, pale to brown in colour. **Conidia are usually curved with a bulge in the centre and tapering towards the ends occasionally almost straight, pale olive green to golden brown colour and are 6-14 septate.** The perfect stage of the fungus is *C. miyabeanus.* It produces perithecia with asci containing 6-15 septate, filamentous or long cylinderical, hyaline to pale olive green ascospores. It produces C25 terpenoid phytotoxins called **ophiobolin A, (or Cochliobolin A), ophiobolin B (or cochliobolin B) and ophiobolin I. Ophiobolin A is most toxic**. These 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 incidence.

Mode of Spread and Survival

The infected seeds are the most common source of primary infection. The conidia present on infected grain and mycelium in the infected tissue may viable for 2 to 3 years. The fungus may survive in the soil for 28 months at 30°C and 5 months at 35°C. 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, Arundo donux,* and *Echinochlora colonum.*

Management

Field sanitation-removal of collateral hosts and infected debris in the field. Crop rotation, adjustment of planting time and proper fertilization are suggested. **Use of slow release nitrogenous fertilizers is advisable.** Grow disease tolerant varieties *viz.,* **Co44, Cauvery, Bala 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 or Captafol 40 g for 8 cents nursery. Spray the crop in the main field with Edifenphos 500 ml or Mancozeb 1 kg or Captafol 625 g/ha.

3. <u>Narrow brown leaf spot</u>

Cercospora janseana

(Sexual stage : *Sphaerulina oryzina)*

Symptoms

The fungus produces **short, linear brown spots mostly on leaves and also on sheaths, pedicels and glumes**. The spots appear in large numbers during later stages of crop growth.

Pathogen

Conidiophores are produced in small groups and brown in colour. Conidia are hyaline or sub hyaline, cylindrical and 3-5 septate.

Management

Spray Carbendazim 250 g or Mancozeb 1 kg/ha.

4. <u>Sheath rot</u>

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.

Pathogen

The fungus produces whitish, sparsely branched and septate mycelium. Condiophore is slightly thicker than the vegetative hyphae. Conidia are hyaline, smooth, single celled and cylindrical in shape.

Favourable Conditions

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

Mode of Spread and Survival

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

Management

Apply recommended doses of fertilizers. Adopt optimum spacing. Spray Carbendazim 250g or Edifenphos 1 lit or mancozeb 1 kg or Chlorothalonil 1 kg/ha at boot leaf stage and 15 days later. Soil application of gypsum in 2 equal splits (500 kg/ha) reduce the sheath rot incidence.

5. Sheath blight

Rhizoctonia solani

(Sexual stage : *Thanetophorus cucumeris)*

Symptoms

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

Pathogen

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

Favourable Conditions

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

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. It infects more than 188 crop species in 32 families. Sclerotia spread through irrigation water.

Management

Avoid excess doses of fertilizers. Adopt optimum spacing. Eliminate weed hosts. Apply organic amendments. Avoid flow of irrigation water from infected fields to healthy fields. Deep ploughing in summer and burning of stubbles. Spray Carbendazim 250 g or Chlorothalonil 1 kg or Edifenphos 1 lit/ha. Seed treatment with *Pseudomonas fluorescens* @ of 10g/kg of seed followed by seedling dip @ of 2.5 kg or products/ha dissolved in 100 litres and dipping for 30 minutes. Soil application of *P.fluorescens* @ of 2.5 kg/ha after 30 days of transplanting (This product should be mixed with 50 kg of FYM/Sand and then applied. Foliar spray at 0.2% concentration commencing from 45 days after transplanting at 10 days interval for 3 times depending upon the intensity of disease.

6. Grain discolouration

Drechslera oryzae, D. rostratum, D.tetramera, Curvularia lunata, Trichoconis 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 discolouration, the extend 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 discolouration 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.

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 bootleaf stage with Mancozeb 1 kg or Iprobenphos 500 ml or Carbendazim 250 g/ha. Store the grains with 13.5-14% moisture content.

7. <u>False smut</u>

Ustilaginoidea virens

(Syn : *Claviceps oryzae - sativa)*

Symptoms

The fungus transforms individual grains into **greenish spore balls of velvetty appearance**. Due to the development of the fructification of the pathogen, the ovaries are transformed into large **velvetty green masses**. Usually only a few spikelets in a panicle are affected.

Pathogen

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

Favourable conditions

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

8. <u>Udbatta disease</u>

Ephelis oryzae

(Sexual stage : *Balansia oryzae-sativa)*

Symptoms

The symptoms are evident at the time of panicle emergence. The infected panicle inside the sheath is matted together by the mycelium. **The entire ear head is converted into a straight compact cylindrical dark spike like structure**. The spikelets are cemented

to the central rachis and the size is remarkeably reduced. No grain is formed. The entire spike is covered by greyish stroma and convex pycnidia are immersed in the stroma.

Pathogen

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

Management

Hot water seed treatment at 45OC for 10 min. effectively controls the disease. Removal of collateral hosts like *Isachne elegans, Eragrostis tenuifolia* and *Cynadon dactylon.*

9. **Stack burn disease**

Trichoconis padwickii

(Syn : *Alternaria padwickii*)

Symptoms

Leaves and ripening grains are affected. In the leaves, circular to oval spots with dark brown margins are formed. The central portion of the spot turns to light brown or almost white and has numerous minute dots. On the glumes reddish brown or pale brown spots appear. The kernels may shrivel and become brittle when severe spotting occurs.

Pathogen

Conidia are elongately fusoid, 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 g/kg. Hot water treatment at 54°C for 15 minutes is also effective. Burn the stubbles and straw in the field.

10. **Bunt or Kernel Smut or black smut**

Tilletia barclayana

Minute black pustules or streaks are produced in the grains which burst open at the time of ripening. The grains may be partially or entirely replaced by the fungal spores. The sorus pushes the glumes apart exposing the black mass of spores. Only a few flowers are infected in an inflorescence. The fungus survives as chlamydospores for one or more years under normal condition and 3 years in stored grains.

11. **Stem rot**

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 sclerotia are seen in the rotting tissues. The culm collapses and plants lodge. If the diseased tiller is opened, profuse

mycelial growth and large number of sclerotia can be seen. The sclerotia may be seen in the stubbles after harvest.

Pathogen

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

Favourable Conditions

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

Mode of Spead of Survival

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

Management

Use of recommended doses of fertilizer. Deep ploughing in summer and burning of stubbles. Avoid flow of irrigation water from infected fields to healthy fields.

12. **Foot rot or Bakanae disease**

Fusarium moniliforme

(Sexual stage : *Gibberella fujikuroi)*

Symptoms

The infected seedings in nursery are lean and lanky, much taller than healthy seedlings and die after some time. In the main field, the affected plants have tall lanky tillers and have 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 micro conidia. Micro conidia are hyaline, single celled and oval in shape. Macro conidia are slightly sickle shaped, narrow at both ends and two to five celled. The fungus produces phytotoxin called 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.

13 Bacterial leaf blight *Xanthomonas oryzae p.v. oryzae*

Symptoms

The disease is usually noticed at the time of heading but in severe cases occur earlier also. Seedlings in the nursery show circular, yellow spots in the margin, later enlarge,

coalesce and cause drying of foliage. **"Kresek"** symptom is seen in seedlings, 1-2 weeks after transplanting. The bacterium enters through the cut wounds in the leaf tips, becomes systemic and cause death of entire seedling.

In grown up plants water soaked, translucent lesions appear usually near the leaf margin. The lesions enlarge both in length and width with a **wavy margin and turn straw yellow within a few days, covering the entire leaf**. As the disease progresses, the lesions cover the entire leaf blade which may turn white or straw coloured. Lesions may also be seen on leaf sheaths in susceptible varieties. Milky or opaque dew drops containing bacterial masses are formed on young lesions in the early morning. They dry up on the surface leaving a white encrustation. The affected grains have discoloured spots surrounded by water soaked areas. **If the cut end of leaf is dipped in water, bacterial ooze makes the water turbid.**

Pathogen

The bacterium is strict aerobe, **gram negative, non spore forming, rod shaped with size ranging from 1-2 x 0.8-1.0um with monotrichous polar flagellum** of 6-8 um. The bacterial cells are capsulated and are joined to form an aggregate mass. Colonies are circular, convex with entire margins, whitish yellow to straw yellow later and opaque.

Favourable Conditions

Clipping of tip of the seedling at the time of transplanting, heavy rain, heavy dew, flooding, deep irrigation water, severe wind, temperature of 25-30OC and 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 like *Leersia spp. Plantago najor, Paspalum dictum,* and *Cyanodon dactylon.* The pathogen spreads through irrigation water in dry season and also through typhoons and rain storms.

Management

Burn the stubbles. Use optimum dose of fertilizers. Avoid clipping of tip of seedling at the time of transplanting. Avoid flooded conditions. Remove weed hosts. Grow resistant cultivars like IR 20 and TKM 6. Spray Streptomycin sulphate and tetracycline combination 300 g + Copper oxychloride 1.25 g/ha.

14. Bacterial leaf streak *Xanthomonas campestris p.v. oryzicola*

Symptoms

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, bacteria ooze out and

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

Management

Similar to bacterial leaf blight.

15. Tungro disease Virus

Symptoms

Infection occurs both in the nursery and in the main field. Plants are markedly stunted. Leaves show yellow to orange discoloration and interveinal chlorosis. Yellow discoloration is commonly seen in "Japonica" varieties, while "Indica" varieties show orange discoloration. Young leaves are often mottled with pale green to whitish interveinal stripes. The plants may be killed if infected early. Tillering is reduced with poor root system. The infected plants have few spikelets and panicles are small with discoloured grains. Tungro infected plants can be chemically identified by **Iodine Test.** Ten cm long leaf tip is cut in the early morning before 6 A.M. and dipped in a solution containing 2 g Iodine and 6 g Potassium Iodide in 100 ml of water for 30 minutes. Tungro infected leaves show dark blue streaks.

Pathogen

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 130 x 30 nm made up of a single species of coat protein of MW 36 K and a single molecule of circular double - standed DNA of 8.3 KbP. RTSV has a isomatric capsid, 30 nm in diameter comprising two to three polypeptide species and a single speices of polyadenylated single - standed RNA of about 10 KbP.

Mode of Spread and Survival

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. The leafhoppers viz, *Nephotettix virescens, N. nigropictus, N. parvus, N.malayanus* and *Recilia dorsalis* transmit the virus in a non-persistent manner. There is no latent period in the vector and infectivity is retained for a maximum period of 6 days after acquisition of the virus.

Management

Summer deep ploughing and burning of stubbles. Destory weed hosts of the virus and vectors. Grow disease tolerant cultivars like IR50 and Co45. Control the vectors in the nursery by application of carbofuran 170 g/cent, 10 days after sowing. Spray Phosphamidan 500 ml or Fenthion 500 ml or Monocrotophos 1 lit/ha or Neem oil 3 per cent in the main field 15 and 30 days after transplanting to control leaf hoppers.

Grassy stunt Virus

The infected plants are markedly stunted and have excessive tillering and has an erect growth habit. The leaves become short, narrow, pale green or pale yellow and have numerous small dark brown spots. On older leaves these spots spreading give a bronze colour to the plants. The plants may produce a few small panicles which bear dark brown and unfilled grains.

Mode of Spread and Survival

The virus is transmitted in a persistent manner by the **brown planthopper, *Nilaparvata lugens*.** It has a latent period of 5 to 28 days in the vector.

16. Rice dwarf Virus

Symptoms

The virus infected plants show marked stunted growth with chlorotic or whitish specks on the leaves. The size of specks varies often and form interrupted streaks along the veins and distal part of infected leaves show diffuse yellowing. The number of tillers may be reduced with retarded growth. The diseased plants may survive until harvest time, remaining more or less green. Plants which are infected at early stage produce no eartheads, if produced, may have small unfilled grains.

Pathogen

The partially purified virus particle have a hexagonal outline and are 70 nm in diameter with a surrounding envelope. The virus particle contains double stranded RNA or of double helical in nature.

Favourable Conditions

High population of *Nephotettix cincticeps, Recllia dorsalis* and *N. nigropictus.* The presence of gramineous weeds like *Echinochloa crusgalli, Glyceria acutifolia* and *Panicum miliaceaaum.*

Mode of Spread and Survival

The virus is found to survive in the gramineous weeds. The virus is transmitted through the egg masses of leafhoppers from one generation to another. (Trans ovarial transmission).

Management

Destory the weed host which harbour the virus and the vectors. Spray Phosphamidon or Fenthinon 500 ml or Monocrotophos 1 lit/ha.

Ragged stunt Virus

Symptoms

Formation of ragged leaves with irregularly edged portions, stunting of plants, vein swelling, delay in flowering, production of nodal branches and incomplete emergence of panicles are the main diagnostic symptoms.

Mode of Spread and Survival

The virus is transmitted in a persistent manner by the brown planthopper, *Nilaparvata lugens* but not through seed. It has a latent period of 3 to 35 days in the vector.

17. Rice yellow dwarf

Mycoplasma Like Organism (MLO)

Symptoms

The infected plants are stunted and have yellowish green to whitish green leaves. There is excessive tillering and leaves became soft and droop slightly. Plants are usually sterile but some may produce small panicles with unfilled grains. If plants are infected early they usually die before maturity, and even if they do survive no panicles are produced or only a small number with no grains. With later infection there may be little sign that the plant is diseased, but regrowth from stubble will show typical chlorosis.

Mode of Spread and Survival

The MLO is transmitted by *Nephotettix virescens* and *N. nigropictus* with a latent period of 25-30 days. It survives on several grass weeds.

Management

Deep ploughing during summer and burning of stubbles. Rice varieties like IR62 and IR64 are resistant to the disease. The management practices followed for Rice Tungro disease may be adopted for this disease also.

DISEASES OF MAIZE

(Zea mays)

1. Downy mildew/Crazy top

Peronosclerospora sorghi

Symptoms

The most characteristic symptom is the development of chlorotic streaks appear on the leaves and the plants exhibit a stunted and bushy appearance due to the shortening of the internodes. White downy growth can be seen not only on the lower surface of leaf but also on the chlorotic streaks. The downy growth also occurs on bracts of green unopened male flowers in the tassel. Sometimes miniature to large leaves has been noticed in the tassel. These leaves bear well defined downy growth. **Proliferation of auxillary buds on the stalk of tassel as well as the cobs is very common (Crazy top).**

Pathogen

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

Favourable Conditions

Low temperature (21-33OC), high relative humidity, (90 per cent), water logging condition and light drizzling. Young plants are highly susceptible.

Mode of Spread and Survival

The primary source of infection is through the oospores present in the soil and also the dormant mycelium present in the infected maize seeds. Secondary spread is through air-borne conidia. The oospores survive in the soil as well as in the infected plant debris.

Management

Deep ploughing. Crop rotation with pulses. Adjust the time of sowing. Rogue out infected plants. Treat the seeds with Metalaxyl compound (Apron 35SD) at 6g/kg. Spray the crop with Metalaxyl + Mancozeb 2kg or Mancozeb 1.25 kg/ha on 20th day after sowing. Grow resistant varieties and hybrids *viz.* Co1, CoH1 and CoH2.

2. Leaf blight

Helminthosporium maydis

(Syn : H. turcicum)

Symptoms

The fungus affects the maize plant at young stage. Small yellowish round to oval spots are seen on the leaves. The spots gradually increase in area into bigger elliptical spots and are straw to grayish brown colour in the centre with dark brown margins. The spots coalesce to form bigger spots and gives blighted appearance. The surface is covered with olive green velvetty masses of conidia and conidiophores.

Pathogen

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

Favourable Conditions

Optimum temperature for the germination of conidia is 8 to 27OC provided with free water on the leaf. Infection takes place early in the wet season.

Mode of Spread and Survival

It is a seed-borne fungus. It also infects sorghum, wheat, barely, oats, sugarcane and spores of the fungus are also found to associate with seeds of green gram, black gram, cowpea, varagu, Sudan grass, Johnson grass and Teosinte.

Management

Treat the seeds with Captan or Thiram at 4 g/kg. Spray Mancozeb 1.25 kg or Captan 1 kg/ha.

3. **Rust** *Puccinia sorghi*

Symptoms

Circular to oval, elongated cinnamon-brown powdery pustules are scattered over both surface of the leaves. As the plant matures, the pustules become brown to black owing to the replacement of red uredospores by black teliospores.

Pathogen

Uredospores are globose or elliptical finely echinulate, yellowish brown with 4 germ pores. Teliospores are brownish black, or dark brown, oblong to ellipsoidal, rounded to flattened at the apex. They are two celled and slightly constricted at the septum and the spore wall is thickened at the apex.

Favourable Conditions- Cool temperature and high relative humidity.

Mode of Spread and Survival

Presence of alternate hosts *viz., Oxalis corniculata* and *Euchlaena mexicana.*

Management

Remove the alternate hosts. Spray Mancozeb at 1.25 kg/ha.

4. **Head smut-** *Sphacelotheca reiliana*

Symptoms

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. Under such conditions the Individual spikelets on tassels may form shoot like growths, or the entire tassel may develop into leaf like structures. The smutted plants are stunted in growth, produce little yield and remain greener than that of the rest of the plants.

Pathogen

Smut spores (chlamydospores) are produced in large numbers which are reddish brown to black, thick walled, finely spined, spherical or slightly irregular.

Favourable Conditions

Low temperature favours more infection and this fungus also infects the sorghum crop and *vice versa.*

Mode of Spread and Survival

The smut spores retains its viability for two years. The fungus is externally seed-borne and soil-borne. The major source of infection is through soil-borne chlamydospores.

Management

Field sanitation. Crop rotation with pulses. Treat the seeds with Captan or Thiram at 4 g/kg.

5. **Charcoal rot** *Macrophomina phaseolina (Rhizoctonia bataticola)*

Symptoms

The fungus attacks roots of seedlings and young plants. The affected plants first exhibit the wilting symptoms. The stalk of the infected plants can be recognized by grayish streaks and become weak. The pith become shredded and grayish black minute sclerotia develop abundantly on the vascular bundles. Shreading of the interior of the stalk often causes stalks to break in the region of the crown. The crown region of the infected plant become dark in colour. Shreadding of root bark and disintergration of root system are the common features.

Pathogen

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

Favourable Conditions- High temperature and low soil moisture (drought)

Mode of Spread and Survival

The fungus has a wide host range, attacking sorghum, cumbu, ragi and pulses. It survives for more than 16 years in the infected plant debris. The primary source of infection is through soil-borne sclerotia.

Management

Long crop rotation with crops that are not natural host of the fungus. Irrigate the crops at the time of earhead emergence to maturity. Treat the seeds with Carbendazim or Captan at 2 g/kg. Grow disease tolerant varieties viz., SN-65, SWS-8029, Diva and Zenit.

Minor diseases

Brown spot *Physoderma maydis.* Water soaked lesions, which are oval, later turn into light green and finally brown.

Stalk rot *Erwinia dissolvens.* Dark brown lesion and rotting of stalk at crown level about 6”-12” from ground level. Lodging of plants.

Mosaic Virus. Formation of mosaic symptoms. Plants produce smaller ear head and immature seeds

DISEASES OF SORGHUM

(Sorghum bicolor)

1. Downy Mildew

Peronosclerospora sorghi

Symptoms

The fungus, *Peronosclerospora sorghi* is the cause of a systemic downy mildew of sorghum. It invades the growing points of young plants, either through oospore or conidial infection and as the leaves unfold they show various types of symptoms. The first few leaves that show symptoms are only partially infected with green or yellow colouration of the infected portion. Abundant downy white growth is produced on the lower surface of the leaves, consists of conidiophores and conidia. Normally three or four leaves develop the chlortic downygrowth type of symptoms. Subsequent leaves show progressively more of a complete bleaching of the leaf tissue, sometimes in streaks or stripes. As the infected bleached leaves mature they become necrotic and the interveinal tissues disintegrate, releasing the resting spores (oospores) and leaving the vascular bundles loosely connected to give the typical shredded leaf symptom.

Pathogen

P. sorghi is systemic in young host plant in the form of intercellular, non-septate mycellium. It is an obligate parasite. Conidiophores emerge through the stomata in single or in clusters which are stout and dichotomously branched. Conidia are single celled, hyaline, globose and thinwalled. Oospores are typically produced abundantly in parallel bands between fibro vascular strands of the shredded leaf tissue which are more or less round, thickwalled and deep brown in colour.

Favourable Conditions

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

Mode of Spread and Survival

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

Management

Crop rotation with other crops like pulses and oilseeds. Avoid the secondary spread of the disease by roguing out the affected plants since the wind plays an important role in the secondary spread of the disease from the infected plants. Grow moderately resistant

varieties like Co25 and Co26. Seed treatment with Metalaxyl (Apron 35 SD) at 6 g/kg of seed. Spray Metalaxyl 500 g or Mancozeb 1 kg or Ziram 1 kg or Zineb 1 kg/ha.

2. Leaf blight

Exerohilum turcicum

(Syn : *Helminthosporium turcicum*)

Symptoms

The leaf blight pathogen also causes seed rot and seedling blight of sorghum. The disease appears in the form of small narrow elongated spots in the initial stage. But in due course they extend along with the length of the leaf becoming bigger. On older plants, the typical symptoms are long elliptical necrotic lesions, straw coloured in the centre with dark margins. The straw coloured centre become darker during sporulation. The lesions can be several centimeters long and wide. Many lesions may develop and coalesce on the leaves, destroying large areas of leaf tissue, giving the crop a distinctly burnt or blasted appearance.

Pathogen

The mycelium is localised in the infected lesion. Conidiophore emerges through stomata and are simple, olivaceous, septate andgeniculate type. Conidia are olivaceous brown, 3-8 septate and thick walled.

Favourable Conditions

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

Mode of Spread and Survival

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

Management

Use disease free seeds. Treat the seeds with Captan or Thiram at 4 g/kg. Spray Mancozeb 1.25 kg or Captafol 1 kg/ha.

3. Rectangular Leaf spot

Cercospora sorghi

Symptoms

The symptoms appear as small leaf spots which enlarge to become rectangular lesions (which can be 5-15 mm long by 2 to 5 mm wide) on the leaf and leaf sheath. Usually the lower leaves are first attacked. The lesions are typical dark red to purplish with some what lighter centres. The lesions are mostly isolated and limited by veins. Severe spotting results in premature drying of leaves. The colour of the spots vary from red, purple, brown or dark depending upon the variety.

Pathogen

Mycelium of the fungus is hyaline and septate. Conidiophores emerge in clusters through stomata, which are brown and simple, rarely branched. Conidia are hyaline, thin walled, 2-13 celled and long obclavate.

Favourable Conditions

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

Mode of Spread and Survival

The conidia survive upto 5 months. The disease spreads through air-borne and seed-borne conidia.

Management

Use disease free seeds. Treat the seed with Captan or Thiram at 4 g/kg. Spray Mancozeb 1.25 kg or Captafol 1kg/ha.

4. <u>Anthracnose and red rot</u>

Colletotrichum graminicolum

Symptoms

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

Pathogen

The mycelium of the fungus is localised in the spot. Acervuli with setae arise through epidermis. Conidia are hyaline, single celled, vacuolate and falcate in shape.

Favourable Conditions

Continuous rain, temperature of 28-30OC and high humidity.

Mode of Spread and Survival

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

Management

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

5. Rust

Puccinia purpurea

Symptoms

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

Pathogen

The uredospores are pedicellate, elliptical or oval, thin walled, echinulated and dark brown in colour. The teliospores are reddish or brown in colour and two celled, rounded at the apex with one germ pore in each cell. The teliospores germinate and produce promycelium and basidiospores. Basidiospores infect *Oxalis corniculata* (alternate host) where pycnial and aecial stages arise after infection.

Favourable Conditions

Low temperature of 10 to 12OC favours teliospore germination and a spell of rainy weather favours the onset of the disease.

Mode of Spread and Survival

The uredospores survive for a short time in soil and infected debris. Presence of alternate host helps in perpetuation of the fungus.

Management

Remove the alternate host *Oxalis comiculata.* Spray the crop with Mancozeb at 1.25 kg/ha.

6. Grain smut/Kernel smut / Covered smut / Short smut

Sphacelotheca sorghi

Symptoms

The individual grains are replaced by smut sori which can be localized at a particular part of the head or occur over the entire inflorescence. The sori are oval or cyclindrical and are covered with a tough white cream to light brown skin (peridium) which often persists unbroken upto threshing. The size, colour and degree of breakage of the sori varies considerably with race of the fungus and the sorghum cultivar. Ratoon crops exhibit higher incidence of disease.

Loose smut/Loose kernel smut

Sphacelotheca cruenta

Symptoms

The affected plants can be detected before the ears come out. They are shorter about a foot than the healthy plants with thinner stalks and marked tillering. The ears come out much earlier than the healthy. The glumes are hypertrophied and the earhead gives a loose appearance than healthy. The sorus is covered by a thin membrane which ruptures very early, exposing the spores even as the head emerges from the sheath. The size of the sorus varies with the variety of the host.

Long smut

Tolyposporium ehrenbergii

Symptoms

The presence of long smut can be discovered only by a close examination of the ears in the field. This disease is normally restricted to a relatively a 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 among which are found several darkbrown filaments which represent the vascular bundles of the infected ovary.

Head smut

Sphacelotheca reiliana

Symptoms

The head is completely replaced by a large gall (sorus). The galls are at first covered by a whitish grey membrane of fungal tissue, which ruputres, often before the head emerges from the boot leaf, to expose a mass of brown-black powder (smutspores) along which are embedded long, thin, darkcoloured filaments which are the vascular bundles of the infected head. Sometimes smaller sori develop on the leaves and lower part of the peduncle.

Management for all smuts

Treat the seed with Captan or Thiram at 4 g/kg. Use disease free seeds. Follow crop rotation. Collect the smutted ear heads in cloth bags and dip in boiling water.

Ergot or Sugary disease

Sphacelia sorghi

Symptoms

The disease is confined to individual spikelets. The first symptom is the secretion of honey dew (creamy stickly liquid) from infected florets. Under favourable conditions, long, straight or curved, cream to light brown, hard sclerotia develop. At the base of the affected plants white spots can be seen on the soil surface, denoting the drops of honey dew which

had fallen on the soil. Often the honey dew is colonised by *Crerebella sorghivulgaris* which gives the head a blackened appearance.

Pathogen

The fungus produces septate mycelium. The honey dew is a concentrated suspension of conidia, which are single celled, hyaline, elliptic or oblong in shape and slightly constricted in the middle.

Favourable Conditions

A period of high rainfall and high humidity during flowering season. Cool night temperature and cloudy weather aggravate the disease.

Mode of Spread and Survival

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

Management

Adjust the date of sowing so that the crop does not flower during September-October when high rainfall and high humidity favour the disease. Spray any one of the following fungicides at emergence of earhead (5-10 per cent flowering stage) followed by a spray at 50 per cent flowering and repeat the spray after a week, if necessary. Ziram (or) Zineb (or) Mancozeb (or) Carbendazim at 1 kg/ha.

7. <u>Head mould/Grain mould/Head blight</u>

More than thirty two genera of fungi were found to occur on the grains of sorghum.

Symptoms

If rains occur during the flowering and grain filling stages, severe grain moulding can occur. Fungi from many genera have been isolated from the infected sorghum grains and the most frequently occurring genera are *Fusarium, Curvularia, Alternaria, Aspergillus* and *Phoma. Fusarium semitectum* and *F.moniliforme* develop a fluffy white or pinkish colouration. *C. lunata* colours the grain black. Symptom varies depending upon the organism involved and the degree of infection.

Favourable Conditions

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

Mode of Spread and Survival

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

Management

Adjust the sowing time. Spray any one of the following fungicides in case of intermittent rainfall during earhead emergence, a week later and during milky stage. Mancozeb 1 kg/ha. or Captan 1 kg + Aureofungin-sol 100 g/ha.

8. Phanerogamic parasite

Witch Weed

Striga asiatica and Striga densiflora

It is a partial root parasite and occurs mainly in the rainfed sorghum. It is a small plant with bright green leaves, grows upto a height of 15-30 cm. It always occurs in clusters of 10-20/host plant. *S. asiatica* produces red to pink flowers while. *S. densiflora* produces white flowers. Each fruit contains minute seeds in abundance which survive in the soil for several years. The root exudates of sorghum stimulate the seeds of the parasite to germinate. The parasite then slowly attach to the root of the host by haustoria and grow below the soil surface and produce underground stems and roots for about 1-2 months. The parasite grows faster and appears at the base of the plant. Severe infestation causes yellowing and wilting of the host leaves. The infected plants are stunted in growth and may die prior to seed setting.

Management

Regular weeding and interculture operation during early stages of parasite growth. Spray Fernoxone (sodium salt of 2, 4-D) at 450g/500 litre of water.

DISEASES OF PEARL MILLET/CUMBU

(Pennisetum americanum)

1. Downy mildew

Sclerospora graminicola

Symptoms

Infection is mainly systemic and symptoms appear on the leaves and the inflorescenses. The first symptoms can appear in seedlings at three to four leaf stage. The affected leaves show patches of light green to light yellow colour on the upper surface of leaves and the corresponding lower surface bears white downy growth of the fungus. The yellow discolouration often turns to streaks along veins. The downy growth seen on infected leaves consists of sporangiophores and sporangia. As a result of infection young plants dry and die ultimately. Symptoms may appear first on the upper leaves of the main shoot or the main shoot may be symptom free and symptoms appear on tillers or on the lateral shoots.The inflorescences of infected plants can be completely or partially malformed with florets converted into leafy structures, the ear head gives the typical symptom of green ear. Infected leaves and inflorescences produce sporangia over a considerable period of time under humid conditions and necrosis begins. The dry necrotic tissues from infected plant contain masses of oospores.

Pathogen

The mycelium is systemic, nonseptae, intercellular in the parenchymatous tissues. Short, stout, hyaline sporangeiophores arise through stomata and branch irregularly, ends with characteristic stalks bearing the sporangia. Sporangia are hyaline, thin walled and elliptical, bear prominent papilla. Oospores are round in shape, surrounded by a smooth, thick and yellowish brown wall.

Favourable Conditions

Very high humidity (90 per cent), presence of water on the leaves and low temperature of 15-25OC favour the formation of sporangiophore and sporangie. Under such conditions, 35,000 sporangia are liberated from one sq. inch of leaf area.

Mode of Spread and Survival

The oospores remain viable in soil for 5 years or longer giving rise to the primary infection on the host seedling. Secondary spread is through sporangia which are produced during rainy season. It is also believed that the dormant mycelium is present in embryo of infected seeds.

Management

Deep ploughing so as to bury the oospore deeply. Rogue out infected plants. Adopt crop rotation. Use excess seed rate. Grow resistant varieties like X5, WCC-75 and Co7. Treat the seeds with Metalaxyl (Apron 35SD) at 6g/kg. Spray Mancozeb 1kg or Metalaxyl+Mancozeb (Ridomil MZ) at 2kg/ha on 20th day after sowing in the field.

2. Smut *Tolyposporium penicillariae*

Symptoms

The pathogen infects few florets and transforms them into plump sacs (sori) containing black powder (smut spores). In the early stage, the sori are larger and greener than normal healthy grains and when the sori mature they become dark brown and are easily broken and release millions of black smut spore balls.

Pathogen

The fungus mostly confined to the sorus. The sori contains spores which are usually in balls and are not easy to separate. Each spore is angular or round and light brown coloured.

Favourable Conditions

High humidity and successive cropping with cumbu.

Mode of Spread and Survival

It survives spore balls in the soil and serve as primary source of inoculum. The secondary spread is by air-borne conidia.

Management

The damage caused by the fungus is negligible. However, removal and destruction of affected earhead will help in controlling the disease.

3. Rust

Puccinia penniseti

Symptoms

Symptoms first appear mostly on the distal half of the lamina. The leaf soon becomes covered by uredosori which appear more on the upper surface. Sometimes, the necrotic spots appear around the group of pustules. The pustules may be formed on leaf sheath. stem and even on peduncles. Later, telial formation takes place on leaf blade, leaf sheath and stem. While brownish uredinia get exposed at maturity, the black telia remain covered by the epidermis for a longer duration.

Pathogen

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

Favourable Conditions

Closer spacing. Presence of abundant brinjal plants and other species of *Solanum* viz., *S.torvum, S. xanthocarpum* and *S. pubescents.*

Mode of Spread and Survival

Air-borne uredospores are the primary sources. The uredial stages also occur on several species of *Pennisetum.*

Management

Spray with Wettable Sulphur 2.5 kg or Captafol 1 kg or Mancozeb 1.25 kg/ha.

4. Ergot or Sugary disease

Claviceps fusiformis

Symptoms

The symptom is seen by exudation of small droplets of light pinkish or brownish sticky fluid (honey dew) from the infected spikelets. Under severe infection many such spikelets exude plenty of honey dew which trickle along the earhead. This attracts several insects. In the later stages, the infected ovary turns into small dark brown sclerotium which is just projecting out of the spikelet.

Pathogen

The pathogen produces septate mycelium which produce conidiophores and are closely arranged. Conidia are hyaline and one celled. The sclerotia are small and dark grey but white inside. Sclerotia are 3-8 mm long and 0.3-15 mm broad.

Mode of Spread and Survival

Sclerotia are viable in soil for 6-8 months. The primary infection takes place by germinating sclerotia present in the soil. Secondary spread is by insects or air-borne conidia. 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 September when high rainfall and high relative humidity favour the disease spread. Immerse the seeds in 10 per cent common salt solution and remove the floating sclerotia. Remove collateral hosts. Spray with Carbendazim 500g or Mancozeb 1.25 kg or Ziram 1kg/ha when 5-10 per cent flowers have opened and again at 50 per cent flowering stage.

Minor diseases

Grain mould Fusarium and *Curvularia* spp. Complex of several fungi.

Grains covered with white, pink or black moulds.

Blast *Pyricularia setariae.* Diamond shaped to circular lesions with dark brown margins and chlorotic haloes.

Zonate leaf spot Gloeocercospora sp. Rough circular lesions with alternating concentric bands of straw and brown colour, often coalescing over the leaf surface.

Banded leaf *Rhizoctonia* spp. Patch work of light and dark and Sheath discoloured areas often bearing fluffy to light Brown fungal mats

DISEASES OF FINGER MILLETS AND SMALL MILLETS

RAGI *(Eleusine coracana)*

1. Blast

Pyricularia grisea

Symptoms

The pathogen attacks the crop from seedling stage to the time of grain formation. The lesions are spindle shaped and are of different size. In the beginning, the spots have yellow margin with grayish green centre. Under humid conditions, an olive grey overgrowth of fungus can be seen on the centre of the spot. Later the centre become whitish grey and disintegrate. The lesions on the seedlings are about 0.3 to 0.5 cm in breath and 1-2 cm in length. Stem infection causes blackening of the nodal region. Maximum damage is caused by the neck infection. The neck region turns black and shrink. 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 shrivelled grains are formed.

Pathogen

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

Favourable Conditions

Optimum temperature is 25-30OC. High relative humidity (92-95 per cent) and continuous drizzles. Presence of collateral hosts like Tenai, bajra, wheat, barley and oats.

Mode of Spread ans Survival

The fungus is seed-borne and the primary infection takes place through the seed-borne conidia and also the other crops serve as source of inoculum. The secondary spread is through air-borne conidia.

Management

Treat the seeds with Captan or Thiram at 4g or Carbendazim at 2 g/kg. Grow moderately resistant varieties like Co10, Co11, Co12, Co13, PR202 and HR374. Spray with Iprobenphos (IBP) or Edifenphos 500 ml or Carbendazim 250 g/ha, first spray immediately after noticing the symptoms and second spray at flowering stage.

2. Seedling blight or Leaf blight

Helminthosporium nodulosum

(Sexual stage : *Cochliobolus nodulosum*)

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 affect 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. *C. nodulosum* produces spherical perithecia and asci contain 1 to 8 ascospores.

Favourable Conditions

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

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 and black in colour.

Favourable Conditions

High soil moisture and high temperature (more than 30OC)

Mode of Spread and Survival

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

Management

Spot drench with Copper oxychloride at 0.25 per cent. Crop rotation with non host plants.

4. **Mottle streak and streak Virus**

Symptoms

The virus affected plants are stunted and pale green in colour. Chlorotic streaks are formed on the leaves and it is continuous in the case of streak and it is discontinous in mottle streak. Early infection leads to reduction of tillers and grain formation.

Favourable Conditions

The disease is high in April-May sown crops due to high population of vectors *viz., Cicadulina bipunctella* and *C. cinai.*

Management

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

Minor diseases

Downy mildew *Sclerospora macrospora* Green ear symptom. Proliferation of spikeles.

Smut *Melanopsicum eleusinis* Grains are converted into enlarged greenish to dirty black sori

Bacterial disease *Xanthomonas eleusineae* Minute red leaf spots. Stunted growth and ears are not formed.

Phanerogamic parasite *Striga asiatica*

TENAI *(Setaria italica)*

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 *Dectylotacnium aegyptium.*

Management

Treat the seeds with Captan or Thiram 4 g Carbendazim 2 g/kg. Spray the crop with Iprobenphos (IBP) 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. Some times lesions also appear as blotches and the rotting of the secondary roots may also occur.

Pathogen

The conidiophores are simple, erect, cylindrical, brown, slightly swollon 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 germpores. 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.

5. <u>Downy mildew or Green ear</u>

Sclerospora graminicola

Symptoms

Primary infection causes chlorosis of the plant and the leaves turn whitish. The terminal spindle fails to unroll, becomes chlorotic and later turn brown and get shredded. Whitish bloom of sporangiophores and sporangia develop on the surface of the affected leaves under humid conditions. The affected plants rarely comes to flowering. If the infection is mild, the plants may develop ears but the floral parts are proliferated into green leafy structures called green ear.

Pathogen

The sporangiophores are quite and branch heavily. The sporangia are hyaline, broadly fusiform or ovate in shape. Oospores are also produced in the infected host tissue. They are spherical with smooth well and dark brown in colour.

Mode of Spread and Survival

Primary infection is mainly from soil-borne oospores or from oospores on the grains. The oospores are able to survive upto 8-10 years.

Favourable Conditions

Rainy weather, low temperature (15-25OC), high humidity (90 per cent) and high soil moisture.

Management

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

KUTHIRAIVALI *(Echinochloa frumentacea)*

1. **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.

VARUGU *(Paspalum scrobiculatum)*

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 epispore.

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.

PANIVARAGU *(Panicum miliare)*

1. **Rust**

Uromyces linearis

Symptoms

Numerous, narrow, minute, brown pustules arranged in linear rows appear on the upper surface of the leaves.

Pathogen

Uredia are erumpent and brown in colour. The fungus also attacks the other hosts like *Panicum ripens* and *P. antidotale.* The fungus is spread through air-borne uredospores.

Leaf spot

*Helminthosporium oryzae*The fungus which attacks the rice also attacks this crop and produce brown rectangular spots.

SAMAI *(Panicum miliaceum)*

1. **Smut** *Sphacelotheca destruens*

Symptoms

The entire inflorescence is converted into a sorus containing spores (Chlamydospores) and fibrous vascular bundles. The sorus is covered by a white or grey membrane. Abnormal development of hairs is evident on the leaf sheaths of infected plants. The smut spores are round or angular and yellowish brown.

Mode of Spread and Survival

Externally seed-borne and survive for more than 8 years.

DISEASES OF PIGEON PEA/REDGRAM

(Cajanus cajan)

1. Wilt

Fusarium oxysporum f. sp. udum

Symptoms

The diseases may appear from early stages of plant growth (4-6 week old plant) upto flowering and podding. The disease appears as gradual withering and drying of plants, as if they were suffering from drought. In the beginning, yellowing of leaves and blackening of portion of stem appear, starting from collar to branches which gradually result in drooping and premature drying of leaves, stems, branches and finally death of plant. Vascular tissues exhibit brown discolouration. Often only one side of the stem and root system is affected resulting in partial wilting.

Pathogen

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

Favourable Conditions

Soil temperature of 17-25OC. Continuous cultivation of redgram in the same field.

Mode of Spread and Survival

The fungus survives in the infected stubbles in the field. The primary spread is by soil-borne chlamydospores and also by seed contaminant. Chlamydospores remain viable in soil for 8-20 years. The secondary spread in the field is through irrigation water and implements.

Management

Treat the seeds with *Trichoderma viride* at 4 g/kg. Avoid successive cultivation of redgram in the same field. Follow long crop rotation with tobacco. Adopt mixed cropping of sorghum in the field. Grow resistant lines like ICP 8862, ICPL 88046, ICPL 227, BWR 254, DPPA 85-14, DPPA 85-15 and GPS 52.

2. Dry root rot

Macrophomina phaseolina

(Sclerotial stage : *Rhizoctonia bataticola)*

Symptoms

The disease occurs both in young seedlings and grown up plants. Infected seedlings can show reddish brown discoloration at collar region. The lower leaves show yellowing, drooping and premature defoliation. The discolored area later turns to black and sudden death of the plants occur in patches. The bark near the collar region shows shredding. The 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. The fungus also produce dark discoloration of

sub-epidermal tissue in lower part stem. Large number of brown dots seen on the stem portion represent the pycnidial stage of the fungus.

Pathogen

The fungus produces dark, brown, filamentous hyphae and constrictions are seen in hyphal branches at the junction with main hyphae. Sclerotia are jet black, smooth, hard, minute, globose and 110-130u in diameter. The pycnidia are dark brown and ostiolated. Conidiophores (phialides) are hyaline, short, obpyriform to cylindrical, develop from the inner walls of the pycnidium. The conidia (Pycnidiospores) are hyaline, single celled and ellipsoid to ovoid.

Favourable Conditions

Prolonged drought followed by irrigation. High temperature of 28-35OC.

Mode of Spread and Survival

The primary spread of the disease is by seed borne sclerotia. Secondary spread is by soil-borne sclerotia and 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 4 g/kg or pellet the seeds with *Trichoderma viride* at 4 g/kg. Apply heavy doses of farm yard manure or green leaf manure like *Gliricidia maculata* at 10 t/ha or Apply Neemcake at 250 kg/ha. Grow resistant varieties like Co4.

3. **Powdery mildew** *Leveillula taurica*

Symptoms

White powdery growth of the fungus can be seen on the lower surface of leaves. The corresponding areas in upper surface show pale yellow discoloration. The white powdery mass consists conidiophores and conidia of the fungus. In severe cases, the white growth can be seen on the upper surface also. The severe infection of the fungus leads to premature shedding of leaves and plant remains barren.

Pathogen

The fungus is intercellular and absorbs nutrition through haustoria. The conidiophores, which arise through stomata, are hyaline, long, non septate, slender and rarely branched and bear single conidium at the tip. The conidia are hyaline, single celled and elliptical or clavate. The fungus also produce black, globose cleistothecia with simple myceloid appendages. They contain 9-20 cylindrical asci. Each ascus contains 3-5 ascospores which are also hyaline and unicellular.

Favourable Conditions

Dry humid weather following rain fall.

Mode of Spread and Survival

The fungus survives in the soil as cleistothecia and ascospores from asci infect the

first lower most leaves near the soil level. Secondary spread is by air-borne conidia.

Management

Spray Carbendazim 500g or Wettable sulphur 1.5 kg/ha at the initiation of the disease and repeat after 15 days.

4. **Stem blight -** *Phytophthora drechsleri f. sp. cajani*

Symptoms

Initially purple to dark brown necrotic lesions girdle the basal portion of the stem and later may occur an aerial parts of the seedlings. Initially lesions are small and smooth, later enlarging and slightly depressed. Infected tissue become soft and whole plant wilts. In the adult plants, infection is mostly confined to basal portions of the stem. The infected bark become brown and the tissue softening causing the plant to collapse. The infected branches may break off in wind. The upper portions of the infected twigs eventually wilt and dry. In leaf, localized yellowing starts from the tip and margin and gradually extends towards the mid-rib. The centre of the spots later turn brown and hard. The spots increase in size and cover a major portion of the lamina, leading to drying.

Pathogen

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

Favourable Conditions

Soils with poor drainage, low lying areas, heavy rain during the months of July-September and high temperature (28-30OC).

Mode of Spread and Survival

The fungus survives in the soil and plant debris in the form of oospores. Primary infection is from oospores and secondary spread by zoospores from sporangia. Rain splash and irrigation water help for the movement of zoospores.

Management

Treat the seeds with Metalaxyl at 7 g/kg. Spray Metalaxyl at 500 g/ha. Adjust the sowing time so that crop growth should not coincides with heavy rainfall.

5. **Leaf spot** *Cercospora indica*

Symptoms

Small, light brown coloured spots appear on leaves. The spots later become dark brown and the infected portions drop off leaving shot hole symptoms. When several spots join together, irregular necrotic blotches develop and premature defoliation occurs. In severe cases, black lesions develop on petioles and stem.

Pathogen

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.

Mode of Spread and Survival

The fungus survives in the infected plant tissues. The disease is spread by air-borne conidia.

Management

Remove the infected plant debris and destroy. Spray Mancozeb 1 kg or Carbendazim 500 g/ha soon after the appearance of symptom and repeat after a fortnight.

6. Sterility Mosaic Virus

Symptoms

The disease attack can be seen in all stages of crop growth. Leaves show typical mosaic mottling symptoms. Yellow patches intermingle with green colour of leaf. The green portions exhibit puckering symptom. In severe cases, leaves become smaller and cluster near tip because of shortened internodes and stimulation of auxillary buds. The plants are generally stunted and do not produce pod. Plants infected at early stages (upto 45 days) of crop growth show near complete sterility and yield loss upto 95 per cent. As plants become older (after 45 days), their susceptibility to the disease decreases and such plants show partial sterility. If pods develop, the seeds may be small, shrivelled and immature. Because the infected plants show sterility and the leaves show the 'mosaic' symptom, the name of the disease is sterility mosaic.

Mode of Spread and Survival

The disease is transmitted by an Eriophyid mite *Aceria cajani.* The self-sown redgram plants and perennial types of redgram serve as sources of infection.

Management

Rogue out infected plants upto 40 days after sowing. Spray Monocrotophos at 500 ml/ha soon after appearance of the disease and if necessary, repeat after 15 days. Grow resistant genotypes/cultivars like ICP 7035, VR3, Purple 1, DA11, DA32, ICP 6997, Bahar, BSMR 235, ICP 7198, PR 5149, ICP 8861 and Bhavanisagar 1.

7. Yellow Mosaic Virus

Symptoms

Small yellow patches or spots appear on young leaves. The area of yellow discoloration slowly increases and newly formed leaves may completely turn yellow. Infected plants are stunted and mature later and produce very few flowers and pods. The pods are small and distorted. The early infection of virus leads to heavy yield loss.

Mode of Spread and Survival

The virus survives in weed hosts and perennial redgram plants. The disease is transmitted by white flies *(Bemisia tabaci)*.

Management

Rogue out the diseased plants upto 45 days after sowing. Remove weed hosts periodically. Spray Monocrotophos or Methyldemeton at 500 ml/ha immediately on noticing the disease and repeat after 15 days, if necessary.

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

Management

Remove and destory infected plant debris. Spray Carbendazim 500g or Wettable sulphur 1.5 kg or Tridemorph 500 ml/ha at the initiation of disease and repeat 15 days later. Grow resistant varieties like LBG17, PDU10, ICI12/2 and PLU 322.

2. Anthracnose

Colletotrichum lindemuthianum

(Sexual stage : *Glomerella lindemuthianum)*

Symptoms

The symptom can be odserved in all aerial parts of the plants and at any stage of crop growth. The fungus produces dark brown to black sunken lesions on the hypocotyl area and cause death of the seedlings. Small angular brown lesions appear on leaves, mostly adjacent to veins, which later become greyish 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, thinwalled, single celled conidia with oil globules. The perfect stage of the fungus produces perithecia with limited number of asci, 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 (15-20OC) 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 1 kg/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. The 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 by air-borne conidia.

Management

Remove and burn infected plant debris. Spray Mancozeb at 1 kg/ha or *Carbendazim* 250 g/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 21-26OC 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 destory. Spray Mancozeb 1 kg or Carbendazim 500 g or Wettable sulfur 1 kg/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 with in week. Dark brown lesions are seen on the stem at ground level and bark shows shredding symptom. The affected plants can be easily pulled

out leaving dried, rotten root portions in the ground. The rotten tissues of stem and root contain a large number of black minute sclerotia.

Pathogen

The fungus produces dark brown, septate mycelium with constrictions at hyphal branches. Minute, dark, round sclerotia of size 110-130u are produced in abundance. The fungus also produces dark brown, globose ostiolated pycnidia on the host tissues. They pycnidiospores (conidia) are thin walled, hyaline, single celled, elliptical and measure 10-42X6-10u.

Favourable Conditions

Day temperature of 30OC and above 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 seed-borne and soil-borne sclerotia. The secondary spreads is through pycnidiospores which are air-borne.

Management

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

6. <u>Yellow mosaic Virus</u>

Symptoms

Initially small yellow patches or spots appear on green lamina. The young leaves are the first to show the symptoms. The yellow discoloration slowly increases and newly formed leaves may completely turn yellow. The infected plants normally mature later and bear a very few flowers and pods. The pods are small and distorted. The early infection causes death of the plant before seed set.

Favourable Conditions

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

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 upto 40 days after sowing. Remove the weed hosts peiodically. Increase the seed rate (25 kg/ha). Grow resistant black gram variety like VBN-1, PDU 10, IC12/2 and PLU 322. Cultivate the crop during rabi season. Follow mixed cropping

by growing two rows of maize (60 x 30 cm) or sorghum (45 x 15 cm) or cumbu (45 x 15 cm) for every 15 rows of black gram or green gram.

7. Leaf crinkle Virus

Symptoms

The symptom appears initially in young leaves. The enlargement of 4th or 5th leaf is seen four or five weeks after sowing. Later crinkling and curling of the tips of leaflets are seen. The petioles as well as internodes are shortened. The infected plant gives a stunted and bushy appearance. Flowering is delayed, infloresence, if formed, are malformed with small size flower buds and fail to open. The age of the plant is prolonged with dark green leaves till harvest.

Favourable Conditions

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

Mode of Spread and Survival

The virus is seed-borne and primary infection occurs through infected seeds. White fly, Bemisia tabaci, is the vector, helps in the secondary spread. The virus is also transmissible.

Management

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

8. Leaf curl

(Tomato Spotted Wilt Virus)

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, extends to the petiole. Plants affected in the early stages of growth develop top necrosis and die. The 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 hosts *viz., Acanthospermum hispidum,Ageratum conyzoides, Amaranthus viridis, Calotropis gigantea, Lagasca mollis, Trianthema portulacastrum, Cassia tora, Cleome gynandra, Solanum nigrum* and *Datura metal* and other legume hosts.

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 upto 30 days after sowing. Remove the weed hosts which harbour virus and thrips. Spray Monocrotophos or Methyl demeton at 500 ml/ha on 30 and 45 days after sowing.

DISEASES OF COWPEA *(Vigna unguiculata)*

1. **Wilt**

Fusarium oxysporum f. sp. tracheiphilum

Symptoms

Symptoms do not appear until the plants are about six weeks old. Initially a few plants are noticed with pale green flaccid leaves which soon turn yellow. Growth is stunted, Chlorosis, drooping, premature shedding or withering of leaves with veinal necrosis often occurs and finally plant dies within 5 days. Brownish, purple discoloration of the cortical area is seen, often extends throughout the plant.

The fungus produces falcate shaped macroconidia which are 4-5 septate, thin walled and hyaline. The microconidia are single celled. hyaline and oblong or oval. The chlamydospores are also produced in abundance.

Favourable Conditions

Temperature of 20-25OC and moist humid weather.

Mode of Spread and Survival

The fungus survives in the infected stubbles in the field. The primary spread is through chlamy dospores and seed contamination. The secondary spread is through conidia by irrigation water.

Management

Treat the seeds with Carbendazim or Thiram at 2 g/kg or treat the seeds with *Trichoderma viride* at 4 g/kg. Spot drench with Carbendazim at 0.5 g/litre.

2. **Bacterial leaf spot**

Xanthomonas vignicola

Symptoms

The disease attacks at any stage of plant growth. The seedling infection starts as red lesion at cotyledon leads to withering. In adult plants yellow circular spots appear on leaves, enlarge and become angular and encircled by a yellow halo. The infection can be seen on the petiole, stem and pods as brown linear lesions. The affected twigs show twig blight and infected pods contain shrivelled seeds.

Favourable Conditions Heavy rainfall, high relative humidity (above 80 per cent) and temperature of 20-25OC.

Mode of Spread and Survival

The bacterium is seed-borne and cause primary infection in the field. The wind, rain splash and insects help in the secondary spread.

Management

Collect the seeds from healthy plants and use disease free seeds.

3. **Aphid-borne mosaic Virus**

Symptoms

The affected plants are stunted and the leaves show variable amounts of dark green

vein banding, leaf distortion and blistering. The affected leaves are leathery. The infected plants produce a few pods which are small and distorted.

Favourable Conditions

The presence of weed hosts and other legume hosts.

Mode of Spread and Survival

The virus is transmitted aphid vectors viz., *Aphis craccivora, A. fabae, A. gossypii, Macrosphum euphorbiae* and *Myzus persicae.* The virus survives in weed hosts and other legume crops.

Management

Remove the infected plants upto 30 days after sowing. Rogue out the weed hosts periodically. Grow resistant varieties like Co.6. Spray thrice with Monocrotophos or Methyl demeton at 500 ml/ha at 15 days intervals.

DISEASES OF SOYBEAN

SOYA BEAN *(Glycine max)*

Dry root rot	*Macrophomina phaseolina*	Refer Black gram
Yellow mosaic Virus		Refer Black gram
Wilt	*Fusarium oxysporum*	Refer Cowpea
Leaf spot	*Cercospora sojana*	Well defined spots often bound by veins

DISEASES OF GROUNDNUT

(Arachis hypogaea)

1. Tikka leaf spots

Early leaf spot : *Cercopora arachidicola*

(Sexual Stage) : *Mycosphaerella arachidis)*

Late leaf spot : *Phaeoisariopsis personata*

Syn : cercospora personata

(Sexual stage) : *Mycosphaerella berkeleyii)*

Symptoms

The disease occurs on all above ground parts of the plant, more severely on the leaves. The leaf symptoms produced by the two pathogens can be easily distinguished by the following characters.Both the fungi produce lesions also on petiole, stem and pegs. The lesions caused by both species coalesce as infection develops and severely spotted leaves shed prematurely. The quality and yield of nuts are drastically reduced in severe infections.

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

The fungus is intercellular and do not produce haustoria and become intracellular when host cells die. The fungus produces abundant sporulation on the upper surface of the leaves. Conidiophores are olivaceous brown or yellowish brown in colour, short, 1 or 2 septate, unbranched and geniculate and arise in clusters. Conidia are sub hyaline or pale yellow, obclavate, often curved 3-12 septate, 35-110 x 2.5 - 5.4 um in size with rounded to distinctly truncate base and sub-acute tip. The perfect stage of the fungus produces perithecia as ascostromata. They are globose with papillate ostiole. Asci are cylindrical to clavate and contain 8 ascospores. Ascospores are hyaline, slightly curved and two celled, apical cell larger than the lower cell.

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

The fungus produces internal and intercellular mycelium with the production of haustoria. The conidiphores are long, continuous, 1-2 septate, geniculate, arise in clusters and olive brown in colour. The conidia are cylindrical or obclavate, short, measure 18-60 x 6-10um, hyaline to olive brown, usually straight or curved slightly with 1-9 septa, not constricted but mostly 3-4 septate. The fungus in its perfect stage produces perithecia as ascostromata which are globose or brodly ovate with papillate ostiole. Asci are cylindrical to ovate, contain 8 ascospores. Ascospores are 2 celled and constricted at septum and hyaline.

Favourable Conditions

Prolonged high relative humidity for 3 days, low temperature (20OC) with dew on leaf surface, heavy doses of nitrogen and phosporus fertilizers and deficiency of magesium in soil.

Mode of Spread and Survival

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

Management

Remove and destory the infected plant debris. Eradicate the volunteer groundnut plants. Keep weeds under control. Treat the seeds with Carbendazim or Thiram at 2g/kg. Spray Carbendazim 250g or Mancozeb 1 kg or Chlorothalonil 1 kg/ha and if necessary, repeat after 15 days. Grow moderately resistant varieties like ALR.1.

2. Rust

Puccinia arachidis

Symptoms

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

Pathogen

The fungus produces both uredial and telial stages. Uredial stages are produced abundant in groundnut and production of telia is limited. Uredospores are pedicellate, unicellular, yellow, oval or round and echinulated with 2 or 3 germpores. Teliospores are dark brown with two cells. Pycnial and aecial stages have not been recorded and there is no information available about the role of alternate host.

Favourable Conditions

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

Mode of Spread and Survival

The pathogen survives as uredospores on volunter 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. Rainsplash and implements also help in dissemination. The fungus also survives on the collateral hosts like *Arachis marginata, A. nambyquarae* and *A. prostrate.*

Management

Avoid monoculturing of groundnut. Remove volunteer groundnut plants and reservoir hosts. Spray Mancozeb 1 kg or Wettable sulphur 2.5 kg or Tridemorph 500 ml or Chlorothalonil 1 kg/ha. Grow moderately resistant varieties like ALR.1

3. <u>Collar rot or seedling blight or crown rot</u>

Aspergillus niger and A. pulverulentum

Symptoms

The fungus is both seed-borne and soil-borne and so the infection can be seen at any stage from sowing onwards. The disease usually appears in three phases.

i. Pre-emergence rot : Seeds are attacked by soil-borne conidia and rotten of seeds prevents the seeds to germinate. The seed are covered with black masses of spores and internal tissues of seed become soft and watery.

ii. Post-emergence rot : The pathogen attacks the emerging young seedling and cause circular brown spots on the cotyledons. The symptom spreads later to the hypocotyl and stem. Brown discoloured spots appear on collar region. The affected portion become soft and rotten, resulting in the collapse of the seedling. The collar region is covered by profuse growth of fungus and conidia and affected stem also show shredding symptom.

iii. Crown rot : The infection when occurs in adult plants show crown rot symptoms. Large lesions develop on the stem below the soil and spread upwards along the branches causing drooping of leaves and wilting of plant.

Pathogen

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 globose and have two rows of hyaline phialides viz., primary and secondary phialides. The conidial head are dark brown to black. The conidia are globose, dark brown in colour and produce in long chains.

Favourable Conditions

Deep sowing of seeds, high soil temperature (30-35OC) and low soil moisture.

Mode of Spread and Survival

The pathogen survive in plant debris in the soil, not necessarily from a groundnut crop. Soil-borne conidia cause disease carry over from season to season. The other primary source is the contaminated seeds. The fungi are carried on the seed surface or under the testa.

Management

Select good quality seeds. Treat the seeds with Carbendazim 2 g or Thiram 4g/kg. Avoid deep sowing of seeds. Destroy the crop debris by burning.

4. **Root rot**

Macrophomina phaseolina

Symptoms

In the early stages of infection, reddish brown lesion appears on the stem just above the soil level. The leaves and branches show drooping, leading to death of the whole plant. The decaying stems are covered by whitish mycelial growth. The death of the plant results in shredding of bark. The rotten tissues contain large number of black or dark brown, thick walled sclerotia. When infection spreads to underground roots, the sclerotia are formed externally as well as internally in the rotten tissue. Pod infection leads to blackening of the shells and sclerotia can be seen inside the shells.

Pathogen

The fungus produces hyaline to dull brown mycelium. The sclerotia are thick walled and dark brown in colour.

Favourable Conditions

Prolonged rainy season at seedling stage and low lying areas.

Mode of Spread and Survival

The fungus remains dormant as sclerotia for a long period in the soil and in infected plant debris. The primary infection is through soil-borne and seed-borne sclerotia. The secondary spread of sclerotia is aided by irrigation water, human agency, implements, cattle etc.

Management

Treat the seeds with Thiram 4g or Carbendazim 2g/kg or treat the seeds with *Trichoderma viride* at 4g/kg. Spot drench with Carbendazim at 0.5 g/lit.

5. **Rossetts VirusSymptoms**

The affected plants are characterized by the appearance of dense clump or dwarf shoots with tuft of small leaves forming in a rossette fashion. The plant exhibits chlorosis and mosaic mottling. The infected plants remain stunted and produce flowers, but only a few of the pegs may develop further to nuts but none bear seeds.

Mode of Spread and Survival

The virus can survive on the volunteer plants of groundnut and other hosts. The virus is transmitted by *Aphis craccivora* in a persistent manner.

Management

Use heavy seed rate and rogue out periodically the infected plants. Spray Monocrotophos or Methyl demeton at 500 ml/ha.

6. Peanut spotted wilt or bud necrosis or groundnut ring mosaic

Tomato spotted wilt virus (TSWV)

Symptoms

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

Mode of Spread and Survival

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

Management

Adopt spacing of 15x15 cm. Remove and destory infected plants upto 6 weeks after sowing. Monocrotophos 500 ml/ha, 30 days after sowing either alone or in combination with AVP (Anti-Viral Principle) extracted from sorghum or coconut leaves. Spray the crop with 10 per cent AVP at 500 lit/ha, ten and twenty days after sowing.

EXTRACTION OF 10 PER CENT AVP

Dried sorghum or coconut leaves are cut and powdered. To one kg of leaf powder, two litres of water is added and heated to 60^0C for one hour. It is then filtered through muslin cloth and diluted to 10 litres and sprayed. Five hundred litres of extract is required to cover one hectare.

DISEASES OF SESAME

(Sesamum indicum)

1. Root rot or stem rot or charcoal rot

Macrophomina phaseolina

Syn : cercospora personata

(Sclerotial stage : *Rhizoctonia bataticola)*

Symptoms

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

Pathogen

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

Favourable Conditions

Day temperature of 30OC and above and prolonged drought followed by copious irrigation.

Mode of Spread and Survival

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

Management

Treat the seeds with *Trichoderma viride* at 4g/kg or *Pseudomonas fluorescens* 10 g/kg or treat the seeds with carbendazim of Thiram at 4g/kg. Apply farm yard manure or green leaf manure at 10t/ha or neem cake 250 kg/ha. Spot drench with Carbendazim at 0.5 g/litre.

2. Leaf blight

Alternaria sesami

Symptoms

Initially small, circular, reddish brown spots (1-8mm) appear on leaves which enlarge later and cover large area with concentric rings. The lower surface of the spots are greyish

brown in colour. In severe blighting defoliation occurs. Dark brown lesions can also be seen on petioles, stem and capsules. Infection of capsules results in premature splitting with shriveled seeds.

Pathogen

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

Favourable Conditions

Low temperature (20-25OC), high relative humidity and cloudy weather.

Mode of Spread and Survival

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

Management

Treat the seeds with Thiram or Carbendazim at 2g/kg. Spray Mancozeb at 1kg/ha.

3. Leaf spot

Cercospora sesami

Symptoms

The disease first appears on the leaves as minute water-soaked lesions, which enlarge to form round to irregular spots of 5-15 mm diameter on both the leaf surface. The spots coalesce to form irregular patches of varying size leading to premature defoliation. The infection is also seen on stem and petiole forming spots of varying lengths. Dark linear spots also occur on pods causing drying shedding.

Pathogen

The hypha of the fungus is irregularly septate, light brown and thick walled. Conidiophores are produced in cluster and are 1-3 septate, hyaline at the tip and light brown coloured at base. Conidia are elongated, 7-10 septate, hyaline to light yellow, broad at the base and tapering towards the apex.

Mode of Spread and Survival

The fungus is externally and internally seed-borne. The fungus also survives in plant debris. Primary infection may be from the seed and infected debris. The secondary spread is through wind-borne conidia.

Management

Treat the seeds with carbendazin or Thiram at 2g/kg. Spray with Mancozeb at 1Kg/ha.

4. Wilt

Fusarium oxysporum f.sp. sesami

Symptoms

The disease appears as yellowing, drooping and withering of leaves. The plants gradually wither, show wilting symptom leading to drying. The infected portions of root and

stem show long, dark black streaks of vascular necrosis.

Pathogen

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

Mode of Spread and Survival

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

Management

Treat the seeds with Thiram or Carbendazim at 2g/kg or pellet the seeds with *Trichoderma viride* at 4g/kg. Apply heavy doses of green leaf manure or farm yard manure.

5. Stem blight

Phytophthora parasitica var. sesami

Symptoms

Black coloured lesions appear on the stem near the soil level. The disease spreads further and affects branches and may girdle the stem, resulting in the death of the plant. Leaves may also show water-soaked patches and spread till the leaves wither. Infection may be seen on flowers and capsules. Infected capsules are poorly developed with shrivelled seeds.

Pathogen

The fungus produces non-septate, hyaline mycelium. The sporangiophores are hyaline and branched sympodially and bear sporangia. The sporangia are hyaline and spherical with a prominent apical papilla. The oospores are smooth, spherical and thick walled.

Favourable Conditions

Prolonged rainfall, low temperature (25OC) and high relative humidity (above 90 per cent)

Mode of Spread and Survival

The fungus can survive in the soil as dormant mycelium and oospores. The seeds also carry the fungus as dormant mycelium, which causes the primary infection. Secondary spread of the disease is through wind-borne sporangia.

Management

Treat the seeds with Captan or Thiram at 2g/kg. Avoid continuous cropping of sesamum in the same field. Remove and destroy infected plant debris.

6. Powdery mildew

Erysipha cichoracearum

(Syn : *Oidium acanthosperml)*

Symptoms

Initially greyish-white powdery growth appears on the upper surface of leaves. When several spots coalesce, the entire leaf surface may be covered with powdery coating. In severe cases, the infection, the infection may be seen on the flowers and young capsules, leading to premature shedding. The severaly affected leaves may be twisted and malformed. In the advanced stages of infection, the mycelial growth changes to dark or black because of development of cleistothecia.

Pathogen

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

Favourable Conditions

Dry humid weather and low relative humidity.

Mode of Spread and Survival

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

Management

Remove the infected plant debris and destroy. Spray Wettable sulphur at 2.5 kg/ha or dust Sulphur at 25 kg/ha and repeat after 15 days.

7. Bacterial leaf spot

Xanthomonas campestris p.v. sesami

Symptoms

Initially water-soaked spots appear on the undersurface of the leaf and then on the upper surface. They increase in size, become angular and restricted by veins and dark brown in colour. Several spots coalesce together froming irregular brown patches and cause drying of leaves. The reddish brown lesions may also occur on petioles and stem.

Pathogen

The bacterium is bacilliform with a monotrichous flagellum. It is gram negative and non-spore forming.

Mode of Spread and Survival

The bacterium survives in the infected plant debris and is seed-borne. The secondary spread is by rain water.

Management

Remove and burn infected plant debris. Spray Streptomycin sulphate or Oxytetracycline hydrochloride at 100g/ha.

8. Bacterial leaf spot

Pseudomonas sasami

Symptoms

The disease appears as water-soaked yellow specks on the upper surface of the leaves. They enlarge and become angular as resticted by veins and veinlets. The colour of spot may be dark brown with shiny oozes of bacterial masses.

Pathogen

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

Mode of Spread and Survival

The bacterium remains viable in the infected plant tissues. It is internally seed-borne and secondary spread through rainsplash and storms.

Management

Keep the field free of infected plant debris. Spray with Streptomycin sulphate or Oxytetracycline hydrochloride at 100g/ha.

9. <u>Phyllody</u>

Mycoplasma Like Organism

Symptoms

The disease manifests itself mostly during flowering stage, when the floral parts are transformed into green leafy structures, which grow profusely. The plants bear cluster of leaves and malformed flowers at the tip. The flower is rendered sterile. The veins of phylloid structure is thick and prominent. Stamens also become leaf like to certain extent. Anthers become green and do not dehisce. Ovary is transformed into a elongated out growth resembling a shoot. The plant is stunted with reduced internodes and abnormal branching gives a bushy appearance.

Mode of Spread and Survival

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

Management

Remove all the reservoir and weed hosts. Avoid growing sesaumu near cotton, groundnut and grain legumes. Rogue out the infected plants periodically. Spray Monocrotophos or Dimethoate at 500ml/ha to control the jassids.

DISEASES OF CASTOR

(Ricinus communis)

Seedling blight

Phytophthora parasitica

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 and causes withering and death of seedling. In mature plants, the infection initially appears on the young leaves and spreads to petiole and stem causing black discoloration and severe defoliation.

Pathogen

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

Favourable Conditions

Continuous rainy weather, low temperature (20-25OC), low lying and ill drained soils.

Management

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

1. **Rust**

Melampsora ricini

Symptoms

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

Pathogen

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

Mode of Spread and Survival

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

Management

Rogue out the self-sown castor crops and other weed hosts. Spray Mancozeb at 1kg/ha or dust Sulphur at 25kg/ha.

2. Leaf blight

Alternaria ricini

Symptoms

All the aerial parts of plants viz., leaves, stem, inflorescences and capsules are liable to be attacked by the fungus. Irregular brown spots with concentric rings form initially on the leaves and covered with fungal growth. When the spots coaleasce to form big patches, 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, sesamum etc. The secondary spread takes place through wind-borne sporangia. premature defoliation occurs. The stem, inflorescence and capsules are also show dark brown lesions with concentric rings. On the capsules, initially brown sunken spots appear, enlarge rapidly and cover the whole pod. The capsules crack and seeds are also get infected.

Pathogen

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

High atmospheric humidity (85-90 per cent) and low temperature (16-20OC)

Mode of Spread and Survival

The fungus also survives on hosts like *Jatropha pandurifolia* and *Bridelia hamiltoniana.* The pathogen is externally and internally 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.

3. Brown leaf spot

Cercospora ricinella

Symptoms

The disease appears as minute brown specks surrounded by a pale green halo. The spots enlarge to greyish white centre portion with deep brown margin. The spots may be 2-4 mm in diameter and when several spots coalesce, large brown patches appear 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

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.

Minor diseases

Powdery mildew ***Leveillula taurica*** White cottony growth on the lower surface of leaves with yellow discolouration on upper surface.

DISEASES OF TOBACCO

(Nicotiana tabacum)

1. Damping off

Pythium aphanidermatum

Symptoms

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

Pathogen

The fungus produces thick, hyaline, thinwalled, non-septate mycelium. It produces irregularly lobed sporangia which germinate toproduce vesicle containing zoopores. The zoospores are kidney shaped and biflagellate. Oospores spherical, light to deep yellow or yellowish brown coloured, measuring 17-19um in diameter.

Favourable Conditions

Over crowding of seedling, ill drained nursery beds, heavy shade in nursery, high atmospheric humidity (90-100 per cent), high soil moisture, low temperature (below 24OC) and low soil temperature of about 20OC.

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 seed beds 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 over crowding of seedlings by using recommended seed rate (1 to 1.5g/2.5m2).

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

2. Black shank

Phytophthora parasitica var. *nicotianae*

Symptoms

The pathogen may affect the crop at any stage of its growth. Eventhough all parts are affected, the disease infects chiefly the roots and base of the stem. Seedlings in the nursery show black discolor of the stem near the soil level and blackening of roots, leading the wet rot in humid condition and seedling blight in dry weather with withering and drying of tips. The pathogen also spreads to the leaves and causes blighting and drying of the bottom leaves. In the transplanted crop, the disease appears as minute black spot on the stem, spreads along the stem to produce irregular black patches and often girdling occurs. The upward movement leads to development of necrotic patches on the stems. The infected tissues shrink, leaving a depression and in advanced condition the stem shrivels and plant wilts. When the affected stem is split open, the pith region is found to be dried up in disc-like plates showing black discolouration. On the leaves large brown concentrically zonate patches appear during humid weather, leading to blackening and rotting of the leaves.

Pathogen

The fungus produces hyaline and non-septate mycelium. The sporangia, which are hyaline, thin walled, ovate or pyriform with papillae, develop on the sporangiophores in a sympodial fashion. Sporangia germinate to release zoospores which are usually kidney shaped, biciliate and measure 11-13 x 8-9um. The fungus also produces globoose and thick walled chlamydospores, measuring 27-42um in diameter. Oospores are thickwalled, globose, smooth and light yellow coloured, measuring 15-20um in diameter.

Favourable Conditions

Frequent rainfall, high soil moisture and high population of rootknot nematodes *Meloidogyne incognita* var. *acrita.*

Mode of Spread and Survival

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

Management

Burn the seed beds with paddy husk or groundnut shell at 15-20 cm thick layer. Provide adequate drainage in the nursery. Drench the nursery beds with 1 per cent Bordeaux mixture or 0.2 per cent Copper oxychloride, two days before sowing. Spray the beds two weeks after sowing with 0.2 per cent Metalaxyl or 0.2 per cent Captafol or 0.2 per cent Copper oxychloride or 1 per cent Boreaux mixture and repeat after 10 days. Select

healthy, disease free seedlings for transplanting. Remove and destroy the affected plants in the field. Spray Mancozeb 1 kg or Copper oxychloride 1 kg or Ziram 1 lit/ha. Spot drench with 0.4 per cent Bordeaux mixture or 0.2 per cent Copper oxychloride.

3. Frog eye spot

Cercospora nicotianae

Symptoms

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

Pathogen

The mycelium is intercellular 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, thinwalled and 2-12 septate.

Favourable Conditions

Temperature of 20-30OC, high humidity (80-90 per cent), close 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 the crop with 0.4 per cent Bordeaux mixture or Thiophanate Methyl 750g/ha or Carbendazim 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 cover 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 ecotophytic and produce 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.

Favourable Conditions

Humid cloudy weather, low temperature (16-23OC), 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 wind blown conidia.

Management

Apply balanced ferilizers. 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. Apply Sulphur dust to the soil at 40kg/ha. (Mix 1 part of Sulphur with 3 parts of fine sand or ash and apply). Spray Dinocap at 375 ml or Carbendazim at 500g/ha.

5. 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 latter become angular when restricted by the veins and veinlets. 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 cover 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 and 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.

6. Mosaic

Tobacco Mosaic Virus (TMV)

Symptoms

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

Pathogen

Tobacco mosaic is caused by *Nicotiana virus I (Marmor tabaci* var. *vulgare).* It is a rod shaped particle measuring 300 X 150-180um with a central hollow tube of about 4um diameter. It is madeup of centrally placed Ribo Nucleic Acid molecules (RNA) covered with a protein coat. It is capable of remaining infective when stored dry for over 50 years.

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

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

host plants for two seasons. Grow resistant varieties like TMV RR2, TMV RR 2a and TMV RR3.

7. **Leaf curl**

Niccotiana Virus 10 *(Ruga tabaci)*

Symptoms

The virus may attack the plant at any stage. When young plants are infected the entire plant remains very much dwarfed. Three forms of leaf curl expression are observed. The leaf margins curl downward towards the dorsal side and show thickening of veins with enation on the lower surface. Crinkle form shows curling of whole leaf edge towards dorsal side with enation on the veins and the lamina arching towards the ventral side between the veinlets. The transparent symptom shows the curling of leaves towards the ventral side with clearing of the veins and enations are absent.

Pathogen

The virus is spherical and measuring 35um in diameter. The virus is Nicotiana virus 10 or Ruga tabaci.

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 Methyldemeton at 0.1 to 0.2 per cent to control the vectors.

8. **Phaneroganic parasite**

Broom rape

Orobanche cernua var. *desertorum*

Symptoms

The affected tobacco plants are stunted and show withering and drooping of leaves to wilting. These are the indicates of underground parasitism of the tobacco roots by the parasite. The young shoot of the parasite emerges from the soil at the base of the plants 5 -6 weeks after transplanting. Normally, it appears on clusters of 50-100 shoots around the base of a single tobacco plant. The plants which are attacked very late exhibit no external symptoms but the quality and yield of leaves are reduced.

Parasite

It is a total root parasite. It is an annual, fleshy flowering plant with a short, stout stem, 10-15 inches long. The stem is pale yellow or brownish red in colour and covered by small, thin, brown scaly leaves and the base of the stem is thickened. White-coloured

flowers appear in the leaf axils. The floral parts are well developed with a lobed calyx, tubular corolla, superior ovary, numerous ovules and a large four-lobed stigma. The fruits are capsules containing small, black, reticulate and ovoid seeds.

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

Management

Rogue out the tender shoots of the parasite before flowering and seed set. Spray the soil with 25 per cent Copper sulphate. Spray 0.1 per cent Allyl alcohol. Apply few drops of kerosene directly on the shoot. Grow decoy or trap crops like chilli, motherbean, sorghum or cowpea to stimulate seed germination and kill the parasite.

DISEASES OF BANANA

1. *Fusarium* wilt/Panama disease – *Fusarium exysporum* f.sp.*cubense*
2. Sigatoka leaf spot – Yellow Sigatoka - *Mycosphaerella musicola*
   – Black Sigatoka - *Mycosphaerella fijiensis*
3. Moko disease / Bacterial wilt – *Ralstonia solanaceaum (Burkholderia solanaceaum)*
4. Tip over / Rhizome rot / top rot – *Erwinia carotovora carotovora*
5. Bunchy top – Banana bunchy top virus (BBTV)
6. Banana Mosaic / Infectious chlorosis – Cucumber Mosaic Virus (CMV)
7. Banana bract mosaic – BBMV
8. Post Harvest Disease

1. Cigar end rot – *Verticillium thoebromae*
2. Anthracnose – *Colletotrichum musae*
3. *Deightoniella* leaf spot / Black spot / fruit spot – *Deightoniella torulosa*
4. Crown / Stem end rot – *Botryodiplodia theobromae (Lasiodiplodia theobromae)*
5. Main stalk rot – *Ceratostomella paradoxa*
6. Pitting disease – *Pyricularia grisea*

Minor Diseases

1. Freckle leaf spot – *Physlostictinia musarum*
2. Bacterial leaf spot – *Xanthomonas musicola*

3. Cordana leaf spot – *Cordana musae*

1. *Fusarium* wilt/Panama disease – *Fusarium exysporum* f.sp.*cubense*

- ❖ It is the most destructive and wide spread disease of banana.
- ❖ First recorded from Australia in 1874 (Bancroft 1876).
- ❖ The disease had attained alarming proportion in Panama and Costa Rica in 1904.
- ❖ In 1917, the cultivation of Gros Michel banana in Panama was completely abandoned because of the disease which was later replaced by red banana.
- ❖ Now the disease has been reported in all the banana growing countries.
- ❖ In India the disease was first reported from Chinsurah in 1911
- ❖ It is presently prevalent in Tamil Nadu, Kerala, Karnataka, Bihar, Assam, Andhra Pradesh, West Bengal and Maharashtra.

Symptoms:

- ❖ Plants will not exhibit any external symptoms initially up to 4-6 month even though 2-3 months old seedlings are also affected.
- ❖ The infected plants show characteristic yellowing of older leaves.
- ❖ The yellowing develops along the margin as a band and spreads towards the midrib.
- ❖ Yellowing of leaves spreads upwards and finally heart leaf alone remains green.
- ❖ Leaves wilt, dry and break at the base of the petiole and hang around the pseudostem
- ❖ This forms a skirt of dead leaves around the plants.
- ❖ Often all the leaves collapse except the heart leaf which alone remains upright.
- ❖ After 4-6 weeks, the pseudostem remains with dead leaves hanging around it.
- ❖ More number of daughter suckers are produced from the infected plants.
- ❖ The pseudostem shows longitudinal splitting at the base above the soil level.
- ❖ The internal symptoms are more reliable for diagnosis.
- ❖ When the infected pseudostem cut transversely, dark brown or black discoloration of water conducting tissues are seen.
- ❖ Gum pockets are also seen in water conducting tissues due the accumulation of gel in response to infection by the host.
- ❖ When the infected corm cut transversely, central portion of the affected corm is dark brown
- ❖ In the corm yellow, red, brown black or purplish dots or streaks run in all directions through the strands radiating from the centre of the corm.
- ❖ The corm smells like rotten fish.
- ❖ Roots of diseased rhizomes are black in colour.
- ❖ The affected plants may show dwarfing.
- ❖ The bunch may show various abnormal developments.
- ❖ It ripens prematurely or irregularly.

- The fingers become bottle necked.
- The flesh becomes pithy, acrid and yellow.

Mechanism of Wilt infection

- The pathogen gains entry into the water – conducting xylem vessels and proliferate causing water blockage.
- The blockage of xylem vessel is also caused by the gel-like materials produced by the host in response to invasion of the pathogen.
- Yellowing and loss of control of stomatal function in leaves are caused by the upward translocation of toxins (Propionic aldehyde, fusaric acid, dopamine etc) produced by the pathogen.
- The closing of xylem vessels by tyloses and destruction of roots contribute significantly towards wilting.

Casual Organism: *Fusarium oxysporum* f.sp *cubense*.

The causal agent of Fusarium wilt or Panama disease of banana is the fungus *Fusarium oxysporum* f. sp. *cubense* (E.F. Sm.) W.C. Snyder & H.N. Hansen (*Foc*). *Foc* is an anamorphic fungus without a known sexual stage (teleomorph). Sexual stage (teleomorph) has not been found even in isolates carrying genes Mat 1 and Mat 2 (Fourie *et al*., 2011).
Its taxonomic position is as follows:
Domain: Eukaryota
Kingdom: Fungi
Phylum: Ascomycota
Class: Ascomycetes
Subclass: Sordariomycetidae
Order: Hypocreales

Morphology

Mycelium – Intracellular in xylem vessels – Mycelium is white or tinted rose or violet

There are three types of asexual spores formed; macroconidia, microconidia and chlamydospores (Nelson, 1991).

Macroconidia are formed from monophialides on branched conidiophores in sporodochia, and to a lesser extent from monophialides on hyphae (Leslie and Summerell 2006). Macroconidia are four to eight celled, falcate to erect to almost straight, sickle-shaped, thin-walled and delicate, with foot-shaped basal attenuated apical cells (Jones 2000). 27-55µm x 3.3-5.5µm

Microconidia abundantly borne on false heads on short monophialides in sporodochia, are one or two celled, hyaline, oval- to kidney shaped. The dimensions of the microconidia are 5-16µm x 5-16 × 2.4-3.5 µm.

Chlamydospores are thick-walled, asexual, globose 7-11µm, usually formed singularly or in pairs, but may also be found in clusters or short chains (Jones 2000; Leslie and Summerell 2006). Oval or globose

Pathogenic races of *Fusarium oxysporum* f. sp. *cubense*

Four physiological races exist based on the pathogenicity on different genotypes of banana and related hosts.

Race: 1 attacks Gros Michel (AAA), Manzano/Apple/Latundan (Silk, AAB), Pisang awak (Karpooravalli (ABB) and Pome (Pachanadan, Ney Poovan, Virupakshi (AAB) and Rasthali (Silk) (AAB) Worldwide (Central America)

Race: 2 attacks Bluggoe (Monthan, Sakkia (AAB)

Race: 3 attacks only *Heliconia* sp

Race: 4 not exist in India. Attacks Cavendish varieties – resistant to race 1 - and mildly resistant to race 2

Mode of survival and spread of the pathogen:

- The pathogen is soil borne. It is able to survive as chlamydospores in previously colonized tissues and in soil where it can persist for long periods, latent or as an endophyte of host weeds (Hennessey *et al.* 2005).
- It survives in soil as chlamydospores for more than 30 years.
- The primary spread of the disease is through infected planting material, rhizomes and suckers.
- The infected trash is an important source of infection.
- Secondary spread is also through the movement of soil, running water, farm machinery and implements and root to root contact.
- There is also the possibility of dissemination by insect vectors, especially the banana weevil borer *Cosmopolites sordidus* (Coleoptera: Curculionidae).

Epidemiology:

- The infection entry is facilitated by mechanical wounds and root damage caused by burrowing nematode *Radopholus similis*
- Survival is influenced by soil moisture, soil texture and soil Ph etc.

- Survival and growth is more in acidic, light textured loam and sandy loam soils than in clayey and alkaline soil with high calcium content.
- Depth of penetration is more in sandy loan than in silt loam soil.
- Clay minerals retard the spread of the disease.
- Survival of the pathogen in the field soil is best at 25% saturation, while increase in soil moisture the survival ability is decreased.
- Growth of the pathogen and disease development in the roots occurs at 21-27^{0}C but not at 34^{0}C
- Symptom development declines during winter months.
- Saturated, poorly drained soils have been correlated with greater disease incidence.
- Low levels of Zinc and high Ca: Mg and K: Mg ratios are associated with more severe disease.
- In soils submerged under 5cm depth of water, the survival was reduced to only one month.
- There is a positive correlation between the amount of inoculum present in the proximity of the roots and extent of root infection.
- During drought period there is considerable reduction in the number of newly diseased plants but soon after heavy rains there is rapid increase in the disease incidence.
- Mechanical wounds caused by tillage operations also help in dispersal of the pathogen.
- The disease is severe in stiff, acid clay and alluvial soils with poor drainage and aeration.

Management:

- Selection and use of resistant varieties Res. Var: Raja bale, Red banana, Walha, Dwarf Cavendish, Robusta, Grand Nain, Nendran.
- Sus Var: Gros Michel, Monthan, Karpooravalli, Rasthali.
- Use of planting materials collected from disease free fields.
- Use of disease free planting materials
- Infected plants should be completely removed and destroyed.
- The pits have to be treated with lime @1-2kg/pit.
- Crop rotation with paddy or Sugarcane for 3-4 years in infected fields.
- Proper drainage facilities.
- Flood fallowing for 6-24 months.
- Dipping of suckers in carbendazim 0.1% for ½ h before planting
- Soil drenching with carbendazim 0.1% at 5, 7 and 9 months after planting.
- Paring and prolinage with carbofuran 3G @40g per suckers for the control of burrowing nematodes.

- Avoidance of root injury through tillage.
- Regular optimum irrigation.
- Plant quarantine. Avoidance of transport of infected planting materials to areas where the disease is not present.
- Deep tillage before planting – the heavily infected upper (20-30cm) soil could be buried to a depth of 60-90 cm and the pathogen could be destroyed by biological and other soil factors.
- Soil organic amendments with crops like legumes, sorghum or sugarcane trash reduce the disease incidence.
- The tissue culture plants are more susceptible hence the Tissue Culture banana should be planted only in pathogen free soils.
- Application of lime reduced the survival of the pathogen to 2 months.
- Capsule application with Carbendazim 50 WP @ 60 mg per capsule on 5,7 and 9 months after planting .

Biological Control:

- Sucker treatment with commercial formulation of Pseudomonas fluorescens @10g per sucker.
- Soil application @2.5 kg /ha
- Capsule application@50mg per sucker at 3, 5 and 7th month after planting.

2. Sigatoka Leaf Spots: 1. Yellow Sigatoka: *Mycosphaerella musicola*,
2. Black leaf streak or Black Sigatoka: *Mycosphaerella fijiensis*

The most important leaf spot disease of banana is caused by three species of the ascomycete genus *Mycosphaerella*.

Yellow Sigatoka, or banana leaf spot, was first recorded in Java in 1902 and the first major epidemic in commercial plantations occurred in the Sigatoka in the Fiji Islands in 1913 and hence the name was given. The disease now occurs in all major banana-producing countries.

In 1963, a more severe Sigatoka-like leaf spot was detected in Sigatoka Valley of Vita Levu in the Fiji Islands and was named 'black leaf streak' because of the dark brown to black spots and streaks formed on affected leaves. Black leaf streak is caused by *Mycosphaerella fijiensis*

A third serious disease of banana is black Sigatoka caused by *Mycosphaerella fijiensis* var. *difformis*. It was first recorded in Honduras in I972 and reached epidemic levels in commercial plantations in 1973-74. Recent studies of the fungi causing black leaf streak and black Sigatoka indicate that there is no consistent morphological difference between

Mycosphaerella fijiensis and *Mycosphaerella fijiensis* var. *difformis* which are now regarded as synonyms. Thus, the disease caused by what is now called *Mycosphaerella fijiensis* is commonly known as black Sigatoka

- The disease occurs throughout the world.
- Defoliation by *Mycosphaerella* diseases reduces the quantity and quality of bananas
- It is also one of the most destruction diseases of banana.
- In the absence of control measures, the disease can reduce bunch weight by up to 50% and cause a 100% loss of production due to deterioration in the quality of the fruit (length and thickness).
- In India the disease is prevalent in all the major banana growing states.
- In India the leaf spot is caused by the yellow Sigatoka pathogen

Symptoms:

- Starts with pale yellow or greenish yellow streaks parallel to veins on the leaves.
- These enlarge to become linear oblong, muddy brown to black spots (up to 1-2cm).
- The centre of the spots become light grey and necrotic surrounded by dark brown or black border with yellow halo.
- The matured spots are elliptical giving eye – spot appearance.
- On the upper surface of the spots fructification of the fungus appears as black specks.
- The spots often coalesce to form large irregular patches of dried tissues.
- In severe conditions the whole leaf dries up from the tip.
- The petiole collapses and the leaf hangs down from the pseudostem.
- Rapid drying and defoliation also seen.
- In case of Dwarf Cavendish except 2 or 3 leaves all the leaves dry out.
- In severe cases, the quality of fruits is drastically reduced.
- Small angular fingers premature ripening peels splitting are commonly seen.
- The bunch starts ripening during the transit affecting the export quality.

Casual Organism: *Mycospharella musicola, Mycosphaerella fijiensis*

Conidia – narrow, elongated, multi septate, hyaline

Perithecia – Dark brown to black globose, ostiolate, amphigenous, erumpent,

Asci- Oblong, clavate,

Ascospores – Hyaline, 2- celled obtuse – ellipsoid with slightly broader upper cell.

YELLOW SIGATOKA	BLACK SIGATOKA

More common in cooler environments	More common in warmer environments
Inoculum consists of both conidia (water-dispersed) and ascospores (wind-dispersed)	Windborne ascospores are the major inoculum
Conidia first appear in the matured spot stage	Conidia first appear in early streak stage
Produce more than 30,000 condia per spot	Produce about 1200 condia per spot
conidia not dislodged by wind	Conidia both water- and wind-dispersed
Matured ascospores are produced 4 weeks after the appearance of streaks	Matured ascospores are produced 2 weeks after the appearance of streaks

Mode of spread and survival:

The pathogen survives on dry infected leaves on the field soil.

The conidia and ascospores spread through rain water and wind.

Epidemiology:

- The dissemination of conidia takes place only by the action of rain or dew.
- A film of water is required for conidia formation.
- Conidia collection takes place early in the morning when leaves are covered with dew.
- Perithecia are produced during warm, humid weather.
- The disease spread is favoured by warm (23-25^{0}C) and rainy humid weather.
- Below 21^{0}C reduction in the infection even if other conditions are favourable.
- The disease is common on poor, badly, drained soils and in shaded areas.
- Closer spacing heavy weed population or grass cover, failure to remove the suckers enhances the disease spread.
- Banana grown in partially shade especially under coconut tends to be less affected.
- Rainy days favour disease development and spread.
- RH (morning) and minimum temperature positively correlated on disease incidence.

- RH (evening) and max temperature negatively correlated on disease incidence.
- For every 1% increase in RH 5 points increases in disease severity index in Coimbatore.

Management:

- Removal and destruction of infected leaves.
- Improved drainage.
- Field sanitation i.e. efficient weed control.
- Regular removal of suckers.
- Proper spacing and fertilizer application.
- Spraying of any one of the fungicides like mancozeb (0.2%), Tridemorph (0.2%) Carbendazim (0.1%) Chlorothalonil (0.2%), Thiophanate. Methyl (0.1%) Propiconazole (0.1%), Hexaconazole (0.1%) along with spreading agents like teepol or sandovit or Triton, AE Continuous use of same systemic fungicides will cause resistance or tolerance in pathogenic population. Hence alternate spray of contact and systemic fungicides at 10-15 days interval should be preferred.

3. **Moko disease / Bacterial Wilt: *Burkholderia solanacearum***

- The disease was first noticed in British Guyana in 1840.
- The disease is widely distributed and occurs in most banana growing regions.
- In India the disease was first reported in west Bengal in 1968 and then in southern states of Tamil Nadu and Kerala in 1979.
- It is reported to occur on cv. Robusta and Poovan in TN causing 75-100% loss.

Symptoms:

- The affected plants show rapid wilting and collapse of the leaves.
- The newly emerged three leaves turn pale green or yellow and collapse near the point of lamia and petiole.
- Most leaves collapse within 3-7 days.
- In Cavendish banana the lower leaves become yellow.
- This spreads to upwards.
- Later the leaves acquire a white tinge – yellow and become dry and flaccid and readily droop.
- The petiole breaks at its junction with pseudostem and droop around the pseudostem.
- If the diseased suckers planted, the terminal leaf become necrotic and the plants die.
- The suckers in infected plants also affected.
- The affected suckers are blackened get twisted and remain stunted.

- The internal symptoms indicate that moko is a typical vacular infection or vascular disease.
- When the pseudostem, corm and peduncle cut transversely all the vascular strands are pale yellow to dark brown or bluish black.
- In some advanced condition, a reddish brown ring of vascular tissue may be seen.
- The vascular discoloration tends to be central.
- Opaque greyish brown or dirty white, slimy bacterial ooze comes out from the cut opened pseudostem.
- The fingers of the infected plants become distorted or turn yellow.
- The pulp turns into a very characteristic dark brown colour with dry rot symptoms.

The distinguishing symptoms between moko and Panama

Panama	Moko
No external symptoms upto 4-6 months	Suckers are blackened get twisted and stunted.
No symptoms on newly emerged leaves and are not collapsed	Newly emerged leaves turn pale green or yellow and collapse
Leaves turn yellow	Leaves turn white tinge yellow
Wilting of leaves starts from older leaves	Wilting of leaves starts from youngest leaves
More number of suckers are produced and all the suckers are affected	A few suckers are affected
Longitudinal splitting of psedostem seen above soil level	No longitudinal splitting of pseudostem
Dark brown or black discoloration of water conducting tissues in pseudostem	Pale yellow to dark brown or bluish black discoloration of water conducting tissues in pseudostem
Reddish tinge is present in pseudostem	Absent
Reddish brown ring of Vascular tissues in the corm is absent	Present
Central portion of the affected corm is dark brown.	Central portion of the affected corm is Yellowish brown
In the corm purplish streaks run radiating from the centre in all direction.	In the corm no purplish streaks run radiating from the centre in all direction.

The vascular discoloration in the corm is peripheral	The vascular discoloration in the corm tends to central.
No bacterial ooze comes out from pseudostem	Opaque grayish brown or dirty white slimy bacterial ooze comes out from pseudostem
No soft rot of corm or pseudostem	Soft patches of bacterial rot is seen in the corm and pseudostem
The flesh become pithy acrid and yellow in colour	The flesh turns into a very characteristic dark brown with dry tor symptoms.

Causal organism: *Ralstonia solanacearum* (*Burkholderia solanacearum*)

Moko disease is caused by the bacterium *Ralstonia solanacearum* (Yabuuchi, Kosako et al. 1995). Initially when it was identified by Rorer in 1911 he named the causal agent *Bacillus musae* however after the bacterium was shown to cause symptoms on solanaceous plants it was reclassified as *B. solanacearum* (Ashby 1926). Then *Pseudomonas solanacearum then Burkholderia solanacearum*. This group has now been renamed *Ralstonia solanacearum*. The bacterium is described as an aerobic, gram-negative, non-fluorescent rod belonging to the rRNA homology group II and single polar flagellate (Palleroni 1984).

Ralstonia solanacearum is a species complex and is traditionally classified into five races based on differences in host range and into biovars based on biochemical properties.

Moko disease, a bacterial wilt of banana, is recognised as *Ralstonia solanacearum* race 2, biovar 1.

Mode of spread and survival:

- The bacterium survives through infected suckers and in soil for 12-24 months.
- Other host plants include ornamentals e.g. *Heliconia*, red ginger, cocoa, dasheen, tomato, potato, castor bean ("Oil nut") and several weeds.
- The disease is spread through infected rhizome cutting machetes, insects, soil, and irrigation water.

Management:

- Strict plant quarantine and phytosanitary measures against movement of infected suckers from one place to other.
- The suckers should be collected from disease free fields.

- Use of disease free suckers.
- Removal and destruction of weed hosts like *Heliconia*
- Disinfection of pruning machetes using formalin diluted with water at 1:3 ratio.
- Early detection and rapid destruction of infected plants.
- Removal of male flowers after the emergence of female hand.
- Better drainage should be provided in the field.
- Avoidance of irrigation water passing from infected field to healthy fields.
- Keeping the soil fallow and weed free for 6-12 months.
- Crop rotation with non hosts like sorghum sugarcane and rice. (In TN 3 year rotation of banana, Sugarcane and rice (two crops) effective.
- Soil and planting materials can be fumigated with methyl bromide.
- Bacterization of planting materials, soil and capsule application of *P. fluorescens*.

4. *Erwinia* Head Rot / Tip Over / Rhizome Rot: *Erwinia carotovora* sub sp. *carotovora*

The disease is wide spread in banana growing areas of the world. In India it is present in Tamil Nadu, Kerala and Andhra Pradesh. The disease was first noticed in cv, Monthan and Pachanadan in 1985 in Coimbatore, Tamil Nadu. It is most commonly observed during hot summer and monsoon season. The disease is predominantly observed in tissue culture plants at 3-5 months after planting.

Symptoms:

- The affected plants show discoloration and soft rot of rhizome and suckers.
- Rotting of collar region followed by epinasty of leaves which dry out suddenly.
- The pseudostem tips over breaking across the rotted stem at the ground level.
- Infected plants can be pushed over easily and it comes out from collar region leaving the corm in the soil.
- Severe splitting of pseudostem is common in late stage of infection and being locally called vedivazhai (Split banana) in Kerala
- Yellowish to reddish bacterial ooze is seen when collar region is cut open.
- Bunch fails to develop.
- In severely infected soil newly planted rhizome rots
- Dark water soaked areas develop in the rhizome.
- Cavities are formed in Rhizomes and the rotten corm emits foul smell.

Casual Organism: *Erwinia carotovora* sub sp. *carotovora*

- Gram negative bacteria with peritrichous flagella.

Mode of spread and survival:

- The pathogen is soil borne enters through wounds.
- The disease also spread through infected suckers.

Management:

- Selection of disease free suckers for planting.
- Use of bigger sized suckers (>500g)
- Avoid planting during rainy season.
- Provide adequate drainage system.
- Banana should not be grown along with onion. Soft rot pathogen increases.
- Soil drenching with 0.2% Bleaching powder at planting and 3rd month of planting.

5. Bunchy top – Banana Bunchy top virus

- The disease is also called as **strangles**, or **curly top** or **cabbage top.**
- It is one of the threatening diseases of banana occurring throughout the world.
- It was first reported in *Musa* sp. in 1879 in Fiji and thereafter from almost all the banana growing countries of the world.
- In India it was first reported from Bengal in 1925, Bihar in 1940, Assam in 1941 and Kerala in 1943.
- Now the disease is prevalent throughout the country.
- It was reported as an introduced disease to our country from Sri Lanka.
- ICAR has categorized bunchy top as one of the diseases of national importance due to drastic and non bearing of fruits.
- Bunchy top disease almost wiped out many precious varieties like Hill banana (Virupakshi) in TN, Nendran and Planthodan of Kerala, Harissal and Lal Velchi of Maharashtra.

Symptoms:

- The disease initially shows series of dark green dots or streaks along the secondary veins on the underside of the lamia, mid rib, petiole and leaf sheath. This symptom is referred to as "**Morse code streaking**" because the streaks are irregular and resemble a series of "dots" and "dashes."
- Dark green, **hook-like** extensions of veins can be seen in the narrow, light-green zone between the midrib and the lamina
- A powdery bloom covers the mid rib and petiole.
- The leaves are very small, narrow, erect and brittle in texture
- Leaves show marginal chlorosis and upward rolling.
- Petiole incompletely elongated.
- The leaf margin become wavy
- Transverse of wrinkling occurs along the length of lamia.

- The affected plants become very dwarf.
- The internodes get shorten.
- Bunching of leaves occurs at the apex of the pseudostem forming a **rosette or bunchy top.**
- Opening of young leaves is earlier than older leaves giving a **funnel – shape** at the top.
- Pale whitish streaks may be seen along the length of lamia.
- Mottling on petiole, pseudostem and flower.
- In early infection no bunches are produced.
- In late infection, bunches are very small and malformed.
- The emergence of bunches are chocked by pseudostem and may split it.
- The rhizomes show decaying.
- The affected plants do not die for at least 1or 2 years.

Casual organism: Banana Bunchy Top Virus (BBTV) OR Banana Virus 1 (or) Musa Virus 1

The virus is isometric, non – enveloped, circular, single stranded DNA virus, 18-20 nm in dia.

Classified in the genus Babuvirus, within the family Nanoviridae

Mode of spread and survival:

- The virus survives in infected plants and perennial hosts.
- Infected suckers are primary source of spread.
- The virus is not sap transmissible.
- 'Transmission by banana aphid *Pentalonia nigronervosa*
- Non persistant virus.
- All the stages of aphid can acquire and transmit the virus.
- Nymphs are most efficient than adults.
- Single aphid can transmit the virus. The minimum no of aphids required for maximum infection is 20.
- Acquisition feeding period is 3h.
- The transmission feeding period in 30min.
- The aphids remain infective up to 13 days.

6. Banana Mosaic / Infectious chlorosis – Cucumber Mosaic Virus (CMV)

- It is also called as **Heart rot** or **Mosaic disease** or **virus sheath rot**.
- The disease was first recorded in NSW of Australia in 1930.
- In India it was first observed in 1949 in Maharashtra and Gujarat.

❖ Now it occurs in almost all banana growing states of the country.

Symptoms:

❖ The disease is characterized by the presence of typical mosaic like or discontinuous linear chlorotic streaks in bands extending from margin to mid rib along the veins.

❖ Raised veins, rolling of leaf margin, twisting and bunching of leaves at the crown

❖ Newly emerged leaves are rigid and erect.

❖ The leaves are narrow and small.

❖ Plants remain stunted.

❖ Plants seldom produce bunches.

❖ Early infection leads to sheath rot and heart rot phase especially in Cavendish and robusta clones.

Casual Organism: Cucumber Mosaic Virus (CMV)

❖ The virus is isometric and 26nm in dia possess tripartite ssRNA genome. Non persistent virus.

❖ The host range of the virus exceeds 800 spices.

Transmission:

❖ Not sap transmissible.

❖ Primary spread through infected daughter suckers.

❖ Sec. spread through aphids like *Aphids gossypi*. *A. craccivora*, *Rhophalosiphum maidis, Myzus persicae* etc.

❖ *Cucumis sativus* var. khira serves as a reservoir of the virus.

7. Banana Bract Mosaic virus: Banana Bract Mosaic Virus (BBMV)

❖ The disease is also considered as a disease of economic importance.

❖ This is commonly known as Kokkan disease since 1966.

❖ Presently the virus is a major constraint in southern states of India.

❖ Nendran, Robusta, Poovan, Ney Poovan, Rasthali, Karpooravalli, Monthan and Red banana are highly susceptible.

Symptoms:

❖ Spindle shaped pinkish to reddish streaks are seen on pseudostem, midrib, peduncle.

❖ In Nendran variety, the orientation of leaf changes giving the appearance of **traveler's palm**.

❖ Spindle shaped waxy coating appears on lower side of the leaves corresponding with spindled shaped mosaic pattern on the upper surface.

- In Nendran variety, very short or long peduncle, abortion of bunches and raised corky growth on the peduncle also observed.
- In Ney Poovan and Nendran varieties, necrotic streaks are seen on leaf, pseudostem, petiole mid rib and fingers.
- In Red banana and Robusta leaf stripping symptom is common.
- In Robusta, fingers stop to develop and become pencil shaped and locally called as **pencil kai** (Pencil sized fruit).
- In Kerala farmers named the disease as **Pola roga** means **disease of pseudostem** in cv. Nendran.

Casual Organism: Banana Bract Mosaic Virus (BBMV)

- The virus is flexuous rod – shaped measuring 750 nm x 15 nm in size.
- Belongs to PVY group.

Transmission:

- Transmission by *Pentalonia nigronervosa, Aphis gossypii, A. craccivora*
- Non persistent virus

Banana streak Virus

The disease was first noticed on Poyo belong to Cavendish sub group (AAA) and was first reported in1986 in Dwarf Cavendish and Giant Cavendish. The yield loss was 48%.

Symptoms:

- Initially small, golden yellow dots appear on the leaves.
- Later they extend to form long chlorotic streaks.
- The chlorotic streaks become necrotic giving a blackish appearance on lamia.
- Necrotic streaks are also seen on mid rib pseudostem and petiole.
- Fruits get distorted, plant vigour reduced.
- Virus particles are bacilliform 119 x 30 nm.
- Transmitted by the citrus mealy bug *Planococcus citri* as non persistent manner.
- The disease is controlled by planting virus free suckers and control of vector.

Management of virus diseases of banana:

- Selection of suckers from healthy garden.
- Use of disease free suckers for planting.
- Plant quarantine. Prohibition of movement of infected suckers to healthy areas.
- Periodical Removal and destruction of infected plants.
- Use of fernoxone 80WP (2,4-D) or Gramaxone 20 EC applied through capsule applicator or psedostem injection 5 ml (125 mg/lit)

- Control of insect vector using systemic insecticides like carbofuran 3G @ 40g/ Phosphamideon 1ml/lit / Methyl dematon 1ml / li
- Heat treatment of suckers at 40^0C for one day for banana mosaic.
- Avoidance of cucurbitaceous and leguminous intercrops for banana mosaic.
- Eradication of weed hosts for banana mosaic.

Post Harvest diseases

1. **Cigar end rot – *Verticilluim theobromae***

Wide spread in banana growing areas. In India it has been reported from Andhra Pradesh and Madhya Pradesh. It can cause serious loss of fruits.

Symptoms:

- It attacks even the immature fingers.
- Sometimes few fingers or all the fingers in the bunch are affected.
- Infection starts from the tip of the fingers.
- The disease causes blackening and shrinkage of the skin and folding of tissues in the fingers.
- The corrugated necrotic portion is covered with powdery mass of conidiophores and conidia
- This gives the appearance of grey ash of a cigar end. Hence it is called as **Cigar end rot**.
- There is a clear demarcation between infected tissues and healthy tissues.
- Normally less than 2cm length to one third of fruit is affected.
- The internal pulp is reduced to dry fibrous condition known as dry rot.

Casual organism: *Verticilluim theobromae*

Conidia are hyaline, oblong to cylindrical.

2. **Anthracnose/ Black rot/ Fruit rot – *Gloeosporium musarum* (*Collectorichum musae*)**

It is a serious disease in all banana growing areas especially in Bihar, Karnataka and Tamil Nadu. All the varieties are susceptible. Causes severe infection on table varieties.

Symptoms:

- The pathogen attacks the plants at all stages of their growth.
- There are two types of anthracnose infection.
- They are latent and non latent.
- The latent infection originates in the field on uninjured green fruits, when the fruit reaches maturity typical lesions develop on the fruit.

- ❖ Non latent infection starts during or after harvest and develops without dormant period.
- ❖ The disease starts as small, black circular specks on flowers and fruits.
- ❖ These specks increase in size and become sunken dark-brown spots with diffused edges
- ❖ Spots coalesce forming larger lesions with bright moist salmon – pink coloured spore masses.
- ❖ The skin of the fruit turns to black, shrivels and covered with pink acervali.
- ❖ The latent infected fruits ripe prematurely
- ❖ This causes discoloration and rotting of the pulp.

Casual organism: *Gloeosporium musarum* (*Collectorichum musae*)

- ❖ Mycelium is septate and coloured
- ❖ Acervali – round or elongated, erumpent, setae absent
- ❖ Conidiophores – Cylindrical, tapered towards apex hyaline, septate, branched
- ❖ Conidia – small, hyaline, single celled, aseptate, oval to elliptical, guttulate,

Mode of spread:

- ❖ Air – borne conidia

Epidemiology:

- ❖ The opt. temperature for disease development is 30- 35^{0}C.
- ❖ Conidia germinate at 85.7 – 100% RH.
- ❖ The disease is more abundant during rainy season.
- ❖ The disease is more severe in June – September when the temperature is high accompanied by showers for number of days.

3. **Crown / Stem end rot – *Botryodiplodia theobromae* (*Lasiodiplodia theobromae*)**

- ❖ The fungus invades the fruit through wounds.
- ❖ The pulp turns rapidly into black watery mass.
- ❖ Greyish black mycelial growth is seen on the fruit surface.
- ❖ The skin becomes black, soft and wrinkled.
- ❖ The skin later gets encrusted with pycnidia
- ❖ Maximum damage to fruits occur at 25 – 30^{0}C and 7^{0}C

4. **Deightoniella leaf and fruit spot / Black spot/ Black tip/ Fruit speckle: *Deightoniella torulosa* (Syn*. Helminthosporium torulosum)***

- ❖ The disease is noticed in many places of TN on Robusta variety.
- ❖ The disease occurs in three forms namely Black spot on leaves, black tip or black end on fruits and speckle (fruit spot) on fruits.

- The disease first appears as round pin point black spots on the main veins of lamina close to the leaf margin.
- These increase in size.
- The spots are separated from healthy tissue by a narrow bright yellow peripheral band.
- The centre of the spots dry and the pale brown areas extend to the edge of the leaf margin.
- The infected fruits show a black tip below the perianth advancing along the fruit.
- The diseased areas are surrounded by a narrow grey or yellowish margin.
- The diseased areas may crack when the fruits develop.
- At later stage pale brown mycelial wefts of the fungus is seen on the surface of the fruit.

Casual Organism: *Deightoniella torulosa*

Conidiophores arising singly or in small groups, brown, 40–170 × 6–10 µm, straight or slightly flexuous, occasionally branched, thick-walled, with up to 6 successive percurrent proliferations near apex, swollen at apex and at point of proliferation to 13–16 µm giving a 'beaded' appearance.

Conidia single, distoseptate, olivaceous, smooth, obpyriform to obclavate, straight or slightly curved, tip rounded, basal cell with a dark protruding scar.

- Conidia spread by air
- Infection requires dew or rain water and is more severe during wet weather.

5. **Main stalk rot / Black head – *Ceratostomella paradoxa***

- The main stalk decay rapidly.
- The affected tissues become soft and black.
- Emit a characteristic sweet smell.
- Black mycelial growth is seen on the rotting area.
- Infection spreads to hands, fruit stalk and to stem end of the fruits causing black lesions.
- The affected fingers drop off
- The disease spreads to fruit causing uneven black discoloration on the skin.
- The disease causes premature ripening of fruit.
- The pulp is changed to dark brown soft wet mass

6. **Pitting disease – *Pyricularia grisea***

- Round sunken pits appears on the finger stalk, crown pads and fruits.

- The sunken centre is surrounded by reddish brown zone with greenish, narrow water – soaked halo.
- The finger stalk infection leads to fruit drop.
- The disease causes serious post harvest loss of quality.

Management of post harvest diseases:

- Eradication alternate hosts like *Heliconia bihal*, *H. brasiliensis*, *Bambusa* sp, *Musa* spp for the control of cigar end rot
- Young bunches should be opened up to light and air by removing the bract.
- Avoiding of closer spacing.
- Removal of distal floral remnants perianth and male buds regularly bud after all the hands are opened.
- Removal of infected materials.
- Covering the bunch with polythene sleeves having 4-6% ventilation immediately after all the hands is opened.
- Harvesting of bunches at correct stage of maturity
- Handling of fruits without causing any wounds, bruise or scratch on fruit skin.
- Preharvest spray with Prochloroz 0.2% (or) Carbendazim (0.1%) Chlorothalonil 0.2% four times at fortnightly intervals.
- Dehanding, delatexing and dipping in fungicidal solution (TBZ, Benomyl, Carbendazim),
- Vacuum packing or irradiation can also be followed.

Minor Diseases

Cordana leaf spot – *Cordana musae*

- The spots are oval pale brown or yellow in colour.
- The centre of the spots is necrotic with delicate concentric zonation.
- The spots are surrounded by yellow halo.
- The spots coalesce to form larger infected zones.
- The underneath of such spots become grayish brown without clear border.
- As the spots grow light fawn coloured lesions with deeply coloured zonation become prominent.
- The underside of the spots becomes smoky grey due to large number of conidiophores.

Freckle Leaf spot: *Phyllostictinia musarum*

- Common in many districts of Tamil Nadu.
- Numerous minute dark brown raised spots are formed on the lower surface of leaf.

- Similar spots are also seen on the fruits giving unsightly appearance.

Bacterial leaf spot – *Xanthomonas musicola*

- First reported in TN.
- Widespread on Monthan, Peyan, Poovan and Rasthali varieties.
- Chlorotic linear streaks appear along the veins.
- Streaks coalesce to form large chlorotic patches.

DISEASES OF GUAVA

1. Wilt – Fusarium oxysporum f.sp psidii; *Fusarium solani*; *Macrophomina phaseolina*; *Cephalosporium* sp
2. Anthracnose – *Gloeosporuim psidii*
3. Fruit canker – *Pestalotiopsis psidii*
4. Red rust – *Cephaleuros virescens*
5. Sooty mould -
6. Post Harvest Diseases
    1. *Phytophthora* fruit rot – *Phytophthora nicotinae* var. *parasitica*
    2. Dry rot – *Diplodia natalensis*
    3. *Phomopsis* fruit rot/ Stylar end rot – *Phomopsis psidii*
    4. Anthracnose – *Gloeosporium psidii*
    5. Fruit canker / Scab – *Pestalotiopsis psidii*
    6. Soft watery rot: *Botryodiplodia theobromae* Pat
    7. Botryosphaeria rot: *Botryosphaeria ribis* Gross. & Duggar

1. Wilt – many fungi

Wilt is the most important disease of guava.

Causes serious loss in guava production

First reported in Allahabad, UP in 1935

Occurs in almost all the guava growing states

More severe in UP and WB

Wilted guava plants have also been reported from Florida, U.S.A. (Webber, 1928), Taiwan (Leu and Kao, 1979), Cuba (Rodrigvez and Landa, 1977), South Africa (Vos et al., 2000), Brazil (Junqueira et al., 2001), Pakistan (Ansar et al., 1994), Bangladesh (Hamiduzzamanet al., 1997), and Canberra, Australia (Lim and Manicom, 2003)

In general, losses due to wilt in guava around Lucknow area vary from 5-60 per cent (Misra and Shukla, 2002). In West Bengal, the disease reduced the yield by 80 percent

Symptoms:

- First external symptom of the disease is yellowing with slight curling of leaves at the terminal branches
- Later drowsiness of plants with yellow to reddish discolouration of the leaves
- Premature defoliation
- Twigs become bare and fail to put forth new flushes and eventually dry up.
- Small sized fruits
- Fruits remain under developed, hard and stony
- Later, the entire plant becomes defoliated and eventually dies.
- Finer roots show black streaks on removing the bark.
- The cortical regions of the stem and roots show discolouration and rotting.
- Vascular tissue becomes light brown in colour.
- Partial wilting is also very common
- Wilted plants later show bark splitting.

Casual organism:

The exact causal agent of the disease is still not fully understood. Various pathogens were involved with the affected plants viz. *Fusarium oxysporum* f. sp. *psidii*, *F. solani*, *Macrophomina phaeseolina*, *Cephlosporium* sp., *Gliocladium roseum* and *Verticillium albo-atrum* etc. have been reported by different workers.

Mode of Spread and survival:

- The disease is soil-borne
- The pathogens survive on dead root bits in soil.
- Spread through movement of plants containing sick soil in virgin areas.
- Short distance spread is by irrigation water

Epidemiology:

- Severity of the disease is more in rainy season.
- Highest incidence is noticed during September- October
- Trees of 6 years of age and above are generally affected.
- More disease in clay loam and sandy loam compared to heavy soil types.

Management:

- Field sanitation
- Removal and destruction of wilted plants
- Forming trenches around the wilted plants
- Avoiding injuries to roots while transplanting
- Maintaining tree vigour with adequate irrigation, manuring, interculture etc.,
- Incorporation of organic maures or green manures
- Use of resistant root stocks like jamun, *Psidium* cattleianum var. *lucidum* and a cross of *Psidium malle* x P. *guajava*

- The pits may be treated with formalin and kept covered for about 3 days and transplanting after two weeks.
- Biological control by *Aspergillus niger* strain AN-17 is found effective. *Aspergillus niger* multiplied in FYM @ 5 kg/pit, applied in the pits while planting new plants.
- In older plants *Aspergillus niger* enriched FYM can be applied @ 10 kg plant1.
- Partially wilted plants recovered fully after rejuvenation / heavy pruning of the tree
- Trunk injection of 0.1% 8 Quinolonol sulphate
- Soil application of lime or Gypsum @ 4lb / trees
- Severe pruning of affected branches followed by soil drenching with 0.2% Benlate or Carbendazim 4 times in a year and foliar spraying with Methyl dematon and Zinc sulphate in a balanced manner.

2. **Anthracnose: *Gloeosporuim psidii***

Common in all the guava growing regions of India

Causes die back, twig blight, wither tip and fruit rot

Symptoms:

Die back phase:

- Young shoots, leaves and fruits are readily attacked, while they are still tender.
- The growing tips turn dark brown
- The black necrotic areas extend backward causing dieback of the plant.
- The plant begins to die backwards form the tip of a branch.
- The fungus develops from the infected twigs and then petiole and young leaves.
- These droop down or fall leaving the dried twigs without leaves.
- The disease appears in epidemic form, during August to September.

Fruit and leaf infection phase:

- Pin-head spots are first seen on unripe fruits, which gradually enlarge.
- They gradually enlarge to form sunken circular, dark brown to black spots or rough blisters with acervuli in concentric rings.
- Creamy spores are produced in masses in moist weather.
- Several spots coalesce to form bigger lesions.
- The infected area on unripe fruits become corky and hardy, and often develops cracks.
- Unopened buds and flowers are also affected which causes their shedding.
- On leaves, the fungus causes necrotic lesions at the tip or on the margin.
- These lesions are usually ashy grey and bear fruiting bodies.

Casual Organism: *Gloeosporuim psidii*

Acervuli - irregular in shape and approximately 500 µm in diameter. Setae are one to four septate, brown, slightly swollen at the base, and tapered at the apex.

Conidiophores - hyaline to faintly brown

Conidia - hyaline, unicellular, and either cylindrical with obscure ends or ellipsoidal with a rounded apex and a narrow, truncate base. They form on in

Mode of spread and survival:

- Survive on mummified fruits and infected tissues of twigs and branches on the tree.
- The conidia are disseminated by wind or rain

Epidemiology:

- The disease develops more rapidly at 30^0C and 96.1% RH on both ripe and unripe fruits.
- Closer planting without canopy management
- Lack of timely harvesting
- Availability of free water in the farm of dew or rains encourages spore production and its dispersal around canopy.

Managements:

- Orchard sanitation
- Pruning and burning of infected twigs.
- Spraying captafol or Zineb or Benomyl
- Pre and post harvest spray with Benlate 0.1%
- Post harvest dip in Benlate or TBZ is also helpfull

3. **Fruit canker/ grey blight / leaf spots / scab: *Pestalotiopsis psidii***

Symptoms:

- The disease generally occurs on green fruits and rarely on leaves.
- Small rusty brown angular spots appear on the leaves.
- Spots enlarge with dark brown margin and grey or ashy centre with acervuli.
- On fruits minute brown or rust coloured, circular necrotic areas develop.
- Later the infection tears open the epidermis in a circinate manner.
- The margin of the lesion is elevated with depressed centre showing crater –like appearance.
- In older cankers, white mycelium consisting of numerous spores is noticeable.
- In severe cases the fruits break open to expose seeds.
- The fruits become under developed hard malformed and mummified which drop off.

Casual Organism: *Pestalotiopsis psidii*

Spread and survival

- Survive on infected leaves, twigs and fruits

- Conidia air borne

Management:

- Orchard sanitation
- Pruning and burning of infected twigs.
- Spraying captafol or Zineb or Benomyl
- Pre and post harvest spray with Benlate 0.1%
- Post harvest dip in Benlate or TBZ is also helpfull

4. **Algal leaf and Fruit Spot: *Cephaleuros virescens***
    - Orange, rust- coloured, dense, silky tufts appear on both surfaces of leaves
    - They turn reddish-purple in colour as they mature
    - If tufts are scraped away, a thin gray-white or dark-coloured necrotic spot remains
    - Bark on twigs and branches may be cracked
    - Young stems and fruit may also be attacked
    - Lesions are usually smaller than leaf spots.
    - As fruits enlarge, lesions get sunken.
    - They are darkish green to brown or black to colour.
    - Cracks develop on older blemishes as a result of enlargement of fruits
    - The pathogen sporulates readily during the period of high rainfall (July-September)
    - The disease incidence is greatest during September.
    - Spray Copper oxychloride (0.3%) 3-4 times at an interval of 15 days when initial symptoms noticed
5. **Cercospora Leaf Spot: *Cercospora sawadae* Yamamoto**
    - Water soaked, brown irregular patches on the lower surface
    - Yellowing on the upper surface of the leaf.
    - Older leaves are mostly affected.
    - Severely affected leaves curl and subsequently drop off
    - Spray mancozeb 0.2% at monthly interval
6. **Sooty mould** *Phragmocapnias betle, Scorias philippensis, Tichomerium grandisporum, Limacinula musicola, Aithaloderma clavatisporum, Tripospermum* sp., *Polychaeton* sp., *Leptoxyphium* sp. *and Conidiocarpus* sp.
    - Sooty mould proliferates in abundance on the foliage
    - Blackish brown velvety thin membranous covering on the leaves
    - In severe infection, the foliage appears black due to heavy infection.
    - The affected leaves curl and shrivel under dry conditions
    - The disease is controlled by controlling the insects by suitable insecticides.
    - Foliar spraying of wettable sulphur + chlorpyriphos + Gum Acacia (0.2 + 0.1 +0.3%) at 15 days interval has been found very effective.

7. **Damping off of Seedlings: *Rhizoctonia solani* Kuhn**

- Both pre emergence and post emergence phases are observed.

Pre emergence phase

- The infected seeds and seedlings show water soaked discoloration
- The seed becomes soft and ultimately rots.
- The affected young seedlings are killed before they reach the soil surface.

Post emergence phase

- Hypocotyle at ground level or upper leaves are discolored into yellowish to brown colour
- It spreads downwards and later turn soft and finally rot and constrict.
- The affected seedlings ultimately topple down and die.
- Strands of mycelium may appear on the surface of the plants under humid conditions.

Management:

- Diseased seedlings should be removed and burnt.
- Excessive use of water and close planting should be avoided
- Seedbeds should be prepared with proper drainage arrangement.
- Dipping of guava seeds in captan / thiram (0.2%) is advocated
- Drenching of soil with Copper oxychloride (0.3%) helps in reducing the diseases intensity in nursery.

8. **Guava rust: *Puccinia psidii* G. Wint.**

Symptoms

- Orange to red pustules appear on leaves young shoots, flowers and/or fruit
- Leaves distorted
- Defoliation
- Reduced growth

9. **Post Harvest Diseases**

1. ***Phytophthora* fruit rot – *Phytophthora nicotinae* var. *parasitica***

Occurs in Tamil Nadu, AP, Rajasthan, Punjab, Maharashtra and KK

The disease incidence varies from 8-30%, depending upon the weather and foliage conditions

Symptoms:

- The symptom starts at calyx disc (stylar) end of the fruit during rainy season
- Whitish cottony growth develops and covers almost entire surface rapidly during humid weather.
- The skin becomes little soft turns light brown to dark brown.

- Affected fruits emit a characteristic unpleasant smell.
- Affected fruits either remain intact on tree or drop off.
- The young or half matured fruits become shrunken, dirty brown, hard and either remain intact as mummified fruit or drop off.

Epidemiology

- Rain and the wind are important for spread.
- Sporangial production is more on the surface of diseased tissues when the temperature is near 25^0 C
- This is an important source of inoculum in the development of epidemics.
- Drops of rain are necessary for the liberation of sporangia from the infected plant material or soil.
- Under high relative humidity, the fruits near the soil level covered with dense foliage are most severely affected.
- The fallen fruits are badly affected.
- Cool, wet environmental conditions with high soil moisture favour disease development.
- High humidity, temperature from 28-32^0C, poorly drained soils and injuries are important for initiation of disease.
- Close plantation.

Management:

- Spraying Dithane Z-78 (0.2%) or Ridomil or Aliette (0.2%) or Copper oxychloride (0.3%) are found effective to control foliar infection.
- Soil drenching with Copper oxychloride (0.3%) or Ridomil or Aliette (0.2%)
- Avoiding unnecessarily dense plant canopy

2. **Anthracnose: *Gloeosporuim psidii***

Symptoms:

- Pin-head spots are first seen on unripe fruits, which gradually enlarge.
- They gradually enlarge to form sunken circular, dark brown to black spots or rough blisters with acervuli in concentric rings.
- Creamy spores are produced in masses in moist weather.
- Several spots coalesce to form bigger lesions.
- The infected area on unripe fruits become corky and hardy, and often develops cracks.

Managements:

- Orchard sanitation
- Pruning and burning of infected twigs.
- Spraying captafol or Zineb or Benomyl

- ❖ Pre and post harvest spray with Benlate 0.1%
- ❖ Post harvest dip in Benlate or TBZ is also helpfull

3. **Fruit canker / scab: *Pestalotiopsis psidii***

Symptoms:

- ❖ The disease generally occurs on green fruits.
- ❖ On fruits minute brown or rust coloured, circular necrotic areas develop.
- ❖ Later the infection tears open the epidermis in a circinate manner.
- ❖ The margin of the lesion is elevated with depressed centre showing crater –like appearance.
- ❖ In older cankers, white mycelium consisting of numerous spores is noticeable.
- ❖ In severe cases the fruits break open to expose seeds.
- ❖ The fruits become under developed hard malformed and mummified which drop off.

Management:

- ❖ Orchard sanitation
- ❖ Pruning and burning of infected twigs.
- ❖ Spraying captafol or Zineb or Benomyl
- ❖ Pre and post harvest spray with Benlate 0.1%
- ❖ Post harvest dip in Benlate or TBZ is also helpfull

4. **Dry fruit rot: *Diplodia natalensis***

- ❖ The disease initially appears as light brown spots at stalk end.
- ❖ The infection spreads very quickly covering the entire surface.
- ❖ The affected young and mature fruits become dark brown to almost black and ultimately dry up.
- ❖ Numerous pycnidia appear as pin head like structure on the rind of dried fruits.
- ❖ Spraying Ziride 0.3% Controls.

5. ***Phomopsis*** **fruit rot /Stylar end rot: *Phomopsis psidii* De camara and *P. destructim***

- ❖ Serious losses up to 10% occur
- ❖ The disease affects the unripe fruits at the stylar end.
- ❖ Discoloration appears in the region lying just below and adjoining the persistent calyx.
- ❖ Small circular white or light brown water soaked lesions are produced.
- ❖ Such area gradually increases in size and turn dark brown
- ❖ Later the affected area becomes soft
- ❖ Covers the entire surface quickly.
- ❖ The pulp becomes soft.
- ❖ The skin gets wrinkled and loose.
- ❖ Diseased area is pulpy and light brown in colour

- ❖ Later mycelial growth and pycnidia can be seen on the fruit surface.
- ❖ Spray Copper oxychloride (0.3%) or carbendazim or Thiophonate methyl (0.1%) before onset of winter fruiting.
- ❖ Post harvest dip in carbendazim 0.1% controls the disease.

6. **Soft watery rot: *Botryodiplodia theobromae* Pat**

Symptoms

The infection starts as a brownish discoloration mostly at the stem end

It gradually proceeds downwards in an irregular wavy manner.

Finally the whole fruit may get involved.

The decay takes in the form of a soft, watery break down

In advanced cases, numerous small pycnidia are produced over the entire surface of the fruit

Avoidance of wounding

Captan found effective against the fungus

Applications of *Bacillus subtilis* and *Streptosporangium pseudovulgare* on guava fruits have been found effective.

7. **Botryosphaeria rot: *Botryosphaeria ribis* Gross. & Duggar**

Symptoms

- ❖ The infection usually occurs at or near the distal end in the region of persistent calyx.
- ❖ The rot begins with a translucent zone around the distal end
- ❖ It becomes brown in colour.
- ❖ With the progress of the disease, the lesion becomes dark black and wrinkled with dry skin with translucent margins
- ❖ Spraying with copper oxychloride (0.3%)/ dithane M 45 (0.2%) at 15 days interval controls

Phanerogamic Parasites: *Loranthus* sp.

Symptoms:

- ❖ Guava trees are commonly affected in the neglected orchards.
- ❖ They are commonly present on trunk or branches of the tree
- ❖ Makes the tree weak.
- ❖ The foliage of the infected host plant is sparse and reduced in size
- ❖ Its bearing capacity and quality of fruit is considerably lowered.
- ❖ The point at which the guava host is penetrated is usually characterized by swollen growths called “burrs”.
- ❖ The burrs help in the identification of sites at which the parasite has entered the host

Management

- ❖ The affected branches should be cut sufficiently to completely eradicate the haustoria.
- ❖ Cuttings out affected portion of tree for enough below burrs to remove haustoria
- ❖ Cut surface is treated with wound dresser viz. Copper oxychloride (0.39%) paste/spray to prevent the secondary pathogens infecting through wounds.
- ❖ Spraying of emulsion of diesel (30-40%) in soap water is recommended found affective.

DISEASES OF POMEGRANATE

1. *Cercospora* leaf and fruit spot – *Pseudocercospora punicae (Cercospora punicae*)

- ❖ Leaf spots are subcircular to irregular, 1- 4 mm diam
- ❖ Initially brown, dingy gray to pale tan and eventually brown to dark brown at the margin.
- ❖ Black elliptic spots appear on the twigs.
- ❖ The affected areas in the twigs become flattened and depressed with raised edge.
- ❖ The affected fruits show small irregular black spots, which later coalesce into large spots.
- ❖ Conidiophores olive brown, short, fasciculate sparingly septate.
- ❖ Conidia hyaline to pale olivaceous cylindric, sub fusoid to sub - clavate septate.
- ❖ Primary source of inoculums is infected leaves, diseased plant debris.
- ❖ The pathogen spreads through wind borne conidia.
- ❖ The disease is serious during September to November.
- ❖ The diseased fruits should be collected and destroyed.
- ❖ Controlled by thiophanate - Methyl 0.1% or Chlorothalonil 0.2% or Mancozeb 0.2%

2. **Bacterial blight – *Xanthomonas axonopodis* pv. *punicae***

Bacterial blight has been of wide occurrence in India, resulting in economic losses in all major pomegranate growing areas of Maharashtra, Karnataka, Andhra Pradesh, Tamil Nadu, Himachal Pradesh and Rajasthan. Outside India it is reported from Pakistan and South Africa. The disease affects all plant parts, but is most destructive on fruits.

Symptoms

- ❖ The disease initially appears as irregular to circular, translucent, small, dark water soaked spots on leaves.
- ❖ Spots turn to dark brown to black oily and glistering are surrounded by prominent yellow hallo or water soaked margins.
- ❖ Later they become necrotic at the centre.
- ❖ Spots coalesce to form large patches.

- Severely infected leaves turn yellow and drop off prematurely.
- Stem infection seen around the nodes which leads to girdling and cracking of nodes.
- On fruits disease starts with water soaked lesions on the skin surface
- Lesions turn dark brown to black, irregular, slightly raised with oily appearance
- Spots enlarge and merge with each other to cover large area
- Fruits split open with L or Y-shaped cracks under severe stages of disease.
- The spots may be covered with thin shining white encrustation consisting of bacteria.

Casual Organism: *Xanthomonas axonopodis* pv. *punicae*

Gram-negative, aerobic, rod – shaped, with single polar flagellum

Mode of spread and survival

- This bacterium can survive on infected plant debris lying in and around the orchards for more than 8 months
- It can also survive for several years on stem cankers on plants or in dormant buds; these are major sources of primary inoculum
- Survives on fallen leaves and on the trees (120 days)
- Wind splashed rain, insects and contaminated pruning tools help in spreading the disease locally
- Long distance spread via infected plants, twigs, and fruits

Epidemiology

- Warm and humid condition favours the disease
- Disease build up is rapid during rainy season.
- High disease severity is observed from July to October.
- Temperatures between 25 to 35oC coupled with humidity above 50%, rains and wind favour rapid disease development.

Management

- Orchard sanitation
- Removal and destruction of infected plant parts by pruning
- Use disease free planting materials
- Spray Bordeaux mixture (0.5% except 1% just after pruning and rest period), altered with spray of streptocycline (5g/10 l) or 2-bromo, 2-nitro propane-1, 3-diol (5g/10 l) mixed with copper based formulations like copper oxychloride or copper hydroxide (20-25g/ 10 l) and spreader sticker (0.5ml/l) at 10-15 days interval depending on weather conditions.

3. **Wilt: *(Ceratocystis fimbriata, Xyleborus fornicates and X. perforans Fusarium oxysporum, Rhizoctonia solani and Meloidogyne incognita)***

Wilt is the second most important disease of pomegranate, adversely affecting pomegranate cultivation in India. The disease has been reported from Maharashtra, Karnataka, Andhra Pradesh and Himachal Pradesh. Pomegranate vascular wilt is mainly caused due to a fungus *Ceratocystis fimbriata* occasionally by other agents like *Fusarium solani and F. oxysporum.* Root rot organisms *viz. Macrophomina phaseolina, Phytophthora nicotianae, Rhizoctonia bataticola;* root-knot nematode *Meloidogyne incognita* and shot hole borer -*Xyleborus fornicates and X. perforans* - are the other agents which, alone or in association with *C. fimbriata* result in drying and wilting of plants.

Symptoms

- Initially yellowing of leaves is observed in some twigs or branches, followed by drooping and drying of leaves, finally the entire tree dies.
- Dark bluish-black/grey/brown discolouration of the wood is seen if the pathogen is *Ceratocystis, in Fusarium* browning of only xylem is observed.
- Pin holes are observed in the bark and wood when shot hole borers are associated with wilt.
- *Macrophomina* sp. destroy the feeder roots and result in root rots
- *Rhizoctonia* sp. cause girdling of stems in nursery or young plants-resulting in wilting
- In nematode infestations infected plants form knots on the roots

Mode of Spread survival

- Survive as conidia of *C. fimbriata,* in soil, and plant debris which are primary source of inoculum.
- *C. fimbriata*, can also be carried on through air, irrigation /rain water.

Management

- Use disease free planting materials
- Avoid water stagnation and create proper drainage.
- Follow recommended spacing of 4.5 m × 3.0 m in the orchard
- On observing first symptoms of wilt due to fungal pathogens immediately soil drench with chlorpyriphos 20EC (2.5-4ml/l) + carbendazim 50WP (2g/l) or propiconazole 25EC (2g/l). Use 5-8 l solution/tree. Also drench at least 2-3 healthy plants on all the four sides around the infected plant/s, repeat the drenching 3-4 times at 20 days interval.
- For root knot nematodes apply carbofuran 3G @ 40g/plant or phorate 10G @ 25g/ plant in wet soil. Drenching with azadirachtin 1% @ 2ml/l.
- Plant *Tagetes erecta* (African marigold) between plants in a row, or in a ring, on the border of plant basin, for more than 4-5 month.

- For shot hole borer (*Xyleborus* spp.), 10 litres preparation containing red soil (4kg) + Methyl parathion 4% dust (25g) + Chlorpyriphos 20EC (20ml) + Copper oxychloride (25 g) needs to be applied on plant base up to 1-2 ft. from second year onwards

4. ***Collectotrichum*** **Leaf and Fruit Spot –** ***Collectotrichum gloeosporioides***
   - The disease appears as small regular to irregular dull violet or black spots on the leaves.
   - The spots are surrounded by yellow margin.
   - Infected leaves turn yellow and fall off.
   - The pathogen spreads wind borne conidia.
   - High humidity and temperature of 20 – 27^{0}C prevailing during August – September favours for severe infection.
   - Controlled by carbendazim 0.1% or Topsin M 0.1% or Mancozeb 0.2% at fortnightly interval.
5. **Fruit spot –** ***Pestalotiopsis versicolor***
   - Brown or rust – coloured spots develop on fruits.
   - Spots coalesce and cause necrotic patches.
   - Centre of the lesions depressed inward with raised margin.
   - Severe infections tear open the rind and course discoloration of seeds.
6. ***Cladosporium*** **fruit rots:** ***Cladosporium oxysporum***
   - Olive brown spots develop on the fruits later the entire fruit rots.
7. ***Aspergillus*** **fruit rots –** ***Aspergillus niger; A.flavus; A.niveus; A.versicolor***
   - Starts as brown discoloration and become blackish brown and slimy and depressed.
   - Rotten fruits emit fermented odour.
   - Rotten area is covered with fungal growth.
8. **Soft Rot –** ***Rhizopus arrhizus***
   - 'First small spots appear on the fruits.
   - They increase and coalesce.
   - Infection restricted to rind.
   - The internal content decay into a pulpy mass
   - Under dry condition crackings develops at the point of infection
9. ***Collectotrichum*** **Fruit rot –** ***Collectotrichum gloeosporioides***
10. **Black heart:** ***Alternaria alternate***
11. **Phytophthora fruit rot:** ***Phytophthora*** **spp.**
12. **Botrytis grey mould:** ***Botrytis cineria***

DISEASES OF PAPAYA

1. Stem rot / Collar rot / Foot rot / Fruit rot / Root rot – *Pythium aphanidermatum*, *Rhizoctonia solani*
2. Anthracnose – *Colletotrichum gloeosporioides*
3. Powdery mildew – *Oidium caricae*
4. Phytophthora blight / Fruit rot / Stem rot – *Phytophthora nicotianae* var. *parasitica*
5. *Asperisporium* Black spot - *Asperisporium caricae*
6. *Cornyspora* Brown spot - *Corynespora cassiicola*
7. Papaya Mosaic - Papaya Mosaic Virus
8. Papaya Ring Spot – Papaya Ring Spot Virus
9. Papaya leaf curl – Tobacco leaf curl virus
10. Fruit rots – Many fungi

1. Foot rot / Stem rot – *Pythium aphanidermatum* and *Rhizoctonia solani*

The diseases is most serious all over the tropical and sub tropical region of the world. In India, the disease appears during rainy season and is prevalent throughout the country. The disease is able to annihilate the entire plantation within a season under favourable conditions and makes the soil unfit for papaya planting.

Symptoms:

- The disease occurs in both young and old plants and young seedlings in the nursery.
- Foot rot or collar rot or root rot is common in 2-3year old trees.
- Water soaked patches appear on the stem at collar region.
- The patches enlarge and girdle the base of the stem.
- The affected tissues turn dark brown or black and rot.
- The terminal leaves turn yellow wilt and drop.
- Fruits become shriveled and drop off.
- The entire plant topples down and dies due to disintegration of parenchymatous tissues.
- The internal tissues of the bark become dry brown and honey comb appearance.
- The rotting may extend upward and downward upto the roots.
- The roots also rot and are destroyed.

Casual Organism: *Pythium aphanidermatum* and *Rhizoctonia solani*

Pythium aphanidermatum

Mycelium – intracellular, branched, thick, hyaline Coenocytic,

Sporangia – Lobed toruloid vesicles formed, encysted zoospores

Oogonia – Spherical, Smooth walled borne in terminal hyphae

Antheridia – Broadly clavate, terminal or intercalary monoclinous

Oospores – Apleurotic, single

Mode of spread and survival:

- The pathogen is soil inhabitant.
- ***Pythium aphanidermatum*** survives in plant debris in soil producing oospores and chlamydospores
- ***Rhizoctonia solani*** survives in plant debris in soil producing sclerotia

Epidemiology:

- A number of factors viz., inoculum density, soil moisture, temperature, pH, light intensity, soil microbes determine the disease development and intensity.
- Papaya residue left out in the soil helps in increasing inoculum level and thus the disease incidence.
- Other predisposing factors are higher nitrogen application, soil conditions, excess soil moisture due to irrigation and heavy rain forming water logged condition.
- Younger plants are more susceptible than older plants.

Management:

- Use of disease free healthy seedlings
- Avoiding injuries to root and collar region of the stem at the time of planting
- Optimum irrigation
- Provision of adequate drainage facilities
- Avoiding direct contact of stem and irrigation water
- Removal and destruction of infected plants
- Seed treatment with captan @ 4 g / kg or *P. fluorescens* @ 10 g / kg
- Soil drenching with Bordeaux mixture 1% or COC 0.25% or Metalaxyl 0.1%
- Soil application of *P. fluorescens* @ 2.5kg /ha

2. **Powdery Mildew – *Oidium caricae***

The disease is prevalent in whole of north India and Maharashtra and Karnataka also.

Symptoms :

- The disease appears as of white powdery patches on both surfaces of the leaves
- More common on lower surface of the leaves.
- Affected areas become chlorotic and sometimes are surrounded by dark margin.
- Flower stalks and fruits are also affected with similar powdery growth.
- The affected portions of leaves later turn yellow and necrotic.

Casual Organism: *Oidium caricae*

Obligate parasite, hyaline septate ectophyte,

Conidia hyaline, granular

Mode of spread and survival

- The pathogen survives in infected plants parts.

- Wind –borne conidia

Epidemiology:

- Maximum disease incidence recorded during September – November with a peak in October.
- The disease decreases with a fall in temperature and an increase in sun shine hours.
- The development of powdery mildew is promoted by high humidity (80-85%) and a temperature range of 24-26°C.

Management:

- Removal and destruction of affected plant debris
- Spraying wettable sulphur 0.3% or Carbendazim 0.1% or Triademefan 0.1% or Benomyl 0.1% or Topsin M 0.1%

3. **Anthracnose: *Colletotrichum gloeosporioides***

Anthracnose is prevalent throughout India.

Anthracnose rot attains serious status during transit and storage causing economic losses.

Symptoms:

- The disease attacks leaves, petiole flower and fruits.
- Initiates as minute dark brown specks on leaves
- They enlarge in size and are surrounded by chlorotic margins
- Spots coalesce covering the entire lamina.
- Shot holes are formed at the centre.
- Necrotic spots are produced on stem.
- Pustules like spots are produced on petioles.
- Flower infection causes flower drop.
- On fruits small circular water soaked dark spots appear.
- The spots enlarge as the fruits mature forming circular sucken lesions.
- The lesions coalesce forming sunken, brown rotting patches.
- The surface of spots has zones formation.
- Acervuli appear as concentric rings.
- The fungus invades fruit tissues producing rot and turning them soft and dark.
- The whole fruit become dirty dark brown and rots.

Causal Organism: *Colletotrichum gloeosporioides*

Perithecia aggregated, globose to obpyriform, dark brown to black, 85-300 µm in diameter; the ostioles are periphysate and paraphyses are present.

The asci are 8- with short stalks, clavate to cylindrical, thickened at apex, 35-80 x 8-14 µm.

The ascospores are hyaline, unicellular, narrowly oval to cylindrical to fusiform.

Acervuli are produced on lesions, and usually setose.

Conidiophores are cylindrical phialidic.

The conidia are cylindrical with obtuse ends, 9-24 x 3-6 µm, unicellular, hyaline or faintly brown. Appressoria are 6-20 x 4-12 µm, ovate to obovate, sometimes lobed

Mode of spread and survival:

- The pathogen survives in infected plants parts.
- Spread through conidia by wind and rain splash.

Management:

- Field sanitation
- Removal and destruction infected leaves.
- Spraying carbendazim 0.1% or Daconil 0.2% or Chlorothalonil 0.2% at 15 day intervals.

4. **Phytophthora Blight/ Fruit rot/ Stem rot: *Phytophthora nicotianae* var. *parasitica***

Symptoms:

- Small, water soaked discolured spots appear on the stem around fruit and leaf scar, and on the fruits.
- The spots enlarge and girdle the stem causing wilting of the plants.
- The fruits are attacked at any stage primarily the matured fruits.
- The infected fruits shrivel, turn dark brown become mummified and finally fall down.
- A whitish mycelial mass develop on the rotting fruits and stem.

Mode of spread and survival:

- The pathogen survives in infected plants parts and soil as oospores.
- The pathogen spreads through wind or rain splash.

Epidemiology

- The optimum temp for disease development is 27.8^0C

Management:

- Removal and destruction of infected plants and fruits,
- Good drainage.
- Spraying COC 0.25% or Mancozeb 0.2%
- Ridomil MZ 0.2% Aliette 0.2% highly effective.

5. ***Asperisporium*** **Black spot -** ***Asperisporium caricae***

Symptoms:

- The first symptoms are scattered small spots, visible on both leaf surfaces.
- On the upper surface, the lesions are round or angular, 1-4 mm in diameter, pale yellow, with dark margins.
- Later the lesions become necrotic and whitish.
- Black pustules are produced on the abaxial surface of the leaf.

- ❖ The lesions are covered with masses of fungal spores which appear as dark dots.
- ❖ The disease is more intense on the lower leaves.
- ❖ Severe infection causes yellowing and premature shedding of leaves.
- ❖ The pustules also occur on fruits.
- ❖ The lesions are superficial and do not enter the flesh of the fruit.
- ❖ The damage to fruit is entirely cosmetic.

Causal Organism: *Asperisporium caricae*

Sporodochia hypophyllous, dark blackish brown to black. Stroma well-developed, erumpent.

Conidiophores closely packed together and covering the surface of the stroma, usually unbranched, hyaline to olivaceous brown, with several prominent conidial scars at the apex, up to 45 x 6–9 µm.

Conidia solitary, ellipsoidal, pyriform or clavate, 1-septate, hyaline to mid pale brown, verrucose,

14–26 x 7–10 µm

Mode of spread and survival:

- ❖ The pathogen survives in infected plants parts.
- ❖ Spread through conidia by wind and rain splash.

Management:

- ❖ Field sanitation
- ❖ Removal and destruction infected leaves.
- ❖ Spraying carbendazim 0.1% or Daconil 0.2% or Chlorothalonil 0.2% at 15 day intervals.

6. Corynespora* Brown spot - *Corynespora cassiicola

- ❖ The symptoms occur on the stem, fruits, petioles and leaves.
- ❖ On the upper surface, the lesions are greyish to whitish, sunken, with dark margin and yellow halo.
- ❖ Sometimes the centre of the lesion shows crack.
- ❖ On the lower surface, the lesions are brown, necrotic, and sunken, with brown-reddish margins.
- ❖ On petioles the lesions are similar to those of the leaves.
- ❖ On fruits, black, rounded, sunken, dried dark velvet lesions occur.
- ❖ Lesions coalesce and cover extensive areas of the fruit surface.
- ❖ Causes post-harvest rot of fruits, entering the fruits through wounds.

Causal Organism: *Corynespora cassiicola*

Conidiophores hypophyllous, erect, simple, sparingly septate, dark brown, up to 600 x 4–11 µm, with successive cylindrical proliferations variable in length, 10–100 µm, lighter in colour towards the apex.

Conidia solitary, obclavate to cylindrical, straight or often slightly curved, tapering towards the apex, pale olivaceous brown, smooth, 4–20 pseudoseptate, 32–220 x 8–22 µm, up to 520 µm long in culture, hilum conspicuous

Mode of spread and survival:

- The pathogen survives in infected plants parts.
- Spread through conidia by wind and rain splash.

Management:

- Field sanitation
- Removal and destruction infected leaves.
- Spraying Dithane M-45 (0.2%)

Papaya Ring spot: Papaya Ring spot Virus (PRSV)

Serious disease in India

One of the limiting factors in papaya cultivation

Incidence varies from 20 – 100%

Loss 5-20%

Symptoms:

- Vein clearing, severe mosaic mottling, puckering and blistering
- Deformation of leaves into thread like structure called **shoe – string** symptoms.
- The margins and distal parts of young leaves roll downwards and inwards.
- The diseased plants are stunted
- The diseased plants are denuded with tuft of small deformed leaves at the apex.
- The petiole length shortened.
- The number of lobes in the leaves increased which become thin and distorted.
- On the fruits, numerous, circular, coalescent concentric ring spots are seen.
- Various degrees of apocarps are also common.
- If affected early no fruit formation
- If affected late a few fruits are produced.
- Fruit size and weight also reduced

Casual Organism:

Belongs to genus *Potyvirus* of the family *Potyviridae*. Filamentous flexuous rod measuring roughly 760 – 800 nm long 12nm wide ss RNA. The virus particle or virion consists of a nucleocapsid, Virus particles typically contain 94.5% protein and 5.5% nucleic acid by weight, and have no outer membrane (non-enveloped).

Transmission:

Mechanical inoculation, grafting and by aphids - *Aphis gossypii* and *Myzus persicae* – must efficient vector.

Neither soil nor seed transmitted.

Alternate hosts – *Cucurbita maxima C. moschata*, *C.pepo* var *pattypan*, *Luffa aegyptiaca* etc.

Papaya leaf curl – Tobacco Leaf Curl Virus (TLCV) and Nicotiana Virus 10

- The disease is characterized by severe curling,
- Crinkling, vein clearing and distortion of leaves.
- The leaf lamina is very much reduced.
- The margins are rolled downwards and inwards in the form of inverted cups.
- The veins get thickened and turn dark green.
- The leaves become leathery and brittle.
- Petioles get twisted into Zig – Zag manner.
- The affected trees fail to flower or bear fruits.

Casual Organism: Tobacco Leaf Curl Virus (TLCV) and Nicotiana Virus 10

Belongs to Gemini – virus group

Transmitted by only grafting and white fly *Bemisia tabaci*.

Alternate hosts: Tobacco, tomato, sunnhemp, chilli, Petunia, Zinnia hollyhock, *Datura stramonuim*.

Papaya Mosaic: Papaya Mosaic Virus, Carica virus1

- The disease produces typical mosaic symptoms showing chlorosis with dark green blisters on the leaves.
- Vector (Aphids): *Myzus persicae, Aphis malvae, A. medicaginis, A. gossypii* and *Macrosiphum sonchi.*

Management of virus diseases:

- Raise papaya seedlings under insect-proof conditions.
- Use disease free seedlings.
- Raise sorghum / maize as barrier crop before planting papaya.
- Removal and destruction infected plants.
- Field sanitation i.e. removal of weeds which act as reservoir hosts or alternate hosts.
- Avoiding intercropping with alternate hosts
- Control insect vectors – Monocrotophos 0.05% or Dimethoate 0.03%
- Transplanting in September and November supplemented with 50g of Neemcake; 100g, of N; 100g of P_20_5 and 200 g of K_2O is recommended for the control of PRSV.
- Use of cross protection with mild strain of PRSV also found to be helpful.

Post harvest diseases

1. Anthracnose – *Colletotrichum gloeosporioides*

2. Phytophthora blight – *Phytophthora nicotianae* var. *parastica*
3. Watery fruit rot – *Rhizopus stolonifer*
4. *Macrophomina* fruit rot – *Macrophomina phaseolina*
5. Stem end rot – *Botryodiplodia theobromae*
6. *Ascochyta* fruit rot – *Ascochyta caracae*
7. *Phomopsis* fruit rot – *Phomopsis caracae papayae*

Watery fruit rot – *Rhizopus stolonifer*

- Irregular water soaked lesions are produced.
- They enlarge and covered with white and dark brown fungal growth.
- The fruits collapsed and become watery and emit a foul odour.

Macrophomina* Fruit rot – *Macrophomina phaseolina

- Causes 5-20% loss during summer
- Small water soaked circular specks on the fruits.
- They rapidly enlarge and become sunken.
- The pathogen advances deep into the fruit and causes rotting and disintegration of fruits.
- The fruit pulp turns brown to black.
- The tissues harden and dotted with sclerotic.

Stem end rot – Botryodiplodia thobromae

- The disease mostly attacks ripe and half ripe fruits.
- The rot begins as dark green, water soaked spots.
- The affected portion becomes shriveled and turns dark brown and roughened due to erumptent pycnidia confluent.
- The spots are surrounded by a dark green water soaked area.
- Infection of fruit stalk results in fruit drop.

***Ascochyta* fruit rot – Ascochyta caricae**

- Attacks half grown or mature fruits.
- Small circular water – soaked spots are produced.
- They become sunken and brownish black.
- The rot spreads outwards and irregularly.

Phomopsis* fruit rot – *Phomopsis caracae papayae

- The affected area becomes soft and pulpy.
- The rotten area turns brown to black,
- Later they get depressed and cracks.

Minor Diseases

Phyllosticta* leaf spot – *Phyllosticta sulata

- They spots are small and round or irregular (or) oval or elongated.
- The spots are white at centre and bounded by yellowish or brownish margin which gradually merges into the green colour.
- Shot holes are seen.
- Spreads though wind.
- Spraying Bordeaux mixture.

Cercospora* leaf spot – *Cercospora papayae

- The spots are sub circular to irregular.
- Ash coloured on the upper surface.
- Immarginate and in district in lower surface.

Chocolate spot – Strain of *Colletotrichum* glolosprioides

Chocolate spot: Post-harvest, superficial lesions, seldom slightly sunken, irregular to rounded, up to 1 cm in diameter, well defined, with characteristic reddish-brown colour. As the fruit ripens the lesions can either remain superficial or grow and become sunken, resembling anthracnose. Sometimes these symptoms together with latex exudation on the centre of the lesion can be observed several days before harvest.

DISEASES OF TOMATO

Tomato has become one of the common vegetables all over the country and is grown extensively in many parts almost the year and tomato plants are severely affected by various diseases of microbial origin. There are > 20 diseases of tomato reported from different parts of the country.

1. **Damping off: *Pythium aphanidermatum*; *P. indicum*; *P. debaryanum*; *P.ultimum***

The disease is most common in many parts of India. Tomato seedlings are highly susceptible to Damping off. The disease is responsible for poor germination and stand of seedlings in the nursery. In nursery beds the disease may start in patches and in the course of 2-4 days the entire lot of seedlings may be destroyed.

Symptoms:

Damping off of tomato and other vegetables occurs in two phases (i) Pre emergence damping off (ii) Post emergence damping off.

Pre emergence Damping off

- ❖ It is a decay of germinating seeds
- ❖ The radical and plumule when they come out of the seed 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
- The affected seedlings become chlorotic and pale green and suddenly collapse
- Dark-brown water-soaked lesions develop at the collar region that rapidly spread over the entire seedling.
- Affected portion shows shrinking and brown discolouration due to rotting
- Eventually, the lesions girdle the hypocotyl, causing seedlings to wilt and die.
- Brown, water-soaked lesions that start on the roots and later extend up the hypocotyl characterize post-emergence Damping-Off.
- The root cortex becomes macerated and easily sloughs off.
- The affected seedling become collapsed and toppled down
- Post emergence mortality of seedlings very conspicuous

Causal organism: *Pythurin aphanidermatum*; *P. indicum*; *P. debaryanum*; *P.ultimum*

Mycelium - hyaline, coenocytic freely branched – reproduction asexually and sexually. Sporangia - irregular, lobed – terminal or intercallery – germinate by producing tubes with vesicle

Zoospores - biflagellate – encysted in ½ hr - germinate to produce germ tube – mycelium

Sexual – oogonuim and anthridium – oospore – zoosporangium – zoospores – mycelium.

Mode of survival and spread

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

Epidemiology

- Damping-off is generally most severe under conditions of high soil moisture and/or compaction, overcrowding, poor ventilation and cool, damp, cloudy weather.
- Water-splashing moves infested soil from diseased to healthy plants.

Management

- Partial sterilization of the soil by burning trash or by steam sterilization done
- Provision of better drainage
- Avoidance of same bed for repeated sowing
- Improving the soil texture by adding soil organic amendments
- **Preparation of** raised **nursery** beds.
- Thin sowing using optimum seed rate

- **Optimum irrigation**
- Application of more well decomposed FYM
- Seed treatment with captan or thiram @ 3g or *Trichoderma viride* @ 4g per kg of seed
- Soil drenching with Bordeaux mixture 1% or copper oxychloride 0.2% @ 4l / sq. metre.
- Spray 0.2% Metalaxyl when there is cloudy weather

2. **Early Blight: *Alternaria solani***

Early blight is also called as *Alternaria* leaf blight or target spot. Early blight is a common leaf-spotting fungal disease of tomato. This is a common disease of tomato occurring all over world. In severe attacks the loss may be upto 80%. In India more severe in June – July crop compared to winter crop.

Symptoms

- The fungus causes leaf blight and may also cause seedling disease, collar rot, stem canker or fruit rot.
- When seedlings are affected dark brown cankers may develop and girdle stems of seedlings at the soil line resulting in seedling disease and collar rot.
- The older leaves are first attacked and the disease progresses upwards.
- The first symptom to appear is small, brown to black, round to angular spots on the leaves.
- As these spots enlarge become irregular, dark brown to black lesions with a series of dark concentric raised rings of necrotic tissues.
- It gives the characteristic symptom of target effect or target appearance.
- The spots are surrounded by yellow halo due to the production of a toxin called **alternaric acid**.
- When several lesions coalesce together, larger area affected giving blight appearance.
- Severely affected leaves eventually turn brown, shrivel and fall down prematurely.
- Severe infestations of this disease can cause 100% defoliation of the plant.
- Extensive defoliation exposes fruits to sunscald and increases fruit rot.
- Stem lesions on older plants usually remain confined to one side of the stem.
- Stem and petiole show sunken and dark necrotic lesions.
- Fruits are also infected.
- Dark brown – black, sunken, dry, leathery lesions occur most frequently on the stem end of the fruit and also have a zonate or “target-like” appearance.
- Spots are velvety appearance and may be covered by a blackish “dust,” consisting of numerous microscopic spores of the fungus.

- Infected fruit often drops off soon after infection.

Causal organism: *Alternaria solani*

Mycelium: septate, branched, light brown which become dark at age, intercellular and later intracellular

Conidiophores - dark coloured emerge through stomata.

Conidia - born in chain, club – shaped, muriform with 5-10 cross septa and 1-5 longitudinal septa with long beak.

Mode of survival and spread

- The fungus survives on infected debris in the soil, on seed, on volunteer tomato plants and other solanaceous hosts, such as Irish potato, eggplant, and black nightshade.
- Survives in infected plant debris in soil upto 3 years
- The early blight fungus is spread by wind and splashing rain,
- Seed borne also

Epidemiology

- Infection occurs rapidly under warm, humid conditions.
- Thousands of spores are produced in spots of infected leaves that are capable of causing more infections.
- Plants under stress from nitrogen deficiency, heavy fruit load, or other factors are most susceptible to the disease.
- Relatively warm temperatures, abundant rainfall, and high relative humidity favor disease development.
- Water stress also predisposes infection
- Opt. temp 28 – 30^0C
- The disease becomes serious in frequent rains followed by warm and dry weather.

Management

- Use of disease free seeds
- Field sanitation
- Crop rotation
- Avoid diseased transplants
- Regular irrigation to avoid water stress
- Clipping of lower leaves upto 20 cm ht
- Avoid overhead irrigation
- To prevent rain splash, use mulch.
- Avoid composting diseased plant material.
- Seed treatment with Captan / Thiram (4g / kg).
- Spraying Mancozeb 0.2% or Ziram 0.2% Captan 0.3% Chlorothalonil 0.2%

- ❖ Maintain adequate but not excessive soil fertility.
- ❖ Proper irrigation and fertilizer management to maintain plant vigour

3. **Late blight:** *Phytophthora infestans*

It is prevalent in hilly regions of TN and Karnataka Where rainfall is abundant. It is very destructive and fast-spreading.

Symptoms

- ❖ Affects leaves, stem and fruits
- ❖ Indefinite, pale green to brownish, irregular water soaked greasy lesions appear at the tip and margin of the leaves.
- ❖ In moist weather enlarge rapidly with central necrotic tissue changing to dark brown or black.
- ❖ A pale green halo is often observed around the leaf spots as they enlarge.
- ❖ The lesions cover whole leaf and spread to petioles.
- ❖ During wet weather, lesions on the abaxial surface of the leaf may be covered with a gray to white moldy growth.
- ❖ As the disease progresses, the foliage turn yellow then brown, curls, shrivels, and dies.
- ❖ When several lesions coalesce together, larger area affected giving blight appearance.
- ❖ On petioles and stems, lesions begin as indefinite, water-soaked spots that enlarge rapidly into brown to black linear elongated lesions that cover large areas of petioles and stems.
- ❖ Affected stems and petioles may eventually collapse at the point of infection, leading to death of all distal parts of the plant.
- ❖ Green fruits are sensitive to this disease
- ❖ Brown to green olivaceous greasy spots develop at stem end of the fruits.
- ❖ When the infection spreads the fruits rot and become covered with whitish mycelium.
- ❖ Infected fruits rapidly deteriorate into foul-smelling masses.

Causal organism: *Phytophthora infestans*

Mycelium: Hyaline endophytic coenocytic highly branched inter- and intracellular Sporangiophores: hyaline, branched arise in cluster through stomata.

Sporangia pear- shaped hyaline papillate, multinucleate, zoospores biflagellate (low temperature) germ tube (high temperature).

Amphigynous antheridium, oogonium spherical.

There are several races of the pathogen found. In India 77 races carrying 8 gene complexes have been identified.

Mode of survival and Spread

P. infestans survives in plant debris or on volunteer tomato plants and on perennial weeds such as nightshade; where both mating types are present (A1 and A2),

P. infestans generates the thick-walled oospores that are long term survival propagules.

Dispersal of the pathogen is by wind, rain, irrigation water or human assisted via movement of infested or infected materials such as seed or tools, contacts among leaves and leaf eating insects etc.

Epidemiology

- Excessive RH (>90%) with suitable temperature are important.
- Opt temp for germination of sporangia by zoospores (direct germination) 12 – 13^0 C and by germ tubers (indirect germination) 21 – 24^0 C.
- Opt temp for growth of the mycelium 16 - 18^0 C.
- Daytime temperatures between 60 and 70°F, night temperatures between 50 and 60°F, and relative humidity near 100% are the ideal conditions for infection and spread of late blight disease.
- Predisposing factors include cool, wet weather and high relative humidity, and large, densely planted crops of tomato.

Late blight disease forecasting systems

1) BLITECAST system

Based on temperature, relative humidity, and rainfall, this is a regional disease forecasting system used by potato and tomato farmers in the North-eastern United States.

2) NoBLIGHT system (Maine)

This is similar to BLITECAST, but differs in the calculation of severity values for relative humidity; see Johnson 2005.

3) HYRE system

This is based on temperature and rainfall.

4) WALLIN system

This is based on temperature and relative humidity.

Management

- Seed material should be selected from disease free crop in disease free area.
- Use of disease free transplants
- Field sanitation
- Wider spacing
- Reduced nitrogenous fertilizers
- Avoid use of sprinkler irrigation
- Avoid working with plants when foliage is wet to avoid spreading of disease

- ❖ Spraying 1% Bordeaux Mixture in early stages at fortnightly interval when the crop is at 15-20 cm ht. If weather is more favourable once in 7 days. Later mancozeb 0.25% is effective.
- ❖ Other fungicides recommended are defoliation, zineb 0.2% and Duter 0.1 – 0.2% Daconil 0.1%, COC (0.25) @ 1.5 – 2.5 kg / ha

4. ***Septoria* leaf spot / Defoliation disease**: *Septoria lycopersici*

It occurs in TN, HP, JK and Karnataka. The disease is as important as the early blight for causing defoliation.

Symptoms

- ❖ Plants are attacked in all stages of growth.
- ❖ The disease first appears on the upper surface of the lower leaves after the fruit set.
- ❖ Several small round water soaked spots appear on the lower leaves.
- ❖ As the spots enlarge they develop dark brown margin and sunken grayish white centres
- ❖ The light-coloured centre of the spots is the most distinctive symptom.
- ❖ Minute, black dot like fruiting bodies (pycnidia) of the fungus develop at the centre of the spots under favourable conditions.
- ❖ Heavily infected leaves turn yellow, wither, and eventually fall off.
- ❖ Complete defoliation of the affected plants is noticed during rainy weather.
- ❖ Stem and flowers are also affected.
- ❖ If infected in early growing season, plants can become 100% defoliated before fruit set.
- ❖ During favourable conditions, the disease can cause extensive defoliation, resulting in sun scalding of fruit and reduction in yield.

Causal organism: *Septoria lycopersici*

Pycnidia are immersed amphigenous globose honey yellow to brown

Conidia hyaline filiform 2-6 septa.

Mode of survival and Spread

- ❖ The fungus survives in infected tomato debris remaining in the soil from a previous tomato crop or weed hosts like horsenettle.
- ❖ It may also be introduced on seed. Both externally and internally seed borne
- ❖ Spread by rain splash and windblown water hands and clothing of tomato pickers.

Epidemiology

- ❖ Persistent dew or long periods of high humidity at 25^0C,
- ❖ muggy weather or drizzling for 2-3 days
- ❖ The disease is favored by moderate temperatures and extended periods of high relative humidity.

Management

- Use of disease free seeds
- Field sanitation
- Crop rotation
- Avoid diseased transplants
- Regular irrigation to avoid water stress
- Clipping of lower leaves upto 20 cm ht
- Avoid overhead irrigation
- To prevent rain splash, use mulch.
- Avoid composting diseased plant material.
- Seed treatment with Captan / Thiram (4g / kg).
- Spraying Mancozeb 0.2% or Ziram 0.2% Captan 0.3% Chlorothalonil 0.2%
- Maintain adequate but not excessive soil fertility.
- Proper irrigation and fertilizer management to maintain plant vigour
- Avoid composting diseased plant material.

5. Leaf Mould: *Passalora fulva* (*Fulvia fulva*)

It is the most severe foliar pathogen of glass house crop and also causes major losses in field crop in tropical and subtropical areas during cooler periods when RH is high.

Symptoms

- The first visible symptoms are pale green or yellowish or chlorotic spots on the upper surface of older leaves.
- Spots are not distinct as they lack a clearly defined border, in contrast with most other foliar diseases.
- Thus affected leaves may look like they have a general yellowing due to a nutrient deficiency.
- They spread quickly under favourable conditions of high humidity.
- The pale green or chlorotic spots turn to distinctive yellow spots.
- An olive-green to tan, velvety fungal growth appears on the lower surface of the spots
- Spores are produced on the lower surface beneath the chlorotic areas producing a characteristic velvety, olive green mould consisting of light grey fructification becoming brown or olive green.
- As the disease progresses the leaves wilt and die.
- Under favorable conditions, complete defoliation can occur.
- Occasionally, the fungus attacks stems, blossoms and fruits.
- Green and mature fruit can have a black, leathery rot on the stem end.

Causal organism: *Passalora fulva* (*Fulvia fulva*)

Mode of survival and spread

- Air – borne and sometimes seed borne also.
- The pathogen can survive in soil at least one year as spores or in infested plant tissue from the previous planting.
- This pathogen can be seed-borne.
- The spores are easily moved by wind and splashing water or rain and tools.

Epidemiology

Opt. temp 22 – 24^{0}C and high humidity

The fungus is dependent on high relative humidity and high temperature for disease development.

Management

- Use of disease free seeds
- Field sanitation
- Crop rotation
- Avoid diseased transplants
- Avoid overcrowding of plants
- Clipping of lower leaves upto 20 cm ht
- Avoid overhead irrigation
- To prevent rain splash, use mulch.
- Seed treatment with Captan / Thiram (4g / kg).
- Spraying Mancozeb 0.2% or Captafol 0.2% Chlorothalonil 0.2% or Carbendazim or thiophanate- methyl 0.1%

6. Botrytis* blight or Gray mould: *Botrytis cineria

Symptoms

- Occurs both in the greenhouse and in the field.
- Affected leaves show light tan or gray spots
- The infected areas become covered by a brown to gray fuzzy fungal growth.
- Infected leaves and blossoms wither and die.
- The fungus proceeds into the stem producing tan, elliptical cankers with concentric rings.
- Stem cankers cause wilting of vines
- *Botrytis* may develop on dying flowers and subsequently infect the fruit calyx.
- White circular (halo) spots appear on the green fruit and are termed "ghost spots."
- The lesions on ripe fruits appear as irregular light brown to gray spots.
- Later, a dark gray, velvety growth develops on the fruit surface, followed by a watery, soft rot.
- Infected fruit become soft and watery, and the flesh turns light gray.

Causal organism: *Botrytis cineria*

Mycelium is septate and branched, hyaline but become dark in color upon age.

Conidiophores are branched and bear conidia at the apex.

Conidia are continuous or one septate, oblong and dark.

Mode of survival and spread

- The fungus overwinters as sclerotia or as mycelium in plant debris and may be seed borne.
- The fungus is easily dispersed long distances by wind.
- Also disseminated shorter distances by splashing and windblown rain

Epidemiology

- High relative humidity is necessary for prolific spore production.
- The fungus has a very wide host range that includes many vegetable crops.
- Fungus sporulation and infection, is favored by cool, wet and humid weather.
- The fungus requires a water film of several hours from rain, dew, fog, or irrigation for spore germination, and a longer period of surface wetness for symptom development.
- Optimum relative humidity for spore production is about 90%, and most spores are produced during the night when the temperature is lower and the relative humidity is higher than during the day.
- Temperatures of 17-23℃ are ideal for disease development.
- Plants approaching maturity are more susceptible. The fungus can also penetrate dead flower tissue or dying foliage.
- Excessive application of nitrogen makes plants such as young transplants more susceptible to gray mold.
- Dense plant canopies will limit air movement within the crop, thus creating conditions for extended surface wetness at night and subsequent increased gray mold severity.

Management

- Use of disease free seeds
- Field sanitation
- Crop rotation
- Avoid diseased transplants
- Avoid overcrowding of plants
- Avoid overhead irrigation
- Avoid excessive application of nitrogen
- To prevent rain splash, use mulch.
- In the greenhouse, maintain a relative humidity of less than 80%, especially during night.

- ❖ Good drainage
- ❖ Seed treatment with captan / thiram (4g / kg).
- ❖ Spraying Mancozeb 0.2% or Captafol 0.2% Chlorothalonil 0.2% or Bordeaux Mixture1%

7. **Powdery Mildew: *Leveillula taurica***

More serious in seedlings in Bangalore region of Karnataka. Occurs during July – October

Symptoms

- ❖ Whitish floury patches appear on the lower surface of the leaves stems
- ❖ Corresponding upper surface shows bright yellow spots
- ❖ Infected leaves may be dwarfed, stiff, and narrow followed by drying of leaves.
- ❖ The fungus progressively attacks new leaves, spreading over leaves, stems, twigs, and even the fruit.
- ❖ Terminal growth of the affected shoot is stunted or killed.
- ❖ The fruit yield is reduced and the affected fruits are smaller in size.

Causal organism: *Leveillula taurica*

Oidiopsis type, endophytic,

Mycelium: hyaline, septate, inter and intracellular,

Conidiophores, hyaline septate, arise through stomata

Conidia cleavate hyaline

Mode of survival and spread:

- ❖ Survives as ascospores in infected plant debris which serve as primary inoculum.
- ❖ The secondary spread is through air borne conidia.

Epidemiology

- ❖ Dry weather coupled with low humidity is favourable for initiation and spread.
- ❖ Infection in favoured by temperature range of 18 – 24^0 C and RH 70 – 100%.

Management

- ❖ Field sanitation
- ❖ Destruction of collateral weed hosts.
- ❖ Spraying wettable Sulphur 0.2% or karathane or Carbendazim or Benomyl or thiophanate- methyl 0.1%

8. **Buck Eye Rot: *Phytophthora parasitica***

Fruit rot or buckeye rot is a serious disease in all the tomato growing areas. The disease is serious is hilly tract of TN, UP & KK. Spoils 30 – 40% fruits before harvest. All the commercial varieties are susceptible. Loss is 35 – 40 %. Fruit rot upto 90%

Symptoms

- The pathogen does not affect the foliage and thus the disease is distinct from late blight.
- Buckeye rot is a destructive fruit rot of green or ripe fruit.
- The fruits which contact the soil or where soil frequently splashes onto fruits are frequently affected
- Small grayish-green or pale brown circular water-soaked spots are produced on immature and mature green fruit at blossom end.
- The spot rapidly enlarges to cover up to half of the fruit diameter.
- The rot then appears brown with definite concentric or target-like appearance that resembles **buckeye chestnut**.
- The affected fruits shrink and mummify
- White flocculent superficial mycelial growth develops profusely in warm and humid conditions.
- The fruits remain firm and retain the shape till they are spoiled by other saprophytes.
- Over time, infected fruit will become soft and mushy.

Causal organism: *Phytophthora parasitica*

Similar to late blight pathogen *Phytophthora infestans*

Mode of survival and spread:

- The fungus survives as chlamydospores in the soil and infected plant debris.
- The secondary spread by surface water and rain splash

Epidemiology

- The fungi are common in many soils.
- The disease is most severe in poorly drained areas.
- The disease is most common during periods of prolonged warm, wet weather
- Peppers are also susceptible to this disease.

Management

- Use of disease free seeds
- Use deep tillage to reduce soil compaction and improve internal soil drainage.
- Field sanitation
- Rotate with corn or other grass crops
- Avoid diseased transplants
- Avoid overcrowding of plants
- Avoid overhead irrigation
- Mulch beneath plants to reduce soil contact with fruit.
- Staking plants and removing foliage and fruits upto a height 15-30 cm from ground level

- Good drainage
- Seed treatment with captan / thiram (4g / kg).
- Spraying Mancozeb 0.2% or Captafol 0.3% or Chlorothalonil 0.2% or Bordeaux Mixture1% or copper oxychloride 0.25% or Ridomil M2 (0.25%) will give fairly good control of buckeye rot.

9. **Anthracnose: *Colletotrichum phomoides, C. coccodes, C. dematium*, and *C. gloeosporioides***

Symptoms

- Anthracnose is a common and widespread rot of ripe or overripe tomato fruit.
- Green fruits are infected but do not show symptoms until ripening.
- Fruit becomes more susceptible as they approach maturity.
- At first, infected fruit show small, circular, slightly sunken, water soaked spots.
- These spots enlarge, become darken, depressed and have concentric rings.
- Masses of the pink fruiting bodies of the fungus can be seen on the surface of the lesions in moist weather.
- During humid weather, masses of buff-colored conidia are extruded from the fruiting structure giving the lesion creamy-pink coloration.
- Under warm and humid conditions, the fungus penetrates the fruit, completely destroying it. Secondary rotting organisms may invade anthracnose lesions to completely rot.

Causal organism: *Colletotrichum phomoides, C. coccodes, C. dematium*, and *C. gloeosporioides*

Mode of survival and spread

- The fungus survives the winter on diseased tomato vines, in the soil, and in seeds.
- These acervuli survive in soil for three years and cause infections either directly or by producing secondary spores.
- The fungus then spreads from infected to healthy fruit as spores are splashed by rain or overhead irrigation, or by pickers working wet plants.

Epidemiology

- Anthracnose is favored by warm rainy weather, overhead irrigation, and heavy defoliation from foliar disease.

Management

- Use of disease free seeds
- Field sanitation
- Avoid overcrowding of plants
- Avoid overhead irrigation
- Use drip irrigation to reduce moisture levels on fruit and humidity in the plant canopy.

- Mulching prevents splashing of spores from the soil onto the fruits.
- Keep the garden weed free
- Staking plants
- Harvest fruit as soon as possible after ripening.
- Seed treatment with captan / thiram (4g / kg).
- Seed treatment by soaking in hot water (122 ºF) for 25 minutes to destroy the fungus.
- Spraying Mancozeb 0.2% or Captafol 0.3% or Chlorothalonil 0.2% will give fairly good control of anthracnose.

10. Fusarium* wilt: *Fusarium oxysporum* f.sp. *lycopersici

This is one of the most common and important wilt diseases of tomatoes around the world

Symptom

- *Fusarium* usually enters its host through feeder roots and subsequently multiplies and colonizes the food and water conducting vessels of the plant.
- Infection may occur at any time during the life of the plant.
- The affected plants show clearing of veinlets and chlorosis followed by yellowing of lower leaves
- Symptoms can be seen on one side of a leaf midrib, or on a single branch, or on several branches on one side of the plant, or on all the lower branches.
- Drooping of the petioles downwards called epinasty symptom.
- The drooping starts from bottom and proceeds to top until all the foliage is killed and the plant dies.
- The leaves gradually wilt and die.
- In due course entire plant wilts.
- The affected plants are stunted in growth.
- Affected plants die early and produce few, if any, fruit.
- Fruits ripe prematurely
- Dark brown or reddish brown or black discolouration of vascular tissues in the form of streaks can be seen from the base of the stem upto the root tip.
- Microscopic examination of sections of the infected tissues would reveal the fungal hyphae both as inter and intracellular.
- In the final stage rotting of roots may be observed but no bark shredding.
- The discoloration can extend from the roots upto the stem through the branches and into the petioles of the plant.

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

Mycelium: Hyaline at first creamy at later, septate, inter and intracellular

Microconidia produced in chain, hyaline spherical

Macroconidia: sickle shaped, 3-5 celled, hyaline

Chlamydospores terminal or intercalary, spherical

It attacks only *Lycopersicon* spp.

Mode of survival and spread

- The fungus survives as chlamydospores and persists indefinitely in infested field soil.
- The fungus is also seed borne and is thought to spread long distances in this manner
- Persists on plant debris and weed hosts
- The fungus spread by anything that moves soil about, including irrigation.

Epidemiology

- The disease is most serious in sandy soils and at temperatures between 80-90^{o} F.
- Soils become infested by planting infected transplants and from movement of infested soil by wind and water erosion or on farm implements.
- It is favoured by sandy, acid soils, low soil moisture, hot weather, low RH, and short day length
- The disease is most severe when air and soil temperatures are between 78^{0} and 90^{o} F
- More likely to occur in poorly drained soil.
- Soil temperature of 20-30º C, hot and dry periods followed by rains,
- Alternating high and low soil temperature and humidity levels
- Presence of root knot nematodes increase the disease development

Management

- Use of disease-free seeds, transplants and soil.
- Field sanitation
- Application of Lime in acid soils to raise the pH to 6.5-7
- Use nitrate forms of nitrogen fertilizer and avoid ammonium forms.
- Avoid flood irrigation because these water supplies can be contaminated with the fungus and spread the disease.
- Long-term rotations with cereals (five to seven years out of tomato) may help.
- Crop rotation should be used in conjunction with varietal resistance to maintain sustainable control and limit the development of new races.
- Control of nematodes with carbofuran or Nemagon
- Seed treatment with Thiram or Carbendazim or *Trichoderma viride*
- Soil mulch with polyethylene sheets in hottest months
- Apply FYM or organic amendments
- Apply 2.5 kg/ha of *Trichoderma viride* commercial formulation with 50kg farmyard manure
- Soil fumigation with methyl bromide in controlled condition

- ❖ Spot drenching with Carbendazim 0.1%

11. Verticillium Wilt: *Verticillium albo-atrum*, *V. dahliae*

These pathogens cause economically important disease of tomato.

Symptoms

- ❖ The affected plants are stunted in growth.
- ❖ Yellow blotches develop on lower leaves;
- ❖ Yellowing of lower leaves progress slowly upward
- ❖ There is necrosis of veins surrounded with chlorotic or tan-colored areas at the margins of leaf tips.
- ❖ This discoloration may become characteristic V-shaped or fan-shaped and more yellow over time.
- ❖ The leaves rapidly turn completely yellow, wither, and drop off
- ❖ Unlike *Fusarium* wilt, symptoms of *Verticillium* wilt do not progress along one side of a leaflet, branch, or plant.
- ❖ Vascular brown discolouration can be seen on cutting through stem or roots.
- ❖ Many adventitious roots develop at the base of the stem.

***Fusarium* wilt**	***Verticillium* wilt**
No interveinal chlorosis. Develops more intense yellowing of leaves	Develops interveinal chlorosis with less intense yellowing of leaves
Slow development	Rapid development
Dark brown or black vascular tissue	Light brown vascular tissue
Prefers light sandy soil	Prefers heavy soil
More localized distribution within an area	Wide distribution
Requires higher optimum temperature	Requires low optimum temperature

Causal organism: *Verticillium albo-atrum*, *V. dahliae*

Mycelium first prostrate and hyaline become floccose and white to grey.

Microsclerotia –dark resting spores.

Mode of survival and spread

- ❖ The fungus is soil-borne and can persist for many years in soil and plant debris.
- ❖ The fungus survives as microsclerotia in infected crop debris and can attack many other plants.

Epidemiology

- ❖ This disease is favoured by long-term tomato cultivation, cool weather and high soil pH. Verticillium wilt is increased by root wounding from cultivators or root-knot nematode feeding.

Management

- ❖ Field sanitation
- ❖ Long-term rotations with cereals and pulses
- ❖ Removal of weed host plants
- ❖ Seed treatment with Thiram or Carbendazim or *Trichoderma viride*
- ❖ Soil mulch with polyethylene sheets in hottest months
- ❖ Apply FYM or organic amendments
- ❖ Apply 2.5 kg of *Trichoderma viride*/ha commercial formulation with 50kg farmyard manure
- ❖ Soil fumigation with methyl bromide in controlled condition
- ❖ Spot drenching with Carbendazim 0.1%

12. **Bacterial Wilt: *Ralstonia solanacearum***

Bacterial wilt is a devastating disease of tomato, tobacco, and potato. It is also called as **southern bacterial wilt**. Wide spread worldwide. Most severe in tropical and subtropical climates with high rainfall. In India total loss has seen reported. In Philippines 15% loss has been reported in India it is a threat to tomato in KK, MP Maharashtra and WB.

Symptoms

- ❖ Sudden drooping of leaves with rapid wilting and death of plants without yellowing or spotting of the foliage
- ❖ The lower leaves may droop first before wilting occurs.
- ❖ Downward curling of leaves occurs.
- ❖ There may be excessive production of adventitious roots along the stem above the soil.
- ❖ The pith appears as water soaked in early stages; later, the pith will turn brown and sometimes become hollow.
- ❖ The discoloration of pith distinguishes this disease from *Fusarium* and *Verticillium* wilt.
- ❖ The vascular system become yellowish brown
- ❖ Stems cut cross-wise may ooze a thick, off-white to yellowish liquid.
- ❖ Sticking the cut end of an infected stem partially into a glass of water reveals a milky-white stream of bacteria in the water within three to five minutes.
- ❖ The root system may also develop a brownish rot as the plant dies.

Causal organism: *Ralstonia solanacearum*

Rod shaped Gram negative 1-4 flagellate

Three major races

Race I infects tomato, potato, chilli, egg plant etc.

Race 2 Infects bananas and *Heliconia* sp.

Race 3 infects potato alone.

Mode of survival and spread

- The bacterium can survive in the soil for months, especially in well-drained soils.
- It is spread by soil and water movement or by diseased plants or infected debris or equipment.
- Bacteria infect plants through the roots or stem, most often where tissue has been injured by cultivating, or by some other physical means such as nematodes.

Management

- Use of disease-free seeds, transplants and soil.
- Field sanitation
- Avoid flood irrigation because these water supplies can spread the disease.
- Long-term rotations with cereals.
- Grow on raised beds to promote drainage
- Application of Bleaching powder @ 15 kg / ha before transplanting.
- Soil fumigation with methyl bromide in controlled condition

13. Bacterial spot: *Xanthomonas euvesicatoria* (*Xanthomonas axonopodis* pv. *vesicatoria*

Bacterial spot reduces tomato yield and quality by defoliation and spotting of fruit.

Symptoms

- Leaves, stems, and fruit may be infected at any growth stage
- Small, brown, water soaked, circular to irregular spots appear on stem and leaves.
- As lesions enlarge, they often become surrounded by a yellow halo.
- They become dried looking sunken and grayish brown in colour.
- Leaf spots appear greasy when leaves are wet
- If spots are numerous, they coalesce, and leaves wither and turn brown
- A general yellowing of heavily spotted areas on leaves occurs, followed by leaf scorch.
- Bacterial ooze may form and dry to form a creamy film
- Spots on green fruit first appear as black, raised, pimple-like dots surrounded by water-soaked areas.
- As the spots enlarge they become gray-brown and corky scabby with sunken, pitted centres irregular margins and raised haloes

Causal organism: *Xanthomonas euvesicatoria* (*Xanthomonas axonopodis* pv. *vesicatoria*

Rod shaped, Gram positive

Mode of survival and spread

- The bacterium overwinters on the surface of seeds (seed borne), in infected debris, and in soil.
- The organism survives in alternate hosts, on volunteer tomato plants and on infected plant debris.

Epidemiology

- Rainfall and humidity levels promote disease development.
- Warm, rainy weather favors rapid spread of bacterial spot.
- Moist weather and splattering rains are conducive to disease development.
- Prolonged heavy rainfall, high RH and temp around 24^0C

Management

- Use of disease-free seeds and transplants
- Field sanitation
- Crop rotation
- Avoid working in plantings when foliage is wet.
- Soaking seed in a solution of one part household bleach (5.25% sodium hypochlorite) and two parts water for one minute followed by rinsing and drying. (or) Disinfect suspect seed with 1% bleach for 10 minutes.
- Hot-water treatment involves soaking seed at exactly 122°F for 25-30 minutes followed by cooling and drying.
- Using drip irrigation reduces bacterial spread and the leaf wetness periods that favor infection compared to sprinkler irrigation
- Spraying with Agrimycin-100 (100 ppm) thrice at 10 days intervals effectively controls the disease.

14. Bacterial canker: *Clavibacter michiganensis* sub sp*. michiganensis*

One of the major diseases of tomato in the world. Yield reduction up to 70% in USA.

Symptoms

- Bacterial canker can be difficult to diagnose because a variety of symptoms may occur and the canker symptom (stem lesion) is not always produced.
- Plants affected in early stage wilt and die and the disease may be confused with bacterial wilt.
- Plants that are infected early are killed while those that become infected later develop leaf scorch and fruit symptoms.
- Infected older plants develop a leaf scorch (browning) at the margin and begin die back.
- The petioles droop down.
- The leaves eventually become brittle and dry, and drop from the plant.
- Long brownish stripes appear on the stem, shoots and petioles

- They may split open to show creamy white or yellow or reddish brown discoloured cavities in the pith and bacterial exudation.
- Eventually the pith becomes reddish brown in color and mealy in texture.
- Whitish cankers develop on stems and petioles under certain conditions.
- Infected fruits show small round raised white spots which later develop light brown roughened centres surrounded by a white halo resembling the characteristic **bird's eye**.

Causal organism: *Clavibacter michiganensis* sub sp*. michiganensis*

Coccoid and curved or straight rods, Non motile, Gram positive

Mode of survival and spread

- Survive for short periods in soil, contaminated greenhouse structures, and wooden tomato stakes
- The bacterium survives for longer periods in plant debris, Solanaceous weeds and volunteer tomato plants.
- The bacterium can survive in soil on infested plant material for at least 1 year.
- The bacterium is carried on seed (**Internal and external seed borne**) and on transplants.
- In the field, the bacterium is spread by splashing water and wind-driven rain; contaminated equipment; and by pruning, staking, and harvesting activities.

Epidemiology

- Disease development is favored by moderately high temperatures (75 to 85^0 F) and wet, humid conditions.
- Presence of root knot nematode increases the severity of the disease.

Management

- Use of disease-free seeds and transplants
- Field sanitation
- Crop rotation with non-host crop helps in reducing the disease incidence.
- Avoid working in plantings when foliage is wet.
- Using drip irrigation reduces bacterial spread and the leaf wetness periods that favor infection compared to sprinkler irrigation
- Avoid overhead irrigation
- Sanitize tools such as pruning shears and stakes.
- Use mulch to prevent rain splash.
- Hot water treatment of seeds at 50°C for 25 minutes is effective.
- Seed treatment with 5% HCL for 5 hr (or) hot water at 560C for 30 min (or) 1.05% Na hypchlorate for 40 min
- Spraying streptomycin followed by CuSO4

Virus diseases

15. Mosaic: TMV (ToMV), CMV, PVX, PVY

Symptoms

- Tobacco mosaic virus (TMV), also known as tomato mosaic virus (ToMV)
- Symptoms of the disease are variable and depend on the virus strain present, the tomato variety grown, and temperature.
- Generally the virus causes mottled areas of light and dark green color called **mosaic mottling**
- Infected leaves may also be small, curled, and malformed.
- Sometimes younger leaves become distorted crinkled.
- In severe cases entire leaf area become pale yellow to white with scattered small dark green islands.
- Sometimes the leaflets become indented resulting in "**fern leaf**" symptoms.
- Affected plants are stunted.
- Affected stems are brittle, easily broken, and contain brown areas in the pith and cortex.
- Ordinarily the fruit does not show any marked disfiguration.
- Some strains (referred to as the acuba strains) may cause a striking yellow mosaic, whereas other strains may cause leaf malformation and "fern leafing," suggestive of cucumber mosaic virus infection.
- The most pronounced symptom produced by CMV is the extremely distorted, **extreme filiformity** or **shoe string-like** leaves
- Both the number and size of the fruit are reduced.
- Occasionally fruit mottling and bronzing, develop.
- At times, fruit may develop a "brown wall" or "internal browning" defects.
- Crop losses upto 20% of the world production
- Many host range *N. glutinosa, N. tabacum, D.stramonum, G. Globosa*, etc.,
- TMV is spread primarily by humans handling infected plants and mechanically transmitting the virus to healthy plants via the sap.
- TMV can be seed borne in tomato upto 94%
- CMY is transmitted by aphids in a non persistent manner.
- Management: Use of disease free seeds
- Seed treatment with 10% Trisodium phosphate Na_3PO_4 for 20 min plus hot water treatment at 70 ^{0} C for 2 – 4 days.

16. Tomato spotted wilt: Tomato Spotted Wilt Virus (TSWV)

Tomato spotted wilt, caused by the virus *Tomato spotted wilt virus* (TSWV), is one of the most economically devastating diseases of tomato around the world. TSWV was first discovered in Australia in 1919. Tomato production losses of 75–100% from tomato spotted wilt have been reported in Hawaii. This is the most serious virus disease of tomatoes in recent years, causing widespread damage to both commercial and home garden plantings. Infected fruit cannot be sold.

Symptoms

- The tomato plants are affected at all stages of growth but seedlings are more vulnerable.
- Produce symptoms on leaves stem and fruits.
- The first observable symptoms are small, chlorotic lesions on the leaflets that often have a darker green "halo."
- These chlorotic lesions may coalesce and become necrotic, giving the foliage a "bronzed" appearance.
- These necrotic regions spread to terminal shoots, causing them to "wilt."
- Tops of plants may suddenly die back (snuff-top) and some plants may wilt on one side, suggesting a wilt disease.
- Leaves curl inwards and downwards
- Stunting of plants
- Cessation of growth
- Under favourable condition bronzing leads to necrosis which continues and leads to death of the plant.
- Early-infected plants produce few or no fruit while later-infected plants produce discoloured fruit.
- Green fruit may have slightly raised bumps with faint, light green to whitish circles.
- The circles and rings are more obvious as the fruit ripens, staying yellowish while the fruit turns red
- The most conspicuous symptoms are discolored blotches or concentric rings on the fruit
- Disease infection leads to reduction in size of the fruits.
- The virus infection causes asymmetrical growth of plant. i.e. one side dry and other side will not dry.

Early blight or *Alternaria* leaf spot	**TSWV**
Necrotic lesions with concentric rings	Necrotic lesions without concentric rings
Yellow halo present surrounding the lesions	No Yellow halo

No bronzing	Bronzing present
No death of plant or asymmetrical growth	Death of plant or asymmetrical growth is present
Brown necrotic lesion in the form of concentric rings are present on fruits	Brown necrotic lesion in the form of concentric rings are absent on fruits
Raised bumps with faint, light green to whitish circles are absent on Green fruit	Green fruit may have slightly raised bumps with faint, light green to whitish circles.
Yellowish circles and rings are absent on the red fruits	Yellowish circles and rings are more obvious on the red fruits
Discolored blotches or rings are absent on the fruits	The most conspicuous symptoms are discolored blotches or rings on the fruit

Causal organism: Tomato Spotted Wilt Virus *(*TSWV*)*

Isometric particles 70 – 90nm in dia

Transmission

TSWV is transmitted by several species of thrips, including common blossom thrips (*Frankliniella schultzei*), western flower thrips (*F. occidentalis*), chilli thrips (*Scirtothrips dorsalis*), and onion thrips (*T. tabaci*).

Nympus acqure the virus from inf. palnt but adults transmit the virus. Long latent period in vector. 4-18 days. Min Ac. feeding period 15 min % of transmission increased by length of feeding period. Seed transn. also recorded but only 1% also sap transmissible.

Management

- New crop should not be planted near older crops or other susceptible crop (egg plant, chilli potato)
- Field sanitation – removal of infected plants, alternate hosts etc.
- Raising barriers crop with *Crotalaria junacel*
- Using antiviral principles.
- Leaf extracts of sorghum, prosopis, *Mirabilis jalapa* contain AVP which effectively control the disease.
- Leaf extracts of 10% is prepared.
- 1 Kg of dried leaf powder – 5 l water – 60^0C – 30 min - filter – add 5 lit of water.
- Sprayed 15, 25, 35 and 45 DAP.
- Checking the population of thrips can reduce the disease incidence. 2-3 foliar sprays with Dimethoate (0.05%) or Monocrotophos (0.05%) at 10 days intervals controls the thrips population.

17. Tomato yellow leaf curl: Tomato yellow leaf curl virus (TYLCV)

Tomato yellow leaf curl is a most destructive viral disease of tomato caused by Tomato yellow leaf curl virus (TYLCV). In tropical and subtropical regions, total losses of tomato crops have been reported. TYLCV is widespread and can be found in most places where tomato is grown. Incidence is more in summer months since the vector population will be more. Losses are high when crops are affected during the half of their growing period. (20 DAP – 92% yield less; 35 DAP – 74% yield less and 50 DAP – 29% yield less.

Symptoms

- Characteristic symptoms are dwarfing of plants.
- Interveinall chlorosis, mosaic mottling, vein clearing and crinkling of leaves
- Smaller leaves than normal, upward rolling giving them a cup-like appearance, puckering of the leaves.
- On the under surface of the leaves there will be small leaf like out growth called enations
- The veins become thickened.
- The leaves become leathery and brittle.
- Reduction in of nodes and internodes length
- The infected plants remain stunted
- The infected plants look pale and produce more lateral branches giving a bushy appearance.
- Abscission of flowers and fruit is common.
- If tomato plants are infected early in their growth, there may be no fruit formed.

Causal organism: Tomato yellow leaf curl virus (TYLCV)

Gemini virus group

Transmission

No sap transmission Transmission by white flies *Bemissia tabaci*. A single viruliferous insect is enough for successful transmission. Females are more efficient than males because females are more voracious feeder. Minimum acquisition feeding period is 30 min. Incubation periods in the body of the vector are 6 hr. Inoculation feeding period is 30 min.

It has wide host range including tobacco, chilli papaya *Acanthospermum hispidum*. *Physalis Crotalaria* etc.

Management

- The affected plants should be removed and destroyed.
- Alternate or collateral hosts harbouring the virus should be removed
- Checking the white fly population can reduce the disease incidence.

- Soil application of granular insecticide like Furadan (1 kg a.i./ha) at the time of sowing seeds in the nursery bed checks whitefly population. Another dose of Furadan (1.5 kg a.i./ha) is given one week after transplanting.
- 2-3 foliar sprays with Dimethoate (0.05%) or Monocrotophos (0.05%) at 10 days intervals controls the white fly population.
- Barrier crops like maize, jowar, bajra are good to protect the crop from TLCV infection. Five or six rows of these crops all around the main tomato plot should be sown at least 50-60 days before transplanting of tomato. These crops check incoming viruliferous whiteflies from entering into tomato crop.
- Use of polythene mulching or reflective mulches (aluminum or silver-colored) in the soil just before transplanting of tomato. Polythene sheets of white, blue, grey and black colours are effective.
- Combined application of polythene mulching and Furadan application in the soil is recommended.

18. Tomato Big bud: *Candidatus* Phytoplasma

- Infected plants are scattered in field
- The first indication of infection appears at the tips of the actively growing shoots.
- The youngest fruit truss, assumes an upright position.
- The buds on the truss also point in a vertical direction,
- Vein clearing and chlorosis of leaves
- Stunted growth with shortened internodes and thickened stem.
- Proliferation of auxiliary shoots with numerous small more rounded, thin leaves giving rosette appearance.
- The calyx segments remain united almost to the tips, and the whole calyx enlarges to form like a bladder with a toothed opening at the top.
- The affected leaves become yellow-green and roll along their margins.
- The size of the leaves reduces as the disease advances.
- Fruit that is well developed but still green at the time of infection becomes hard and tough and colours extremely slowly or not at all.
- Occasionally leaves and auxillary shoots show purple pigmentation.
- Transmitted by leafhopper *Orosius argenatatus,* grafting, dodder etc
- Controlled by rouging and insecticide spraying.

DISEASES OF BRINJAL

Damping off: *Pythurin aphanidermatum*; *P. indicum*; *P. debaryanum*; *P.ultimum*

The disease is most common in many parts of India.

Brinjal seedlings are also susceptible to Damping off.

The disease is responsible for poor germination and stand of seedlings in the nursery.

In nursery beds the disease may start in patches and in the course of 2-4 days the entire lot of seedlings may be destroyed.

Symptoms:

Damping off of tomato and other vegetables occurs in two phases (i) Pre emergence damping off (ii) Post emergence damping off.

Pre emergence Damping off

- It is a decay of germinating seeds
- The radical and plumule when they come out of the seed 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
- The affected seedlings become chlorotic and pale green and suddenly collapse
- Dark-brown water-soaked lesions develop at the collar region that rapidly spread over the entire seedling.
- Affected portion shows shrinking and brown discolouration due to rotting
- Eventually, the lesions girdle the hypocotyl, causing seedlings to wilt and die.
- Brown, water-soaked lesions that start on the roots and later extend up the hypocotyl characterize post-emergence Damping-Off.
- The root cortex becomes macerated and easily sloughs off.
- The affected seedling become collapsed and toppled down
- Post emergence mortality of seedlings very conspicuous

Causal organism: *Pythurin aphanidermatum*; *P. indicum*; *P. debaryanum*; *P.ultimum*

Mycelium - hyaline, coenocytic freely branched – reproduction asexually and sexually. Sporangia - irregular, lobed – terminal or intercalary – germinate by producing tubes with vesicle

Zoospores - biflagellate – encysted in ½ hr - germinate to produce germ tube – mycelium

Sexual – oogonuim and anthridium – oospore – zoosporangium – zoospores – mycelium.

Mode of survival and spread

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

Epidemiology

- Damping-off is generally most severe under conditions of high soil moisture and/or compaction, overcrowding, poor ventilation and cool, damp, cloudy weather.
- Water-splashing moves infested soil from diseased to healthy plants.

Management

- Partial sterilization of the soil by burning trash or by steam sterilization done
- Provision of better drainage
- Avoidance of same bed for repeated sowing
- Improving the soil texture by adding soil organic amendments
- **Preparation of** raised **nursery** beds.
- Thin sowing using optimum seed rate
- **Optimum irrigation**
- Application of more well decomposed FYM
- Seed treatment with captan or thiram @ 3g or *Trichoderma viride* @ 4g per kg
- Soil drenching with Bordeaux mixture 1% or copper oxychloride 0.2% @ 4l / sq. m
- Spray 0.2% Metalaxyl when there is cloudy weather

Fusarium Wilt: *Fusarium solani*

The common disease is common in Tamil Nadu. The disease affects brinjal in all stages of its growth.

Symptoms

- It attacks seedlings as well as matured plants.
- Leaves turn chlorotic.
- The leaves become flaccid and hang down.
- In the course of a week time, the plant wilts and dries up.
- The roots exhibit varying degrees of rotting.
- If the bark of the stem is peeled, Vascular browning is seen as brown streaks at the base of the stem and roots.
- Whitish fungal growth is seen on base of stem and roots.

Causal organism: *Fusarium solani*

Mycelium is septate and hyaline.

Microconidia are hyaline, wedge shaped, 2-celled and are formed in chain.

Macroconidia are falcate and septate.

Chlamydospores are globose, intercalary or terminal.

Mode of survival and spread

- The fungus survives as chlamydospores and persists indefinitely in infested field soil.
- Persists on plant debris and weed hosts
- The fungus spread by anything that moves soil about, including irrigation.

Epidemiology

- The disease is most serious in sandy soils and at temperatures between 80-90^{o} F.
- Soils become infested by planting infected transplants and from movement of infested soil by wind and water erosion or on farm implements.
- It is favoured by sandy, acid soils, low soil moisture, hot weather, low RH, and short day length
- More likely to occur in poorly drained soil.
- Alternating high and low soil temperature and humidity levels
- Presence of root knot nematodes increase the disease development

Management

- Use of disease-free seeds, transplants and soil.
- Field sanitation
- Avoid flood irrigation because these water supplies can be contaminated with the fungus and spread the disease.
- Long-term rotations with cereals (five to seven years out of tomato) may help.
- Control of nematodes with carbofuran or Nemagon
- Seed treatment with Thiram or Carbendazim or *Trichoderma viride*
- Apply FYM or organic amendments
- Apply 2.5 kg of *Trichoderma viride*/ha commercial formulation with 50kg farmyard manure
- Spot drenching with Carbendazim 0.1%

***Verticillium* Wilt (*Verticillium albo-atrum*, *V. dahliae*)**

It is now recognized as one of the most destructive disease in TN. Losses caused by the disease extend from about 5-10% in midly affected crops to 90% in severely affected crops.

Symptoms

- Although seedlings can be infected, symptoms usually are not observed until plants are older.
- A yellowing of lower leaves followed by wilting is the first sign of disease.
- A characteristic symptom of infection is a V-shaped lesion that develops on older leaf tips that later expands to cover the leaf.

- Brown, necrotic tissue within lesions is surrounded by a large, irregular area of yellowing due to a systemic leaf toxin produced by the fungi.
- As the disease progresses, stunting and chlorosis become severe with diurnal wilting.
- Wilting can be asymmetric, with sections of the plant remaining turgid.
- Permanent wilt and plant death follow.
- Dissecting through the crown of affected plants reveals dark-brown vascular discoloration, which can extend into the pith and up into the stem and branches.
- Fruit that form are small and deformed with internal discoloration.

Causal organism: *Verticillium albo-atrum*, *V. dahliae*

Mycelium first prostrate and hyaline become floccose and white to grey.

Microsclerotia –dark resting spores.

Mode of survival and spread

- The fungus is soil-borne and can persist for many years in soil and plant debris.
- The fungus survives as microsclerotia in infected crop debris and can attack many other plants.

Epidemiology

- This disease is favoured by long-term tomato cultivation, cool weather and high soil pH.
- Verticillium is favored in its development if the soil is alkaline.
- *Verticillium* wilt is increased by root wounding from cultivators or root-knot nematode feeding.

Management

- Field sanitation
- Long-term rotations with cereals and pulses
- Removal of weed host plants
- Seed treatment with Thiram or Carbendazim or *Trichoderma viride*
- Soil mulch with polyethylene sheets in hottest months
- Apply FYM or organic amendments
- Apply 2.5 kg of *Trichoderma viride*/ha commercial formulation with 50kg farmyard manure
- Soil fumigation with methyl bromide in controlled condition
- Spot drenching with Carbendazim 0.1%

Leaf spot: *Alternaria melongenae* and *Alternaria solani*

Symptoms

- Irregular, brown and necrotic spots with concentric rings are seen on leaves.
- Spots coalesce to cover large necrotic areas.

- Leaf dries and then fall down.
- Fruit infection shows dark brown and sunken spots.
- Fruit turn yellow and drop.

Causal organism: *Alternaria melongenae* and *Alternaria solani*

Mycelium: septate, branched, light brown which become dark at age, intercellular and later intracellular

Conidiophores - dark coloured emerge through stomata.

Conidia - born in chain, club – shaped, muriform with 5-10 cross septa and 1-5 longitudinal septa with long beak.

Mode of survival and spread

- The fungus survives on infected debris in the soil, on seed, on volunteer tomato plants and other solanaceous hosts, such as Irish potato, tomato, and black nightshade.
- Survives in infected plant debris in soil upto 3 years
- The fungus is spread by wind and splashing rain,
- Seed borne also

Management

- Field sanitation
- Regular irrigation to avoid water stress
- Avoid overhead irrigation
- To prevent rain splash, use mulch.
- Avoid composting diseased plant material.
- Seed treatment with Captan / Thiram (4g / kg).
- Spraying Mancozeb 0.2% or Ziram 0.2% Captan 0.3% Chlorothalonil 0.2%
- Proper irrigation and fertilizer management to maintain plant vigour

Cercospora* Leaf Spot: *Cercospora melongenae

This is common but minor disease in tropics.

Symptoms

- Symptoms appear on the leaves, petioles, and stems of eggplant.
- Initially, small, circular to oval chlorotic spots with light to dark tan centres appear
- They may develop angular or irregular shapes.
- Elliptical to oval lesions may occur on the leaf blades, veins, and petioles.
- Appear more abundantly on the lower surface.
- They are brown to steel-gray on the upper surface and light brown on the lower surface (Anonymous 2011).
- Conidiophores appear in fascicles (clusters) of 3 to 12. pale to medium brown, paler towards the apex, occasionally septate, and unbranched,

- Conidia, hyaline mildly curved (Mycobank Database 2012).
- Soil as well as seed borne
- Survives in plant debris and affected fruits.
- Spread through air, water and insects etc.

Management

- Maintain proper field sanitation
- Use disease-free transplants
- Irrigate in the morning to reduce humid and damp conditions overnight.
- Avoid over-irrigation to reduce relative humidity.
- Avoid overhead sprinkler irrigation in order to minimize leaf wetness and spread of the pathogen in splashing water droplets.
- Increase the spacing between plants to improve aeration and drying of wet foliage.
- Keep plants adequately fertilized.
- Spraying Mancozeb 0.2% or Ziram 0.2% Captan 0.3% Chlorothalonil 0.2%

Phompsis* blight and fruit rot: *Phomopsis vexans

Common throughout the world. Although it appears as damping off, Leaf blight, the most destructive phase of the disease is the fruit rot. The pathogen attacks only brinjal. In India fruit rot phase causes heavy damage in the field and also during transit.

Symptoms

- The disease affects brinjal from seedling to maturity in one or other form of the disease.
- In nursery it causes **damping off of seedlings** also.
- After transplanting the leaves which come into contact with soil first affected.
- Defined, circular or irregular, grey to brown spots with light coloured centre develop on leaves.
- At later stage numerous fruiting bodies, called pycnidia, can be observed as small, black pimples, embedded in the host tissue.
- The affected leaves turn yellow and die.
- The petiole and stems also affected.
- Stem symptoms include brown or dark sunken lesions slightly above the soil surface, and can result in cankers.
- Stem lesions are dark brown becoming grey at the centre as black pycnidia develop.
- Mostly the stem base is attacked and is characterized by constriction of the base or a grey dry rot.
- The stem is girdled

- The skin peels off and the inner tissues are exposed in strong wind.
- The infected plants toppled down due to breaking of the main stem.
- The fruits are attacked while on the plant.
- Pale sunken oval spots develop on the surface of the fruits.
- These subsequently enlarge and become depressed
- With one lesion or several spots coalescing, large portions of the fruit are affected
- The spots contain many black pycnidia arranged in concentric rings.
- The internal portion of the fruits rots.
- The whole fruits will be mummified due to dry rot.

Causal organism: *Phomopsis vexans*

Mycelium is septate

Pycnidia with or without beak erumpent, brown to black globose or irregular with ostiole,

Pycnidia produce two types of conidia. *viz*. Alfa conidia and beta conidia.

Alfa conidia are hyaline, sub cylindrical, single celled, fusoid, curved aseplate.

Beta conidia (stylospores) are filiform, hyaline and one-celled.

Perithecia are in clusters containing clavate, sessile, 8-spored asci.

Ascospores are bluntly fusoid and 2-celled.

Mode of survival and spread

- This fungus can survive in plant debris or in mummified fruit in the soil.
- Seed produced on plants grown in affected fields can be infested with fungal spores and may initiate disease on seedlings.
- The major means of spread is by rain splashing or overhead irrigation splash tools and insects.

Epidemiology

- Disease develops fast in wet weather with temperature around 26^0 C storage rot is maximum at 25^0 C.
- Disease is favored by hot and wet weather.
- The optimum temperature for fungal growth is 29°C and it grows well up to 32°C.

Management

- Remove and destroy all infected plant material to reduce initial inoculum.
- Use disease-free seed and transplants
- A 3-4 year crop rotation is beneficial, since the fungus does not infect other crops.
- Weed control is advisable since pathogen can survive on solanaceous weeds such as nightshades.
- Deep summer ploughing

- Mulch and furrow irrigate to help reduce splashing of water and soil.
- Hot water seed treatment at 50^0 C for 30 min
- Spraying difolaton 0.2% or Captan 7-10 days interval – five sprays.

Sclerotinia blight: *Sclerotinia sclerotiorum*

In Tarai region of N. India, it is a more serious disease.

Symptoms

- Dark -green, water-soaked lesions develop on foliage, stems and fruit.
- Stem infections girdle the stem at the soil line, causing plants to wilt and die.
- Fruits are infected directly from the soil surface or through the peduncle rot quickly
- Affected fruits rot and turn into a watery mass..
- In advanced stages, white, cottony mycelium blankets affected tissue, and sclerotia form on the surface.

Causal organism: *Sclerotinia sclerotiorum*

Mycelium hyaline much branched septate, inter and intracellular,

apothecia – funnel shaped cup

produces asci – ascospores.

Mode of survival and spread

- Soil borne. Survives in soil & plant debris as sclerotia.
- Secondary spread through air borne ascospores

Epidemiology

- Dec – Jan more prevalent.
- Dew, fog and frequent rain generally favor disease development.
- Optimum temperature 15.5 to 21^0 C

Management

- Field sanitation
- Deep summer ploughing
- Use wide row spacing
- Good drainage
- Flooding & Drain if possible
- Crop rotation with rice or beat onion spinach or maize, small grains and grasses
- Spraying ziram or carbendazim have been reported to give some relief.

Bacterial Wilt: *Ralstonia solanacearum*

In many areas of India considered as one of the very serious disease.

Symptoms

- Symptoms are sudden drooping of leaves with rapid wilting and death of plants without yellowing or spotting of the foliage
- The lower leaves may droop first before wilting occurs.

- Downward curling of leaves occurs.
- Sometimes there is an excessive production of adventitious roots along the stem above the soil line.
- The pith appears as water soaked in early stages; later, the pith will turn brown and sometimes become hollow.
- The vascular system become yellowish brown
- Stems cut cross-wise may ooze a thick, off-white to yellowish liquid.
- Sticking the cut end of an infected stem partially into a glass of water reveals a milky-white stream of bacteria in the water within three to five minutes.
- The root system may also develop a brownish rot as the plant dies.

Causal organism: *Ralstonia solanacearum*

Rod shaped Gram negative 1-4 flagellate

Three major races

Race I infects tomato, potato, chilli, egg plant etc.

Race 2 Infects bananas and *Heliconia* sp.

Race 3 infects potato alone.

Mode of survival and spread

- The bacterium can survive in the soil for months, especially in well-drained soils.
- It is spread by soil and water movement or by diseased plants or infected debris or equipment.
- Bacteria infect plants through the roots or stem, most often where tissue has been injured by cultivating, or by some other physical means such as nematodes.

Management

- Use of disease-free seeds, transplants and soil.
- Field sanitation
- Avoid flood irrigation because these water supplies can spread the disease.
- Long-term rotations with cereals.
- Grow on raised beds to promote drainage
- Application of Bleaching powder @ 15 kg / ha before transplanting.
- Soil fumigation with methyl bromide in controlled condition

Little leaf – *Candidatus* Phytoplasma

First reported from India in 1938. It occurs only in India & Sri Lanka. In almost all the states of the country it has become a serious problem facing brinjal cultivation. Loss upto 100% in diseased plants.

Symptoms

- The most characteristic symptom is the reduction in size of leaves and petiole.
- The petioles are so short that the leaves appear to be sticking to the stem.

- The leaves become thin, narrow, soft glabrous and pale green in colour.
- The internodes also shortened.
- The auxiliary buds stimulated to produce >100 branches.
- The number of leaves produced will be more giving a bushy appearance.
- In early infection no flowers are formed
- Even if formed the floral parts are modified into green leaf like structures.
- After infection no fruits are formed.
- In late infection the fruit size is very much reduced.

Causal organism: Candidatus Phytoplasma

The phytoplasma is transmitted by the jassid *Hishimonas phycitis*

Acquisition feeding period 1 hr

Latent period in the vector 16 days

Inoculation feeding period <1 hr

No transsovarious transmission.

Alternate hosts – Potato, Tomato (Big bud) chillies, *Datura, Cathranthus roseus, Argemon mexicana*

Management

- Roughing of infected plants
- Eradication of weed hosts.
- Spraying systemic insectides like malathion, Metasystex to control vectors,
- Pusa purple cluster, Arka sheel Aushy, Manjari Gota and Banarus Giant are moderately resistant to resistant in field.

Mosaic: Potato virus Y (PVY) Tobacco mosaic virus (TMV)

Potato Virus Y

Mosaic mottling of leaves and stunting of plants are the characteristic symptoms

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.

Tobacco Mosaic Virus

Conspicuous mottling of leaves

Leaves also develop blisters in advanced cases.

Severely infected leaves become small and misshapen.

Plants infected early remain stunted.

PVY is easily sap transmitted. It is transmitted in the field through aphids, *Aphis gossypii* and *Myzus persicae* and perpetuates on weed hosts like *Solanum nigrum* and *S. xanthocarpum.*

TMV is transmitted by sap, contaminated implements and clothes, soil debris and hands of labour. It can perpetuate on many cultivated plants like cucurbits, legumes, pepper, tobacco, tomato and weed hosts. The virus survives in plant debris

---

DISEASES OF BHENDI

1. **Powdery mildew:** *Erysiphe cichoracearum*

It is very severe in this crop. Normally occurs during September – December.

Symptoms

- The infection is first seen on older leaves.
- The disease symptoms appear as subtle, small, round, whitish spots on leaves and sometimes stems.
- The spots enlarge and coalesce rapidly
- White or grayish powdery growth or patches resembling talcum powder on the upper surface of older leaves or other plant parts.
- The fungal growth is diffused without any marked boundary
- The talc-like powder is composed of fungal mycelium, conidiophores and conidia.
- Heavily infected leaves become yellow, and then become dry up and brown.
- The affected leaves may roll upward,appear scorched and fall off prematurely
- Severe infection causes very much reduction in fruit yield.

Causal organism: *Erysiphe cichoracearum*

Mycelium: ectophytic external, hyaline, septate haustoria are button-shaped

Conidiophores short hyaline

Conidia are single celled, hyaline, barrel shaped and in long chains basipetally.

Cleistothecia are globose and dark brown myceloid appendages.

The asci are pedicellate, ovate or ellipsoid.

The number of ascospore is usually 2 rarely 3 per ascus.

The ascospores are single celled, hyaline and oval to sub cylindrical

Mode of spread and survival

- Fungus overwinters on plant debris or alternate host
- Spread through airborne conidia

Epidemiology

- Disease is favoured by warm, dry weather with cool nights that result in dew formation

Management

- Removal of weed host plants
- Field sanitation

- ❖ Plants should be well manured and applied fertilizers to maintain the vigour of plants
- ❖ Spraying Sulphur dust 8-10kg / ac or Wettable Sulphur 0.3% (or) Benlate 0.1% or tridemorph 0.1% or dinocap 0.1% or propiconazole 0.05 % or Rubigon 0.025% or Bavistin (0.1%)

2. ***Cercospora* leaf spot:** *Cercospora abelmoschi* , *Cercospora malayensis*

Symptoms

- ❖ *Cercospora abelmoschi* causes no definite spots but grows as a sooty black to dark olivaceous mould on the lower surface of the leaves.
- ❖ Severely affected leaves dry up.
- ❖ *Cercospora malayensis* produces dark brown circular or irregular spots with grey centre.
- ❖ Both the leaf spots cause severe defoliation

Causal organism: *Cercospora abelmoschi* , *Cercospora malayensis*

Conidiophores are pale to medium olivaceous brown, multiseptate, some times branched, geniculate and irregular.

Conidia are obclavate to cylindric, olivaceous brown and straight to curved.

Mode of spread and survival

- ❖ The fungus survives in the diseased crop material.
- ❖ Spread thro air borne conidia.

Epidemiology

- ❖ Both the leaf spots are common during humid seasons

Management

- ❖ Removal of weed host plants
- ❖ Field sanitation
- ❖ Plants should be well manured and applied fertilizers to maintain the vigour of plants
- ❖ The leaf spot disease can be controlled by spraying with any one of the fungicides viz, carbendazim 0.1% or Mancozeb 0.2% or Difolatan 0.2%.

3. **Wilt: *Fusarium oxysporum* f.sp*. vasinfectum***

Symptoms

- ❖ The infected plants appear as stunted
- ❖ Leaves become yellow before the appearance of typical wilt symptoms
- ❖ The leaves show epinasty and vein clearing.
- ❖ They lose turgor.
- ❖ Finally the plant dies.
- ❖ If a diseased stem or root is cut longitudinally the vascular tissues appear as dark streaks.

4. Vein Clearing or Yellow vein mosaic:

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 at all the stages of crop growth and severely reduces growth and yield. If the plants are affected early there will be a total loss. The extent of damage declines with delay in infection of the plants in a field. Plants affected 50 and 60 DAS suffer a loss of 84 and 49% respectively.

Symptoms

- The disease is initially characterized by yellowing of the entire network of the veins in the leaf lamina.
- Then the leaves turn completely yellow
- In severe infections the younger leaves also turn completely yellow
- The leaf size becomes reduced.
- In extreme cases, the infected leaf becomes totally light yellow or cream coloured and there is no trace of green colour.
- The veins and veinlets are thickened.
- The affected plants become stunted in growth.
- Due to infection flowering may be restricted.
- The fruits if formed they are small in size, malformed, yellowish green in colour, fibrous and harder than the healthy fruits.

Causal organism: Yellow vein mosaic virus.

The causal agent is the single-stranded DNA *Bhendi yellow vein mosaic virus* (BYVMV), which is associated with a beta satellite, both of which are required for infection. BYVMV belongs to the genus *Begomovirus*, family *Geminiviridae*.

Transmitted by *Bemisia tabaci*

Weed Hosts: *Croton sparsiflora, Malvastum tricupsidatun, Ageratum* sp, *Hibisus tetraphyllus*

Commelina nudiflora

Management

- Rouging and destruction of infected plants
- Raising of new crops near older crops should be avoided.
- Removal of weed hosts.
- Growing resistant varieties like Pusa Savani, Arka Anamika, Parbhani Kranti, Arka Abhay and Varsha Uphar.
- Use of yellow sticky traps
- Spraying systemic insecticides starting soon after emergence of the plants
- Raising border crops – maize, jowar or Bajra with spraying in insecticides

DISEASES OF CRUCIFERS

Diseases of Cabbage and Cauliflower

Damping off: *Pythium aphanidermatum*; *P. debaryanum*

Symptoms

Damping off of tomato and other vegetables occurs in two phases (i) Pre emergence damping off (ii) Post emergence damping off.

Pre emergence Damping off

- It is a decay of germinating seeds
- The radical and plumule when they come out of the seed 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
- The affected seedlings become chlorotic and pale green and suddenly collapse
- Dark-brown water-soaked lesions develop at the collar region that rapidly spread over the entire seedling.
- Affected portion shows shrinking and brown discolouration due to rotting
- Eventually, the lesions girdle the hypocotyl, causing seedlings to wilt and die.
- Brown, water-soaked lesions that start on the roots and later extend up the hypocotyl characterize post-emergence Damping-Off.
- The root cortex becomes macerated and easily sloughs off.
- The affected seedling become collapsed and toppled down
- Post emergence mortality of seedlings very conspicuous

Causal organism: *Pythurin aphanidermatum*; *P. debaryanum*

Mycelium - hyaline, coenocytic freely branched – reproduction asexually and sexually. Sporangia - irregular, lobed – terminal or intercallery – germinate by producing tubes with vesicle

Zoospores - biflagellate – encysted in ½ hr - germinate to produce germ tube – mycelium

Sexual – oogonuim and anthridium – oospore – zoosporangium – zoospores – mycelium.

Mode of survival and spread

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

Epidemiology

- Damping-off is generally most severe under conditions of high soil moisture and/or compaction, overcrowding, poor ventilation and cool, damp, cloudy weather.
- Water-splashing moves infested soil from diseased to healthy plants.

Management

- Partial sterilization of the soil by burning trash or by steam sterilization done
- Provision of better drainage
- Avoidance of same bed for repeated sowing
- Improving the soil texture by adding soil organic amendments
- **Preparation of** raised **nursery** beds.
- Thin sowing using optimum seed rate
- **Optimum irrigation**
- Application of more well decomposed FYM
- Seed treatment with captan or thiram @ 3g or *Trichoderma viride* @ 4g per kg of seed
- Soil drenching with Bordeaux mixture 1% or copper oxychloride 0.2% @ 4l / sq. metre.
- Spray 0.2% Metalaxyl when there is cloudy weather

Club root: *Plasmodiophora brassicae*

Club root, caused by the slime mold *Plasmodiophora brassicae*, was first recognized in Europe during the 13th century. It has since become the most serious disease of crucifer crops worldwide. Club root is a most persistent and devastating disease of cruciferous crops which affects nearly all cultivated, as well as many wild and weed members of the cabbage family. Potentially most dangerous disease of Cruciferaceae. Very common in temperate region. Once the disease has appeared in the area if becomes difficult to raise a profitable crop of crucifers in that area. Affected plants are usually a total loss. Most cruciferous weeds are susceptible and can serve as reservoirs of the pathogen from one year to another.

Symptoms

- Symptoms become apparent only after the pathogen has advanced considerably in the root system.
- Clubroot may develop extensively on plant roots before the first sign (an abnormal wilting and yellowing of leaves, especially on warm days) is noticed aboveground.
- If the soil is moist, these symptoms may not become apparent until water stress occurs.
- Infection in the nursery stage results in the death of seedlings
- Infected plants at first show pale green to yellow leaves.
- Later, infected plants show wilting in hot, sunny midday, which recover during night.

- The aerial parts of the plants shows stunting, chlorosis, reduction in size of head, flagging of leaves and non specific unhealthy symptoms
- Infected older plants remain alive but stunted and fail to produce marketable heads.
- Most characteristic symptoms are seen on the roots.
- When the roots are examined the characteristic symptoms consist of small or large, spindle or spherical or knobby or club shaped swellings due to hypertrophy and hyperplasia which cause enlarged thick fleshy growths of the roots.
- The clubs are generally widest in the middle and taper toward the ends.
- Hypertrophy causes malfunctioning of xylem, which results in flagging of the leaves.
- The malformation may be seen either at the base of the tap root or at the lateral roots or the entire root system.
- The infected plants ultimately die.
- Older and larger clubbed roots disintegrate before the end of the season because of invasion by saprophytes.

Causal Organism: *Plasmodiophora brassicae*

The fungus is slime mold.

It produces resting spores inside the host cells. They are hyaline spherical, gives rise to zoospores

Zoospores are irregular amoeboid with uninucleate protoplasm with two flagella. Devoid of cell wall. These zoospores penetrate the cell wall enter and multiply causing hyperplasia and hypertrophy.

Pathogen: Obligate endoparasite

Thallus is Plasmodium (Naked slimy mass of protoplasm containing numerous nuclei) gives zoosporangia / resting spores released into soil when decayed and produce zoospores.

i. Primary phase occurs in root hairs

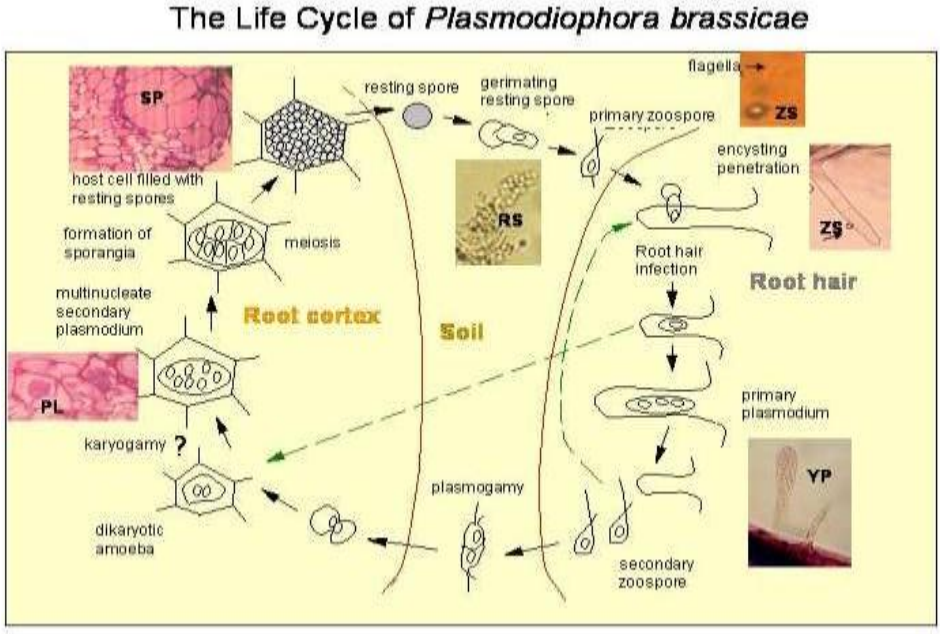
ii. Sec. phase occurs in cortical cells of roots

Primary phase – Resting spores germinate and produce zoospores (primary) with two flagella. Encysted zoospores attach with root hair, myxamoeba enters the root hair cell, divides to several uninucleate amoebae and nuclei divides and form multinucleate plasmodia which again divide and form thin walled zoosporangia which contain 4-8 uninucleate zoospores, escapes in the soil, fuse as gametes and form quadriflagellate binucleate zoo

Sec. phase – Quadriflagellate binucleate zoospores (secondary) reinfect the root and form binucleate plasmodium, penetrate the root cortex – plasmodium repeatedly divides and host cells hypertrophied and the plasmodium transformed into masses of resting spores released into soil as the root decay.

Disease cycle

Within the infected plant roots, the organism develops rapidly, causing an increase in the number and size of cells, which results in "clubbing." During the development of the organism in the plant, new zoospores are produced; these are capable of infecting the same plant or adjacent plants and, thus, repeating the cycle. Eventually, resting spores are formed within the diseased plant tissue, and these are released into the soil when the plant roots disintegrate.



Mode of survival and spread

- *P. brassicae* spores can remain viable in the soil and infected plant debris in soil for at least 20 years, even in the absence of a susceptible host. After the roots decay, spores are released into the soil where they remain dormant until conditions are suitable for germination, e.g. when another cruciferous crop is grown.
- The fungus can be spread through contaminated soil from place to place.
- Farm machinery, boots, animals, the dung of animals that have eaten diseased roots, and compost containing remains of a diseased crop are some of the means of spreading club root.
- The resting spores of the fungus can be spread from field to field by infested soil, contaminated water supplies, infected transplants, infested soil on farm machinery, and even by roving animals such as cattle.
- The most important means of spread is on the roots of infected transplants, contaminated irrigation water and surface flood water. Reports indicate that dam

water, especially water from dams receiving run off from infected fields, can become contaminated with the fungus.

- Spore dispersal by wind is also possible, especially under dry conditions.

Epidemiology

- The disease is favoured by wet acid soil
- Optimum soil temperature is 20 -25^0 C for disease development; although infection can also occur at temperatures as low as 12°C.
- High soil moisture is more important for disease development.
- Sensitive to alkaline condition
- The germination of resting spores requires moist, acid soil and can occur over a wide temperature range of 12°- 27° C.
- Disease development is favored by high soil moisture and soil temperature between 18°-25° C.
- Although clubroot has been found in soils exhibiting a wide pH range from 4.5-8.1, the disease is primarily associated with acid soils.
- No spore germination at pH 7.2 or above.
- Development of the disease is favored by a low soil pH (below 7.0), cool soil, and excess soil moisture.
- Cool, wet and acidic soils favour the development and spread of the disease.
- Clubroot is more prevalent on poorly-drained soils, particularly low-lying areas.
- Clubroot is more pronounced in acidic soils (pH<7.0), although at high spore concentration levels the disease can develop in alkaline soils (pH>7.0).

Management

- Eradication of crucifer weeds.
- Field sanitation
- Long rotations (7 years or longer) with non-cruciferous crops
- Avoid over-watering the soil and using practices that flood the soil, such as furrow irrigation.
- Improve drainage in poorly drained soils and low lying areas
- Use of only disease-free transplants
- Use of seedlings from disease free area
- Raise the soil pH to 7.2 by application of hydrated lime 6 week before planting and keeping the soil uniformly wet. Lime inhibits disease development, but will not prevent a disease outbreak if the spore load in the soil is sufficiently high. In some soils, large amounts of lime may be required. Be careful, because such applications may result in nutritional problems or encourage diseases such as common scab in potatoes and white rot in onions.

- The quantity of lime is determined by initially measuring the pH of the soil. Suppose a soil is with pH 5 it requires 2.5 t / ha.
- Soil application of PCNB 0.1%
- Solarisation is only economic to use on heavily-infested fields during periods of high temperature
- Seed treatment with cabendazim 2g/kg
- Nursery drenching with carbendazim or chlorothalonil 0.1%.
- Spot drenching with carbendazim/chlorothalonil 0.1%.
- Soil fumigation with chloropicrin, methyl bromide
- Application of formalin 2 % @ 10 lit/sq. m. of seed bed drenching the soil with PCNB 1.0% is found effective
- Seedling dipping in 0.1% $HgCl_2$ (or) 0.8% Benlate or Carbendazim for 15-20 minutes

Downey Mildew: *Hyaloperonospora parasitica* (synonym: *Peronospora parasitica*)

It attacks cabbage, cauliflower, turnip and radish. Very serious on cauliflower. The disease is very serious in nursery and it can also appear in field planting.

Symptoms

- Plants can be infected at any stage of development.
- In seed beds, cotyledons and primary leaves are invaded resulting in fungal growth visible on the underside of the leaf.
- Later a slight yellowing develops opposite to the fungal growth on the upper side of the leaf.
- The young leaf or cotyledon, when yellow, may drop off.
- In older plants, purplish brown spots appear on the under surface of the leaves, stem and pods.
- These may remain as small or enlarge in size.
- The upper surface of leaves shows irregular chlorotic or yellowish translucent spots delimited by veins.
- Under cool, moist conditions, grayish white fluffy downy growth appear on the under surface of the lesion.
- As the lesions enlarge and dry out they become necrotic and the leaves become tan and papery
- In severe infection the leaves shed
- The affected cauliflower curds look brownish to blackish at the top.
- The stem also get affected showing dark brown lesion which later develop downy growth.
- Numerous sunken black spots appear on cabbage head.

Causal Organism: *Hyaloperonospora parasitica* (synonym: *Peronospora parasitica*)

It is an obligate parasite. The mycelium is coenocytic, intercellular and is with ovate haustoria. Sporangiophores arise in groups through stomata, dichotomously branched and bear sporangia. The sporangia are greyish, ovoid and thin walled provided with papilla at the distal ends. Oospores are not common.

Mode of survival and Spread

- The fungus overwinters mainly in roots or in decaying portions of diseased plants on cruciferous weeds, and occasionally on crop seed.
- Survives as thick-walled spores which remain in the soil after the crop decays.
- On growing plants, spores are blown about by wind and splashed by rain.

Epidemiology

- The fungus thrives well in cool moist environment. Chiefly in humid region.
- Moisture and temperature are important in the spread and reproduction of this fungus.
- High relative humidity during cool or warm, but not hot, periods promotes its growth and sporulation.
- Presence of a water film on the foliage from fog, drizzling rain, or dew allows spores to germinate, infect, and produce more spores on a susceptible host in as few as 4 days.
- Rapid development and spread of the disease occur at 10 – 15 ^{0}C
- Heavy humidity, fog, drizzling rains or dews that remaining on plants until mid morning for 4 days is ideal for development and spread.
- Abundant sporulation and rapid disease development occur at greater than 98% relative humidity, when leaves are wet, and at 8-16°C.

Management

- Field Sanitation
- Crop rotation
- Clean seed beds.
- Use of disease free transplants
- All the weeds serving as alternate host to the fungus should be destroyed.
- The crop should be irrigated judiciously to avoid periods of high humidity.
- Seed treatment with hot water or organomercurials .
- Spraying Maneb 0.2% Ridomil MZ 72 0.25%
- Spraying the seedlings in the nursery beds with Copper Oxychloride (0.3%) is effective
- In the field, the crop is sprayed with Copper Oxychloride (0.5%).

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

White rust: *Albugo candida* (*Cystopus candida*)

Symptoms

- All aerial parts are affected
- Symptoms manifest as chlorotic or necrotic spots on upper leaf surfaces.
- White or creamy yellowish raised blisters or pustules are seen on lower surface of the leaves (Armstrong 2007).
- Other symptoms include chlorosis, necrosis, defoliation, swelling and distortion of stems and flowers, stunted growth, and leaf curling (Babadoost 1990).
- The leaves may become thick fleshy and inrolled
- Severe infection reduces the leaf size.
- Spots coalesce together causing drying.
- The systemic infection affects stem and inflorescence
- If the disease becomes systemic, an infected plant may form swollen, twisted floral parts called “stagheads” (Armstrong 2007).
- The inflorescence shows various deformations.
- They become thick fleshy and greenish leading to sterility.
- Floral parts show swelling and distortion due to hyperplasia and hypertrophy. This is known as staghead.
- The petals become sepals like; stamens become leaf like; Ovules and pollen grain are atrophied. Ovary is sterile

Causal Organism: *Albugo candida* (*Cystopus candida*)

It is an obligate parasite. Mycelium is intercellular. Knob like haustoria, hyphe collect beneath the epidermis Sporangiophores are club-shaped hyaline and produce short chain of sporangia basipetally. Sporangia are single celled, spherical, hyaline and are arranged in chain.

Mode of survival and Spread

- Oospores serve as primary inoculum for this disease and can survive for many years in soil and plant debris or as a contaminant of seeds.
- Sporangia produced in pustules are spread by wind, rain or insects to neighbouring plants.

Epidemiology

- Cool moist weather favours development of the disease.
- Under optimum conditions, between 16 and 25° C and high relative humidity (Damicone and Roberts n.d.), the sporangia germinate to produce biflagellate, motile,

infective zoospores (Reddy 1996). If there is no free moisture available on the host surface, the sporangia may simply re-infect the host plant without producing zoospores. If free water exists on leaf surfaces, the zoospores drop their flagella, encyst, and form germ tubes, which enter leaves through the stomata to cause systemic infections.

- Infection is generally favoured by cool (13-18°C), wet weather in the form of prolonged dews or fog.

Management

- Field sanitation
- Destroy the oospores on surface of seed by hot water treatment at 52°C for 20 minutes.
- Removal of Cruciferous weeds
- Spray 0.8% Bordeaux mixture or copper oxychloride 0.25% or Chlorothaloni0.1% or metalaxyl 0.1 % or Mancozeb 0.2 %.

Alternaria leaf spot and Blight: *Alternaria brassicae* and *A. brassiciola*

It is a destructive disease on seed crop. The leaf spot phase reduces plant vigour and renders the crop unsalable or unusable where leaves are the edible portion of the plant. Besides the leaf spot phase, edible flower parts of cauliflower curds, seedlings, and flower parts related to seed production can be diseased.

Symptoms

- On seedlings, the symptoms are small dark spots on the stem or black streaks on cotyledons and hypocotyls that cause damping-off or stunting of the plant.
- On older plants, the bottom older leaves are infected first.
- Symptoms appear as small circular dark coloured spots on the leaves.
- Spots spread rapidly to form circular lesions upto 1-3 inches in diameter.
- The enlargement of the spots has characteristic concentric rings (target spots) and possibly surrounded by yellow halos.
- Lesion centres may break apart, giving a shot hole appearance to the leaf
- Under favourable conditions, the lesions become covered with sooty black mass of spores.
- Infected leaves soon turn yellow and drop.
- Several spots cause premature death of leaves.
- Petiole and stem also affected showing linear spots.
- The disease infects the base of heads and cause browning of cabbage and cauliflower heads
- Flower clusters may also become infected during seed production, affecting seed quality.

Causal Organism: *Alternaria brassicae* and *A. brassiciola*

Conidiophores are thick and brown. Conidiophores are thick and geniculate. Conidia are both transversely (11-15 septa) and vertically septate (0-3 septate), coloured and beaked. Conidia are mostly solitary or chains up to 4.

Mode of survival and Spread

- Survives in plant debris in soil
- Cruciferous weeds may also harbour these fungi
- Primarily seed borne via mycelium within the seed or transitory spores on the seed (both internally and externally seed borne)
- Conidia are disseminated by wind and water.

Epidemiology

- Occurs during warm, moist conditions
- Bright sunshine, frequent dews or showers, and temperatures between 60 and 90 °F favor disease development.
- Disease development is favored by free moisture on plant surfaces and temperatures between 20-27°C.

Management

- Use of disease free seeds
- Proper crop rotation
- Seed treatment with Thiram 2-3g/ kg
- Seed treatment with hot water (50^{O}C for 30 minutes)
- Spraying at full bloom, pod set and pre- harvest stage with Captan (0.2%) or Mancozeb or zimeb 0.2% or Copper Oxychloride (0.5%) for the control of disease.

Black leg of cabbage: *Phoma lingam* (sexual stage: *Leptosphaeria macutans*)

Black-leg is of major concern in crucifer production. It is a serious disease in temperate region.

Most destructive one.

Symptoms

- Symptoms manifest as oval, sunken, light-brown cankers with purple-to-black margins near the base of stems.
- These lesions extend below the soil surface, causing a black rot of lower stem and roots.
- Cankers enlarge and girdle stems, causing plant collapse.
- Lesions may also develop on cotyledons and hypocotyls of young seedlings causing Damping-off several weeks before transplanting
- Lesions appear on leaves as pale, irregular spots.
- Leaf spots gradually enlarge, becoming circular to oval with ashy gray centers.

- Under favorable conditions, small black fruiting structures (pycnidia) develop in stem cankers and leaf spots.
- Vascular tissues may turn black in color prior to external rot symptoms.
- The fungus interferes with water conduction in tissues causing wilting and debilitating plants.
- Severely infected plants are stunted and often wilt.
- The leaves remain attached and the plant turns a dull blue-red color.
- The root system may be destroyed, although new roots may form above the stem cankers, allowing the plant to remain alive.
- Infection can spread to the base of leaves of cabbage heads in storage where brown to black spots develop.
- Flower stalk and siliques and seed also affected

Causal Organism: *Phoma lingam* (sexual stage: *Leptosphaeria macutans*)

Mycelium septate, branched, hyaline at young and dark with age.

Two types of pycnidia can be found; one on infected, live plants (thinner-walled pycnidium with a neck) and the other on crop residues (thicker-walled pycnidium with a narrow ostiole). through infected plantings during wet conditions when pycnidia are present.

Pycnospores hyaline one celled.

Mode of survival and Spread

- Fungus survives on plant debris or living plants as pseudothecia, which release ascospores that may be windborne for long distance.
- The fungus survives in association with infected plant residues until the plant debris decomposes (1 to 4 years)
- It can survive for years as mycelia in infected seed.
- Sources of the fungus also include cruciferous weeds
- Survives in plant debris in soil for 3 years
- Seed borne
- Secondary spread by air borne ascospores
- Pycnidiospores are dispersed by splashing rain or irrigation water
- Disease also spreads by workers or equipment moving

Management

- Use disease-free or treat seeds with hot water.
- Hot water treatment of seeds at 50^0 C for 30 minutes is recommended.
- Practice a four year rotation in seedbeds and fields.
- Avoid cultivation in fields where crucifers have been continuously grown for 2 years.
- Seedlings before transplanting should not be dipped in water

- ❖ Rogue diseased plants from seedbeds.
- ❖ Improve soil drainage and air circulation.
- ❖ Control brassica weeds.
- ❖ Incorporate crop debris promptly after harvest to hasten decay.
- ❖ Avoid working in the fields when wet.
- ❖ Seed infection can be prevented by spraying the seed plants with copper oxychloride or with an organo mercuric compound.
- ❖ Seed treatment with Captan or Thiram 4g/kg of seed, followed by seed treatment with *Trichoderma viride* 4g/kg.
- ❖ Pusa Drumhead, a cabbage cultivar has been reported to be tolerant under field condition.

Cabbage yellows or *Fusarium* wilt of cabbage: *Fusarium oxysporum* f.sp. *conglutinans*

It was found first in the Hudson Valley in 1899. It now occurs in all states where cabbage is grown in warm seasons. Yellows can attack all members of the cabbage family, including cabbage and cauliflower. It is a vascular disease. It is common in warm condition.

Symptoms

- ❖ Plants will develop characteristic symptoms 2 to 4 weeks after transplanting
- ❖ The entire plant appears to have a dull cast, and lower leaves turn yellow-green in color.
- ❖ A lateral warping or curling of the stem and leaves occurs.
- ❖ The lower part of the leaf blade adjoining the petiole or midrib wilts and dies first, resulting in a curve in the midrib.
- ❖ The lower leaves turn yellow first, and then symptoms move to the upper leaves.
- ❖ The wilting of lower leaves may be more apparent on one side of the plant than the other.
- ❖ The yellowed leaves turns brown dies and becomes brittle drop of prematurely
- ❖ Growth is stunted.
- ❖ When stems of infected plants are cut in a cross section, internal necrosis (browning) usually is visible.

Causal Organism: *Fusarium oxysporum* f.sp. *conglutinans*

Mode of survival and Spread

The Fusarium yellows fungus is capable of surviving many years in the soil, even in the absence of host plant debris.

Epidemiology

Infection is favored by warm soil temperatures (80-85^0 F) and does not develop well in temperatures below 61 F.

Fusarium yellows is a "hot weather" disease.

Soil moisture and pH have little reaction on the fungus.

Management

Field sanitation

Crop rotation

Seed treatment

Rhizoctonia* disease of cabbage: *Rhizoctonia solani (Thanetophorus cucumeris*; synonym: *Pellicularia filamentosa)

The fungus causes damping-off and wirestem of cabbage, cauliflower, and other crucifer seedlings in the seedbed; bottom rot and head rot of older plants in the field and storage. Losses occur as reduced stands and lowered yield and quality.

Symptoms

1. **Damping-off.**
   - ❖ Seeds may decay, especially in cold, wet soils.
   - ❖ Stems of young seedlings become light brown and water-soaked near the soil line.
   - ❖ Affected seedlings quickly wilt, topple over, and die.
   - ❖ Damped-off plants commonly occur in circular patches in plant beds or along sections of rows.
2. **Wirestem**
   - ❖ Wirestem is the most common and destructive phase of Rhizoctonia disease.
   - ❖ This disease is more serious in nursery beds.
   - ❖ The affected young seedlings show reddish brown discolouration of the stem near the ground level.
   - ❖ This area gets constricted and the plants bent or twist without breaking.
   - ❖ In some cases, the seedling continues to grow even though the lesion girdles the stem.
   - ❖ The lesion is quite sunken, and the stem resembles a wire, hence the name 'wirestem'.
   - ❖ The girdled seedling eventually dies.
   - ❖ In transplanted plants, the stem above and below the soil line shrivels and darkens, becoming tough and woody or wiry.
   - ❖ In wet soil, the decayed outer tissues of the stem may slough off when the plant is pulled.
   - ❖ Growth of diseased seedlings after transplanting to the field is usually slow.
   - ❖ Severely diseased plants soon die.

- ❖ Even if the plants are able to produce new roots above the affected stem after transplanting, they are generally stunted, unthrifty, and they produce small heads.

3. **Bottom rot**
   - ❖ The lower leaves droop, decay, and turn black, but remain attached.
   - ❖ Some plants may recover and produce heads.
   - ❖ In cabbage, bottom rot usually develops into head rot.
4. **Head rot**
   - ❖ A firm to slimy dark decay of the bases of the outer leaves and heads of cabbage develops between early head formation and maturity.
   - ❖ The outer leaves of the head wilt, become pale, and turn brown to black near the main stem.
   - ❖ As leaves are killed, they dry at the base and remain held in place by the margin of the blade that folds over the top of the head.
   - ❖ Foliage leaves die and drop off, exposing the stem beneath the head.
   - ❖ A cobwebby brown mold may be conspicuous on decayed tissues and between the head leaves in damp weather.
   - ❖ Small brown sclerotia may develop and be visible on parts or the whole surface of the head.
   - ❖ Dark brown sunken spots are produced inside the head.
   - ❖ A firm, persistent, dark decay continues to develop in transit and storage.

Causal Organism: *Rhizoctonia solani (Thanetophorus cucumeris*; synonym: *Pellicularia filamentosa)*

Mode of survival and Spread

- ❖ The fungus survives indefinitely in the soil, primarily as small, hard, chocolate-brown, kernel-like bodies called sclerotia.
- ❖ Crucifer seed infected with *Rhizoctonia* is also a primary source of inoculum;

Epidemiology

- ❖ The strain of *Rhizoctonia solani* causing bottom rot and head rot of cabbage grows at temperatures ranging between 8° to 32°C.
- ❖ Cabbage may become infected at temperatures ranging between 11° to 32°C with an optimum between 25° to 26°C.
- ❖ Cool, cloudy weather, high humidity, wet and compact soil, and overcrowding especially favours development of the Wirestem disease.
- ❖ Damping-off develops most rapidly at temperatures of 75°F (23°C) and above in wet soils following heavy rains or over-irrigation.

Management

- Disinfest the soil before seeding with steam or a soil fumigant.
- Grow plants in well-drained, fertile soil. Fertilize based on a soil test
- Treat the seed with hot water.
- When transplanting, discard all seedlings with discolored stems and roots.
- Do not plant crucifers for at least three years in fields where bottom rot, head rot, and root rot have occurred.
- Cultivate the soil as soon as possible after heavy rains to aerate it and thus make conditions less favorable for infection.
- Avoid covering parts of leaves with soil when cultivating.
- Banking soil around plants creates conditions favorable for development of bottom rot and head rot.
- Harvest crucifer root crops when the soil is comparatively dry so that a minimum of soil will adhere to the branch roots.
- Discard that show severe Rhizoctonia infection.
- A reduction in the inoculum level of *Rhizoctonia* has been reported following cropping with cereals and incorporating the crop residue into the soil.
- seed treatment with antagonist fungal culture of Trichoderma viride (3-4 g/kg of seed) or Thiram (2-3 g/kg of seed) are effective.
- Soil around the affected seedling should be drenched with Dithane M 45 (0.2%) or Bavistin (0.1%) to control the spread of the disease.

Black Rot of crucifer: *Xanthomonas campestris* pv*. campestris*

Black rot is a potentially lethal bacterial disease that affects cruciferous vegetables including cabbage and cauliflower. Black rot occurs worldwide wherever cruciferous plants are grown and makes cruciferous vegetables unfit for the marketplace or the table. Black rot of crucifers is considered the most destructive disease of crucifers worldwide. The disease seldom occurs and causes little damage in cooler temperate climates but can cause severe losses in transplant and field production in warmer subtropical climates. The disease causes high yield and quality losses, especially in tropical and subtropical regions during the rainy season. In warm and wet conditions black rot losses may exceed 50% due to the rapid spread of the disease. The disease is usually most prevalent in low areas and where plants remain wet for long periods. Conditions favouring plant-to-plant spread of the bacterium has led to a total loss of crucifer crops. In India it was first reported in 1929 and then its widespread occurrence all over the country is well recognized. It was introduced from west to our country.

Symptoms

- The disease affects primarily above ground parts of plants at any stage of growth

- On young seedlings, cotyledons are infected showing blackening at the margin.
- Later, the cotyledon shrivels and drops off, but only after the bacteria have passed into the young leaves and stem.
- Affected seedlings turn yellow to brown, wilt, and collapse.
- On older plants Initial symptoms are irregular, dull, yellow blotches that appear on the edges of leaves.
- As the disease progresses, these blotches expand and progresses to form the characteristic wedge or V-shaped lesion associated with this disease with the open part along the edge of the leaf.
- The V-shaped areas are initially yellow, but eventually become brown and necrotic in the center with a yellow border or halo.
- Within the affected area, leaf veins and veinlets turn black forming to a net-like pattern, giving the disease its name – black rot.
- At advanced stages, affected tissue becomes brown and necrotic.
- Black leaf veins may extend from the affected leaf into the main stalk where the darkened vascular system may be visible.
- As the disease progresses into the vascular system, lesions resulting from systemic invasion may appear along leaf midribs and between leaf veins.
- The V-shaped areas enlarge and the entire leaf eventually turns yellow or wilts and falls from the plant.
- A typical cross-section of an infected stem or petiole shows a black ring due to invasion of the water-conducting vessels
- Systemically infected plants may be stunted
- More severe symptoms develop on one side of the plant.
- The lower leaves on infected plants are usually stunted, yellow to brown, and wilted and often drop prematurely.
- Infected plants may consist of only a long, bare stalk topped with a tuft of leaves.
- In affected cabbage, heads are smaller and outer leaves may senescence.
- The disease can progress on cabbage during storage, making the heads unmarketable.
- Plants may quickly rot immediately before or after harvest, due to secondary soft-rotting bacteria.

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

It is a Gram negative and rod-shaped bacterium with single polar flagellum.

Mode of survival and Spread

- The bacteria can overwinter in plant debris, in and on seeds, and in and on weeds.

- The pathogen may survive in diseased crop residue buried in soil for up to 2 years, but not more than 60 days free in soil.
- The major source of these bacteria is infected seeds, which enable long-distance spread of the disease. As few as three infected seeds in 10,000 (0 03%) can cause black rot epidemics in a field.
- The pathogen is spread within and between fields by splashing water, wind, insects, machinery, irrigation or drainage waters, blowing of detached leaves or dust particles and shipping and handling of infected plants.

Epidemiology

- Free moisture is required for infection by the pathogen.
- After infection, symptoms may appear on plants within 7 to 14 days under optimum conditions (25 to 30 degrees C).
- The disease develops best under warm, wet conditions.
- Temperatures of 80 to 86 degrees F favour growth of the bacterium.
- Free moisture, in the form of rain, dew, or fog, is required for infection to occur.
- The disease may be more severe in low and shaded areas.
- This bacterial disease is common in areas having a warm and wet climate.

Management

- Disease-free seed and transplants
- Use clean, disinfested containers and planting materials.
- Good air and soil drainage
- Field sanitation
- Cultural practices such as deep tillage hasten the decomposition of infested crop residue
- Crop rotation for 3 years
- Provide a balanced fertility program.
- Control of all cruciferous weeds in and around the production area.
- Do not work in fields when plants are wet.
- Destroy or bury all crop debris immediately after harvest.
- Planting should be done on raised beds to facilitate drainage.
- Avoid sprinkler irrigation
- Also, do not overcrowd or plant in poorly drained soil.
- Do not work in the seedbed or fields when plants are wet.
- Use clean or new harvest containers that are smooth and flexible.
- Spraying fields with fixed copper bactericides
- Seed treatment with 0.1% $Hgcl_2$ (or) Aureomycin 0.1% or Agrimycin-100 (100ppm) or Streptocycline (100 ppm) for 30 min (or) hot water 50^0 C for 20 – 25 min

- Soil application stable bleaching powder @ 10 – 12.5 kg / ha.
- Spraying agrimycin 100 or Streptocycline gives effective control.
- Spraying copper oxychloride 0.25% + Agrimycin 100 at 200 ppm.

Stalk rot/Cottony rot/ White blight/ white mould / white rot: *Sclerotinia sclerotiarum*

It is considered to be a limiting factor in cauliflower and cabbage seed production in N. India.

Causes 30% loss in cauliflower. This fungus can cause serious losses in the field, in storage, and under transit and market conditions.

Symptoms

- Generally, damp weather favours the occurrence of the disease.
- Infections may occur on the stem at the ground level, on the leaves at their bases, or where the foliage comes in contact with the soil.
- The infections begin as water-soaked, circular areas, which soon become covered by white, cottony fungal growth.
- The affected tissue becomes soft and watery as the disease progresses.
- The fungus eventually colonizes the entire cabbage head and produces large, black, mustard seed like structures called sclerotia on the diseased tissue.

Cauliflower mosaic virus

Mosaic mottling, vein clearing, vein banding, stunted growth, central leaves are smaller, small sized head and poor quality

Spread by aphids *Brevicoryne brassicae*, *Myzus persicae*

DISEASES OF SWEET POTATO

White rust

The disease is of world wide occurrence and attacks other members of Ipomola. Irregular yellow specks appear on the lower surface of the leaves. Latter they become white blisters and rupture to expose the white powdery mass of spores. Corresponding to this yellowing or browing can be seen on the upper surface of the leaves. Severe infection causes defoliation.

Black rot

Occurs wherever sweet potatoes are grown primarily a storage rot disease. Also occur in the field as well as in seed beds.

All underground parts of the plant are affected. Black spots appear on the young sprouts which eventually girdle the plant . This result in yellow and sickly appearance of the leaves the blackening extends to roots and stem. Dark circular depressed spots are grayish black when dry and dark greenish. Black when moist at the centre small black fruiting bodies develop.

C. Organism: Mycelium septate branched brown with age. Microconidia are hyaline one celled cylindrical. Macroconidia or chlamydospores olive brown obovate oval terminal or in chain perithecia flask shaped with long beak ascospores elliptical.

Spread: Chlamydosporesa nd perithecia in crop residue & tuber.

F. Condition: Infection occurs in field at 10 -30 C with opt at 25 C Amount of disease increases with increase in soil moisture at storage opt temp 23 – 27 ^{0}C.

- **Control**: Use of disease free planting materials
- Field sanitation.
- Soil should be well drained.
- Heat treatment at 43 C for 24hr.
- Dipping the seed materials in Hg CL2 at 1:1000 (or) TBZ 3.32 g ai / l
- Cutting should be planted in soil free from pathogen for disease free crop.

Storage rots

Soft rot : The disease begins as soft rot which progresses rapidly decaying the whole tuber within 4-5 days. When the skin inbroken a straw coloured liquid drips out of it. If the skin is not broken the affected tubers lose moisture and become shriveled and mammidfied when the skin is ruptured during the progress of the diseases fugal growth can be seen covering the affected.

Surface consisting of sporangiophores and sporangia of the fungus the tissues turn brown with mild odour.

F. Condition: Most rapid decay of tuber occurs at 15 – 23 C and RH 75 – 84%

- **Control**: Aovid wound or bruises on the tubers during digging and storing.
- DRY IN SUN FOR 1-2 hr before storage.
- Storages should be clean and washed with 2.5% CUSO4 solution.
- The temp of the stores should be held at 26.5 – 30 C with 90% RH for 10 – 14 days then declimed to 13C & 85 -90% RH.Clearing of tubers before storage.

Dry rot

It is of minor importance in the field but it may cause serious damage in the storage. In the field the rot begins at the stem end of the root. The diseased roots are shrunken and wrinkled. The surface is covered with minute mounds. The internal decayed tissue is coal black in colour.

Charcoal rot

This rot is relatively slow in storage. The tissues becomes chocolate to cinnamon brown and later dark reddish brown in colour. Minute balck sclerotia develop on the affected tissues giving black appearance. The decay in spongy at first turning to hard mummified consistency.

DISEASES OF BEANS

Anthracnose

- This is an imported disease.
- It causes greater loss in cool temperature regions than in the tropics.

Symptoms:

- All aerial parts are affected.
- Most striking symptoms appear on immature pods.

- On leaves initially blackened dead portions of veins appear on the under surface of the leaves.
- Later similar spots appear on the upper surface of the leaves.
- Lesions are also formed on petioles stems and cotyledons.
- On pods black sunken cankers with lighter or grey central areas are seen.
- The central area shows pinkish mass of spores during central areas are seen.
- The infection may go deep into the seed.
- The seeds of infected pods show brown to light chocolate coloured sinken cankers on the seed coad.

C. Organisms: Colletotrum lindemuthiamum

Similar to that of C. gloeosporioides.

Mode of spread and survival:

The fungus survive on infected seeds and plant debris.

The fungus is seed borne.

Epidemiology:

Optimum temp 17 – 27 C ; RH 90 – 100%

In hills of North India maximum intensity reaches in August – September.

The disease is severe in cool wet season or when over head irrigation is used.

Management:

- Use of disease free seeds.
- Crop rotation for three years.
- Field sanitation.
- Avoiding over head irrigation.
- Sowing between mid April to Mid May.
- Seed treatment with Carbendazim 2g/kg.
- Foliar spraying with Carbendazim 0.1% or Mancozeb 0.2%or Zineb 0.2%.
- Use of resistant varieties wells Red kidney sea way, maniton. In HP 2 accessions AB 136 and G 2333.

Rust

- The disease attacks mostly leaves rarely the stem petioles and pods.
- Initial symptoms are minute whitish slightly raised pustules are produced on under surface of the leaves.
- Later they become distinct reddish brown circular sori or pustules.
- On formation or teleuospors they turn dark brown or black.
- Severe infection gives a rusty appearances to the leaves.
- Severely infected leaves may turn yellow and dry and fall off.
- **C. Organism:** Uromyces phaseoli typical
- Autoecious long cycle rust.
- Uredospores – globose or ellipsoid single celled echimulated,

- Teleospores – globose or broadly ellipsoid single – celled
- pedicellate chestunut brown smooth with few varicose marks.

Mode of spread – Avi borne conidia

Epidemiology:

- Cloudy and humid days with leaf wetness in the morning favour germination of spores and infection.
- A medium to fairly high temperature is required for heavy infection.
- Long day hours favour the disease.

Management:

- Field sanitation.
- Crop rotation.
- Wider spacing.
- Foliar spraying with Mancozeb 0.2% or Triadimefan 0.05%.

Augular Leaf spot

In India the severity of the disease has been reported upto 70% resulting in > 56% pod loss.

Symptoms:

- Angular spots appear in between veins and vein lets on the leaves.
- The spots are initially dark grayish on upper surface and light grayish on lower surface of the leaves.
- Later the spots turn to dark brown covered with grey mould on the lower surface.
- Severely infected leaves fall off prematurely.
- On pods the spots are circular having reddish brown centre with well defined ashy black border.
- Severely infected pods either bean no seeds or produce shriveled seeds.

Mode of Spread and Survival:

- The pathogen perpetuates through seed and plant debris.
- Both seed and soil borne.
- The disease is severe in wet weather.

Management:

- Crop rotation 3-4yr.
- Disease free seeds.
- Deep ploughing.
- Wider spacing.
- Well drained weed free soil.
- Application of carbendazim 0.1% or Tridemorph 0.1%
- Use of res var EC 77007, EC 10037, EC 10039.

Powdery mildew.

- Similar to PM of Pea

Ashy stem blight / Ashy grey stem

- Also called as dry root rot or charcoal rot.

Symptoms:

- Black sunken canker appears near the base of the cotyledons.
- It spreads downward into the stem and first pain of the infolded leaves.
- This results in damping off and death of young seedlings.
- On older stem reddish brown shallow cankers are produced.
- They become grey at centre with numerous minute black pycnidia.
- Infected plants are killed.
- Root infection causes blackening of roots and hence called as charcoal root.
- Leaf infection shows irregular dead areas at the tip marked by pycnidia.
- The infection spreads down the veins reaches the petiole causing defoliation.

C. Organism: Macrophomina phaseolina

Seed and soil borne.

Warm weather conditions of 32 – 34 C favourable for disease incidence.

Management:

- Disease free seeds.
- Field sanitation.
- Removal and destruction of infected plants.
- Avoidence of excessive soil moisture.
- Seed treatment thiram or carbendazim.
- Soil drenching with COC or Carbendazim.

Common Blight

It is a major constrain in bean production in tropics.

Yield loss upto 84% reported.

Symptoms:

- The disease affects leaves stem pod and seeds.
- Starts as water soaked spots on the under surface of the leaves.
- Enlarge to form irregular sunken reddish to brownish spots with yellow halo.
- Reddish discolouration of veins and mid ribs.
- Reddish streaks are formed on the stem .
- Severe infection results indefoliation.
- On pods water soaked spots surrounded by district zones of reddish brown or brick red band of tissues are formed.
- The seeds are discoursed and shriveled .
- Seedling arises from infected seeds exhibits wilting.

C. Organism: Xanthomonas axonopodis pv. phaseoli.

Mode of spread and survival:

The bacterium overwinters in seed plant debris and weed hosts.

Primarily seed borne sec spread through wind splashed rain.

Epidemiology:

Warm and humid condition favour disease development.

Management:

- Disease free seeds.
- Field sanitation.
- Deep ploughing to bury the plant debris.
- 2-3 yr crop rotation.
- Spraying COC + Mancozeb.
- Seed treatment with streptomycin sulphate 0.5% or Hot water treatment at 50 C for 10min.
- Res var Goliath waxy, Ruse 6, Tara valley.

Common Mosaic- Bean Common Mosaic Virus (BCMV or BV 1)

One of the most commonly prevalent viral disease.

Causes considerable loss amounting to 26 – 84%.

First reported in 1963 in India.

Symptoms:

- The leaves have irregular shaped light yellow and green areas frequently in a mosaic pattern.
- During early stage of infection the leaves appear crinckled chlorotic, stiff with downward curling.
- In advanced stage the leaf size is reduced with filform leaflets, the plants are stunted and appear bushy.
- The pods are under sized and have few seeds.
- Vascular necrosis of the root stem leaves and pods is also common.

C. Organism:

Bean common mosaic virus

Seed transmission.

Also transmitted by aphids. Aphis craccivora, A. fabae, Myzuz persical.

Management:

- Use of disease free seeds.
- Adjusting planting date coinciding with reduced vector actively.
- Barrier cropping with maize.
- Closer spacing.
- Removal and destruction of infected Plants.

- Spraying Monocrotophos 0.05% or Dimethoate 0.05%
- Res . var Pusa Parvati Top crop Masterpiece Premier PDR 14.

Yellow Mosaic Virus – Bean Yellow Mosaic Virus

- Bean yellow mosaic virus small chlorotic spots surrounded by yellow halo appear on leaves.
- These spots enlarge and the leaves appear yellowish.
- Young leaves become glossy stiff and curled upwardly.
- The mottling of contrasting yellow and green areas become more intense. This distinguish from common mosaic.
- The disease causes stunting bunches and delayed maturity.

C. Organism: Bean Yellow mosaic Virus

Transmn by aphids Aphids fabae M. persical. Macrosiphum euphorbial.

Management: Refer common Mosaic except for res var.

Res. var Black Turtle soup.

DISEASES OF COCONUT

1. Bud Rot – *Phytophthora palmivora* and *Phytophthora katsurae*
2. Basal stem rot / Thanjavur Wilt – *Ganoderma lucidum*
3. Stem bleeding – *Thielaviopsis paradoxa*
4. Grey leaf spot – *Pestalotia palmarum*
5. Root (Wilt) Disease – Phytoplasma

Bud rot: *Phytophthora palmivora* and *Phytophthora katsurae*

This is one of the worst diseases of coconut. It is found in many parts of India. First reported in India in 1906 by E.J. Butler. Common killer / fatal disease. The seedlings and young trees are more susceptible.

Symptoms:

- Affects palms of all ages, but young palms of less than 20 years of age are more susceptible especially during rainy / monsoon season
- The first external symptom is pale green or yellowish green discolouration of the heart leaf or central shoot.
- At the base of the heart leaf irregular, sunken, water soaked brown spot appears.
- The disease is characterized by rotting of the terminal bud and surrounding tissues quickly
- The infected portion will be degenerated to slimy mass emitting foul smell.
- The affected heart leaf comes off easily when it is pulled.

- Within a few days the infection spreads to adjacent older leaves which become yellow and then brown.
- In adult palm, the first externally visible symptom is withering of the spear leaf, which subsequently turns pale in colour, bends over at the base and hangs down
- Withering and yellowing of one or two younger leaves surrounding the spindle
- Rotting of the tender leaf base and soft tissues of the crown into a shiny mass of decayed material that emit a foul smell which attracts flies
- Rotting progresses downwards, affects the meristem and kills the palms
- The internal tissues show discolouration assuming a pale pink with a brown border
- Drooping of successive leaves and falling of leaves one by one, leaving only mature leaves in the lower whorls at the crown
- Young nuts cease to develop and fall off prematurely.
- Severely affected palms are killed due to death of growing buds.
- Retained nuts on the palm grow to maturity
- The palm ultimately succumbs to the disease, if not checked

Causal Organism: *Phytophthora palmivora* and *Phytophthora katsurae*

Oomycetous – Peronosporales – Pythiaceal

Mycelium hyaline coenocytic – intercellular

sporangiophores are simple or branched.

Sporangia – Pear –shaped hyaline thin walled formed singly on germination – motile zoospores reniform with two flagella.

Also produces chlamydospores – intercallery or terminal sexual reproduction – Anthernidia – Oogonia – oospores.

Mode of spread and survival

- The fungus survives as oospores, chlamydospores and mycelium in the soil and in the frond base or basal part of the crown which act as primary source inoculums.
- Secondary spread by zoospores through irrigation water, wind and windblown rain

Epidemiology

- Heavy rainfall, high relative humidity of 94-100% and temperature below 24^{o}C for 6 days are highly favourable for the disease spread

Management

- Unless the infection is noticed in the early stages no curative measures could save the palm.
- In early stages of the disease, cleaning and removal of all affected tissues of the crown along with some healthy tissues and application of Bordeaux paste and protect it from rain by providing a mud pot or a polythene covering till normal shoots emerges
- Spray 1% Bordeaux mixture to the spindle leaves and 2-3 innermost whorls of leaves

- Burn all diseased tissues removed from the palm
- As a prophylactic measure, spray 1% Bordeaux mixture on the spindle leaves and on the base of 3-4 innermost whorls of leaves of neighbouring palms
- In palms that are sensitive to copper containing fungicides, spray and spray drench with Mancozeb 0.4% (4g/L)
- Small perforated sachets containing 2g Mancozeb may be tied to the top of the leaf axil. When it rains, a small quantity of the fungicide is released from the sachets to the leaf base, thus protecting the palm
- Adopt control measures for rhinoceros beetle and red palm weevil
- Provide adequate drainage in gardens
- Adopt proper spacing and avoid overcrowding in bud rot prone gardens
- In bud rot prone gardens, prophylactic measures should be done to all palms 2-3 times at 45 days intervals
- Clean the crown before monsoon and follow strict phytosanitation
- Regular application of organic matters favours the establishment of biocontrol agents and suppression of pathogens
- Apply salt and ash mixture or paddy husk after removing the affected portion in the crown and subsequently cover with a mud pot. They absorb moisture and keep the protected portion dry
- Apply bleaching powder on the affected portion

Basal Stem Rot / Thanjavur Wilt / Foot Rot / Ganoderma Wilt / Anabe Roga: *Ganoderma lucidum, Ganoderma applanatum* and *G. boninense*

Thanjavur wilt disease of coconut is a major constraint in the production of coconut not only in TN but also in the neighboring states. The disease was first noticed in Thanjavur district of TN after the cyclone of 1952 and 1955 and hence the name **Thanjavur wilt**.

Symptoms

Palms bear profusely just prior to and at the initiation of symptoms

The diseased palms show the following typical symptoms in different parts of the palm *viz.,* stem, leaves, inflorescence and roots.

a. Leaves

- Initially the leaves in the outer one or two whorls wither and droop with yellowing and browning.
- Yellowing and drooping of leaves progress to younger leaves in quick succession leaving the spindle leaf alone.
- Spindle leaf stand erect with flacidity
- The spindle leaf becomes short and do not unfold properly.

- ❖ The dried outer whorl of leaves remaining hang down around the stem for a long time to form a skirt of dead leaves around the trunk (Plate31)
- ❖ Leaves break or buckle very close to their bases
- ❖ The crown is reduced to a mere handful of short erect yellow leaves (Plate 32).
- ❖ Drooped leaves fall off one by one leaving only a few smaller leaves at the apex.
- ❖ Delayed production of new leaves
- ❖ The newly formed leaves are pale and smaller in size
- ❖ Finally smaller leaves wither and bud decays
- ❖ Crown is easily blown off by wind

b. Stem:

- ❖ Bleeding of reddish brown viscous fluid (honey like) from the base of the stem with the decayed tissues beneath.
- ❖ It is restricted to basal portion of the stem (0.1-1.5 m) from the base
- ❖ Discolouration, necrosis and rotting of internal tissues up to the height of bleeding (exudation) and emitting a bad smell.
- ❖ In advanced stage the basal portion of the stem decays up to the central core.
- ❖ Sometimes wilt without external bleeding.
- ❖ The bark peels off.
- ❖ Apex of the trunk tapers
- ❖ In the advanced phase or in dead palms, the fungus fructifications may be observed at the base of the palm just above the ground level as a shiny, waxy, brown, corky and woody bracket

c. Inflorescence and nuts:

- ❖ Production of new inflorescence is arrested
- ❖ Button shedding is common.
- ❖ The subtended bunches hang down.
- ❖ Shedding of immature buttons and formation of barren nuts
- ❖ When the disease progress slowly, only few normal nuts are produced.
- ❖ Often bear profusely just prior to and at the time of initiation of symptoms.
- ❖ Nut and kernel weight, water content, copra weight and oil content decrease.

d. Roots:

- ❖ Extensive damage of the root system following rotting and disintegration of cortical tissues
- ❖ Decay and death of finer roots (70 %).
- ❖ Production of new roots is very poor.

Causal organism: ***Ganoderma lucidum, Ganoderma applanatum*** **and** ***G. boninense***

The fungus is hetrothalic. Hyphae are hyaline, chlamydospores are intercallery or terminal golden yellow colour granules.

Conidia are round and thin walled.

Mode of spread and survival

- Fungus is soil borne inhabiting in roots of dead as well as living woody plants in the soil.
- The disease spread is mainly through root contact.
- Secondary spread through air borne basidiospores.

Epidemiology

- Trees in the age group of 10 – 30 years are more susceptible.
- Prolonged drought, high soil temperature, low rainfall, extensive flooding, sandy or sandy loam soils, presence of hard pan in the sub-soil and neglected gardens are conditions favourable for the disease incidence

Management:

- The stumps of already infected and dead palms should be removed and burnt.
- The palms with advanced stage of infection and economical palms should be cut along with root system and removed.
- Isolate the affected palm from the healthy ones by digging a trench of 1m deep and 50cm wide at 2.0m away from the bole of the infected palm
- Avoiding repeated ploughing and deep tillage to prevent root injury
- Provision of adequate soil moisture through irrigation or conserve moisture by coconut husk burial during summer
- Flood irrigation should be avoided. (follow basin or drip irrigation)
- Provision of good drainage system
- Intercropping with crops like banana, turmeric etc.,
- Apply FYM or green leaf manure 50kg/palm/year to increase soil organic content.
- Balanced application of fertilizers.
- Soil application of neem cake @ 5 kg / palm /year.
- Apply 200g phosphobacteria+200 g *Azotobacter* / palm / year
- Soil application of *Trichoderma viride* 200g + *P. florescence* 200g fortified in neem cake /compost / vermicompost / other organic wastes / palm/year
- Drench the basin with 40 litres of 1% Bordeaux mixture or 0.1% calixin after soaking soil, at quarterly interval for one year
- Root feeding of 2 g Aureofungin-sol + 1g of Copper sulphate or Calixin 2ml or hexaconazole 2 ml in 100 ml water at quarterly interval
- Avoid growing leguminous crops in and around the garden

Stem bleeding: *Thielaviopsis paradoxa - Conidial stage* (imperfect) *Ceratocystis paradoxa - Ascigerous stage* (perfect)

The disease is mostly found in Thanjavur and Kanyakumari districts of TN and Northern Kerala.

Symptoms

- Dark brown to black fluid exudes through the stem cracks on the basal part of the trunk
- On drying the fluid forms black encrustation with brownish orange margin (Plate38)
- Bleeding patches progress both upwards and downwards and cover major portion / part of the trunk
- Lesions / patches coalesce to form larger patches
- Tissues beneath the bleeding points decay and become yellowish
- Except vascular bundles the other tissues are disintegrated
- Leaf size reduces leading to reduced crown size
- The rate of leaf production slows down
- Bunch production is reduced
- Nuts and buttons shed heavily
- In advanced stages uniform yellowing and dropping of leaves leads to complete death of the palm

Causal Organism: *Thielaviopsis paradoxa - Conidial stage* (imperfect) *Ceratocystis paradoxa - Ascigerous stage* (perfect)

Wound parasite

The fungus is airborne.

Epidemiology

Growth cracks on the trunk, sudden heavy manuring or heavy rains followed by extensive dry period, poor drainage, soil moisture stress, hard pan formation in the soil, imbalance in nutrition, excessive soil salinity, stem injury, lightning attack, insect attack etc. act as predisposing / aggravating factors of this disease

Management:

- Chisel out the affected tissues completely and paint the wound with Calixin 5% (5ml/100ml water)
- Apply coal tar or rubber kote after 1-2 days (Plate42)
- Burn the chiseled out material
- Avoid any mechanical injury to trunk
- Apply neem cake 5 kg per palm in the basin along with other manures and fertilizers

- Root feeding with Calixin 5% (5ml/100 ml water) during September-October,January-February and April-May (Plate43)
- Drench the wet basin with 25 ml of Calixin in 25 litres of water once in 3-4 months
- Trail pepper on coconut to prevent direct exposure of cracks to pathogen
- Irrigate basins regularly during summer months and conserve moisture by coconut husk burial
- Adequate manuring to increase the vigour of the palms
- Apply normal dose of organic manures (50 kg/palm/year) and balanced fertilizers

Grey blight: *Pestalotiopsis palmarum*

It is one of the widely distributed diseases. It reduces considerably the vitality of the palm. The disease is particularly severe in Kerala and Tamil Nadu. Palms of 20-40 year old are highly susceptible

Symptoms:

- Appears on the leaflets of mature leaves of the outer whorls of palm
- Yellowish brown oval spots with brown margins appear on leaflets
- The centre of the spots become greyish white, with dark brown margin
- Many spots coalesce to form large irregular necrotic patches causing extensive blight
- The acervuli of the fungus appears as black minute dots / specks on the lesions
- In advanced stages, the tips and margins of the leaflets dry and shrivel giving a burnt appearance
- Premature shedding of leaves and reduction in the number of leaves prolong the pre bearing age of young palms

Causal Organism: *Pestalotiopsis palmarum*

Mode of spread

Spread is through air borne conidia.

Epidemiology

Disease intensity is severe during rainy season with low temperature and high humidity.

Poor soil nutritional status, especially potash deficiency and poor drainage increases the disease incidence. Neglected gardens are severely affected.

Management:

- Recommended agronomic practices should be followed
- Good drainage facilities.
- Remove severely affected older leaves and burn
- Spray the crown with 1% Bordeaux mixture or 0.3% tilt (3ml/L) or COC or Dithane M 45
- Apply higher dose of potash (25% extra) along with recommended dose of NPK

- Apply increased dose of farmyard manure and composted coir pith (@100 kg/palm/year)
- CGD, COD and their hybrids are tolerant

Root Wilt: *Phytoplasma*

The disease was first reported following the heavy floods of 1882 in Kerala. Now the disease has spreads to almost all coconut growing areas of Kerala and parts of adjoining of TN especially in Coimbatore, Tirunelveli and Kanyakumari districts. Also called as Kerala wilt disease. It is non lethal but debilitating.

Symptoms

- The disease is not a fatal one but a slow decline disease causing drastic yield reduction
- The characteristic and earliest diagnostic symptom is the abnormal inward bending of leaflets termed as ribbing or flaccidity resembling the ribs of mammals (Plate1)
- Flaccidity of leaves of the central and outer whorls
- Whole frond develops a cup like appearance (Plate2)
- Foliar yellowing and marginal necrosis of leaflets of the outer whorls of leaves (Plate3)
- Leaves produced subsequently become smaller and thinner
- Paling of the younger leaves and stunting of the crown due to the reduction in number and size of the leaves
- Deterioration and decay of roots and rootlets from tip backwards and reduction in regeneration of fresh roots
- Root cortex turn brown and dries up in flakes
- Superimposing of leaf rot disease root (wilt) diseased palms
- Drying up of the spathes and necrosis of spikelets from tip downward in unopened inflorescence
- Inflorescence necrosis, production of little or no female flowers, pollen sterility and shedding of immature nuts and buttons
- Reduction in size and number of nuts
- Production of poor quality nut / copra, thinner husk, less firm shell, weaker fibres, uneven thickness of kernel
- Thinner kernel never dries up into copra but remains soft and flexible with very less oil content
- Insipid tender coconut water

  Causal organism: Phytoplasma

  Vectors – Lace wing bug – *Stephanitis typicus*

Plant hopper - *Proutista moesta*

Epidemiology

- Occurs in all major soil types but the spread is faster in sandy, sandy loam, alluvial and heavy textured soils. Laterite soils have lower incidence and intensity of the disease.
- Higher incidence in water logged low lying areas adjacent to rivers and canals and also in neglected gardens

Management

Efficient management of root (wilt) affected palms demand control of all pests and diseases, imparting natural resistance and health to the palms through proper manuring and agronomic practices. A package of management practices for the effective management of root (wilt) disease is given below

- Total eradication of all the disease affected palms in mildly affected areas of Northern Kerala
- In heavily disease affected tracts, rougue out all severely affected uneconomic adult palms yielding less than 10 nuts per year and all diseased palms in the pre-bearing age
- Replant with disease tolerant like CGD varieties or high yielding hybrids like Chandrasankara and CGD X WCT
- Apply balanced dose of NPK fertilizers in the form of urea, rock phosphate and potash

  Average management : 0.34 kg N, 0.17 kg P_2O_5 and 0.68 kg K_2O per palm per year

  Good management : 0.50 kg N, 0.32 kg P_2O_5 and 1.20 kg K_2O per palm per year
- Add organic manures (cattle or green manure) @ 50 kg per palm per year
- Lime : 1 kg per palm per year and MgO (Magnesite Magnesium source) : 100g (In Onattukara region: 500g)
- Grow green manure crops like sunhemp, sesbania, cowpea, calapagonium etc in the coconut basin and incorporate in situ
- Adopt mixed farming by raising fodder crops in the interspaces and maintaining milch cows
- Mixed cropping with cocoa and intercropping with tapioca, yams and elephant foot yam
- Under rainfed conditions apply fertilizers in 2 splits, 1/3 at the time of south-west monsoon and 2/3rd before the north-east monsoon. Under irrigated conditions apply fertilizers in three equal splits during April-May, August-September and December-January

- Apply fertilizers and manures in 10 cm deep circular basins at a radius of 2m from the bole of the palm
- Desilt the channel and strengthen the bunds during summer months when the crop is grown under the bund and channel system
- Adopt strictly all the prescribed prophylactic measures against leaf rot disease, red palm weevil, rhinoceros beetle etc.
- Initial stage of the disease, spray the crown and drench the basin with 500 ppm tetracycline

Leaf Rot: Fungal complex predominantly by *Colletotrichum gloeosporioides, Exserohilum rostratum and Fusarium spp.*

Symptoms

- Affects palms of all ages, especially palms below 25 years
- Water soaked lesions with different shape and colours on the emerging spindle and young tender leaves (Plate7)
- Dark brown spots develop on the tender leaves which later enlarge and dry up as the leaf emerges (Plate8) and leaflets will not open fully (Plate9)
- Successive central shoots are affected, resulting in rotting of all the leaves on the crown
- These lesions enlarge, coalesce leading to extensive rotting of spindle leaves (Plate10)
- Extension of rotting into the interior of spindle resulting in decay and sometime rotting of buds
- Tips of leaflets and midribs often become blackish, shriveled and fall off in wind, giving a 'fan' like or 'arrow leaf' appearance to the leaves (Plate11)
- Successive infection of the emerging spindles results in appearance of symptoms in most of the leaves of the crown
- Extensive lesions and their coalescing results in severe blighting of lamina (Plate12)
- Breaking of ends of leaves which subsequently become yellow and eventually hang, dry and fall of

Causal Organism: Fungal complex predominantly by *Colletotrichum gloeosporioides, Exserohilum rostratum and Fusarium spp.*

Mode ofSpread

Primary spread through air-borne conidia.

Epidemiology

Free water, raindrops, high relative humidity and low temperature especially during monsoon season are highly favaourable. The prevalence of leaf rot due

to *C. gloeosporioides* is maximum during monsoon months where as the incidence due to *E. rostratum* is maximum during winter season

Management

- Remove and destroy severely affected / senile and uneconomic palms
- Replant with healthy seedlings from elite palms and hybrid varieties tolerant to the leaf rot - root (wilt) disease complex. CGD and hybrids of CGD X WCT are tolerant under field conditions
- Adopt all recommended practices viz agronomic, cultural, plant protection etc as applicable to root (wilt) disease endemic region
- Clean the crown in general during pre-monsoon period.
- Remove and destroy the rotten portions from the spear leaf and the 2-3 adjacent leaves
- Pour 300 -1000 ml of fungicidal solution of Hexaconazol (Contaf 5E) - 2ml or 3g mancozeb in 300ml water at the base of spear
- Spray crowns and leaves with 1% Bordeaux mixture or 0.5% Copper oxychloride formulation (5g/L) or 0.4% Mancozeb (4g/L) in January, April-May and September. While spraying, care should be taken to spray the spindle leaf and 2-3 adjacent leaves

Mahali / Fruit Rot / Nut Fall: *Phytophthora palmivora* and *Phytophthora katsurae*

Symptoms

- Female flowers and immature nuts rot and shed especially during rainy season
- Nuts of 2-5 months old are highly susceptible
- Water soaked dark green lesions appear, usually near the fruit stalk of immature nuts, which later turn brown and become irregular in shape (Plate19)
- Lesions spread on the husk and endosperm resulting in their discolouration and rotting
- Nuts fall prematurely (Plate20)
- Fluffy white mycelial growth forms on the fallen nuts or buttons (Plate21, 22)
- Water soaked lesions on the inflorescence and rachis result in rotting and drying of inflorescence and rachis

Causal Organism: *Phytophthora palmivora* and *Phytophthora katsurae*

Spread of Disease

The fungus survives as oospores, chlamydospores and mycelium in the soil, frond base or basal part of the crown.

Epidemiology

Heavy rainfall, high relative humidity of 94-100% and temperature below 24oC are highly favourable for the disease spread. Dissemination of the disease is primarily by wind and wind blown rain and also by contact between bunches, dripping of rain water from the diseased to healthy bunches and also through insects

Management

- Spray 1% Bordeaux mixture or copper oxychloride (5g/L) on the bunches and crown during monsoon and subsequently twice at 40 days interval along with stickers as prophylatic measure in disease prone area
- Collect and destroy the fallen nuts
- In disease prone areas, adopt proper spacing of palms
- Provide proper drainage
- Regular manuring and proper cultural practices reduce the disease incidence

Anthracnose and Leaf Blight: *Colletotrichum gloeosporioides*

Symptoms

- Affect palms of all ages especially on outer whorls of mature leaves
- Burnt or dried appearance of outer whorls of leaves
- Reddish brown oval to irregular sunken lesions with dark brown margin on leaves
- Salmon or reddish brown fruitification of the fungus as dots on the affected leaves
- Lesions coalesce resulting in leaf blight
- Disease progress to mid whorl of leaves causing premature drying and falling of leaves

Causal Organism: *Colletotrichum gloeosporioides*

The disease is air borne

Management

- Remove and destroy severely affected leaves
- Spray 1% Bordeaux mixture or Mancozeb 0.4% (4g/L) on outer and mid whorls of leaves at 45 days intervals
- In severe case, spray 0.3% (3ml/L) tilt / contaf at monthly interval

Mid Whorl Yellowing / Quick Yellow Declining: *Phytoplasma*

Symptoms

- A fatal disease caused by Phytoplasma
- Affects palms of all ages and spread rapidly
- Affected palms die within months (4-10 months)
- Lower and mid whorl leaves exhibit yellowing which subsequently spreads to inner whorls leaving the spindle leaf (Plate44)
- The whole foliage become yellow and necrotic within months (Plate45)
- Developing and opened inflorescence, rachis and flowers become black

- ❖ Female flowers are severely reduced
- ❖ All nuts fall prematurely
- ❖ Basal regions of leaves rot resulting in drooping and falling of leaves in quick succession
- ❖ Sometimes leaves show bronzing and fall off in quick succession without hanging around
- Spear leaves and growing point rots resulting in the death of the palms

  Causal Organism: ***Phytoplasma***

  Vector: Plant hoppers – *Myndus crudus*

  Management
- Remove and destroy diseased palms
- Grow resistant variety – Malayan Yellow Dwarf.
- Adopt management followed for Root (wilt) disease

Inflorescence Blight and Nut Fall: ***Colletotrichum gloeosporioides, Gloeosporium spp.***

Symptoms

- ❖ Serious disease after monsoon and during summer
- ❖ Sunken black or dark brown lesions on rachis
- ❖ Lesions coalesce resulting in drying of rachis from tip downward
- ❖ Inflorescence dries extensively resulting in severe button shedding and immature nut fall
- ❖ Sunken dark grey, brown or black lesions with black to dark brown margins develop anywhere on the soft portion of immature nuts
- ❖ Lesions enlarge to irregular form and coalesce to cover larger parts of the nuts
- ❖ Dark brown or salmon coloured fruiting bodies of the fungus appear on affected parts
- ❖ Longitudinal ruptures develop on the affected fruits, allowing the fungus and other organisms to enter and causing rotting of mesocarp, shell and endosperm

  Causal Organism: ***Colletotrichum gloeosporioides, Gloeosporium spp.***

  Fungus is air borne
- ❖ Remove and destroy dried inflorescence and fallen buttons and nuts
- ❖ Spray 1% Bordeaux mixture or Mancozeb 0.4% (4g/L) on inflorescence and also on developing buttons and nuts as prophylactic measure at 45 days interval

Botryodiplodia Nut Fall: ***Botryodiplodia theobromae*** **(*Lasiodiplodia theobromae*)**

Symptoms

- ❖ Occurs during summer months and affects nuts of all stages
- ❖ Disease appears as black extensive patches at the stalk end of the fruit
- ❖ Later these black patches progresses to cover the major portion or entire nuts without definite margins.

- ❖ Black fruiting bodies of the fungus seen as dots on affected nuts and rotting of nuts
- ❖ Nuts rot and shed prematurely

Causal Organism: *Botryodiplodia theobromae* (*Lasiodiplodia theobromae*)

Airborne

- ❖ Remove and destroy dried inflorescence, fallen button and nuts
- ❖ Spray 1% Bordeaux mixture or Mancozeb 0.4% (4g/L) on inflorescence and also on developing buttons and nuts as prophylactic measure at 45 days interval
- ❖ In diseased gardens, spray the inflorescence and nuts with 0.1% Calixin / Contaf / Tilt after harvesting the mature nuts

Tatipaka Disease

Symptoms

- ❖ First appeared in Tatipaka village of East Godavari district of Andhra Pradesh, following a cyclone in 1949.
- ❖ Palms in the age group of 25 to 60 years are more susceptible.
- ❖ Development of an abnormally large crown with dark green inner leaves and higher yield is the precursor of disease incidence. Subsequently the crown becomes smaller in size producing progressively shorter leaves.
- ❖ The stem begins to taper. The leaves give a fascinated appearance due to improper unfolding of leaflets. The affected tree produces smaller bunches with atrophied barren nuts.
- ❖ The causal agent is suspected to be Phytoplasma.

Differences between Basal stem rot, Stem bleeding and Root wilt diseases.

Description	**Basal Stem Rot / Thanjavur Wilt / Foot Rot / Ganoderma Wilt / Anabe Roga**	**Stem bleeding**	**Root Wilt**
Bleeding symptoms	Seen at the base or collar region. Sometime may not be seen externally.	Seen at the base or any part of the trunk.	Not seen.
	(Reddish brown viscus fluid)	Brownishto black, non viscus fluid)	--
Internal decay	Present upto the central core	Confined to bark tissues.	--

Maximum height of bleeding symptoms	Upto 4-5m	Upto trunk apex	--
Root decay	Extensive root decay present	Not present	Present
Brackets of *Ganoderma*	Produced in the advanced stage of the disease or after the death of the palms	Not produced	Not produced
Crown symptoms	Yellowing, drooping and drying of leaves one after another from outer whorl	Only reduction of crown size and tapering of crown at advanced stages.	Yellowing, Flacidity and inward bending of leaflets. No. & size of leaflets reduced.
Rib like leaf lets	Absent	Absent	Present
Death	Within 6 to 24 months	Longer period	Longer period

DISEASES OF ARECANUT

Mahali/Fruit Rot/Koleroga: *Phytophthora meadii* (major species), *P. arecae, P.heveae*

- This dreaded disease occurs in all the arecanut growing regions receiving heavy rainfall
- The crop loss varies from 10-90%
- Characteristic symptom is rotting and extensive shedding of the immature nuts during south west monsoon
- The first symptom appears as dark green/yellowish water soaked lesions on the nut surface usually near the soft inner perianth region and infected nuts lose its natural green luster
- The lesions gradually spread covering the entire nuts (before or after shedding) which consequently rot and shed
- A felt off white mycelial mass envelopes on entire surface of the fallen nuts
- As the disease advances the fruit stalks and the axis of the inflorescence rot and dry, sometimes being covered with white mycelial mats
- Infected nuts are lighter in weight and possess large vacuoles
- Dark brown radial strands on kernel make them unfit for chewing

- ❖ Infection occurring later in the season results in rotting and drying up of nuts without shedding called 'Dry Mahali'

Causal Organism: *Phytophthora meadii* (major species), *P. arecae, P.heveae*

Mode of Spread and survival

- ❖ The fungus survives as oospores, chlamydospores and mycelium in soil, on fallen nuts, on dried nuts and inflorescence remaining in the crown. The spread of the disease is by wind and windblown rain

Epidemiology

- ❖ Mahali is weather dependent and seen during monsoon season from June-October
- ❖ Continuous heavy rainfall with intermittent bright sunshine hours, low temperature of 20-23^0 C, constant high relative humidity of more than 90%, wind, cloudy weather, plantations situated in valley surrounded by rubber plantations etc. favours the outbreak of the disease

Management

- Prophylactic spray of 1% Bordeaux mixture with stickers once before the onset of south west monsoon followed by second and third applications at 40-45 days interval
- Cover bunches with polythene sheets before monsoon rains
- Collect and destroy all fallen and infected nuts
- Remove and destroy all completely affected inflorescence and immature bunches

Bud Rot or Crown Rot: *Phytophthora meadii, P.heveae, P. arecae*

- ❖ Occurs independently (bud rot) or following severe fruit rot infection (crown rot)
- ❖ Spindle leaf shows discolouration from the natural light green colour to yellow and then to brown followed by withering
- ❖ Infection spreads inside the bud causing rotting of growing bud and the surrounding young leaves
- ❖ The spindle slumps/droops and can be drawn out with a gentle pull
- ❖ Infection spreads to the adjacent leaves, which becomes yellow, droops and drop off one by one leaving a bare stem
- ❖ Secondary organisms colonize the infected tissues and converts it into a slimy mass emitting a foul smell
- ❖ Severe fruit rot infection, especially on bunch and inflorescence stalks, which spreads to the stem and outer leaf sheaths
- ❖ Inner portion of the leaf sheath exhibits water soaked lesions and base of leaf sheath rots resulting in yellowing of outer leaves
- ❖ Disease spreads to subsequent upper leaf sheath base resulting in their rotting and yellowing of middle whorls of leaves

- ❖ Tender portion of the stem and growing bud becomes infected resulting in rotting of the internal tissues of the crown and finally death of the palm

Causal Organism: ***Phytophthora meadii, P.heveae, P. arecae***

Spread of Disease

- ❖ The fungus survives as oospores, chlamydospores and mycelium in soil, on fallen nuts and on dried nuts and inflorescence remaining on the crown. The spread of the disease is by wind and windblown rain

Epidemiology

- Continuous heavy rainfall with intermittent bright sunshine hours, low temperature of 20-23^0 C, constant high relative humidity more than 90%, wind, cloudy weather, plantations situated in valley surrounded by rubber plantations etc. favours for the outbreak of the disease

Management

- ❖ Remove and destroy dead palms, fruit rot affected dried bunches, fallen nuts and crowns of palms affected by bud/crown rot
- ❖ When the spindle leaf starts withering, remove and destroy all affected tissues of the spindle along with healthy tissues
- ❖ Apply Bordeaux paste and protect it by covering with a polythene cover or a mud pot
- ❖ Spray drench the spindle leaf base and the base of the other leaves with 1% Bordeaux mixture

Basal Stem Rot/Foot Rot/Anabe Roga/Ganoderma Wilt: ***Ganoderma lucidum*** **(frequent),** ***G. applanatum*** **(associated)**

- ❖ A fatal disease affecting the palm in the age group of 5-10 years
- ❖ Destroy 5-8 % of the palms in neglected and water logged gardens
- ❖ The diseased palm cannot be identified in the initial stages of the disease
- ❖ The palm in the advanced stage of infection shows symptoms on the crown, stem and root system
- ❖ Leaflets of the outer whorls of the leaves develop yellowish-orange discolouration which gradually spreads to the inner whorls of leaves
- ❖ The pale discolouration spreads to the whole leaf and the entire crown becomes progressively yellow with the dried outer whorl of leaves drooping down and covering the stem
- ❖ Later the inner whorl of leaves also becomes yellow leaving only the spear leaf green
- ❖ Development of inflorescence and nuts is arrested
- ❖ In the advanced stages, spindle gets dried up and the crown topples down leaving the bare stem

- Stem tapers, internodal length is reduced and its internal tissues are completely damaged
- The infected brittle stem breaks off easily during heavy wind
- The base of the stem up to 1m height shows brown disolouration and oozing of a dark fluid
- Bracket shaped fruitification of the fungus develops at the base of the trunk, usually after the death of the palm, or on the stump or on the live palm
- Roots exhibit varying degrees of discolouration and rotting, which become brittle and dry with a musty smell. Uptake of water and nutrients is affected

Causal Organism: *Ganoderma lucidum* (frequent), *G. applanatum* (associated)

Spread of Disease

- The disease is severe in neglected, ill-drained and overcrowded gardens especially with hard, black loamy acid soils of higher iron and calcium contents
- Soil borne, but secondary spread is through air borne spores

Management

- Remove and destroy all severely affected palms and stumps of dead palms, including roots
- Drench the soils with 1% Bordeaux mixture before planting healthy seedlings
- Isolate affected palms by digging deep trenches all around, one metre from the base and drench with 0.1% Calixin / Contaf (1ml/L). Minimum of 10 litres of fungicide solution is required for a bearing palm
- Improve drainage
- Avoid dense planting
- Avoid flood irrigation and water flowing from infected palms to healthy palms
- Avoid repeated ploughing and digging in the diseased gardens
- Balanced manuring and fertilizer application
- Apply 2 kg neem cake/palm/year
- Discourage growing of collateral hosts of the fungus, such as *Delonix* regia and*Pongamia glabra* in the vicinity of gardens
- As a prophylactic measure, apply biocontrol agents *Trichoderma harzianum* fortified in organic manures into the soil
- Root feeding of palms with 100 ml of 2.0 % Calixin (20ml/L) through 2-3 roots in the early stage of infection at quarterly intervals

Stem Bleeding: *Thielaviopsis paradoxa* (*Ceratocystis paradoxa*)

- Young palms of 10-15 years are more susceptible but older palms are rarely infected
- Small discoloured depressions or spots on the basal portions of the stem
- Spots coalesce to form larger patches followed by cracking

- Fibrous layers of the stem disintegrate which eventually hollows up to varying depths along the infected portions
- Dark brown gummy exudates ooze out from cracks
- Stem tapers near the crown
- Crown size and yield is reduced
- Severely affected palms die off

Causal Organism: *Thielaviopsis paradoxa* (*Ceratocystis paradoxa*)

Spread of Disease

- The disease is severe in neglected gardens with poor drainage. Higher water table predisposes the palms to this disease

Management

- Improve drainage
- Balanced manuring/fertilizer application
- Scrap off the affected portion of tissue and smear with hot coal tar or rubber kote mixed with Calixin @ 5ml/kg or Bordeaux paste
- Swab 5% Calixin (5 ml/100 ml) after the removal of the infected portion along with healthy tissues and drench soil with 0.1% Calixin or root feeding of 100 ml of Calixin 2.0% (20ml/L) through 2-3 roots
- Trailing pepper

Yellow Leaf Disease; *Phytoplasma*

- Also known as *Kattuveezhcha* / *Chandiroga* / *Arasiroga*
- Most serious disease of arecanut and is present in all districts of Kerala in moderate to severe form
- The yield loss is as high as 50% over a period of three years immediately following the disease incidence
- Affects palms of all age groups (Plate1,2)

Symptoms

- Symptoms appear as translucent spots of 1-3 mm diameter and characteristic yellowing of the tips of leaflets in two or three leaves of outermost whorls. (Plate3)
- Yellowing gradually extends to the middle of the lamina showing a clear cut demarcation of yellow and green parallel bands on both sides of the midrib of leaflets (Plate4)
- Progressive drying up of tips of the chlorotic leaves
- As the disease progresses the yellowing extends to the whole lamina, leaving only the leaf stalk green

- ❖ Sometime one or two leaflets in any part of the crown or the entire foliage may be affected in the initial stage
- ❖ In the advanced stage, leaves are reduced in size, stiff, pointed, closely bunched rosetted and abnormally puckered
- ❖ Internodal length is reduced, stems tapers and inflorescence not produced (Plate5)
- ❖ Mature and immature nuts shed in large numbers, endosperm becomes soft and black
- ❖ The crown falls off leaving a bare trunk
- ❖ Varying degrees of rotting on roots
- ❖ Lateral root production reduced
- ❖ Tips of absorbing young roots blacken and rot, especially in water logged conditions.
- ❖ Roots exhibit vascular discolouration

Causal Organism: *Phytoplasma*

Vector: Planthopper (*Proutista moesta*)

Managent

- ❖ Apply recommended dose of fertilizers
- ❖ Add extra 160 g of rock phosphate to increase new root formation
- ❖ Apply lime at the rate of 500 g per palm once in 2-3 years during June-July
- ❖ Apply organic manure @ 12 kg each of compost and green leaves per palm
- ❖ Provide irrigation during summer months
- ❖ Avoid water stagnation in the garden by providing drainage facilities
- ❖ Grow cover crops in the garden and apply in situ
- ❖ Remove infected palms
- ❖ Replant the affected gardens with high yielding local varieties like Kasargod local, Dakshin Kannada local, Sirsi local and also with dwarf varieties like Hirahalli dwarf and Mohit nagar
- ❖ Apply 100 g Magnesite and 100g of micronutrient per palm per year
- ❖ Spray Rogor/Dimethoate 30 EC (1.5 ml/L) to control insect vectors

Inflorescence Die-Back and Button Shedding: *Colletotrichum gloeosporioides*

- Wide spread in areca nut plantations often resulting in heavy yield loss
- The disease is seen throughout the year, but becomes severe during summer months from February to May
- Yellowing of rachillae of inflorescence
- Yellowing progresses from the tip of the rachillae towards the main rachis

- As the disease progress, the entire rachillae turn dark brown causing wilting and drying of inflorescence (inflorescence die back)
- Shedding of female flowers (buttons) in large numbers
- Discolouration of the inner soft calyx region of the female flowers and buttons results in their shriveling, drying and shedding
- Concentric rings of light pink coloured mass of spores appear on the discoloured infected inflorescence
- On severe infection the female flowers and buttons shed completely or dries up and remains on the crown

Causal Organism: *Colletotrichum gloeosporioides*

Spread of Disease

- ❖ Spread is through air borne conidia

Management

- ❖ Remove and destroy completely dried inflorescence, buttons and all fallen nuts
- ❖ Spray 0.4% Zineb or Mancozeb (4g/L) twice, first one just after the setting of female flowers and then again at 3-4 weeks interval as prophylactic measure. Bordeaux mixture 1% and 0.3% Captan (3g/L) are also very effective
- ❖ In diseased gardens, spray 0.1% contaf/tilt/topas/bavistin or 50 ppm(50mg/L) of aureofungin sol

Leaf Rot: *Colletotrichum gloeosporioides*

- Seedling and young bearing palms are more susceptible
- Water soaked lesions on margins and tips of young leaves and emerging spindle leaf
- Lesions develop into dark brown sunken spots which enlarges and dries up due to extensive rotting
- Dried/shrivelled tips and margins of leaflets shred off leaving only the mid-veins
- On severe infection, lesions develop on mid-veins, midrib and even to the growing spindle leaf resulting in rotting of spindle leaves and growing bud

Causal Organism: *Colletotrichum gloeosporioides*

Spread of Disease

- ❖ Air borne disease, spreading through conidia

Management

- ❖ Remove and destroy affected portion of the leaves
- ❖ Spray 0.4% Mancozeb(4g/L)/1% Bordeaux mixture on spindle leaf and inner whorls of leaves and also spray drench the axil of inner whorls of leaves with either of the fungicides
- ❖ In severe case, spray the crown with 0.1% Contaf / Tilt(1ml/L) / bavistin (1g/L) at fortnightly intervals

Leaf Blight/Grey Blight: *Pestalotiopsis palmaraum* and *Phomopsis palmicola*

- Seedlings and young palms are affected, especially during summer months
- Characteristic reddish brown or dark brown discoloured spots on mature outer whorls of the leaves
- Spots enlarge to form irregular patches with brown margin and grey centre which have minute black dot like structures
- In advanced stages of infection, extensive blighting of leaves results in stunted growth

Causal Organism: *Pestalotiopsis palmaraum* and *Phomopsis palmicola*

Spread of Disease

- Spread of the disease is through air borne conidia

Management

- Remove and destroy severely affected older leaves
- Apply recommended dose of manures and fertilizers
- Provide shade to the nursery plants
- Spray 1% Bordeaux mixture or 0.4% Mancozeb (4g/L)

Leaf Spot/Anthracnose: *Colletotrichum gloeosporioides*

- Seedlings and palms of all ages are affected
- Outer whorls of the leaves are more susceptible
- Small yellow specks appear on the leaf lamina
- Specks enlarge to form irregular sunken lesions with dark brown margins and light brown centre having minute dot like structures
- Characteristic prominent yellow halo develops around the sunken lesions
- Lesions advance and coalesce to form larges patches resulting in premature yellowing, blighting, drying, drooping and shredding of leaves
- Severe infection causes stunted growth and death of the seedlings

Causal Organism: *Colletotrichum gloeosporioides*

Spread of Disease

- The fungus spread through air borne conidia

Management

- Remove and destroy severely affected leaves
- Provide shade to nursery plants
- Spray 1% Bordeaux mixture/0.4% Mancozeb (4g/L) at fortnightly intervals
- In severe cases, spray 0.1% contaf/tilt (1ml/L)/bavistin(1g/L) at fortnightly intervals

Bacterial Leaf streak: *Xanthomonas campestris* pv *arecae*

- Dark green, water – soaked, translucent limear lesions or stripes produced paralled to mid of the leaflets.

- Later the lesions are covered with abundant creamy white and slimy bacterial exudates on the lower surface.
- On drying the exudates forms a waxy film or Creamy white to yellowish falkes.

DISEASES OF TEA

1. Blister blight: *Exobasidium vexans* Massee

This disease was first reported from Assam in 1868. Since then it spreads to most other tea growing states of North and South India. It also occurs in Burma, Indonesia, Japan, Malaysia, Singapore, Sri Lanka and Taiwan. Since 1946 it has become severe year after year in South India causing heavy damage to the industry.

Blister blight produces losses up to 50%.

Losses due to blister blight extend up to 43% on annual crop basis.

Heavy attack can result in death of the whole plant. Even with fungicide control 11.3% yield loss occur in Sri Lanka, in the peak attack at the altitudinal range of 1500m - <1800 m (FUCHS, 1989).

Symptoms:

- It is a disease of both nursery and planted crop.
- In nursery, seedlings are stunted and produce many thin stems instead of a single stalk.
- Repeated attacks cause death of seedlings.
- Succulent leaves and green shoots of newly pruned tea are highly susceptible
- The fungus attacks the first flush of 2-3 young leaves and kills the young shoots and buds. Thus the new growth is largely ruined.
- Mature leaf is not affected.
- The first symptom is the appearance of small, circular, oily, pale or pinkish, translucent spots on the tender leaves
- These spots enlarge in size in due course upto an inch in diameter.
- Later the spots turn into deep red shiny blisters.
- The spots become depressed into a shallow cavity on the upper surface of the leaves.
- Correspondingly the under surface of the leaf bulges as a concave trough-like depression forming a classic blister – like swelling.
- The lower bulged surface is covered with white fungal growth.
- Leaves become curled and distorted.
- Later, the blisters turn to dark brown and shrink to flattened patch.
- The infection spreads to petiole and young succulent stem resulting in serious damage.

- On stem, spots are produced without blister.
- The fungus penetrates the stem.
- Stem infection leads to goose neck shape, dieback and snapping at the point of infection.
- The leaves and buds above the point of infection wilt and wither

Causal organism: *Exobasidium vexans* Massee

Mycelium – Septate inter & intracellular - 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.

Two kinds of spores – the conidia and the basidiospores are produced

Conidia – borne singly at the tips of long stalks. Hyaline elliptical, single celled or two celled (when mature), straight or slightly curved

Basidia are intermingled with conidial stalks and sterile hyphae. **Paraphyses** single, septate, apically rounded Basidia are long club shaped, clavate, 30 - 35 x 5 - 6 µm. generally bearing two sterigmata

Basidiospores are ovate to oblong, ellipsoid, hyaline initially unicellular, becomes 1-septate at maturity, 13-27 x 4.3-6.5 µm

Sporulation occurs after 10-19 days and spore discharge period extends upto 8 days. Number of spores ejected in 24 hours is 1.3 million/sq.cm and the pathogen completes its life cycle 11- 28 days.

Mode of spread and survival:

- Air borne
- Survives in the pre existing infected bushes

Epidemiology:

- Favourable conditions for infection are cloudy weather (monsoon months); continuous leaf wetness for 11-13 hours coupled with relative humidity >60% and temperature between 17 to 22°C.
- RH plays an important role in the epidemics.
- The disease is favoured by cool day and night with wet or humid conditions.
- More severe outbreaks occur under shade or adjacent to jungle or wind breaks or in damp low lying areas where mist persists.
- Cool moist, relatively still air favours infection.
- Moist and shade are therefore more conductive for the development of severe attacks.
- Temperatures > 24°C are fatal to the disease. Because of that periodical appearance and disappearance of the disease occurs.

Management:

- Removal of affected leaves and shoots by pruning and destruction
- Spraying Bordeaux mixture or Copper fungicides found to be effective.
- Copper fungicides are often used with nickel salts which have eradication effect.
- (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, Bayleton, Baycor offer good disease control.
- Chemical fungicides such as copper oxychloride as protectant (inhibits germination of spores), tridemorph (Calixin), hexaconazole (Contaf 5E) and propiconazole (Tilt 25EC) are recommended for blister blight control in both pruning and plucking fields.

Grey blight: *Pestalotiopsis theae*

It occurs both in North and South India.

In South India, it occurs in Karnataka, Kerala and Tamil Nadu.

Symptoms:

- The disease generally attack older leaves.
- The disease initiates as minute round brown spots.
- Soon the spots enlarge and turn grey with dark brown margin.
- The lesions become necrotic
- Fructification (acervuli) appears as minute black dots in the form of concentric rings.
- The fungus also attacks plucking points causing die back.
- If young leaves affected the leaves become blackened and unfolded.

Brown blight: *Glomerella cingulata*

Symptoms:

- Small, oval, pale yellow-green spots first appear on young leaves.
- Often the spots are surrounded by a narrow, yellow zone.
- As the spots grow and turn brown or gray, concentric rings with scattered, tiny black dots become visible
- Eventually the dried tissue falls, leading to defoliation.
- Leaves of any age can be affected.

Mode of spread and survival:

- Survives in the pre existing infected bushes
- Air borne conidia
- Pathogen gains entry through wounds

Epidemiology:

- The infection is predisposed by sun scorch insect plucking wounds.

- The incidence is more frequent on weak bushes especially if potassium is deficient
- More prevalence in shear harvesting fields during monsoon, stripping and wounds.

Management:

- Spraying of mancozeb at 0.3% or carbendazim or thiophanate methyl 0.05 % at 10-15 days interval

Black rot: *Corticium invisum, C. theae*

- Small, dark brown irregular spots appear on leaves.
- Spots coalesce to form dark brown patches covering the entire leaf lamia.
- Leaves and twigs turn brown.
- Dead leaves are hanging on thin threads from the branches
- The affected leaves drop off.
- Before the leaf turns black, the lower surface shows white powdery appearance.

Causal organism: *Corticium invisum, C. theae*

Mode of Spread:

- Air borne

Epidemiology:

- The disease develops rapidly when the temperature and humidity are high.
- Basidiospores germinate only in wet weather or when the leaf is covered with dew.

Management:

- Pruning in December
- Removal and destruction of affected debris
- Spraying copper oxychloride

Red Rust: *Cephaleuros mycoidea* and *Cephaleuros parasiticus*

The disease is widespread and imported in India, Africa, Sri Lanka and America.

Symptoms:

- On leaves the alga occurs sometime as parasite or some time as epiphytic.
- The alga occurs as orange yellow, roughly 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.
- Under favourable conditions the alga penetrates the leaf tissues.
- The penetration filaments are intercellular.
- They never penetrate host cells but get nourishment by osmosis.
- 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.

Causal organism: *Cephaleuros mycoidea* and *Cephaleuros parasiticus*

The cell contents of the alga orange red, hence the misapplication of the name rust.

Epidemiology:

- Poor soil drainage, imbalanced nutrition, and exposure to relatively high temperature and humidity predispose tea plants to infection
- Rainy season is more favourable for multiplication of alga

Management:

- Removal and destruction of affected plant parts
- Increasing the vigour of bushes by proper fertilizer applicaton
- Spraying with Bordeaux mixture 1%

Root diseases

Primary Root diseases

a. **Brown root disease: *Fomes noxius***

- Common in low elevation area
- Slow spreading and quick killing pathogen
- The roots are encrusted with a mass of earth and small stones cemented to the root by the mycelium
- Mycelium tawny brown resembling sambar skin
- 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
- Infection spreads mainly through root contact
- More common in sandy soils than claying soils/
- The disease originates from decaying stumps leaf out in soil.

b. **Black root disease: *Rosellinia arcuata***

- First identified root disease of tea
- It is a common disease on tea
- Occurs in India and Sri Lanka
- The fungus originates from the heaps of dead leaves.
- The fungal attack usually begins 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.
- The mycelium divides into number stands at the entry point which radiate over the surface of the wood forming a white star upto 1 cm diameter.
- A swollen ring of tissue is formed round the stem above and below the dead patch.

- ❖ Mycelium grows freely through surface soil and organic matter and spreads rapidly in damp weather.
- ❖ Removal of surface mulch around 10 meters
- ❖ Drenching the soil with Dithane M 45/Captan 30 g/10 litres of water.
- ❖ Avoid soil rehabilitation.

c. **Red root disease: *Poria hypolateritia***

- ❖ Fast spreading and slow killing pathogen
- ❖ It occurs in Assam and TN.
- ❖ The fungus is confined to underground parts.
- ❖ Mycelium white, later turns red, in advanced stages may appear black
- ❖ Interwoven with adhering soil; on washing soil goes off – blood red mycelium seen.
- ❖ When the bark is peeled off, characteristic, flat, black rhizomorphs are seen.
- ❖ These strands form branched markings on the surface of the wood.
- ❖ 'The roots show mottled appearance of red and white.
- ❖ The bark is softened.
- ❖ The wood may be discoloured as bluish black.

d. Xylaria root disease: *Xylaria* sp.

- ❖ Roots covered by black, ribbon like mycelial strands;
- ❖ Extensive necrosis of feeder roots;
- ❖ High casualty soon after drought.

SECONDARY ROOT DISEASES

a. ***Armelliella* root rot: *Armelliella mellea***

- ❖ Usually the disease becomes apparent after it has severely damaged the root system
- ❖ The foliages become chlorotic, wilted and droped off.
- ❖ Death of whole plants then follows.
- ❖ Sheets of creamy mycelium are seen beneath the bark along with flattened brown rhyzomorphs.
- ❖ In advanced stages sporophores are produced at the collar region.
- ❖ The sporophores occur in clumps and are pale brown and mushroom – shaped.
- ❖ Infected large root pieces provide main source infection.
- ❖ Rhyzomorphs can grow through soil and reach healthy hosts.

b. **Inter root disease: *Botryodiplodia theobromae***

- ❖ Commonly seen in low and mid-elevations area
- ❖ The disease appears 3 months after pruning.
- ❖ Weak appearance of frame
- ❖ Presence of unhealthy leaves,
- ❖ Failure of bushes to recover after pruning,

- Die back of new shoots
- Presence of white powdery spots with black centres on root surface
- The bark is rough and abnormally thickened,
- The fructifications are minute, black, and spherical embedded in the bark.

c. **Charcoal stump rot: *Ustulina zonata***

- The leaves wither turn brown and drop off.
- Sudden death of bushes
- White fan shaped mycelium on the surface of wood beneath the bark
- Charcoal like encrustation on bark seen in advanced stages

d. **Violet root rot: *Sphaerostilbe repens***

- Leaves turn yellow and droop, gradual death of bushes,
- Presence of enlarged lenticels on root bark;
- Roots become inky black/violet;
- Develop rancid odour- vinegar smell
- White colour mycelium, later turns to purple, seen on wood.
- Avoid planting in water logging areas
- Improved drainage controls

e. **Collar canker: *Phomopsis theae***

- Observed mostly in young tea
- Pathogen invades the stem through open wound.
- Chlorosis, cessation of growth, profuse flowering and canker on stem
- Predisposing factors are deep planting, planting in gravelly soils, mulching closer to collar, wound caused by weeding implements, fertilizer application close to the collar, pegging, low moisture status in bark and surface watering during dry weather.
- Preventive measures include avoid planting of susceptible clones in gravelly soils and drought prone areas, improving organic matter of marginal soils and using plants with good root system. Removal of affected portion by pruning to healthy wood and application of copper fungicide or spore suspension of biocontrol agents like *Trichoderma* and *Gliocladium* to cut ends are the curative measures.

Minor diseases

Pink disease: *Pellicularia salmonicolor*

- A number of silky thread unit to a thin film on stem and leaves.
- Young branches lose the leaves and die back.
- Bark killed in patches.
- The branches become irregularly swollen.
- The fungus forms pink fructification over affected f stem.
- 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.
- The disease is severe after heavy rains.

Thread blight: _Pellicularia koleroga_

- The disease is common in Assam and other parts of India.
- 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.

Twig die back/ Stem canker/Branch Canker: _Macrophoma theicola_

- Cancerous growth around the longitudinal wounds on the branches of tea bush.
- Control measures are to cut off the affected branches
- Spraying any of the systemic fungicides (Tridemorph, hexaconazole and/or calixin) at 0.5 % (50g in 10L) over the infected portions.

Leptothyrium die-back: _Leptothyrium theae_

- Common in higher altitudes
- Pathogen enters through the pruning cuts
- Dieback of shoots below the cut ends
- Infected branches snap off easily.
- Application of copper oxychloride & linseed oil paste on cut ends prevents the disease.

DISEASES OF COFFEE

Collar rot: _Rhizoctonia solani_

The disease occurs on 1-3 months old seedlings in nursery.

Prevalent in all coffee growing areas of India

Seedling loss uto 10-20%

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
- Removal and destruction of affected seedlings
- Seed treatment with carbendazim (1g/kg) carboxin (0.7g/kg)
- Soil drenching with carbendazim 0.05% or Mancozeb 0.05% or Captan 0.05%

Coffee Leaf rust / Oriental leaf disease / Coffee rust: *Hemileia vastatrix*

Coffee originates from high altitude regions of Ethiopia, Sudan and Kenya and the rust pathogen is believed to have originated from the same mountains. The earliest reports of the disease hail from the 1860s. It was reported first by a British explorer from regions of Kenya around **Lake Victoria in 1861** from where it is believed to have spread to Asia and the Americas. 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 his collaborator Christopher Edmund Broome after an analysis of specimens of a “coffee leaf disease” collected by George H.K. Thwaites in Ceylon. Berkeley and Broome named the fungus *Hemileia vastatrix*, *Hemileia* referring to the half smooth characteristic of the spores and *vastatrix* for the devastating nature of the disease.

It is unknown exactly how the rust reached Ceylon from Ethiopia but over the years that followed, the disease was recorded **in India in 1870**, Sumatra in 1876, Java in 1878, and the Philippines in 1889. During 1913 it crossed the African continent from Kenya to the Congo, where it was found in 1918, before spreading to West Africa, the Ivory Coast (1954), Liberia (1955), Nigeria (1962-63) and Angola (1966).

It is the most devastating disease of coffee. Found in every coffee growing region in the world. It has reached epidemic proportion on many occasions resulting heavy economic loss. The severe epidemic of the disease was first reported in Ceylon in 1867. In India it was first reported from **Karnataka in 1870**. In Sri Lanka, severe epidemic of the disease changed the growers to convert coffee plantations to tea or rubber cultivation. Coffee Leaf rust can cause yield losses in excess of 75% where outbreaks are severe. It causes 50 – 60% loss in India.

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 rust infects mainly leaves, but also young fruit and buds.
- The disease initiates as small yellow or blotchy, orange powdery pustules, 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.
- As the spots become older their centre becomes necrotic and the spores are confined to the outer areas.
- Several spots coalesce to form large necrotic patches
- Severely affected leaves shed prematurely.
- Vegetative growth is reduced.
- Die -back of the branches also occur.
- Premature ripening of berries and production of poor-quality, 'light coffee beans

Causal organism: *Hemileia vastatrix*

Spermogonia and aecia unknown.

Uredinia hypophyllous, densely scattered and giving a powdery appearance on yellowish-orange rounded blotches about 3-25 mm in diam., consisting of numerous narrow interwoven ‘feeder” hyphae and ± rounded cells below the stomata, bearing clavate filaments emerging through the stomata, whose tips bear numerous pedicels on which the spores are borne. The **urediniospores** are packed together like segments of an orange, giving each spore a kidney-like shape, curved with short 28-36 x 18-28 µm, wall hyaline,

strongly warted on the convex face, smooth on the straight or concave face, 1 µm thick (Laundon & Waterston 1964b). **Teliospores** are rarely found. **Telia** as the uredinia, hypophyllous; teliospores often produced in uredinia, sometimes in teliosori borne in cluster on short pedicels, 1-celled, more or less spherical to limoniform or spherical or napiform and smooth with aterminal papilla, wall hyaline to yellowish, smooth 16-25 × 19-22 µm, borne.

Although assumed to be heteroecious, the life cycle of *H. vastatrix* is not completely known. It is generally agreed that the rust does not complete its life cycle on the coffee tree, but no alternate host is known (Coutinho et al 1995).

Mode of spread and survival:

- The fungus survives in off season by uredinospores
- Dispersed primarily by wind and rain splash
- When the spores erupt, they enter the air current where they can travel a few centimeters to the next leaf, or hundreds of kilometers to another site (spores have been recorded travelling 1,000 m up in the high altitude air streams).
- There are also documented cases of spores being transported to new sites by small insects such as *Thrips* and parasitoid wasps.
- Spores may also be dispersed or carried from one farm to another by people, insects and animals.
- Not usually seed-borne but coffee berries and seeds may become contaminated by urediniospores that are then carried elsewhere.
- Telespores 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.
- Severe epidemic occurs during July – December.
- Disease incidence is reduced during January – March.
- The disease is also severe in shaded coffee due to high humidity and low light intensity.

Management

- Use of resistant varieties like Sln. 5B, Sln. 8 Sln. 9
- Wider spacing
- Shade managenet
- Pruning of dead and dying twigs after harvest

- Apply balanced nutrients to maintain plant vigour
- Diseased fallen leaves should be collected
- 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 contaf 400 ml or tilt 160 g or opus 42 ml in 200l water during August – September
- Biological control with *Verticillium hemileiae* during winter

Anthracnose: *Colletotrichum coffeanum*

The disease also known as die back or brown blight or black berry or Nilgiri twig disease

In India it occurs in Karnataka and Tamil Nadu.

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.
- Infection on the twigs causes wilting of young leaves exhibiting die back symptoms.
- Berries fail to mature forming kattekai.
- The root system decays and feeder roots die.

Causal organism: *Colletotrichum coffeanum*

Mode of spread and survival:

- The fungus survives on dead tissues on the bark which provides the major sources of infection.
- Conidia dispersed by rain splash.
- Also by birds machinery etc
- Coffee pickers are also a major source of dispersal

Epidemiology:

- Dry period followed by the monsoon rain.
- Inadequate overhead shade
- Prolonged drought and
- Soil water stress favour the disease development.

Management:

- Collection and destruction infected plant debris.
- Good drainage.
- Balanced fertilizer application
- Shade should be maintained.
- Leaf mulching at the base of the plants to conserve soil moisture during dry season

- Use of resistant varieties like Blue Mountain, Geisha, Rume, Sudan
- Spraying of Bordeaux mixture 1%

***Cercospora* leaf spot / Brown eye spot / Berry Blotch / Fruit spot: *Cercospora coffeicola* Berk. & Cooke**

- The disease is severe on nursery seedlings and young crop.
- Lesions begin as small, circular chlorotic spots on the upper leaf surface
- Spots expand to become necrotic with dark brown margin and tan, 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,"
- Necrotic spots increase in size and become irregular in shape and cause leaf blight.
- The margins of the lesions are dark brown to reddish brown or purplish to black in color.
- Affected leaves turn yellow and shed prematurely
- On green berries spots are initially brown, oval or irregular, 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.

  The affected berries shrivel, dry up and shed.

  Causal organism: *Cercospora coffeicola* Berk. & Cooke

  Stromata are slight to 50 µm in diameter, globular, and dark brown.

Conidiophores are in fascicles, 3–30 stalks, pale to medium brown, sometimes branched, multiseptate, mildly to abruptly geniculate, 20–275 x 4–6 µm. The conidial scars are distinct and thickened.

Conidia are hyaline, acicular to obclavate, nearly straight, needle-shaped with an acute apex and truncate or subtruncate base with a conspicuous, thickened hilum, indistinct multiseptate, 40–150 x 2–4 (–7) µm.

Mode of spread and survival

- Survive in debris for 35 weeks.
- Fallen leaves constitute a primary source of infection, especially at the end of the dry season.
- Pathogen dispersal is by spores (conidia) that are windborne (mostly during the daytime)
- Also spread by splashing rain and human contact

- Also seed borne

Epidemiology

- The pathogen sporulates readily under more humid conditions.
- Germination may occur between at temperatures between 8°C and 30°C, although 27°C is optimum.
- the development of brown eye spot is enhanced in plants already under stress
- The highest risk for infection occurs when the temperature range is 20–28°C and there are from 36 to 72 hours of continuous environmental wetness.
- Factors that predispose the plant to infection include

Management

- Maintain adequate plant nutrition In fact, the disease may be effectively prevented or controlled in most locations with a proper fertility regime.
- Sanitation and crop debris 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, root-knot nematodes, and root rot; these predispose coffee plants to infection.
- Provide or ensure adequate soil drainage.
- Grow coffee under shade (35–65%), or in an agroforestry setting.
- Avoid over-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.
- Harvest cherries on time, before disease progresses too far.
- Control weeds (this minimizes plant stress and relative humidity in the plant canopy).
- Avoid planting coffee transplants too deep in soils.
- Spraying of Bordeaux mixture 1% or Captan or Mancozeb 0.5% or Carbendazim 0.01% foltaf 0.4%.

Black rot / Koleroga / thread blight: *Koleroga noxia* Donk

It has been reported from many coffee growing countries including India.

In India it occurs in Karnataka and TN.

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.
- The affected leaves turn black and rot.
- On green berries blackening is seen as a narrow band.
- When the affected leaves and berries dried white fungal web can be seen.
- There will be defoliation and berry drop on the infected.

Causal organism: *Koleroga noxia* Donk

Basidiomycetes

Hyphae hyaline when young and turn light brown with age.

Basidia are simple, oval rounded or pyriform with sterigimata.

Basidiospore are hyaline elongated

Scelerotia are also produced hyaline at first and become brownish later scattered all over the diseased leaves, berries and young shoots.

Mode of spread and survival

- The pathogen spreads by contact form leaf to leaf through vegetative mycelium.
- Pathogen survives as scelerotia in infected plant debris.

Epidemiology

- Usually occurs during monsoon months with high humidity and hanging mist
- Heavy rains favour the disease.
- In south India the disease is severe only in *Coffea arabica*
- It is influenced by south west monsoon period from June – September
- The fungus infects Canthium sp., Crotons, Jasmine, Dahlia

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

Fusarium bark disease / Storey's bark disease: *Fusarium stilbioides*

It is important in South East, Asia, Southern Africa and West Indies.

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 .
- The fungus grows beneath the bark layer which becomes flaky in texture.
- 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.
- The affected suckers liable to break after a heavy crop.
- Under storeys bark type disease cinnamon to tawny olive 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.
- All tissues below the lesion and the cambium are killed.
- The foliage wilts and dies.

Causal organism: *Fusarium stilboides*

Mode of spread and survival:

- The fungus survives on dead debris.
- Infect the damaged coffee berries as secondary invader.

Epidemiology:

- Insect damage (Wood boring beetles) unfavourable cultural conditions like poor soil management, irregular pruning and drought predispose the plant to infection.
- **Management:**

Good soil management practices with adequate mulching for moisture conservation.

- Proper pruning
- Improving soil fertility
- Application of captan or Captafol 0.2% to trunk bases

Coffee Berry disease / Berry blight: *Colletotrichum coffeeanum* var*. virulans* and *Colletotrichum kahawae*

CBD can cause considerable yield losses of up to 75% when not adequately controlled.

Symptoms

- Coffee berry disease also affects ripening berries causing a brown blight phase which appears as dark, sunken anthracnose lesions on the red berry.
- The characteristic symptom of CBD is the development of small, water-soaked lesions on young, expanding berries
- They rapidly become dark brown or black and slightly sunken.
- They enlarge to cover the whole berry, which eventually rots.
- Under humid conditions, masses of pale pink spores become visible on the surface of lesions.
- The berries mummify and remain on the fruit branch
- These symptoms, collectively known as 'anthracnose', are typical of CBD.
- shedding of berries may also be seen.
- If infection occurs at a later stage of fruiting pale, corky scab like lesions may appear on young and mature berries.
- These lesions are known as 'scab' lesions

Causal organism: *Colletotrichum coffeeanum* var*. virulans* and *Colletotrichum kahawae*

Short conidiophores in acervali.

Conidia are small rod – shaped unicellular hyaline.

Setae either present or absent.

Mode of spread and survival

- The conidial spread is through rain splashes.
- The fungus survives on bark of the branches mummified berries and in lesions on affected berries.

Epidemiology:

- Infection takes place in temp. between 12 – 26^{0}C with moisture.
- The disease advances with season and the rainy period.
- Long rainy period with high rain fall are suitable for the development of the diseases.

Management:

- Use of res. Var. Blue Mountain
- Spraying of copper oxide

Sooty mould: *Capnoduim braziliense*

- The leaves and shoots are covered with black sooty fungal growth.
- Heavy attack of the aphids and scale insects predispose for infection.
- By controlling the insects the disease is controlled.
- 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.
- The fungus occurs in several stages.
- The most common stage is the corticum stage.
- 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.

It is often more serious at low altitude.

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.

Casual Organism: *Rosellina* spp.

Perithecia are produced

Conidia – ellipsoid or ovoid, single celled, hyaline to pale brown.

Mode of spread and survival

- Survives in infected plant debris in soil as primary source of inoculums

Epidemiology

- The disease is favoured moist and warm condition.
- Soil pH of 5.2 and soil moisture of 50 – 70% Soil MHC is most optimum.
- Host mortality is increased with size of inoculum

Management

- Removal of stumps and large roots of wild trees for establishing new coffee plantation
- Raising corn or other non host crops for two years before coffee planting
- Shade may be increased in some cases.
- Restacking of plant debris at particular interval to disturb fungal growth prevents the spread of the diseases.

- Removal and destruction of roots of affected trees, and the holes left open for a few months.
- Drench the soil with carbendazim 0.4 % or carboxin 75 WP 0.3% @ 3 liter/plant in the initial stage of wilting
- Application of biocontrol agent *Trichoderma* in affected blocks is useful in reducing disease incidence.

Brown root disease / Stump rot: *Fomes noxius* Corner

- Brown root disease also known as 'Stump Rot,'
- Mostly associated with rotting stumps of shade trees in the plantation.
- In India occurs in Karnataka and Tamil Nadu.
- 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.
- Inner portion of roots shows dark brown to black wavy lines of fungal hyphae.
- Spread through root contact.
- The roots become very brittle and readily snap off.
- Digging trenches of 60 cm deep and 30 cm wide.
- Affected plants should be uprooted and burnt.

Red Root rot disease: *Poria hypolateritia* Berk

- 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 after uptoory of inf. Plant.
- Whenever a shade tree is felled uproot the stump with root system to avoid disease spread infuture.

Santavery root disease: *Fusarium oxysporum* f. sp. *coffeae*

- Sudden wilting yellowing of leaves defoliation and death of aerial parts
- T.S of infected roots shows brown to pinkish discolouration .
- Scrapping of the bark of the stem near ground level also shows internal discolouration.

- Disease is soil borne.
- Soil drenching carbendazim 0.8% or Carboxin 0.4% @ 3-5 l / plant in the initial stage of infection

---

DISEASES OF RUBBER

Abnormal Leaf fall: *Phytophthora palmivora* Butl. *P. meadii* Mc Rae. *P.nicotianae var parasitica* and *P. botryose* (Chee)

Phytophthora infection occurs in several rubber growing countries including India, Sri-Lanka, Burma, Cambodia, Vietnam, Liberia, Costa Rica, Venezuela, Malaysia and Thailand (Edathil *et al*., 2000). The pathogen causes several types of symptom on the plant of which the abnormal leaf fall symptom is most severe in many parts of south India. It induces the shedding of leaves during June – August while general leaf fall occurs in December. Yield loss 9 - 25 %.

Symptoms

The different types of symptoms are as follows.

On Fruits

- The fruit-pods are the first seat of infection and consequently become responsible in spreading the disease to mature leaves and green stems
- Small lesions indicated initially by 'pin head' black globules of latex, usually 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.
- The Infected pods become black or grey, malformed and unopened
- The Infected pods either remain attached on the tree or drop off prematurely.
- The seeds within the pods are shriveled, distorted, unopened, unviable and rotten.

On Leaves

- The fungal growths are washed by rain down onto the leaves, petioles, young shoots and stems where they cause further infections.
- Initially the affected leaves show circular water soaked dull grey lesions with fine droplets of coagulated latex in concentric rings..
- In course of time the lesions enlarge and coalesce to form large, irregular necrotic areas with various shades of black
- The infection eventually spread to petiole.
- One or more black lesion with a drop of white coagulated latex in the centre may develop on the petiole.

- The affected leaves enormously shed prematurely either green or after turning coppery red.

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 from the infected tissues.
- Later in the season cankerous growth develops.
- The bark rots, and a coagulated rubber pad, emanating a foul smell is seen in between the wood and the rotting bark.
- Brownish or dark discoloration of the wood is seen below the bark.
- This infection is called **patch canker or bark canker**.
- In the renewed bark region, small, sunken, vertically parallel depressions are formed due to localised rotting and drying of bark which gets pressed to the wood.
- When infected bark is removed, characteristic, distinct vertical black lines are seen on the wood corresponding to the external depressions.
- In advanced stages of infection the bark splits open, causing gaping wounds (canker) with foul-smelling pads of coagulated latex between the bark and wood.
- Bark rot often reduces yield.
- The renewed bark becomes highly uneven regeneration of the bark giving panel burrs which make subsequent tapping difficult or impossible..
- This is called **bark stripes or black rot or black stripes or Black thread** disease.

Causal organisms: *Phytophthora palmivora* Butl. *P.meadii* McRae. *P.nicotianae var parasitica* and *P. botryose* (Chee)

Mode of spread and survival

- The oospores and chlamydospores produced inside the bark or in soil are primary source of infection.
- These carry the pathogen over adverse conditions.
- The sporangia and zoospores help in secondary spread through rain splash and irrigation water.

Epidemiology:

- High atmospheric humidity, low temperature, high rainfall, and crowded canopy are predisposing factors for the disease initiation and spread of inoculums that result in epiphytotics.
- Disease occurs mostly during rainy season. The fungus requires high humidity for its activity.
- The severity of the disease increases as the rain fall increases over 360 mm in a month.
- A temperature range of 15.6 – 20.0^0 C most favourable for sporangial production and spread of the disease.
- Prolonged wet weather coupled with humid atmospheric conditions favour the disease.
- Disease incidence is less in Kanyakumari District, but increasingly heavy northwards along the southwestern coast.
- In Sri Lanka, severe leaf fall occurs when the following weather conditions prevail for four consecutive days in the presence of infected fruits on the trees.
    1. Temperature less than 28.9^0C during day time.
    2. RH above 80% throughout.
    3. At least 2.5 mm of rain per day.
    4. Less than 3 hr of sunshine per day.
    5. The above periods have been named as **Peries**.

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.
- Aerial spraying of fycol 8 with soya oil @ 5l / ha is done in Kerala.
- Removal of bark canker affected tissues and dressing with organomercurial fungicides or copper fungicides (Bordeaux paste).
- Susceptible clones: PB 86, PB 235, PB 260, PB 311, PB 28/59, RRIM 600, RRIM 628, RRIM 703, RRII 5, PR 255, PR 261 and Tjir 1
- Tolerant clones: RRII 105, PB 217, GT 1 and GL 1

Spray schedule developed by Rubber Research Institute of India

Prophylactic spraying of the foliage prior to the onset of south west monsoon with Bordeaux mixture using high volume sprayers or Oil-based copper oxychloride dispersed in diluent spray oil employing either low volume airblast sprayers from the ground or through aerial application. For micron spraying, based on the tree spread, foliage intensity, planting material used and age of plants, two rounds of spray using about 17 to 22 litres of fungicide

oil mixture per hectare per round (1:6 proportion) with a gap of 10 to 15 days or a single round of spray with about 30-40 litres of fungicide oil mixture per hectare (1:5 proportion) may be necessary. For aerial spraying 6.2 litres of 40% oil based copper oxychloride paste in 37 litres of diluent oil or 8 kg oil dispersible copper oxychloride powder 56% in 40 litres of oil is used per hectare. Spraying should be done as close to the monsoon as possible.

Powdery Mildew / *Oidium* Secondary Leaf Fall: *Oiduim heveae* Steinm.

The disease is more common in India and in other countries. Predominantly noticed on newly formed tender flush during the refoliation period of January to March. The disease is severe in Kanyakumari, Idukki and Wynad district of South India and North Eastern States.

It causes extensive damage to rubber plantation in India.

Symptoms:

- The symptoms are more predominant on tender leaves and shoots.
- It infects mostly young shoots that refoliate after annual wintering.
- 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.
- The fallen leaves form a black carpet on the plantation floor.
- Sometimes petioles and young twigs are also infected.
- Later the petioles also fall off.
- On older leaves initially white patches of the fungus seen which become necrotic lesions later.
- The inflorescence and young fruits are also covered with white powdery masses
- Infected flowers and tender fruits are shed by reducing the fruit set
- The infected tender shoots dry off and die back symptoms appear.

Causal organism: *Oiduim heveae*

The fungus ectophytic

Mycelium – extensively branched, hyaline septate thin walled haustoria produced.

Conidiophores – Short produced in chain

Conidia – Barrel – shaped or cylindrical basipetal

Mode of spread and survival

- The fungus survives as dormant mycelium inside the bark.
- Secondary spread by wind borne conidia

Epidemiology

- The dormant mycelium become in active cloudy days with warm weather and low humidity.
- Cloudy days with light rains and or misty nights with dew formation during refoliation favour serious disease outbreak.
- 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.
- The clones RRII 105, RRII 118, RRII 300, PB 217, PB 235, PB 280, PB 311, PB 5/51, RRIM 605 Tjir 1 and PR 261 are susceptible to the disease.
- Dusting during the refoliation period commencing from bud break in about 10% of the trees, giving 3 to 5 rounds at weekly to fortnightly interval using 11 to 14 kg 325-mesh fine sulphur dust per round per hectare.
- Sulphur mixed with an inert material like Talc (70:30) is commonly used.
- Wettable sulphur or Carbendazim (2.5 g /l) is also effective in nurseries and for young plants as a spray.
- Carbendazim 0.05% a.i. (1g in 1 litre water) is more effective than sulphur for nurseries and young rubber.
- Alternate use of Carbendazim and sulphur is recommended to avoid resistance problem.
- Fogging with fungicide tridemorph such as Calixin 75 EC at 0.5 a.i/ha also provides a good control of the disease.
- For efficiency, dusting may be carried out in the early morning hours when the leaves are moist and the atmosphere calm.
- Power dusters like Skoda Micro spray power 400 or Aspee Turblow tree duster could be used for sulphur dusting.

***Colletotrichum* leaf fall or Secondary leaf fall: *Colletotrichum gloeosporioides* (Penz.) Sacc. *Colletotrichum acutatum* (Sexual stage: *Glomerlla cingulata*)**

In North East India the disease is prevalent throughout the year except during winter.

Symptoms

- Tender leaves during the first ten days of expansion are most susceptible to infection.
- Infection usually starts mostly at the tip of the leaves and spreads towards leaf base.
- 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.

- The spots coalesce and dry up leading to defoliation.
- Sometimes the centre of the spots dry and fall off leaving shot hole.
- Infected leaves become distorted, turn black, shrivel and fall off leaving the petioles on the plant for a short period.
- Lesions are observed on the green portions of stem also causing shoot die back.
- The fungus attacks green pods and cause rotting.

Causal organism: *Colletotrichum gloeosporioides* (Penz.) Sacc. *Colletotrichum acutatum* (Sexual stage: *Glomerlla cingulata*)

Mycelium – Septate hyaline at first and light brown at later.

Conidia – Oblong, single – celled,

Perithecia – Spherical brown

Asci – Oblong, Ascospores are curved unicellular.

Mode of spreads and survival:

- The fungus survives in infected plant debrieses.
- Water disperses the conidia.

Management:

- Spraying with Bordeaux mixture 1% or chlorothalonil and propined such as Daconil and Antracol respectively at 0.2% in water using a knapsack sprayer or a back-pack power mist blower.
- Adequate drainage
- Application of recommended dose of fertilizers
- 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* (Petch) M.B. Ellis

Symptoms:

- Common in nursery or on young plants in the field
- On tender brown leaves disease appears as dark brown water soaked spots.
- The leaves become black and get shriveled and distorted.
- Severely infected leaflets fall off leaving the petioles.
- When light green coloured leaves are infected minute yellow spots which later develop narrow reddish brown margin and a white papery centre are observed
- 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 develops a chocolate brown colour due to sporulation of the fungus.

- The fungus produces minute purple spots.

Causal organism: *Drechslera heveae*

Conidia are curved navicular or fusiform, pale to mid golden brown or reddish brown and smooth, 6 – 11 pseudosepta with hylum.

Mode of spread:

- The spores are disseminated by wind, rain or irrigation splashes

Management:

- Spraying with Bordeaux mixture 1% or mancozeb 0.2% or Carbendazim 0.04%
- Shading the nursery plants reduces the disease incidence.
- Maintain seedlings in vigorous condition through adequate balanced nutrition.

Corynespora leaf spot: *Corynespora cassiicola* (Berk & Curt)

Earlier the disease was confined to nurseries only. From 1996 onwards severe incidence of the disease was observed in mature plantations in the Dakshin Kannnada District of Karnataka and in the adjoining districts of Kerala. In nurseries, disease incidence is noticed during November to May period. On mature trees, disease is observed during the refoliation period from February to May.

Symptoms

- Minute spots (pin head lesions) develop when the leaves are very young
- The spots enlarge and become circular or irregular with a brown or papery centre surrounded by a dark brown ring and yellow halo.
- The papery centre may fall off forming shot holes.
- Young leaflets exhibit shriveling and drying of terminal portion.
- Veins become dark brown due to toxin production by the fungus
- These appear as a **fish bone** or a **railway track marked on a map**.
- Severe infection on midrib causes leaf blight.
- The blighted leaves may remain on branches for some more time giving a **burnt up appearance**.
- Severe disease incidence leads to yellowing and defoliation.
- One lesion is enough to cause defoliation, especially, if it is sited on the main vein.

Causal organism: *Corynespora cassiicola*

Conidia are obclavate cylindrical or slighted curved multiseptate, tapering towards the apex

Management

- Spraying with fungicide Benlate at 0.3%.
- Repeated spraying with Bordeaux mixture 1% or Dithane (Indofil) M-45 0.2% or Bavistin 0.02% is recommended for nursery.
- Shading the nursery reduces the disease incidence.

- Maintain seedlings in vigorous condition through adequate balanced nutrition.
- High volume spraying with mancozeb 0.2% (Dithane/Indofil M-45 2.66 g/l) carbendazim 0.05% (Bavistin 1g/l) at 2-3 weeks interval during refoliation is effective in mature plantation.
- Micron spraying with oil dispersible copper oxychloride 56% (8kg) or oil dispersible mancozeb 70% (7 kg) dispersed in 40 l spray oil per ha is also effective.

Pink disease – *Corticuin salmonicolor* Berk & Br.

The disease is noticed during southwest monsoon period in the traditional rubber growing areas in South India. The disease incidence is very mild in Kanyakumari District due to the weak southwest monsoon and absence of prolonged wet conditions. Disease has been noticed in Assam, Tripura and Meghalaya in north east. Pink disease is the most serious stem disease of affecting mainly the young rubber trees.

Symptoms

- More damaging for plants in the age group of 2 to 12 years
- The stem with brown bark is affected anywhere from the base to branches.
- In young plants the forking region is more vulnerable.
- The early symptom is the cobweb like superficial film of silky white mycelium which glistens in sunlight
- The mycelium grows covering all around the stem, ramifies the bark tissue and damages it.
- When the pathogen damages the latex vessels, the latex exudes.
- Rotting, drying up and cracking of the affected bark follow
- The leaves turn yellow and dry up.
- The dried leaves remain attached to the branch.
- New sprouts develop from below the affected region.
- Light pink coloured pustules appear in parallel lines all over the bark.
- These contain the spores.
- In the advanced stage, pink encrustations appear on the lower shaded regions of the affected branches.
- The disease gets its name from this stage.
- The pink encrustations contain the basidia on which basidiospores are produced.

Causal organism: ***Corticuim salmonicolor***

Mode of spread

Spread – Air blown basidiospores

Rain splash dispersed conidia

Epidemiology

Wet weather and high relative humidity are favourable for disease development.

Management

Affected plant parts should be pruned and burnt

Wound dressing with Bordeaux paste

Prophylactic: Two rounds of spraying with 1% Bordeaux mixture (during May and August) on to the fork and branches reduces disease incidence.

Application of Bordeaux paste to the topmost brown portion of the stem with a 30 cm wide band all around.

Curative: Application of Bordeaux paste in early stage on infected part and up to 30 cm above and below.

In advanced cases, Bordeaux paste may be initially applied and then the affected portion scraped to remove mycelium and decayed bark, followed by a second Bordeaux paste application.

Dried branches should be pruned after disinfection.

Thiram (10g in 1 kg) incorporated in a wound dressing compound or tridemorph 2% (Calixin 25 ml/l) in 1% ammoniated field latex or thiride 0.75% or tridemorph 1% or propiconazole 0.1% (Tilt 4 ml/l) in pidyvyl, china clay and water (1:2:4 by volume) is also effective as rainwash is prevented.

Root Diseases

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.
- Older rhizomorphs become yellow to red.
- 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.

C. Organism: *Fomes lignosus*

Fruiting bodies are bracket – shaped (7-20 cm wide) with yellow margin. They are concentrically banded reddish brown on the upper surface with a pore – studded red – brown lower surface.

Mode of spread and survival:

- Survives in old stumps and large roots for several years
- Spread by the contact of growing roots with rhizmorph growing in the soil.

Management:

- Old dead and infected stumps should be removed and burnt.

- The infected roots are treated with Copper Sulphate 0.2%
- Soil drenching with Brodeaux mixture 1%

Brown root disease: *Phellinus noxius* Corner G.H. Cunn

It occurs in most of the rubber growing countries. It is the major root disease of rubber in India. The disease occurrence is sporadic and often noticed in replanted areas and in sandy or light soils. Low incidence of brown root disease is noticed in Assam and Tripura

Symptoms

- The symptoms appear as loss of shiny appearance of leaves, drooping, yellowing and buckling (curving) of leaflets
- Retardation in growth of trees.
- The leaves later turn to reddish yellow and dry up.
- Occasionally flowering and fruiting is observed.
- The trees die bark.
- On the infected roots the surrounding soil and stones from a hard and thick coating.
- Yellowish 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.
- On old infected stumps large, hard, brownish purple brackets of fungus with grey underside develop.
- Infection is noticed on young nursery plants also.
- When the young trees affected they are killed.

Causal organism: ***Phellinus noxius* Corner G.H. Cunn**

Mode of spread

- The spores blown over by wind spread the disease.
- The spread is more often through root contact with infected roots.

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 (Provisional recommendation).
- When the fungicide dries up, a thin coating with a wound dressing compound may be given.
- Refill the soil and drench the base with fungicide solution.
- The dried up plants may be uprooted, root traced, collected and destroyed.
- As prophylactic, measure the bases of trees neighbouring the affected trees may also be drenched with fungicide solution mentioned above.

Purple root disease: *Helicobasidium compactum*

- ❖ Purple root disease is observed more in North East India though mild scale occurrence of the disease has also been reported from South India.
- ❖ The infection is observed both in nurseries and in the main field.
- ❖ Aerial symptoms are yellowing and drying of plants
- ❖ Infected plants are weak with sparse crowns.
- ❖ The tap root will be badly damaged and its bark often sloughs off exposing wood.
- ❖ Several adventitious roots arise with which the plant survives.

❖ Formation of a brown spongy fruiting body of the fungus around the collar of the infected plants is a distinct symptom

❖ Removal of affected roots and plants

❖ Drenching of fungicides like tridemorph 0.5% (Calixin 6.25ml/L) or propiconazole 0.13% (Tilt 5ml/L)

Poria root disease: *Poria vincta* (Berk.) Cooke

- ❖ The disease occurrence is sporadic
- ❖ Yellow discolouration of leaves is the only external symptom.
- ❖ The affected root shows soil encrustation as in the case of brown root disease but to a limited extent.
- ❖ Around the root the fungus forms a complete mat of pale white in colour intermingled with brick-red and black tinges.

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 (Provisional recommendation).
- ❖ When the fungicide dries up, a thin coating with a wound dressing compound may be given.
- ❖ Refill the soil and drench the base with fungicide solution.
- ❖ The dried up plants may be uprooted, root traced, collected and destroyed.
- ❖ As prophylactic, measure the bases of trees neighbouring the affected trees may also be drenched with fungicide solution mentioned above.

Dry Rot, Stump Rot Collar Rot: *Ustulina deusta* (Hoffm.ex Fr) Lind

- ❖ Affects roots, collar, trunk and branches
- ❖ Copious exudation of latex from the lesions during the rainy season
- ❖ The fungus penetrates the wood and spreads inside.
- ❖ Later fruiting bodies of the fungus appear as soft circular to irregular, grayish white ear lobe like structures which later join together and become grey or black and brittle.
- ❖ As a result the wood becomes soft and powdery.

- Affected trees or branches are killed or blown over.
- Black double lines formed by the fungus are seen inside the wood.
- Ustulina root infections are also noticed.
- The fungus penetrates the bark through wounds and cracks and hence the disease incidence is more following heavy wind.

Management

- Scrape off the fructifications, affected bark and wood showing black lines.
- Apply a wound dressing compound in which Thirum 0.75% a.i (Thiride 10 g/l) is incorporated.
- Hexaconazole 0.02% a.i (Contaf 4 ml/l) is also effective.
- Avoid accumulation of rubber at the base of the tree.
- For root infection see the treatment for brown root disease.

Red root rot: *Ganoderma pseudoferrum*

- The fungus attacks the root and induces wet rot.
- Reddish rhizomorphs are formed on the surface of the diseased roots and form a red skin.
- Adventitious roots develop profusely.
- In advanced stages of decay the wood may be spongy.
- Spread thro root contact

Shrinking root disease: *Sphaerostilbe repens*

- The fungus invades the root system
- Defoliation occurs and the tree is killed
- The fungus spread between the bark and wood of the infected root by means of reddish brown rhizomorph.
- Dead roots are bluish purple and emit foul odour.
- Low lying areas should be avoided for rubber planting.

DISEASES OF COCOA

White thread blight: *Marasmius scandens*

Horse hair blight: *Marasmius equicrinis*

Severe incidence was reported in 6 months old seedlings in Karnataka in 1990. Found in severe form in the plains. 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.
- Spread longitudinally and irregularly along the surface of the stem.

- The fungus enters the leaf at the nodes along the petiole.
- 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
- Horse hair blight forms a tangle of thin black threads through the canopy of leaves
- The dehisced leaves along with healthy leaves remain together and form a dense mass, preventing the development of new flush
- The dead leaves with mycelial mat are easily carried by wind on to the leaves and stems of healthy plants and initiate the disease

Mode of spread and survival

- Through mycelial threads

Epidemiology

- High humidity, less aeration and sun light.

Management:

- Removal of affected branches and leaves by pruning
- Provision of more aeration and sun light
- Spraying with Bordeaux mixture 1 % or copper oxychloride 0.4 % or Mancozeb 0.4 %

Seedling blight / Seedling die back / Black pod rot / Chupon Blight and Twig Dieback / Trunk or Stem Canker: *Phytophthora palmivora, P. megakarya, P. citrophthora* and *P. capsici*

Most destructive of all the fungal disease. First reported from Guyana in W. Indies in 189. In India it was first reported in 1965. Loss may be upto 10% in the world. 30-90% of the total global crop loss is caused by this disease (Bowers et al., 2001). Most common in all the cocoa growing areas of the world. In India most important and very serious disease of cocoa especially during south-west monsoon

Seedling blight/Seedling die back: *Phytophthora palmivora*

It is severe on young seedlings during rainy season. It is observed in nurseries of Karnataka Kerala and TN.

Symptom

- Seedling blight/seedling dieback is 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.
- The disease appears as dark brown to black water soaked linear lesions.
- The lesions extend to petiole and leaves resulting in wilting and subsequent defoliation of seedlings.

- Defoliation and die back of seedlings are noticed in advanced stages of the disease.

Black pod rot Symptoms: *Phytophthora palmivora*

- Pods of all ages are susceptible
- Pods or cherelles (immature pods) may be infected at any place on the surface, but is most often initiated at the tip or stem end.
- One or more small, brown and translucent spots appear anywhere on the pod 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
- Within four to seven days of infection, the lesion enlarges and assumes an elliptical shape
- As the lesion advances, a whitish growth of the fungus consisting of mycelia and sporangia is produced over the dark brown pod surface (Plate4)
- Lesion increases rapidly and covers the whole pod surface (Plate5)
- Under humid conditions a white mould appears on the surface.
- The whole pod and beans are invaded by the fungus and turns black in colour, which subsequently rots and shed in large numbers
- Infected beans are also discoloured.
- Diseased pods eventually become black and mummify.
- The pathogen also will cause a seedling blight in cacao nurseries as a result of infection of the stem and young leaves

Chupon Blight and Twig Dieback: *Phytophthora palmivora*

- 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 to petiole and extends backwards into the twigs or chupons
- The lesion spreads longitudinally in all directions and turns dark brown to black
- When the lesions girdle the stem, the portion above the point of infection wilts showing twig dieback or chupon blight

Stem Canker: *Phytophthora palmivora*

- Affects both seedlings and mature plants
- Occurs on the main trunk, jorquettes and fan branches
- The canker often develops from the pods.
- The infection from the pods spreads to the peduncle and then to the cushion and bark causing canker

- Greyish brown water soaked lesion with a broad dark brown to black margin appears on the bark
- Reddish brown liquid oozes out from the cracks of lesions, forming a rusty deposit
- The canker at the collar region appears as dark brown, irregular, water soaked lesion with oozing of reddish brown liquid
- Infection on the internal bark beneath the outer lesion appears as reddish brown discolouration
- Lesions in the internal bark coalesce, leading to extensive rotting
- The infection spreads from the cortical tissues into vascular tissues reaching wood
- Wood infection appears as greyish brown to black discolouration with black streaks
- When the cankers girdle the main stem or branches, dieback symptoms occur and the tree ultimately dies

Causal organism: *Phytophthora palmivora, P. megakarya, P. citrophthora* and *P. capsici*

In 1976, isolates of *P. palmivora* from cacao were placed into one of four morphological groups (MF1, MF2, MF3, and MF4). Further studies redefined the morphological groups. The MF1 form was considered to be the typical *P. palmivora*, and the MF2 form a variant (atypical) of *P. palmivora* (4). The MF3 form was described as a new species, *P. megakarya* Brasier and Griffin (4), and the MF4 form considered to be *P. capsici* Leonian (39,43). Recently, it has been proposed that isolates of *P. capsici* differing in morphological and pathological attributes should be called *P. tropicalis* (2). *Phytophthora palmivora* is present in most countries and is an important part of the black pod complex, while *P. megakarya* is present only in several countries in West Africa (14). However, *P. megakarya* appears to be more virulent than *P. palmivora* and is becoming the dominant species in West Africa, moving from Nigeria and Cameroon into countries where it has not been reported previously. In the major cacao-growing region of Bahia, Brazil, three species have been implicated: *P. palmivora*, *P. capsici*, and *P. citrophthora* (Smith and Smith) Leonian (17,22,24). *Phytophthora capsici* appears to be the dominant and most important species attacking cacao in Brazil, and also has been reported to occur in other countries in Central and South America, as well as in the West Indies, Indonesia, and India (6). *Phytophthora citrophthora* is the least common species found in Brazil, but is the most virulent (17,24). *Phytophthora heveae* Thompson also has been found to cause black pod in some countries (6).

Mode of spread and survival

- The fungus survives as oospores, chlamydospores and mycelium in soil, on fallen fruits, dried fruits and flower cushions, stem etc.
- The spread of the disease is by wind and windblown rain.

Epidemiology:

- Continuous heavy rainfall with intermittent bright sunshine, low temperature of 22-25^0 C, constant high relative humidity of more than 90%, wind, cloudy weather etc. favour the outbreak of the disease
- Closer spacing and damp locality favour the spread of the disease.

Management:

- Collect and destroy all fallen and completely infected pods at weekly intervals or during each harvest
- Remove the piles of pod husks lying in the garden and destroy by burning
- Give proper spacing between plants.
- Prune the epiphytes and chupons frequently so as to regulate shade
- Provide adequate drainage in gardens
- Harvest the ripe pods frequently to ensure that recently infected pods are not lost completely
- Prophylactic spray of Bordeaux mixture 1% or copper oxychloride 0.3-0.4 % with stickers, first before the onset of heavy south west monsoon and a second and third at 40-45 days interval, especially on pods and flower cushions
- Spray with metalaxyl or fentin acetate or Aluminium ethyl phosphonate.

Charcoal pod rot: *Botryodiplodia theobromae or Lasiodiplodia theobromae*

In Kerala it has become a serious disease. It is found throughout the year. But it is severe during summer months.

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 (Plate6).
- The spots enlarge into larger lesions and cover the entire pod giving a chocolate brown colour (Plate7).
- Severely infected pods become black in color and exhibit a sooty covering all over, consisting of spores of the fungus (Plate8)
- Young pods become mummified and shriveled (Plate9)
- Infected pods dry up and generally remain attached to the plant

Causal organism: *Botryodiplodia theobromae or Lasiodiplodia theobromae*

Mode of Spread

- wound parasite
- Wind borne conidia

Management

- Collect and destroy all fallen/completely dried pods
- Regularly harvest ripened pods

- Remove the piles of pod husks and destroy by burning
- 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
- Spray the pod bunches twice with Contaf / Tilt 0.1% or Bavistin 0.1 % at 2-3 weeks interval after harvesting all mature pods. Subsequently harvest only after 45 days of spraying
- Control of mealy bug and Tea mosquitoes

Cherelle Rot / *Colletotrichum* Pod Rot: *Colletotrichum gloeosporioides*

- It is noticed especially during February-May
- 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
- Sometimes the infection may start from anywhere on pod surface, other than stalk region, as dark brown sunken lesion
- 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

- Collect and destroy all infected and dried cherelles
- Remove and destroy by burning, the piles of pod husks lying in the garden
- Regularly harvest the ripe pods to ensure that recently infested pods are not completely lost
- 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
- Spray the pod bunches twice with Contaf / Tilt 0.1% or Bavistin 0.1 % at 2-3 weeks interval after harvesting all mature pods. Subsequently harvest only after 45 days of spraying

Leaf Blight and Shot Hole: *Colletotrichum gloeosporioides* & *C. theobromicolum*

- Round to slightly irregular chlorotic spots appear on the leaves,
- Which later turn brown with a clear yellow halo around each spot.
- The spots develop anywhere on the leaf lamina but more often on the tip or margins
- The centre of the spots become necrotic, shrivel and drop off forming shot holes

- Spots enlarge or coalesce to form large blighted areas with an even margin and dark brown to black dots representing its fruiting bodies
- Defoliation occurs, when such lesions cover a major portion of the leaf

Management

Spray 1 % Bordeaux mixture / 0.4 % copper oxychloride or 0.4 % Mancozeb (4 g/L)

Vascular Streak Dieback: *Oncobasidium theobromae*

- A single leaf in the second or third flush from the tip of twig becomes yellow, with islets of green patches scattered over the yellowish lamina
- Such leaves fall off prior to the older leaves of the same branch
- Leaves above and below the first fallen ones develop similar symptoms and are lost
- This leads to a distinctive appearance where the youngest and older leaves are still present but all the middle ones have shed
- The bark in the leaf fall region of the branch becomes rough due to swelling of the lenticels
- The auxiliary buds of the fallen leaves sprout and then rapidly die
- At the later stage, dieback symptom appear on these branches
- Because of infection, the xylem vessels turn brownish which appear as streaks within the vascular tissues. Hence, the disease is known as vascular streak dieback
- The disease can be easily diagnosed in the field by stripping the bark or by splitting the affected stem longitudinally and observing the brownish streaks

Management

- Regularly prune and destroy infected branches
- Spray 0.1% Contaf or Tilt or Topas at monthly intervals in severely infected garden

Pink Disease: *Corticium salmonicolor or Pellicularia salmonicolor*

- Oozing of brown liquid from the fork region, the trunk and branches (Plate27)
- White, silky mycelial threads appear and spread around the branch like a “cobweb”
- Shoots wilt, leaves shed and ultimately branches dry up (Plate 28)
- As infection progresses, the trunk/branches become ringed and die (Plate29)
- Characteristic pinkish powdery coating appears on the irregular cracks
- Numerous off shoots are formed below the point of infection (Plate30)

Management

- Remove and destroy completely infected and dried shoots/branches
- Reduce overhead shade
- Prune properly to improve aeration inside the garden.
- Prune smaller infected branches and swab the cut ends with Bordeaux paste or 0.5% Contaf / Calixin in rubber kote or coal tar (5 ml/kg)

- As a preventive measure, spray 1% Bordeaux mixture at regular intervals on forking regions and branches during the rainy season

Witches broom: *Moniliophthora perniciosa* (formerly *Crinipellis perniciosa* (Stahel) Singer (= *Marasmius perniciosus*)

- It causes abnormal growths and lesions on the shoots, branches, floral cushions and fruits.
- It also attacks nursery seedlings.
- Basidiospores penetrate young meristematic tissues in vegetative and floral buds.
- The colonized tissues undergo several physiological and hormonal changes leading to swelling and formation of numerous succulent vegetative branches, known as **brooms**, within flower cushions and on vegetative apical or axillary buds.
- The fungus also infects pods causing green patches and necrotic lesions, uneven ripening, and various deformations.
- Brooms and infected pods become progressively brown and dry

Frosty pod rot or *Moniliophthora* pod rot: *Moniliophthora roreri* (Ciferi & Parodi) Evans et al.,

- Yield losses have been estimated between 25% to total loss of the crop
- It is a devastating disease of cacao pods.
- The pods are highly susceptible during the first 90 days of their growth.
- Early symptoms include discolored areas of swelling on the pods followed by a dense formation of cream-colored powdery growth. so called frosty pod
- The area of the pod with sporulation spreads rapidly,

Root Diseases

Brown root rot *- Phellinus noxius*

Red root rot *- Ganoderma spp.*

Black root rot *- Armillaria spp. & Rosellina arcuata*

- Sudden premature yellowing and drying of slightly off-green leaves is a possible indication of root diseases
- Rhizomorphs are found firmly attached to the infected roots or ramifying into a network with soil encrustation around root
- They are brown / red / black depending on the fungus associated
- The wood of a newly-killed root has brown / black lines and is hard
- Generally, fruiting bodies of the fungi grow at the collars of diseased trees and on decaying exposed roots or stumps
- The disease can be identified based on the fruiting bodies or rhizomorphs grown on dead stumps or decayed roots

Management

- Remove and destroy dead/infected trees including roots.
- Treat soil with 2% CuSO4 and replant after 6 months to one year
- Prune branches, regulate shade and provide adequate spacing for improving aeration.
- Apply organic matter @ 10-15 kg/pit and lime @ 1-2 kg/pit
- Isolate infected trees by digging trenches 1.2 m deep and 0.6 m wide
- Drench soil with 1 % Bordeaux mixture or 0.2 % copper oxychloride or Contaf/Tilt /Bavistin

Swollen shoot – Theobroma Virus / Cocoa Swollen Shoot Virus

It is a serious disease Africa

But it has not been reported in India.

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

Spread

By insect vectors like mealy bug and grafting

Control:

- Removal and destruction of infected trees
- Removal of alternate hosts for virus and vector
- Control of vectors

**************************ALL THE BEST**************************