Development of e-Courses for B.Sc. (Agriculture) Degree Program

PATH 272
DISEASES OF FIELD CROPS AND THEIR MANAGEMENT
Diseases of Field Crops and Their Management

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1. Diseases of Rice

Fungal Diseases

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

**Symptoms**

The fungus attacks the crop at all stages of crop growth. Symptoms appear on leaves, nodes, rachis, and glumes. On the leaves, the lesions appear as small bluish green flecks, which enlarge under moist weather to form the characteristic spindle shaped spots with grey centre and dark brown margin (*Leaf blast*).

The spots coalesce as the disease progresses and large areas of the leaves dry up and wither. Spots also appear on sheath. Severely infected nursery and field appear as burnt. Black lesions appear on nodes girdling them. The affected nodes may break up and all the plant parts above the infected nodes may die (*nodal blast*).

During flower emergence, the fungus attacks the peduncle and the lesion turns to brownish-black which is referred to as rotten neck / neck rot / panicle blast (*neck blast*).

In early neck infection, grain filling does not occur while in late infection, partial grain filling occurs. Small brown to black spots may also be observed on glumes of the heavily infected panicles. The pathogen causes yield losses ranging from 30-61 per cent depending upon the stages of infection.

**Pathogen**

The mycelium is hyaline to olivaceous and septate. *Conidia* are produced in clusters on long septate, olivaceous conidiophores. Conidia are pyriform to ellipsoid, attached at the broader base by a hilum. Conidia are hyaline to pale olive green, usually 3 celled. The perfect state of the
fungus is *M. grisea* producing perithecia. The ascospores are hyaline, fusiform, 4 celled and slightly curved.

**Favourable Conditions**

- Intermittent *drizzles*, cloudy weather, more of rainy days, longer duration of **dew** high relative humidity (93-99 per cent).
- Low night temperature (between 15-20˚C or less than 26˚C).
- Availability of **collateral hosts** and excess dose of nitrogen.

**Forecast** for rice blast can be made on the basis of minimum night temperature range of 20-26˚C in association with a high relative humidity 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 forecasting model was developed named as BLAST. Later several other models have also been developed namely, **PYRICULARIA, PYRIVIEW, BLASTAM, EPIBLA** and **PBLAST**.

**Disease Cycle**

The disease spreads primarily through airborne conidia since spores of the fungus present throughout the year. Mycelium and conidia in the infected straw and seeds are major sources of inoculum. Irrigation water may carry the conidia to different fields. The fungus also survives on **collateral hosts** viz., *Panicum repens*, *Digitaria marginata*, *Brachiaria mutica*, *Leersia hexandra* and *Echinochloa crusgalli*. 
Spores land on leaves, germinate, penetrate the leaf, and cause a lesion 4 days later; more spores are produced in as little as 6 days. Infections from spores arriving from a distance are termed primary infections.

Primary infections generally result in a few widely scattered spots on leaves. Spores arising from the primary infections are capable of causing many more infections. This cycling is called secondary spread. Secondary spread is responsible for the severe epidemics of blast in fields and localized areas.

**Management**

- Grow resistant to moderately resistant varieties CO47, IR 20, ADT36, ADT39, ASD 18 and IR64. Avoid cultivation of highly susceptible varieties viz., IR50 and TKM6 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 **Tricyclazole** at 2 g/kg. or **Pseudomonas fluorescens** @ 10g/kg of seed. Spray the nursery with carbendazim 500mg/L or tricyclazole 300mg/L.
• Spray the main field with Edifenphos 500 ml or Carbendazim 500 g or Tricyclazole 500 g or Iprobenphos (IBP) 500 ml /ha.

Brown Spot - *Helminthosporium oryzae* (Syn: *Drechslera oryzae; Bipolaris oryzae*)
(Sexual stage: *Cochliobolus miyabeanus*)

**Symptoms**

The fungus attacks the crop from seedling to milky stage in main field. Symptoms appear as minute spots on the coleoptile, leaf blade, leaf sheath, and glume, being most prominent on the leaf blade and glumes.

The spots become cylindrical or oval, dark brown with yellow halo later becoming circular. Several spots coalesce and the leaf dries up. The seedlings die and affected nurseries can be often recognised from a distance by scorched appearance. Dark brown or black spots also appear on glumes leading to grain discoloration. It causes failure of seed germination, seedling mortality and reduces the grain quality and weight.

**Pathogen**
*Bipolaris oryzae* produces brown septate mycelium. **Conidiophores** arise singly or in small groups. They are geniculate, brown in colour. **Conidia** are usually curved with a bulged center and tapered ends. They are pale to golden brown in colour and are 6-14 septate. The perfect stage of the fungus is *C. miyabeanus*.

It produces **perithecia** with asci containing 6-15 septate, filamentous or long cylindrical, hyaline to pale olive green **ascospores**. The fungus produces 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 severity.

**Disease Cycle**

Infected seeds and stubbles are the most common source of primary infection.
The conidia present on infected grain and mycelium in the infected tissue are viable for 2 to 3 years. Airborne conidia infect the plants both in nursery and in main field.

The fungus also survives on collateral hosts like *Leersia hexandra* and *Echinochloa colonum*. The brown spot fungus is normally present in areas with a long history of rice culture. Airborne spores that are capable of causing infection are produced in infested debris and older lesions.

**Management**

- Field sanitation-removal of collateral hosts and infected debris from the field.
- Use of slow release nitrogenous fertilizers is advisable.
- Grow tolerant varieties *viz.*, Co44 and Bhavani.
- Use disease free seeds.
- Treat the seeds with Thiram or Captan at 4 g/kg. Spray the nursery with Edifenphos 40 ml or Mancozeb 80 g for 20 cent nursery.
- Spray the crop in the main field with Edifenphos 500 ml or Mancozeb 2 kg/ha when grade reaches 3. If needed repeat after 15 days.
Narrow brown leaf spot - *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.

![Symptoms](image)

**Pathogen**

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

**Management**

Spray Carbendazim 500 g or Mancozeb 2 kg/ha.

Sheath rot - *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 remain within the sheath or emerge partially. The panicles rot and abundant whitish powdery fungal growth is seen inside the leaf sheath.

![Symptoms](image)
Pathogen
The fungus produces whitish, sparsely branched, septate mycelium. Conidia are hyaline, smooth, single celled and cylindrical in shape.

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

Disease Cycle
The disease spreads mainly through air-borne conidia and also seed-borne. Primary source of inoculum is by means of infected plant debris. Secondary spread is by means of air borne conidia produced on the leaf sheath.

Management
- Spray Carbendazim 500g or Edifenphos 1L or Mancozeb 2 kg/ha at boot leaf stage and 15 days later.
- Soil application of gypsum (500 kg/ha) in two splits.
- Application of Neem Seed Kernal Extract (NSKE) 5% or neem oil 3 % or Ipomoea or Prosopis leaf powder extract 25 Kg/ha. First spray at boot leaf stage and second 15 days later.

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.
The infection extends to the inner sheaths resulting in death of the entire plant. Older plants are highly susceptible. Plants heavily infected in the early heading and grain filling growth stages produce poorly filled grain, especially in the lower part of the panicle.

Pathogen

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

Favourable Conditions

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

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

Management

- Grow resistant varieties like Mansarovar, Swarau Dhan, Pankaj etc.
- Apply organic amendments viz., neem cake @ 150Kg/ha or FYM 12.5 tons/ha. Avoid flow of irrigation water from infected fields to healthy fields.
- Deep ploughing in summer and burning of stubbles.
- Spray Carbendazim 500 g/ha
- Soil application of P.fluorescens @ of 2.5 kg/ha after 30 days of transplanting (product should be mixed with 50 kg of FYM/Sand and applied).
- Foliar spray P.fluorescens at 0.2% at boot leaf stage and 10 days later

False smut - *Ustilaginoidea virens* (Syn: *Claviceps oryzae - sativa*)

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

Pathogen

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

Disease Cycle

Grasses and wild rice species are alternate hosts. The main source of inoculum is airborne spores. Ascospores produced from sclerotia act as primary source of infection while chlamydospores are secondary source of infection. Chlamydospores are air-borne, abundant at heading stage.

Favorable conditions

- Rainfall and cloudy weather during flowering and maturity
Udbatta disease - *Ephelis oryzae* (Sexual stage: Balansa oryzae-sativa)

**Symptoms**

Symptoms appear at the time of panicle emergence. The entire ear head is converted into a straight compact cylindrical black spike like structure since the infected panicle is matted together by the fungal mycelium. The spikelets are cemented to the central rachis and the size is remarkably reduced. The entire spike is covered by greyish stroma with convex pycnidia immersed inside.

Pathogen

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

Management

- The pathogen is internally seed borne.
- Hot water seed treatment at 45°C for 10 min. effectively controls the disease.
- Removal of collateral hosts *Isachne elegans, Eragrostis tenuifolia* and *Cynadon dactylon*.

Stackburn disease - *Trichoconis padwickii* (Syn: *Alternaria padwickii*)

**Symptoms**

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

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

Management

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

Bunt or Kernel Smut or black smut - *Tilletia barclayana*

Minute black pustules or streaks are formed on 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.
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 small black sclerotia are seen in the rotting tissues. The culm collapses and plants lodge. The sclerotia are carried in stubbles after harvest.

**Pathogen**

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

**Favourable Conditions**

- Infestation of leaf hoppers and stem borer.
- High doses of nitrogenous fertilizers.

**Disease Cycle**

The sclerotia survive in stubbles and straw those are carried through irrigation water. The fungus over winters and survives for long periods as sclerotia in the upper layers (2-3 inches) of the soil profile. The half-life of sclerotia in the field is about 2 years. Viable sclerotia have been found in fields for up to 6 years after a rice crop. The sclerotia are buoyant and float to the surface of floodwater where they contact, germinate, and infect rice tillers near the water line.

**Management**

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

**Foot rot or Bakanae disease - *Fusarium moniliforme* (Sexual stage: *Gibberella fujikuroi*)**

**Symptoms**

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

![Symptoms](Symptoms.png)

**Pathogen**

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

- The fungus is externally seed-borne.
- Treat the seeds with Thiram or Captan or Carbendazim at 2 g/kg.


Symptoms

The grains may be infected by various organisms before or after harvesting causing discolouration, the extent of which varies according to season and locality. The infection may be external or internal causing discoloration of the glumes or kernels or both. Dark brown or black spots appear on the grains.

The discoloration may be red, yellow, orange, pink or black, depending upon the organism involved and the degree of infection. This disease is responsible for quantitative and qualitative losses of grains.
Favourable Conditions

• High humidity and cloudy weather during heading stage

Disease cycle

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

Management

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

Bacterial Diseases

Bacterial leaf blight - *Xanthomonas oryzae pv. oryzae*

Symptoms

The disease is usually noticed at the time of heading but it can occur earlier also. Seedlings in the nursery show circular, yellow spots in the margin, that enlarge, coalesce leading to drying of foliage. “Kresek” symptom is seen in seedlings, 1-2 weeks after transplanting. The bacteria enter through the cut wounds in the leaf tips, become systemic and cause death of entire seedling.
In grown up plants water soaked, translucent lesions appear near the leaf margin. The lesions enlarge both in length and width with a wavy margin and turn straw yellow within a few days, covering the entire leaf. As the disease advances, the lesions cover the entire lamina which turns white or straw coloured. Milky or opaque dew drops containing bacterial masses are formed on young lesions in the early morning. They dry up on the surface leaving a white encrustation. The affected grains have discoloured spots. If the cut end of leaf is dipped in water, it becomes turbid because of bacterial ooze.

**Pathogen**

The bacterium is aerobic, gram negative, non spore forming, rod with size ranging from 1-2 x 0.8-1.0μm with monotrichous polar flagellum. Bacterial colonies are circular, convex with entire margins, whitish yellow to straw yellow colored and opaque.

**Favorable Conditions**

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

**Disease Cycle**

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

- 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 IR 20 and TKM 6.
- Spray Streptomycin sulphate and tetracycline combination 300g + Copper oxychloride 1.25 Kg/ha.

Bacterial leaf streak - *Xanthomonas oryzae pv. 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, bacterial ooze out and form small yellow band-like exudates under humid conditions. In severe cases the leaves dry up.

**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 IR 20 and TKM 6.
- Spray Streptomycin sulphate and tetracycline combination 300g + Copper oxychloride 1.25 Kg/ha.

Viral Diseases

**Rice Tungro Disease (RTD)** - *Rice tungro bacilliform virus* (RTBV) and *Rice tungro spherical virus* (RTSV)

**Symptoms**

Infection occurs both in the nursery and main field. Plants are markedly stunted. Leaves show yellow to orange discoloration and interveinal chlorosis. Young leaves are sometimes mottled while rusty spots appear on older leaves. Tillering is reduced with poor root system.
Panicles not formed in very early infection, if formed, remain small with few, deformed and chaffy grains.

**Pathogen**

Two morphologically unrelated viruses present in phloem cells. *Rice tungro bacilliform virus* (RTBV) bacilliform capsid, circular ds DNA genome and *Rice tungro spherical virus* (RTSV) isometric capsid ss RNA genome.

**Disease Cycle**

Transmission mainly by the leaf hopper vector *Nephotettix virescens* Males, females and nymphs of the insect can transmit the disease. Both the particles are transmitted semi-persistently, in the vector the particles are noncirculative and nonpropagative. Plants infected with RTSV alone may be symptomless or exhibit only mild stunting. RTBV enhances the symptoms caused by RTSV. RTSV can be acquired from the infected plant independently of RTBV, but acquisition of RTBV is dependent on RTSV which acts as a helper virus. Both the viruses thrive in rice and several weed hosts which serve as source of inoculum for the next. Ratoon from infected rice stubble serve as reservoirs of the virus. Disease incidence depends on rice cultivars, time of planting, time of infection and presence of vectors and favorable weather conditions.
Management

- Field sanitation, removal of weed hosts of the virus and vectors.
- Grow disease tolerant cultivars like Pankhari203, BM66, BM68, Latisail, Ambemohar102, Kamod253, IR50 and Co45.
- Control the vectors in the nursery by application of Carbofuran 170 g/cent 10 days after sowing to control hoppers.
- Spray Phosphomidan 500 ml or Monocrotophos 1lit/ha (2 ml/litre) or Neem oil 3% or NSKE 5% to control the vector in the main field 15 and 30 days after transplanting.
- Set up light traps to monitor the vector population.

Rice Grassy stunt disease - *Rice grassy stunt tenuivirus*

**Symptoms**

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

Pathogen

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

Disease Cycle

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

**Symptoms**

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

**Pathogen**

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

**Disease Cycle**

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

**Management**

- Destory weed host that serve as source of inoculum
- Spray Phosphamidon or Fenthion 500 ml or Monocrotophos 1 lit/ha.

Rice ragged stunt disease – *Rice ragged stunt virus*

**Symptoms**

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

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

Disease Cycle

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

**Rice yellow dwarf disease – Rice yellow dwarf virus**

**Symptoms**

Prominent stunting of plants and excessive tillering are the characteristic symptoms of the disease. Leaves yellowish green to whitish green, become soft and droop. Plants usually remain sterile but sometimes may produce small panicles with unfilled grains.

Pathogen

- Caused by a phytplasma (rice yellow dwarf phytplasma designated as a novel taxon, ‘*Candidatus Phytoplasma oryzae*’)

Disease Cycle

The disease is transmitted by leafhopper vectors *Nephotettix sp. Nephotettix* with a latent period of 25-30 days in the vector. The pathogen survives on several grass weeds.

Management

- Deep ploughing during summer months and burning of stubbles.
- Rice varieties IR62 and IR64 are moderately resistant to the disease.
- The management practices followed for Rice Tungro disease holds good for this disease also.
2. Diseases of Sorghum

Downy Mildew - *Peronosclerospora sorghi*

**Symptoms**

The fungus causes systemic downy mildew of sorghum. It invades the growing points of young plants, either through oospore or conidial infection. As the leaves unfold they exhibit green or yellow colouration. Abundant downy white growth is produced on the lower surface of the leaves, which consists of sporangiophores and sporangia.

Normally three or four leaves develop the chlorotic downy growth. Subsequent leaves show progressively more of a complete bleaching of the leaf tissue 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 an obligate parasite systemic in young plant. The mycelium is intercellular, non-septate. Sporangiohores emerge through the stomata in single or in clusters which are stout and dichotomously branched. Spores are single celled, hyaline, globose and thin walled. Oospores are spherical, thick walled and deep brown in colour.
Favourable Conditions

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

Disease Cycle

The primary infection is by means of oospores present in the soil which germinate and initiate the systemic infection. Oospores persist in the soil for several years. Secondary spread is by air-borne sporangia. Presence of mycelium of the fungus in the seeds of systemically infected plants is also a source of infection. The disease has been known to occur through a collateral host, Heteropogon centortus on which the fungus perpetuates of the host. The breakdown of tissue causes shredding. The oospores either fall to the soil or are wind blown, often within host tissue. They can remain viable in the soil for 5-10 years. Conidia are formed at night in large numbers. The optimum temperature for production is 20-23°C.
Management

- **Crop rotation** with other crops viz., pulses and oilseeds.
- Avoid the secondary spread of the disease by roguing out the infected plants since the wind plays a major role in the secondary spread of the disease.
- Seed treatment with **Metalaxyl** at 6 g/kg of seed.
- Spray **Metalaxyl** 500 g or **Mancozeb** 2 kg or **Ziram** 1 kg or **Zineb** 1kg/ha.

**Leaf blight - *Exerohilum turcicum*** (Syn: *Helminthosporium turcicum*)

**Symptoms**

The pathogen also causes **seed rot** and **seedling blight** of sorghum. The disease appears as small narrow elongated spots in the initial stage and in due course they extend along the length of the leaf. On older plants, the typical symptoms are long **elliptical necrotic lesions**, straw coloured in the centre with dark margins.
The straw coloured centre becomes darker 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 burnt appearance.

**Pathogen**

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

**Favourable Conditions**

- Cool moist weather.
- High humidity (90 per cent)
- High rainfall.

**Disease cycle**

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

**Management**

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

**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 lighter centers. The lesions are mostly isolated and limited by veins. The colour of the spots varies 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 moist weather.
- High humidity (90 per cent)
- High rainfall.

**Disease cycle**

The conidia survive up to 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 2 kg/ha.

**Anthracnose and red rot - *Colletotrichum graminicolum***

**Symptoms**

The fungus causes both leaf spot (anthracnose) and stalk rot (red rot). 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 like acervuli are seen on the white surface of the lesions. 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.

**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 28-30°C.
- High humidity.

**Disease cycle**

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 2 kg/ha.
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. 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.

![Symptoms on leaves and stalk](image)

**Pathogen**

The uredospores are pedicellate, elliptical or oval, thin walled, echinulated and darkbrown 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.

**Favourable Conditions**

- Low temperature of 10 to 12°C favours teliospore germination.
- A spell of rainy weather favours the onset of the disease.

**Disease cycle**

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 2 kg/ha.

**Grain smut/Kernel smut / Covered smut / Short smut - *Sphacelotheca sorghi***

**Symptoms**

The individual grains are replaced by smut sori. The sori are oval or cylindrical and are covered with a tough creamy skin (*peridium*) which often persists unbroken up to thrashing. **Ratoon** crops exhibit higher incidence of disease.

**Loose smut/ kernel smut - *Sphacelotheca cruenta***

**Symptoms**

The affected plants can be detected before the ears come out. They are shorter 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.

**Long smut - *Tolyposporium ehrenbergii***

**Symptoms**

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

**Symptoms**

The entire head is replaced by large sori. The sorus is covered by a whitish grey membrane of fungal tissue, which ruptures, before the head emerges from the boot leaf to expose a mass of brown smut spores. Spores are embedded in long, thin, dark colored filaments which are the vascular bundles of the infected head.

**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 bury in soil.

Ergot or Sugary disease - *Sphacelia sorghi*

**Symptoms**

The disease is confined to individual spikelets. The first symptom is the secretion of honey dew from infected florets. Under favourable conditions, long, straight or curved, cream to light brown, hard sclerotia develop. 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.

Favourable Conditions

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

Disease Cycle

The primary source of infection is through the germination of sclerotia which release ascospores that 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 favor the disease.
• Spray any one of the following fungicides viz., Mancozeb 2 kg/ha (or) Carbendazim at 500 g/ha at emergence of ear head (5-10 per cent flowering stage) followed by a spray at 50 per cent flowering and repeat the spray after a week, if necessary.

Head mould/Grain mould/Head blight
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 occurs. The most frequently occurring genera are *Fusarium*, *Curvularia*, *Alternaria*, *Aspergillus* and *Phoma*. *Fusarium semitectum* and *F. moniliforme* develop a fluffy white or pinkish coloration. *C. lunata* colours the grain black. Symptom varies depending upon the organism involved and the degree of infection.

![Symptoms](image)

**Favourable Conditions**

- Wet weather following the flowering favors grain mould development.
- The longer the wet period the greater the mould development.
- Compact ear heads are highly susceptible.

**Disease cycle**

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.

**Phanerogamic parasite - 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 up to a height of 15-30 cm. The plants occur in clusters of 10-20/host plant. *S. asiatica* produces red to pink flowers while *S. densiflora* produces white flowers. Each fruit contains minute seeds in abundance which survives in the soil for several years.

The root exudates of sorghum stimulate the seeds of the parasite to germinate. The parasite then slowly attaches to the root of the host by haustoria and grows below the soil surface producing 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 intercultural operation during early stages of parasite growth.
- Spray Fernoxone (sodium salt of 2, 4-D) at 450g /500 litre of water.

**Symptoms**
3. Diseases of Wheat

Black or stem rust - *Puccinia graminis tritici*

**Symptoms**

Symptoms are produced on almost all aerial parts of the wheat plant but are most common on stem, leaf sheaths and upper and lower leaf surfaces. Uredial *pustules* (or sori) are oval to spindle shaped and dark reddish brown (rust) in color. They erupt through the epidermis of the host and are surrounded by tattered host tissue. The pustules are dusty in appearance due to the vast number of spores produced. Spores are readily released when touched.

As the infection advances teliospores are produced in the same pustule. The color of the pustule changes from rust color to black as *teliospore* production progresses. If a large number of pustules are produced, stems become weakened and lodge. The pathogen attacks other host (barberry) to complete its life cycle. Symptoms are very different on this woody host. Other spores are *Pycnia (spermagonia)* produced on the upper leaf surface of barberry which appears as raised orange spots. Small amounts of honeydew that attracts insects are produced in this structure. *Aecia*, produced on the lower leaf surface, are yellow. They are bell-shaped and extend as far as 5 mm from the leaf surface.

Brown or leaf rust - *Puccinia triticina (P. recondita)*

**Symptom**
The most common site for symptoms is on leaf blades, however, sheaths, glumes and awns may occasionally become infected and exhibit symptoms. Uredia are seen as small, circular orange blisters or pustules on the upper surface of leaves.

Orange spores are easily dislodged and may cover clothing, hands or implements. When the infection is severe leaves dry out and die. Since inoculum is blown into a given area, symptoms are often seen on upper leaves first. As plants mature, the orange urediospores are replaced by black teliospores. Pustules containing these spores are black and shiny since the epidermis does not rupture. Yield loss often occurs as a result of infection by Puccinia recondita f. sp. tritici. Heavy infection which extends to the flag leaf results in a shorter period of grain fill and small kernels.

**Yellow or stripe rust** - *Puccinia striiformis*

Mainly occur on leaves than the leaf sheaths and stem. Bright yellow pustules (Uredia) appear on leaves at early stage of crop and pustules are arranged in linear rows as stripes. The stripes are yellow to orange yellow. The teliospores are also arranged in long stripes and are dull black in colour.
Pathogen

The uredospores of rust pathogen are almost round or oval in shape and bright orange in colour. The teliospores are bright orange to dark brown, two celled and flattened at the top. Sterile paraphyses are also present at the end of sorus.

Disease Cycle

In India, all these rusts appear in wheat growing belt during Rabi crop season. Uredosori turn into teliosori as summer approaches. The inoculum survives in the form of uredospores / teliospores in the hills during off season on self sown crop or volunteer hosts, which provide an excellent source of inoculum. In India, role of alternate host (Barberis) is not there in completing the life cycle.

The fungus is inhibited by temperatures over 20°C although strains tolerant of high temperatures do exist. The complete cycle from infection to the production of new spores can take as little as 7 days during ideal conditions. The disease cycle may therefore be repeated many times in one season. During late summer, the dark teliospores may be produced. These can germinate to produce yet another spore type, the basidiospore, but no alternate host has been found. Although the teliospores seem to have no function in the disease cycle they may contribute to the development of new races through sexual recombination.
Life cycle of *Puccinia graminis*

**Favourable Conditions**
- Low temperature (15-20°C) and high humidity during November – December favour black and brown rusts.
- Temperature less < 10° favours yellow rusts.

**Disease cycle**
Uredospores and dormant mycelium survive on stubbles and straws and also on weed hosts and self sown wheat crops. Wind borne uredospores from hills are lifted due to cyclonic winds and infect the crop in the plains during crop season.

**Management**
- Mixed cropping with suitable crops.
- Avoid excess dose of nitrogenous fertilizers.
- Spray Zineb at 2.5 kg/ha or Propioconazole @ 0.1%.
Grow resistant varieties like PBW 343, PBW 550, PBW 17

Loose smut - *Ustilago nuda tritici* (*Ustilago tritici*)

**Symptoms**

It is very difficult to detect infected plants in the field until heading. At this time, infected heads emerge earlier than normal heads. The entire inflorescence is commonly affected and appears as a mass of olive-black spores, initially covered by a thin gray membrane. Once the membrane ruptures, the head appears powdery.

Spores are dislodged, leaving only the rachis intact. In some cases remnants of glumes and awns may be present on the exposed rachis. Smutted heads are shorter than healthy heads due to a reduction in the length of the rachis and peduncle. All or a portion of the heads on an infected plant may exhibit these symptoms. While infected heads are shorter, the rest of the plant is slightly taller than healthy plants. Prior to heading affected plants have dark green erect leaves. Chlorotic streaks may also be visible on the leaves.

**Disease Cycle**

Ears of infected plants emerge early. The spores released from the infected heads land on the later emerging florets and infect the developing seed. Infection during flowering is favored...
by frequent rain showers, high humidity and temperature. The disease is internally seed borne, where pathogen infects the embryo in the seed.

Management

Treat the seed with Vitavax @ 2g/kg seed before sowing. Burry the infected ear heads in the soil, so that secondary spread is avoided.

Flag smut - *Urocystis tritici*

**Symptoms**

The symptoms can be seen on stem, clum and leaves from late seedling stage to maturity. The seedling infection leads to twisting and drooping of leaves followed by withering. Grey to grayish black sori occurs on leaf blade and sheath. The sorus contains black powdery mass of spores.
Pathogen

Aggregated spore balls, consisting 1-6 bright globose, brown smooth walled spores surrounded by a layer of flat sterile cells.

Favourable Conditions

- Temperature of 18-24°C.
- Relative humidity 65% and above.

Disease cycle

Seed and soil borne. Smut spores are viable for more than 10 years.

Management

- Treat the seeds with carboxin at 2g /kg.
- Grow resistant varieties like Pusa 44 and WG 377.

Hill bunt or Stinking smut - *Tilletia caries* / *T.foetida*
**Symptoms**

The fungus attacks seedling of 8-10 days old and become systemic and grows along the tip of shoot. At the time of flowering hyphae concentrate in the inflorescence and spikelets and transforming the ovary into smut sorus of dark green color with masses of chlamydospores. The diseased plants mature earlier and all the spikelets are affected.

**Pathogen**

Reticulate, globose and rough walled. No resting period. Germinate to produce primary sporidia which unite to form ‘H’ shaped structure.

**Life cycle**

The spores on the seed surface germinate along with the seed. Each produces a short fungal thread terminating in a cluster of elongated cells. These then produce secondary spores which infect the coleoptiles of the young seedlings before the emergence of the first true leaves. The mycelium grows internally within the shoot infecting the developing ear. Affected plants develop apparently normally until the ear emerges when it can be seen that grain sites have been replaced by bunt balls. In India disease occurs only in Northern hills, where wheat is grown.
Favourable Conditions

- Temperature of 18-20°C.
- High soil moisture.

Disease cycle

Externally seed borne

Management

- Treat the seeds with carboxin or carbendazim at 2g/kg.
- Grow the crop during high temperature period.
- Adopt shallow sowing.
- Grow resistant varieties like Kalyan sona, S227, PV18, HD2021, HD4513 and HD4519.

Karnal bunt - *Neovassia indica*

Symptoms

Symptoms of Karnal bunt are often difficult to distinguish in the field due to the fact that incidence of infected kernels on a given head is low. There may be some spreading of the glumes
due to sorus production but it is not as extensive as that observed with common bunt. Symptoms are most readily detected on seed after harvest.

The black sorus, containing dusty spores is evident on part of the seed, commonly occurring along the groove. Heavily infected seed is fragile and the pericarp ruptures easily. The foul, fishy odor associated with common bunt is also found with karnal bunt. The odor is caused by the production of trimethylamine by the fungus. Seed that is not extensively infected may germinate and produce healthy plants.

**Foot rot -** *Pythium graminicolum* **and** *P. arrhenomanes*

**Symptoms**
The disease mainly occurs in seedlings and roots and rootlets become brown in colour. Seedlings become pale green and have stunted growth. Fungus produces sporangia and zoospores and oospores.

**Favourable Conditions**
Wet weather and high rainfall.

**Disease cycle**
Through soil and irrigation water.

**Management**
- Follow crop rotation.
- Treat the seeds with Carboxin or Carbendazim at 2g/kg.

**Powdery mildew - *Erysiphe graminis var. tritici***

**Symptoms**
Greyish white powdery growth appears on the leaf, sheath, stem and floral parts. Powdery growth later become black lesion and cause drying of leaves and other parts.

**Pathogen**
Fungus produces septate, superficial, hyaline mycelium on leaf surface with short conidiophores. The conidia are elliptical, hyaline, single celled, thin walled and produced in
chains. Dark globose cleistothecia containing 9-30 asci develop with oblong, hyaline and thinwalled ascospores.

Disease cycle

Fungus remains in infected plant debris as dormant mycelium and asci. Primary spread is by the ascospores and secondary spread through airborne conidia.

Favourable Conditions

- Temperature of 20-21°C.

Management

- Spray Wettable Sulphur 0.2% or Carbendazim @ 500 g/ha

Leaf blight - *Alternaria triticina* / *Bipolaris sorokiniana*

Symptoms

Reddish brown oval spots appear on young seedlings with bright yellow margin. In severe cases, several spots coalesce to cause drying of leaves. It is a complex disease, having association of *A.triticina, B.sorokiniana* and *A. alternate.*
**Disease cycle**

Primary spread is by externally seed-borne and soil borne conidia. Secondary spread by air-borne conidia.

**Favourable Conditions**

- Temperature of 25°C and high relative humidity.

**Management**

- Spray the crop with Mancozeb or Zineb at 2 kg/ha.

**Other minor diseases**

Helminthosporium leaf spot: *Helminthosporium* spp.

Tundu or yellow ear rot: *Corynebacterium tritici* + *Anguina tritici*

Seedling blight: *Rhizoctonia solani* and *Fusarium* sp

Sclerotinia rot: *Sclerotinia sclerotiorum*

Molya disease: *Heterodera avenae* ([Nematode](https://www.AgriMoon.Com))
4. Diseases of Pearl millet

Downy mildew - *Sclerospora graminicola*

**Symptoms**

Infection is mainly **systemic** and symptoms appear on leaves and inflorescence. The initial symptoms appear in seedlings at three to four leaf stages. The affected leaves show patches of light green to light yellow colour on the upper surface and the corresponding lower surface bears white downy growth of the fungus consisting of **sporangio phores** and **sporangia**. The yellow discolouration often turns to streaks along veins. 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 inflorescence of infected plants gets completely or partially malformed with florets converted into leafy structures, giving 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 contain masses of **oospores**.
Pathogen

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

Favourable Conditions

- Very high humidity (90%).
- Presence of water on the leaves
- Low temperature of 15-25°C favor the formation of sporangiophore and sporangia.

Disease cycle
The oospores remain viable in soil for 5 years or longer giving rise to the primary infection on seedlings. Secondary spread is through sporangia produced during rainy season. The dormant mycelium of the fungus is present in embryo of infected seeds.

**Management**

- Deep ploughing to bury the oospores.
- Roguing out infected plants.
- Adopt crop rotation.
- Grow resistant varieties WCC-75, Co7 and Co (Cu)9.
- Treat the seeds with Metalaxyl at 6g/kg.
- Spray Mancozeb 2 kg or Metalaxyl + Mancozeb at 1 kg/ha on 20th day after sowing in the field.

**Smut - *Tolyposporium penicillariae***

**Symptoms**

The pathogen infects few florets and transforms them into plump sori containing smut spores. The sori are larger than normal healthy grains and when the sori mature they become dark brown releasing millions of black smut spore balls.

**Pathogen**

The fungus is mostly confined to the sorus. The sori contain spores in groups and are not easy to separate. Each spore is angular or round and light brown.
Favourable Conditions
- High relative humidity.
- Successive cropping with pearlmillet.

Disease cycle
- The pathogen survives as spore balls in the soil and serves as primary source of inoculum. Secondary spread is by air-borne conidia.

Management
- The damage caused by the fungus is negligible.
- Removal and destruction of affected ear head will help in controlling the disease.

Rust - *Puccinia pennisetii*

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. The pustules may be formed on leaf sheath, stem and on peduncles. Later, telial formation takes place on leaf blade, leaf sheath and stem. While brownish uredia are exposed at maturity, the black telia remain covered by the epidermis for a longer duration.

Pathogen
*Uredospores* are oval, elliptic, sparsely echinulated and pedicellate. *Teliospores* are dark brown in colour, two celled, cylindrical to club shaped, apex flattened, broad at top and tapering towards base. The fungus is macrocyclic producing uredial and telial stages on pearlmillet and aecial and pycnial stages on brinjal.
Favourable Conditions

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

Disease cycle

Air-borne uredospores are the primary sources. The uredial stages also occur on several species of Pennisetum, which helps in secondary spread of the pathogen.

Management

Spray with Wettable Sulphur 3 kg or Mancozeb 2 kg/ha.

Ergot or Sugary disease - Claviceps fusiformis

Symptoms

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

The pathogen produces septate mycelium which produces conidiophores and is closely arranged. Conidia are hyaline and one celled. The sclerotia are small (3-8mm x 0.3-15mm) and dark grey but white inside.

Disease cycle

Sclerotia are viable in soil for 6-8 months. The primary infection takes place by germinating sclerotia present in the soil. Secondary spread is by insects or airborne 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 2 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 - Fungal complex
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 spot - *Rhizoctonia* spp.
Patch of light and dark, discoloured areas and often bearing fluffy to light brown fungal mats.
5. Diseases of Maize

Downy mildew/Crazy top
Sorghum downy mildew - *Peronosclerospora sorghi*
Philippine downy mildew - *Peronosclerospora philippinensis*
Crazy top - *Sclerophthora macrospora*

Symptoms

The most characteristic symptom is the development of chlorotic streaks on the leaves. Plants exhibit a stunted and bushy appearance due to shortening of the internodes. White downy growth is seen on the lower surface of leaf. Downy growth also occurs on bracts of green unopened male flowers in the tassel. Small to large leaves are noticed in the tassel. Proliferation of auxiliary buds on the stalk of tassel and the cobs is common (Crazy top).
Pathogen

The fungus grows as white downy growth on both surface of the leaves, consisting 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 (21-33°C)
- High relative humidity (90 per cent) and drizzling.
- Young plants are highly susceptible.

Disease cycle

The primary source of infection is through oospores in soil and also dormant mycelium present in the infected maize seeds. Secondary spread is through airborne conidia. Depending on the pathogen species, the initial source of disease inoculum can be oospores that over winter in the soil or conidia produced in infected, over wintering crop debris and infected neighboring plants. Some species that cause downy mildew can also be seed borne, although this is largely restricted to seed that is fresh and has high moisture content.

At the onset of the growing season, at soil temperatures above 20°C, oospores in the soil germinate in response to root exudates from susceptible maize seedlings. The germ tube infects the underground sections of maize plants leading to characteristic symptoms of systemic infection including extensive chlorosis and stunted growth. If the pathogen is seed borne, whole plants show symptoms. Oospores are reported to survive in nature for up to 10 years.

Once the fungus has colonised host tissue, sporangiophores (conidiophores) emerge from stomata and produce sporangia (conidia) which are wind and rain splash disseminated and initiate secondary infections. Sporangia are always produced in the night. They are fragile and can not be disseminated more than a few hundred meters and do not remain viable for more than a few hours.

Germination of sporangia is dependent on the availability of free water on the leaf surface. Initial symptoms of disease (chlorotic specks and streaks that elongate parallel to veins)
occur in 3 days. Conidia are produced profusely during the growing season. As the crop approaches senescence, oospores are produced in large numbers.

Management

- Deep ploughing.
- Crop rotation with pulses.
- Rogue out infected plants.
- Treat the seeds with metalaxyl at 6g/kg.
- Spray the crop with Metalaxyl + Mancozeb @ 1kg on 20th day after sowing.
- Grow resistant varieties and hybrids viz. CO1, COH1 and COH2.

Leaf blight - *Helminthosporium maydis* (Syn: *H. turcicum*)

Symptoms

The fungus affects the crop 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 in the centre with dark brown margins. The spots coalesce giving blighted appearance. The surface is covered with olive green velvety 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 septa.
Favourable Conditions

• Optimum temperature for the germination of conidia is 8 to 27°C provided with freewater on the leaf.
• Infection takes place early in the wet season.

Disease cycle

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 2 kg or captan 1 kg/ha.

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.

Disease cycle

Primary source of inoculums is uredospores surviving on alternate hosts viz., Oxalis corniculata and Euchlaena mexicana.

Management

• Remove the alternate hosts.
• Spray Mancozeb at 2 kg/ha.

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. The smutted plants are stunted produce little yield and remain greener than that of the rest of the plants.

Pathogen

Smut spores are produced in large numbers which are reddish brown to black, thick walled, finely spined, spherical.

Favourable Conditions

- Low temperature favours more infection and this fungus also infects the sorghum

Disease cycle

The smut spores retain its viability for two years. The fungus is externally seedborne 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.

Charcoal rot - *Macrophomina phaseolina* (*Rhizoctonia bataticola*)

Symptoms

The affected plants exhibit wilting symptoms. The stalk of the infected plants can be recognized by grayish streak. The pith becomes shredded and grayish black minute sclerotia develop on the vascular bundles. Shredding of the interior of the stalk often causes stalks to
break in the region of the crown. The crown region of the infected plant becomes dark in colour. **Shredding** of root bark and disintegration of root system 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)

**Disease cycle**

The fungus has a wide host range, attacking sorghum, pearl millet, finger millet and pulses. It survives for more than 16 years in the infected plant debris. The primary source of infection is through soil-borne sclerotia. The pathogen also attacks many other hosts, which helps in its perpetuation. Since the fungus is a facultative parasite it is capable of living saprophytically on dead organic tissues, particularly many of its natural hosts producing sclerotial bodies. The fungus over winters as a **sclerotia** in the soil and infects the host at susceptible crop stage through roots and proceeds towards stem.

**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

Bacterial Stalk rot - *Erwinia dissolvens*

**Symptoms**

The basal internodes develop soft rot and give a water soaked appearance. A mild sweet fermenting odour accompanies such rotting. Leaves some time show signs of wilting and affected plants topple down in few days. Ears and shank may also show rot. They fail to develop further and the ears hang down simply from the plant.

**Disease cycle**

Borer insects play a significant role in initiation of the disease. The organism is soil borne and makes its entry through wounds and injuries on the host surface. The organism survives saprophytically on debris of infected materials and serves primary inoculum in the next season.

Mosaic - *Maize mosaic potyvirus*

**Symptoms**

Symptoms appear as chlorotic spots, which gradually turn into stripes covering entire leaf blade. Chlorotic stripes and spots can also develop on leaf sheaths, stalks and husks. Moderate to severe rosetting of new growth is observed. Size of stalk, leaf blades and tassel tend to be normal in late infection.
Pathogen

It is caused by *Maize mosaic potyvirus*. Virions are flexuous, 750-900nm long, ssRNA genome.

Disease cycle

It is transmitted in nature by leaf hopper vector, *Perigrimus maidis*.

Brown spot - *Physoderma maydis*

Water soaked lesions, which are oval, later turn into light green and finally brown.
6. Diseases of Sugarcane

**Red rot - Colletotrichum falcatum** (Perfect stage: *Physalospora tucumanensis*)

**Symptoms**

The first external symptom appears mostly on third or fourth leaf which withers away at the tips along the margins. Typical symptoms of red rot are observed in the internodes of a stalk by splitting it longitudinally. These include the reddening of the internal tissues which are usually elongated at right angles to the long axis of the stalk. The presence of cross-wise white patches are the important diagnostic character of the disease. The diseased cane also emits acidic-sour smell. As the disease advances, the stalk becomes hollow and covered with white mycelial growth.

Later the rind shrinks longitudinally with minute black, velvety fruiting bodies protruding out of it. The pathogen also produces tiny reddish lesions on the upper surface of leaves with dark dots in the centre. The lesions are initially blood red with dark margins and later on with straw coloured centres. Often the infected leaves may break at the lesions and hang down, with large number of minute black dots.

**Pathogen**

The fungus produces thin, hyaline, septate, profusely branched hyphae containing oil droplets. The fungus produces black, minute velvety acervuli with long, rigid bristle-like, septate setae. **Conidiophores** are closely packed inside the acervulus, which are short, hyaline and single celled. The **conidia** are single celled, hyaline, falcate, **granular** and **guttulate**. Fungus
also produces large number of globose and dark brown to black perithecia with a papillate ostiole.

Asci are clavate, unitunicate and eight-spored. Large number of hyaline, septate, filiform paraphyses is also present among asci. Ascospores are ellipsoid or fusoid, hyaline, straight or slightly curved and unicellular which measure 18-22 μm x 7-8μm.

Favourable Conditions

- Monoculturing of sugarcane.
- Successive ratoon cropping.
- Water logged conditions and injuries caused by insects.

Disease cycle

The fungus is sett-borne and also persists in the soil on the diseased clumps and stubbles as chlamydospores and dormant mycelium. The primary infection is mainly from infected setts. Secondary spread in the field is through irrigation water and cultivation tools. The rain splash, air currents and dew drops also help in the spread of conidia from the diseased to healthy plants in the field. The fungus also survives on collateral hosts Sorghum vulgare, S. halepense and Saccharum spontaneum. If the conidia settle on the leaves they may germinate and invade the leaves through various types of wounds. Stem infection may take place through insect bores and root primordia. The soil-borne fungus may also enter the healthy setts through cut-ends, and
cause early infection of the shoots. Though the perfect stage of the fungus has been observed in
nature, the role of ascospores in the disease cycle is not understood.

**Management**

- Adopt crop rotation by including rice and green manure crops.
- Select the setts from the disease free fields or disease free areas.
- Avoid ratooning of the diseased crop.
- Soak the setts in 0.1% Carbendazim or Triademefon 0.05% solution for 15 minutes
  before planting.
- Grow resistant varieties CO 62198, CO 7704 and moderately resistant varieties CO 8001,
  CO8201.
- Setts can be treated with aerated steam at 52 °C for 4 to 5 hours and by moist hot air at
  54°C for 2 hours.

**Smut - *Ustilago scitaminea***

**Symptoms**

It is a culmiculous smut. The affected plants are stunted and the central shoot is converted
into a long whip-like, dusty black structure. The length of the whip varies from few inches to
several feet. In early stages, this structure is covered by a thin, white papery membrane. The
whip may be straight or slightly curved.

On maturity it ruptures and millions of tiny black smut spores (*teliospores*) are liberated
and disseminated by the wind. Affected plants are usually thin, stiff and remain at acute angle.
The whip like structure, representing the central shoot with its various leaves, may be produced
by each one of the shoots/tillers arising from the clump.
The smutted clumps also produce mummified arrows in which lower portion consisted of a normal inflorescence with typical flowers and the upper portion of the rachis is converted into a typical smut whip. Occasionally smut sori may develop on the leaves and stem.

**Pathogen**

The fungal hyphae are primarily intercellular and collect as a dense mass between the vascular bundles of host cell and produce tiny black spores. The thin membrane which covers the smut whip represents the host epidermis. The smut spores are light brown in colour, spherical, echinulated and measuring 6.5- 8.5µm in diameter. Smut spores germinate to produce 3-4 celled, hyaline promycelium and produce 3-4 sporidia which are hyaline and oval shaped with pointed ends.

**Favourable Conditions**

- Monoculturing of sugarcane.
- Continuous ratooning and dry weather during tillering stage.

**Disease cycle**

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

**Management**

- Plant healthy setts taken from disease free area.
- Remove and destroy the smutted clump (collect the whips in a thick cloth bag/polythene bag and immerse in boiling water for 1 hr to kill the spores).
- Discourage ratooning of the diseased crops having more than 10 per cent infection.
- Follow crop rotation with green manure crops or dry fallowing.
- Grow redgram as a companion crop between 2 rows of sugarcane.
- Grow resistant varieties like Co 7704 and moderately resistant varieties COC 85061 and COC 8201.

**Sett rot or Pineapple disease - *Ceratocystis paradoxa***

**Symptoms**

The disease primarily affects the setts usually two to three weeks after planning. The fungus is soil-borne and enters through cut ends and proliferates rapidly in the parenchymatous tissues. The affected tissues first develop a reddish colour which turns to brownish black in the later stages. The severely affected setts show internodal cavities covered with the mycelium and abundant spores. A characteristic pineapple smell is associated with the rotting tissues. The setts may decay before the buds germinate or the shoots may die after reaching a height of about 6-12 inches. Infected shoots are stunted.
**Pathogen**

The fungus produces both macroconidia and microconidia. Conidiophores are linear, thin walled with short cells at the base and a long terminal cell. The microconidia are hyaline when young but become almost black at maturity. They are thinwalled, cylindrical and produced endogenously in chains in the long cells of conidiophores and pushed out in succession. Macroconidia are produced singly or in chains on a short, lateral conidiophores. Macroconidia are spherical or elliptical or truncate or pyriform and are hyaline to olive green or black measuring 16-19x10-12 um.

The fungus also produces chlamydospores on short lateral hyphae in chains, which are oval, thick walled and brown in colour. The perithecia are flask shaped with a very long neck. The bulbous base of the perithecia is hyaline or pale yellow, 200-300μm in diameter and ornamented with irregularly shaped, knobbed appendages. The ostiole is covered by numerous pale-brown, erect tapering hyphae. Asci are clavate and measures 25x10μm and ascospores are single celled, hyaline, ellipsoid, more convex on one side, measures 7-10 x 2.5-4μm.

**Favourable Conditions**

- Poorly drained fields.
- Heavy clay soils
- Temperature of 25-30º C
- Prolonged rainfall after planting.

**Disease Cycle**

The fungus survives as conidia and chlamydospores in the soil and in the infected, buried cane tissues. The inoculum moves from field to field through wind-borne conidia or irrigation or rain water. Inside the sett it spreads rapidly through the parenchymatous tissues and causes sett rot.

The insects like cane borer (*Diatraea dyari*) also helps in the spread of the disease. The pathogen also survives on coconut, cocoa, mango, papaya, coffee, maize and arecanut. Insects also play a part in the dissemination of the pathogen.

**Management**

- Soak the setts in 0.05% Carbendazim 15 minutes.
- Use long setts having 3 or 4 buds.
- Provide adequate drainage during rainy seasons.
**Wilt - *Cephalosporium sacchari***

**Symptoms**

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

**Pathogen**

The fungal mycelium is hyaline, septate and thin walled. The conidiophores are simple or branched and produce single celled, hyaline, oval to elliptical microconidia.

**Favourable Conditions**

- High day temperature (30-35°C).
- Low humidity (50-60%).
- Low soil moisture and alkaline soils.
- Excess doses of nitrogenous fertilizers.

**Disease Cycle**

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

**Management**

- Select the seed material from the disease-free plots.
- Avoid the practice of ratooning in diseased fields.
- Burn the trashes and stubbles in the field.
- Grow coriander or mustard as a companion crop in the early stages of crop.
- Dip the setts in 40ppm Boran or Manganese for 10 minutes or in 0.25% Emisan or 0.05% Carbendazim for 15 minutes.
Rust - *Puccinia erianthi* (Syn: *P. melanocephala* and *P. kuehnii*)

**Symptoms**

Minute, elongated, yellow spots (uredia), usually 2-10 x 1-3 mm appear on both the surfaces of young leaves. The pustules turn to brown on maturity. Late in the season, dark brown to black telia appear on the lower surface of leaves. In severe cases, the uredia also appear on the leaf sheath and the entire foliage looks brownish from a distance.

**Pathogen**

The mycelium is hyaline, branched and septate. *P.kuehnii* produces ovoid or pear shaped, single celled uredospores measuring 29-57 x 8-37μm with apical thickening and golden yellow in colour. Teliospores are produced in scanty which are yellow in colour, club shaped, two celled, smooth walled and measuring 24-34 X 18-25μm single celled, dark yellow coloured with 4 equatorial pores.

Teliospores are produced in abundance, which are pale to brick colour, two celled, smooth walled and slightly constricted at septum. Occurrence of pycnial and aecial stages and the role of alternate host are unknown.

**Favourable Conditions**

- Temperature of 30°C.
- Humidity between 70 and 90 per cent.
- High wind velocity and continuous cloudiness.

**Disease Cycle**

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

• Remove the collateral hosts.
• Spray Tridemorph 1 kg or Mancozeb 2 kg/ha.

Gummosis - *Xanthomonas axonopodis pv. vasculorum*

Symptoms

The bacterium produces two distinct types of symptoms. On the mature leaves, longitudinal stripes or streaks, 3-7mm in width and several cm in length, appear around the affected veins, near the tip. Initially these stripes are pale yellow in colour, later turn to brown. The affected tissues slowly dry up.

The infected canes are stunted with short internodes, giving a bushy appearance. When such canes are cut transversely or split open longitudinally, a dull yellow bacterial ooze comes out from the cut ends and bacterial pockets are seen inside the slitted cane. The fibro vascular bundles are deep red and internodal cavities formed in the severe cases are filled with yellow coloured bacterial gums.

Pathogen

The bacterium is a short rod, Gram negative, non spore forming measuring 1.0 to 1.5µm X 0.4 to 0.5µm, with a single polar flagellum. It is facultative anaerobe and it produces yellow slimy growth.

Disease Cycle

The bacterium remains viable in the soil as well as in infected canes. The primary transmission is through naturally affected diseased setts or through soil-borne contamination. The secondary spread may be through wind splashed rain, harvesting implements, animals and insects. The bacterium can survive in the insect's body for a long time and in this way may be transmitted long distances. On entry into the host the bacterium reaches the vascular tissues and becomes systemic. The bacterium also perpetuates on maize, sorghum, pearl millet and other weed hosts, which also serve as sources of inoculum.

Management

• Remove and burn the affected clumps and the stubbles in the field. Select setts from disease free areas.
• Avoid growing collateral hosts like maize, sorghum and pearl millet near the sugarcane fields.

**Red stripe - *Pseudomonas rubrilineans***

**Symptoms**

The disease first makes its appearance on the basal part of the young leaves. The stripes appear as water soaked, long, narrow chlorotic streaks and become reddish brown in few days. These stripes are 0.5 to 1 mm in width and 5-100 mm in length, run parallel to the midrib. The stripes remain confined to lower half of the leaf lamina and whitish flakes spread to growing points of the shoot and yellowish stripes develop, which later turn reddish brown. The rotting may commence from the tip of the shoot and spreads downwards. The core is discoloured to reddish brown and shrivelled and form cavity in the centre. In badly affected fields, a foul and nauseating smell appears.

**Pathogen**

The bacterium is a short rod (0.7 X 1.67 µm), gram negative, non capsulate with a polar **flagellum**.

**Favourable Conditions**

• Continuous ratooning and prolonged rainy weather with low temperature (25°C)

**Disease cycle**
The pathogen remains viable in the soil and infected plant residues. The bacterium also survives on sorghum, pearl millet, maize, finger millet and other species of Saccharum. The bacterium primarily spreads through infected canes. The secondary spread is mainly through rainsplash, irrigation water and insects. Infected parenchymatous cells may collapse and normal functioning of the plant parts may fail. Several grasses, including ragi and bajra, have been reported to be infected by the bacteria and these hosts may also play a role in the perpetuation and spread of the pathogen.

Management

- Whenever the disease is noticed; the affected plants should be removed and burnt.
- Growing resistant varieties Select setts from the healthy fields.
- Avoid growing collateral hosts near the sugarcane fields.

Sugarcane Mosaic - Sugarcane mosaic potyvirus

Symptoms

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

Pathogen

Sugarcane mosaic potyvirus is a flexuous rod, 650-770nm long X 12-15nm with ss RNA genome.

Disease cycle
The virus is mainly transmitted through infected canes used as seed. The virus also infects *Zea mays* and a number of other cereals (*Sorghum vulgare, Pennisetum americanum, Eleusine indica, Setaria lutescens, Echinochloa crusgalli, Stenotaphrum secondatum, Digitaria didactyla*) which serve as potential sources of virus inoculum. The virus also spreads through viruliferous aphids *viz.*, *Melanaphis sacchari, Rhopalosiphum maidis* in a non-persistant manner. The virus is also sap-transmissible. The *incubation period* varies from 7 to 20 days, depending upon the host variety and virus strain. The symptoms may be prominent or masked depending on the environmental conditions and variety.

**Management**

- Roguing of infected plants and use of disease free planting material.
- Chemical sprays to manage the insect vector population in early crop stage.
- Grow mosaic-resistant or, at least, tolerant varieties.
- Breeding mosaic-resistant varieties is needed.
- *Saccharum spontaneum* L. and *S. barberi* (Jesweit) carry resistance to mosaic and so varieties with this background must be preferred.
- Rogue out the diseased clumps periodically. Select setts from the healthy fields as the virus is sett-borne Aerated Steam Therapy (AST) at 56°C for 3 hrs, for setts before planting is advised.

**Grassy shoot - Phytoplasma**

**Symptoms**

The disease appears nearly two months after planting. The disease is characterised by the production of numerous lanky tillers from the base of the affected shoots. Leaves become pale yellow to completely chlorotic, thin and narrow. The plants appear bushy and ‘grass-like’ due to reduction in the length of internodes premature and continuous tillering. The affected clumps are stunted with premature proliferation of auxillary buds. Cane formation rarely occurs in the affected clumps, if formed, thin with shorter internodes having aerial roots at the lower nodes. The buds on such canes usually papery and abnormally elongated.

**Pathogen**
The disease is caused by a phytoplasma. Two types of bodies are seen in ultrathin sections of phloem cells of infected plants. The spherical bodies of 300-400 nm diameter and filamentous bodies of 30-53 mm diameter in size.

Disease cycle

The primary spread of the phytoplasma is through diseased setts and cutting knifes. The pathogen is transmitted secondarily by aphids viz., Rhopalosiphum maydis, Melanaphis sacchari and M. idiosacchari. Sorghum and maize serves as natural collateral hosts.

Management

- Eradication of diseased parts as soon as symptoms are seen.
- Avoid selection of setts from diseased area.
- Pre-treating the healthy setts with hot water at 52°C for 1 hour before planting
- Treating them with hot air at 54°C for 8 hours.
- Spraying the crop twice a month with insecticides.

Ratoon stunting - *Clavibacter xyli sub sp. xyli* (Rickettsia Like Organism - RLO)

Symptoms

Diseased clumps usually display stunted growth, reduced tillering, thin stalks with shortened internodes and yellowish foliage. Orange-red vascular bundles in shades of yellow at the nodes are seen in the infected canes.

Pathogen

The pathogen (*Clavibacter xyli sub sp. xyli*) is a RLO known to be present in the xylem cells of infected plants. They are small, thin, rod shaped or coryneform (0.15 to 0.32μm wide and 1.0-2.7μm long) and Gram positive.
Disease cycle

The primary spread is through the use of diseased setts. The disease also spreads through harvesting implements contaminated with the juice of the diseased canes. Maize, sorghum, Sudan grass and Cynodon serves as collateral hosts for the pathogen.

Management

- Select the setts from disease free fields or from disease free commercial nursery.
- Remove and burn the clumps showing the disease incidence.
- Treat the setts before planting, as specified for grassy shoot disease.

Minor diseases

Damping-off - *Pythium aphanidermatum, P. debaryanum, P. graminicola, P. ultimum*

Germinating seeds and young seedlings are attacked and killed in pre-emergence phase and seedlings show water soaked lesions at collar region, leading to withering and drying in post emergence stage.

Downy mildew - *Peronosclerospora sacchari*

Downy fungal growth with yellow stripes on upper surface, shredding of older leaves, rapid elongation of internodes of affected canes.

Eye spot - *Helminthosporium sacchari*

The water soaked spot develops on leaves, later elongated and turns to form “eye” shaped spot with reddish brown centre surrounded by straw yellow tissues.

Ring spot –*Leptosphaeria sacchari*

The water soaked spots appear on leaves and turns to straw colour later surrounded by a thin reddish brown band and a diffused discoloration zone.

Leaf scald - *Xanthomonas albilineans*
Diseases of Field Crops and Their Management

Whitish lines appear on the leaves, run to the full length of leaves and sheaths. Later leaves wither and dry from tip down-wards, gives a scald appearance to the clump. Sprouting of lateral buds of the matured canes occurs in acropetal fashion.

White leaf - *Phytoplasma*

Sugarcane white leaf is of minor importance and is caused by phytoplasma. The plants exhibit pure white leaves, stripped leaves and mottled leaves. Its vector is *Matsumuratettix hiroglyphicus*. 
7. Diseases of Turmeric

**Rhizome Rot - *Pythium graminicolum***

**Symptoms**

Starting from the margins the leaves get dried up, collar region of pseudo stem becomes soft and water-soaked and plants collapse. The rhizomes decay as a result of the attack of the fungus.

**Disease cycle**

Pathogen is **soil-borne**, therefore primary inoculum comes from soil. Infected rhizomes used for seed purpose may also transmit the disease. Irrigation water from diseased field helps in the spread of the disease.

**Management**

- Seed material should be selected from disease free areas.
- Avoid water stagnation in the field. Light soil may be preferred and drainage facility to be ensured.
- Grow tolerant varieties like Suguna and Sudarshan.
- Crop rotation to be followed.
- Deep plough in summer. Planting is to be done in ridge and furrow method.
- Remove diseased plants and the soil around plants to be drenched with Mancozeb (3gm/lit) or 3gm Ridomil M.Z.
- Spray the crop with Mancozeb (2.5g/lit) or Carbendazim (1g/lit) +1ml sandovit.
• Keep rhizomes in 3g Metalaxyl or 3g Mancozeb mixed in one litre of water for one hour and shade dry before planting.

**Leaf Spot - *Colletotrichum capsici***

**Symptoms**

Oblong brown spots with grey centres are found on leaves. The spots are about 4-5 cm in length and 2-3 cm in width. In advanced stages of disease black dots representing fungal acervuli occur in concentric rings on spot. The grey centers become thin and gets teared. Severely effected leaves dry and wilt. They are surrounded by yellow halos. Indefinite number of spots may be found on a single leaf and as the disease advances; spots enlarge and cover a major portion of leaf blade.

![Symptoms](image)

**Favorable condition**

• The disease is usually appears in October and November
• Relative humidity of 80% and temperatures of 21 – 230C favours the primary infection

**Disease cycle**

The fungus is carried on the scales of rhizomes which are the source of primary infection during sowing. The secondary spread is by wind, water and other physical and biological agents. The same pathogen is also reported to cause leaf-spot and fruit rot of chilli where it is transmitted through seed borne infections. If chilli is grown in nearby fields or used in crop rotation with turmeric, the pathogen perpetuates easily, building up inoculum potential for epiphytotic outbreaks.

**Management**
• Select seed material from disease free areas.
• Treat seed material with mancozeb @ 3g/litre of water or carbendazim @ 1 g/litre of water, for 30 minutes and shade dry before sowing.
• Spray mancozeb @ 2.5 g/litre of water or carbendazim @ 1g/litre; 2-3 sprays at fortnightly intervals.
• The infected and dried leaves should be collected and burnt in order to reduce the inoculum source in the field.
• Spraying Blitox or Blue copper at 3 g/l of water was found effective against leaf spot.
• Crop rotations should be followed whenever possible.
• Cultivate tolerant varieties like Suguna and Sudarshan.

Leaf Blotch  *Taphrina maculans*

**Symptoms**

This disease usually appears on lower leaves in October and November. The individual spots are small 1-2 mm in width and are mostly rectangular in shape. The disease is characterized by the appearance of several spots on both the surfaces of leaves, being generally numerous on the upper surface. They are arranged in rows along the veins. The spots coalesce freely and form irregular lesions. They first appear as pale yellow discolorations and then become dirty yellow in colour. The infected leaves disort and have reddish brown appearance.

**Disease cycle**

The fungus is mainly air borne and primary infection occurs on lower leaves with the inoculum surviving in dried leaves of host, left over in the field. The ascospores discharged from
successively maturing asci infect fresh leaves without dormancy, thus causing secondary infection. Secondary infection is most dangerous than primary one causing profuse sprouting all over the leaves. The pathogen persists in summer by means of ascogenous cells on leaf debris, and dessicated ascospores and blastospores in soil and among fallen leaves.

Management

• Select seed material from disease free areas.
• Treat the seed material with Mancozeb @ 3g/litre of water or Carbendazim @ 1 g/litre of water for 30 minutes and shade dry before sowing.
• Spray mancozeb @ 2.5 g/litre of water or Carbendazim @ 1g/litre; 2-3 sprays at fortnightly intervals.
• The infected and dried leaves should be collected and burnt in order to reduce the inoculum source in the field.
• Spraying Copper oxy chloride at 3 g/l of water was found effective against leaf blotch.
• Crop rotations should be followed whenever possible.

Minor diseases

a. Dry rot - *Rhizoctonia bataticola*
b. Leaf spot - *Cercospora curcuma*
c. Leaf Blight - *Rhizoctonia solani*
d. Brown rot - It is a complex disease caused by the nematode *Pratylenchus* sp. associated with *Fusarium* sp.
8. Diseases of Tobacco

Damping off - *Pythium aphanidermatum*

**Symptoms**

The pathogen attacks the seedlings 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 favorable conditions, the entire seedlings in the nursery are killed within 3 to 4 days. A thick weft of mycelium may be seen on the surface of the soil.

**Pathogen**

The fungus produces thick, hyaline, thin walled, non-septate mycelium. It produces irregularly lobed *sporangia* which germinate to produce vesicle containing zoospores. The *zoospores* are kidney shaped and biflagellate. *Oospores* spherical, light to deep yellow or yellowish brown coloured, measuring 17-19μm 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 24 C) and low soil temperature of about 20°C.

**Disease cycle**

The pathogen 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 at 10 days interval commencing from 20 days after germination.

**Black shank - *Phytophthora parasitica var. nicotianae***

**Symptoms**

The pathogen may affect the crop at any stage of its growth. Even though all parts are affected, the disease infects chiefly the roots and base of the stem. 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 8-9µm. The fungus also produces globose and thick walled **chlamydospores**, measuring 27-42µm in diameter. **Oospores** are thick walled, globose, smooth and light yellow coloured, measuring 15-20µm in diameter.

**Favourable Conditions**

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

**Disease cycle**

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

**Management**

- Cover the seed beds with paddy husk or groundnut shell at 15-20 cm thick layer and burn.
- 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 Captan or 0.2 per cent Copper oxychloride or 1 per cent Bordeaux mixture and repeat after 10 days.
• Select healthy, disease free seedlings for transplanting.
• Remove and destroy the affected plants in the field.
• Spray Mancozeb 2 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.

Frog eye spot - *Cercospora nicotianae*

**Symptoms**

The disease appears mostly on mature, lower leaves as small ashy grey spots with brown border. The typical spots have a white centre, surrounded in succession by grey, brown portions with a dark brown to black margin, resembling the eyes of a frog. Under favorable 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.

**Favorable Conditions**

• Temperature of 20-30°C.
High humidity (80-90 per cent).

Close spacing, frequent irrigation and excess application of nitrogenous fertilizers.

**Disease cycle**

The pathogen is seed-borne and 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.

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

**Symptoms**

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

**Pathogen**

The fungus is *ecotrophic* and produces hyaline, septate and highly branched mycelium. Short, stout and hyaline *conidiophores* arise from the mycelium and bear conidia in chains. The conidia are barrel shaped or cylindrical, hyaline and thin walled. *Cleistothecia* are black, spherical with no ostiole, with numerous densely-woven septate, brown-coloured appendages. They contain 10-15 asci which are ovate with a short stalk. Each ascus contains two ascospores which are oval to elliptical, thinwalled, hyaline and single celled.
Favourable Conditions

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

Disease cycle

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

Brown spot - *Alternaria longipes*

Symptoms

Brown spot in contrast to frog-eye spot is not normally observed in the nursery but is very much prevalent in the field. Initially it appears on lower and older leaves as small brown, circular lesions, which
spread, to upper leaves, petioles, stalks and capsules even. In warm weather (30˚C) under high humidity, the leaf spots enlarge, 1-3 cm in diameter, centres are necroses and turn brown with characteristic marking giving target board appearance with a definite outline. In severe infection spots enlarge, coalesce and damage large areas making leaf dark-brown, ragged and worthless. On leaves nearing maturity, leaf spots are surrounded by bright yellow halo, due to production of toxin ‘alternin’ by the fungus.

Symptoms

Disease cycle

The fungus over summers in the soil as mycelium in the diseased plant debris such as stems of tobacco, weeds and other hosts. Under favourable weather in the next season conidial production starts which infect the lowermost leaves. As the season progresses, repeated infection cycles of the fungus attack healthy tissues of all aerial parts of tobacco of any age under high humidity. There is enormous spore density in the air near the end of the harvesting. Fungus persists as a mycelium in dead tissue for several months.

Management

• Removal and destruction of diseased plant debris can check the primary infection promptly.
• Continuous growing of tobacco after tobacco must be avoided in the heavily infected fields.
• Weekly, spraying of fungicides such as Maneb or Zineb @ 2g/ha or Benomyl or Thiophanate methyl at 1kg/ha.
**Anthracnose - Colletotrichum tabacum**

**Symptoms**

Initially, infection starts on lower leaves as pale-brown circular spots of 0.5 mm diameter with papery depressed centres outlined by slightly raised brown margin. The leaf-spots may remain small with white areas in the centre or coalesce to form large necrotic lesions. Under continuous humid weather, dark brown or black, elongated, sunken necrotic lesions appear on midrib, petiole and stem resulting in petiole and stem rot. Such seedlings do not establish in the field if planted. Primary infection starts from affected bits of aerial parts left in the soil in the previous season. The pathogen is not seed-borne but persists in the soil on dried plant debris.

**Management**

- Raised seed beds and rabbing with farm wastes help in reducing the initial infection
- Removal and destruction of all diseased debris minimises the pathogen in the soil.
- Rogueing diseased seedlings especially with necrotic lesions on stem
- Protective spraying with Bordeaux mixture at 1.0% (2-2-500) or Zineb @ 2 kg/ha

**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.
Symptoms

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

**Pathogen**

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

**Favourable Conditions**

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

**Disease cycle**

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.

**Tobacco mosaic** - *Tobacco mosaic virus (TMV)*

**Symptoms**
The disease begins 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 associated more with the veins, turning into irregular blisters.

The early infected plants in the season are usually 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

The disease is caused by *Tobacco mosaic tobamovirus*. It is a rigid rod measuring 300 X 150-180 nm with a central hollow tube of about 4nm diameter with ssRNA as its genome.

Disease cycle

The virus spreads most rapidly by contact wounds, sap and farm implements and operators. The virus remains viable in the plant debris in the soil as the source of inoculum as the longevity of the virus is very high. It is capable of remaining infective when stored dry for over 50 years. The virus has a wide host range, affecting nearly 50 plant species belonging to nine different families. The virus is not seed-transmitted in tobacco but tomato seeds transmit the virus. No insect vector known to 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 botanical 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.

**Leaf curl - *Tobacco leaf curl virus* (TLCV)**

**Symptoms**

The infections may occur at any stage, when young plants are infected the entire plant remains very much dwarfed. Curling of leaves with clearing and thickening of veins; twisting of petioles; puckering of leaves; rugose and brittle and development of enations are the important symptoms of tobacco leaf curl disease. Three forms of leaf curl expression are observed. First the leaf margins curl downward towards the dorsal side and show thickening of veins with enation on the lower surface. Second *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. Third the transparent symptom shows the curling of leaves towards the ventral side with clearing of the veins and enations are absent.
Pathogen

It is caused by Tobacco leaf curl geminivirus. Virions are geminate, non- enveloped, 18 nm diameter circular ssDNA genome. The virus is a white fly transmitted Geminivirus with ssDNA as genome.

Disease cycle

The virus has a narrow host range in eight plant families. The virus is not transmissible through sap or seed. The whitefly, Bemisia tabaci is the vector. Due to wide host range of the virus many other plants are acting as source of inoculums.

Management

- Remove and destroy the infected plants.
- Rogue out the reservoir weed hosts which harbour the virus and whiteflies. Planting tobacco crop during the crop periods when the vector population is low.
- Spray Methyldemeton at 0.1 to 0.2 per cent to control the vectors.

Phanerogamic parasite

Broom rape - Orobanche cernua var. desertorum

Symptoms

The affected tobacco plants are stunted and show withering and drooping of leaves to wilting. These indicate 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.

Disease cycle

The seeds of the parasite remain dormant in the soil for several years. Primary infection occurs from the seeds in the soil. The seeds spread from field to field by irrigation water, animals, human beings and implements. The dormant seeds are stimulated to germinate by the root exudates of tobacco and attach itself, to the roots by forming haustoria. Later, it grows rapidly to produce shoot and flowers. Orobanche also attacks other 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, moth bean, sorghum or cowpea to stimulate seed germination and kill the parasite.
9. Diseases of Groundnut

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 appearance, spot colour and shapes. 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.
The pathogen 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 µm 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 6-10µm, 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 broadly 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 (20 C) with dew on leaf surface.
- Heavy doses of nitrogen and phosphorus fertilizers
- Deficiency of magnesium in soil.

Disease cycle

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

Management

- Remove and destroy the infected plant debris.
• Eradicate the volunteer groundnut plants.
• Keep weeds under control.
• Treat the seeds with Carbendazim or Thiram at 2g/kg.
• Spray Carbendazim 500g or mancozeb 2 kg or Chlorothalonil 2 kg/ha and if necessary, repeat after 15 days.
• Grow moderately resistant varieties like ALR 1.

**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 ruptures and exposes a powdery mass of uredospores. Corresponding to the sori, small, necrotic, brown spots appear on the upper surface of leaves. The rust pustules may be seen on petioles and stem. Late in the season, brown *teliosori*, as dark pustules, appear among the necrotic patches. In severe infection lower leaves dry and drop prematurely. The severe infection leads to production of small and shriveled seeds.

![Symptoms](image)

**Pathogen**

The pathogen 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 germ pores. 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.

Teliospores

Favourable Conditions
- High relative humidity (above 85 per cent).
- Heavy rainfall.
- Low temperature (20-25°C).

Disease cycle
The pathogen survives as uredospores on volunteer groundnut plants. The fungus also survives in infected plant debris in soil. The spread is mainly through wind borne inoculum of uredospores. The uredospores also spread as contamination of seeds and pods. 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 2 kg or Wettable Sulphur 3 kg or Tridemorph 500ml or Chlorothalonil 2 kg/ha.
- Grow moderately resistant varieties like ALR 1.

Collar rot or seedling blight or crown rot - Aspergillus niger and A. pulverulentum

Symptoms
The disease usually appears in three phases.

i. **Pre-emergence rot**

Seeds are attacked by soil-borne conidia and caused rotting of seeds. The seeds 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 discolored 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 (30-35°C).
- Low soil moisture.

**Disease cycle**

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 infeced seeds. The pathogen is also seedborne in nature.

**Management**

- Crop rotation.
- Destruction of plant debris.
- Remove and destroy previous season's infested crop debris in the field
- Seed treatment with *Trichoderma viride* / *T.harzianum* @ 4 g/kg of seeds and soil application of *Trichoderma viride* / *T.harzianum* at 2.5kg/ha, preferably with organic amendments such as castor cake or neem cake or mustard cake @ 500 kg/ ha.

**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 with 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.

**Disease cycle**

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 and cattle etc.

**Management**

• Treat the seeds with thiram or carbendazim 2g/kg or *Trichoderma viride* at 4g/kg.

• Spot drench with Carbendazim at 0.5 g/lit.

**Rossette -** *Groundnut rosette assistor virus* (GRAV), *Groundnut rosette virus* and *Groundnut rosette satellites*

**Symptoms**

The affected plants are characterized by the appearance of dense clump or dwarf shoots with tuft of small leaves forming in a rosette 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 no seed formation.

![Symptoms](image_url)

**Pathogen**

The disease is caused by a complex mixture of viruses viz., *Groundnut rosette assistor virus* (GRAV), *Ground nut rosette virus* and *Groundnut rosette satellites* is an isometric, not enveloped and 28nm diameter (reported from India) and it gives no overt symptom in groundnut. *Groundnut rosette virus* is with ssRNA genome, which becomes packaged in GRAV virious and thus depends on it for aphid transmission, but produces no overt symptoms in groundnut. The
groundnut rosette satellites are satellite RNAs that control the symptoms and cause the different types of rosette (chlorotic, green and mosaic).

**Disease Cycle**

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

**Management**

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

**Groundnut bud necrosis disease - Groundnut bud necrosis virus** (GBNV- Tospo virus)

**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.
Plant becomes stunted with short internodes and short auxillary shoots. Leaflets show reduction in size, distortion of the lamina, mosaic mottling and general chlorosis. In advanced conditions, the necrosis of buds occurs. Top bud is killed and necrosis spreads downwards. Drastic reduction in flowering and seeds produced are abnormally small and wrinkled with the dark black lesions on the testa.

**Pathogen**

It is caused by *Groundnut bud necrosis virus* (GBNV). The virus particles are spherical, 30 nm in diameter, enveloped, ssRNA with multipartite genome.

**Disease cycle**

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

**Management**

- Adopt plant spacing of 15x15 cm.
- Remove and destroy infected plants up to 6 weeks after sowing.
- Application of 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.

**Minor diseases**

**Stem rot** - *Sclerotium rolfsii*

**Symptoms**

The first symptom is the sudden drying of a branch which is completely or partially in contact with the soil. The leaves turn brown and dry but remain attached to the plant. Near soil on stems white growth of fungus mycelium is appeared. As the disease advances white mycelium web spreads over the soil and the basal canopy of the plant. The sclerotia, the size and colour of mustard seeds, appear on the infected areas as the disease develops and spreads. The entire plant may be killed or only two or three branches may be affected. Lesions on the developing pegs can retard pod development. Infected pods are usually rotted.
Management

- Cultural practices such as deep covering or burial of organic matter before planting, non-dirtling cultivation by avoiding movement of soil up around the base of plants and preventing accumulation of organic debris are extremely useful in reducing the disease.
- Crop rotation with wheat, corn and soyabean may minimize the incidence of stem rot.
- Seed treatment with Carbendazim / Thiram / Captan @ 2-3 g/kg seed.
- Seed treatment with *Trichoderma viride* formulation (4g/kg) followed by application of 2.5kg *Trichoderma viride* formulation mixed with 50kg farm yard manure before sowing.

**Wilt - *Fusarium oxysporum* and *F. solani***

**Symptoms**

Germinating seeds are attacked by the pathogens shortly before emergence. There is general tissue disintegration and the surface of the seedling is covered with sporulating mycelium. Damping off symptoms characterized by brown to dark brown Water soaked sunken lesions on the hypocotyl which later encircle the stem and extend above the soil level. Roots are also attacked, especially the apical portions. The affected seedlings become yellow and wilted. The leaves turn greyish green and the plants dry up and die. The roots and stems show internal vascular browning and discolouration. These fungi are also commonly associated with pod rot.

**Management**

- Seed treatment with systemic fungicides like Carbendazim at 2g/kg seed.

**Anthracnose - *Colletotrichum dematium* and *C. capsici***

**Symptoms**
Small water-soaked yellowish spots appear on the lower leaves which later turn into circular brown lesions with yellow margin 1 to 3 mm in diameter. In some cases lesions enlarge rapidly become irregular and cover the entire leaflet, and extend to the stipules and stems. Brownish grey lesions occur on both the surfaces of leaflets. Infection spreads to stipules, petioles and branches.

**Disease cycle**

The pathogen is seed, soil and air-borne.

**Management**

- Deep summer ploughing.
- Use healthy certified seeds.
- Removal of plant debris.
- Seed treatment with copper oxychloride at 3g/kg seed or carbendazim at 2g/kg seed.

**Yellow mould - Aspergillus flavus**

**Symptoms**

Seed and un-emerged seedlings attacked by the pathogen are rapidly shriveled and dried. Brown or black mass covered by yellow or greenish spores may be seen. Decay is most rapid when infected seeds are planted. After seedling emergence cotyledons already infected with the pathogen, show necrotic lesions with reddish brown margins. This necrosis terminates at or near the cotyledonary axis. Under field conditions the diseased plants are stunted, and are often...
chlorotic. The leaflets are reduced in size with pointed tips, widely varied in shape and sometimes with veinal clearing.

Management

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

**Grey mould - *Botrytis cinerea***

Infection is seen on leaves, stem and underground parts of the groundnut. Initially infection occurs at ground level by a light grey fungal rot which causes death of the plants.

**Bacterial wilt - *Pseudomonas solanacearum***

Infected plants appear unhealthy, chlorotic and wilt under water stress. Dark brown discolouration of xylem is seen. Grey slimy liquid ooze out of the vascular bundles.

**Leaf spot - *Alternaria arachidis* and *A. tenuissima***

**Symptoms**

Lesions produced by *A. arachidis* are brown in colour and irregular in shape surrounded by yellowish halos. Symptoms produced by *A. tenuissima* are characterized by blighting of apical portions of leaflets which turn light to dark brown colour. Lesions produced by *A. alternata* are small, chlorotic, water soaked, that spread over the surface of the leaf. The lesions become necrotic and brown and are round to irregular in shape. Veins and veinlets adjacent to the lesions become necrotic. Lesions increase in area and their central portions become pale, rapidly dry out, and disintegrate. Affected leaves show chlorosis and in severe attacks become prematurely senescent. Lesions can coalesce, give the leaf a ragged and blighted appearance.
**Management**

- Foliar application of Mancozeb (2kg/ha) or Copper oxychloride (2kg/ha) or Carbendazim (500g/ha).

**Indian Peanut Clump Disease - Peanut Clump virus**

Earlier this disease was confused with groundnut rosstte. Now it is recognized as a distinct virus causing clump disease. The leaves turn very dark and plants become severely stunted. The disease is soil borne and transmitted by a fungus, *Polymyxa graminis*. The pH of the soil affects transmission. It is also transmitted by seed. The virus is rod shaped, 190-245nm long x 21nm wide, not enveloped, ssRNA genome.

**Other virus diseases of minor importance occurring on groundnut are:**

Peanut chlorotic streak (caused by *Caulimovirus*, occurs only in India), Peanut green mosaic and mottle (caused by a *Potyvirus*), peanut stunt (caused by *Cucumovirus*), groundnut chlorotic spot (caused by a *Potexvirus*), groundnut eye spot (caused by *Potyvirus*) and groundnut ringspot.
10. Diseases of Castor

Seedling blight - *Phytophthora parasitica*

**Symptoms**

The disease appears circular, dull green patch on both the surface of the cotyledon 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.

![Dead seedling](image)

![Spot on older leaf](image) ![Leaf blight symptom](image)

**Pathogen**

The pathogen 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 (20-25˚C).
• Low lying and ill drained soils.

**Disease cycle**

The pathogen 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.

**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.

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

**Disease cycle**

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

- Rogue out the self-sown castor crops and other weed hosts.
- Spray Mancozeb at 2kg/ha or Propiconazole 1l/ha.

Leaf blight- *Alternaria ricini*

**Symptoms**

All the aerial parts of plants viz., leaves, stem, inflorescences and capsules are liable to be attacked by the pathogen. Irregular brown spots with concentric rings form initially on the leaves and covered with fungal growth. When the spots coalesce to form big patches, premature defoliation occurs. The stems, inflorescences 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.

*Alterneria* leaf spot with concentric rings

**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 %).
- Low temperature (16-20°C)

**Disease cycle**

The pathogen 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 2g/kg.
- Remove the reservoir hosts periodically.
- Spray mancozeb at 2kg/ha.

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.

![Spots on leaf](image)

Pathogen

The pathogen 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.

Disease cycle

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

Management
Spraying with 1% **Bordeaux mixture** or **Copper oxy chloride** @ 0.2% may help to bring the disease under check; but where the cultures of Eri-silk worm are maintained on castor plants, spraying would not be desirable.

- Use of resistant varieties would be the most effective method for combating the disease.
- Spraying twice with Mancozeb 2g/lit or Carbendazim 500g/ha at 10-15 day interval reduces the disease incidence.
- Treat the seed with thiram or Captan 2gm/kg seed.

**Powdery mildew - *Leveillula taurica***

**Symptoms**

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

![Powdery mass covering entire leaf](image)

**Management**

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

**Stem rot - *Macrophomina phaseolina***

**Symptoms**

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

Management

• Grow tolerant and resistant varieties like Jyothi, Jwala, GCH-4, DCH-30 and SHB-145.
• Avoid water logging.
• Destruction of crop debris.
• Selection of healthy seed.
• Providing irrigation at critical stages of the crop.
• Treat the seed with thiram @ 2g/kg or carbendazim at 2g/ kg seed.
• Seed treatment with *Trichoderma viride* formulation at 4g/kg of seed.
• Soil drenching with Carbendazim (1g/1 litre of water) 2-3 times at 15 days interval.

**Bacterial leaf spot - Xanthomonas campestris pv. ricincola**

**Symptoms**

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

Management

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

Wilt - *Fusarium oxysporum*

Symptoms

When seedlings are attacked cotyledonary leaves turn to dull green colour, wither and die subsequently. Leaves are droop and drop off leaving behind only top leaves. Diseased plants are sickly in appearance. Wilting of plants, root degeneration, collar rot, drooping of leaves and necrosis of affected tissue and finally leading to death of plants. Necrosis of leaves starts from margins spreading to interveinal areas and finally to the whole leaf. Spilt open stem shows brownish discolouration and white cottony growth of mycelia much prominently in the pith of the stem.
Management

- Selection of disease free seeds.
- Grow tolerant and resistant varieties like Jyothi, Jwala, GCH-4 DCH-30 and SHB 145.
- Avoid water logging
- Burning of crop debris
- Green manuring and intercropping with red gram
- Treat the seeds with thiram @ 2g/ kg or carbendiazim @ 2g/ kg seed.
- Seed treatment with 4g of *Trichoderma viride* talc formulation.
- Multiplication of 2kg of *T.viride* formulation by mixing in 50kg farm yard manure
- Sprinkling water and covering with polythene sheet for 15days and then applying between rows of the crops is helpful in reducing the incidence.
11. Diseases of Sunflower

Root rot or charcoal rot - *Rhizoctonia bataticola* (Pycnidial stage: *Macrophomina phaseolina*)

**Symptoms**

The pathogen is seed-borne and primarily causes *seedling blight* and *collar rot* in the initial stages. The grown up plants also show symptoms after flowering stage. The infected plants show *drooping* of leaves and death occurs in patches. The bark of the lower stem and roots shreds and are associated with a large number of *sclerotia*. Dark coloured, minute *pycnidia* also develop on the lower portion of the stem.

**Pathogen**

The fungus produces a large number of black, round to irregular shaped *sclerotia*. The pycnidia are dark brown to black with an ostiole and contain numerous single celled, thin walled, hyaline and elliptical *pycnidiospores*. 
Favourable Conditions
- Moisture stress and higher temperature favour development of the disease.

Disease cycle
The pathogen survives in soil and in infected crop residues through sclerotia and pycnidia. The pathogen is seed-borne and it serves as primary source of infection. Wind-borne conidia cause secondary spread. The soil borne sclerotia also spreads through rain splash, irrigation water and implements.

Management
- Closer planting of the seedling should be avoided.
- Optimum nutrition should be provided to maintain the plant vigour.
- Whenever the soil becomes dry and the soil temperature rises then irrigation should be provided.
- Seed treatment with *Trichoderma viride* formulation at 4 g/kg seed.
- In endemic areas long crop rotation should be followed.
- Treat the seeds with Carbendazim or Thiram at 2/kg
- Spot drench with Carbendazim at 500 mg/litre.

Leaf blight - *Alternaria helianthi*

Symptoms
The pathogen produces brown spots on the leaves, but the spots can also be seen on the stem, sepals and petals. The lesions on the leaves are dark brown with pale margin surrounded by a yellow halo. The spots later enlarge in size with concentric rings and become irregular in shape. Several spots coalesce to show bigger irregular lesions leading to drying and defoliation.
Pathogen

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

Favourable Conditions

- Rainy weather.
- Cool winter climate.
- Late sown crops are highly susceptible.

Disease cycle

The fungus survives in the infected host tissues and weed hosts. The fungus is also seed-borne. The secondary spread is mainly through wind blown conidia.

Management

- Deep summer ploughing.
- Proper spacing
- Clean cultivation and field sanitation.
- Use of resistant or tolerant variety like B.S.H.1.
- Application of well rotten manures.
- Practicing crop rotation.
- Planting in mid-September.
- Remove and destroy the diseased plants
- Treat the seeds with Thiram or Carbendazim at 2 g/kg. Spray Mancozeb at 2 kg/ha.
Rust - *Puccinia helianthi*

**Symptoms**

Small, reddish brown pustules (uredia) covered with rusty dust appear on the lower surface of bottom leaves. Infection later spreads to other leaves and even to the green parts of the head. In severe infection, when numerous pustules appear on leaves, they become yellow and dry. The black coloured telia are also seen among uredia on the lower surface. The disease is autoecious rust. The pycnial and aecial stages occur on volunteer crops grown during off-season.

**Pathogen**

The uredosporces are round or elliptical, dark cinnamon-brown in colour and minutely echinulated with 2 equatorial germ pores. Teliosporces are elliptical or oblong, two celled, smooth walled and chesnutt brown in colour with a long, colourless pedicel.

**Favorable Conditions**

- Day temperature of 25.5° to 30.5°C with relative humidity of 86 to 92 per cent enhances intensity of rust attack.
**Disease cycle**

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

**Management**

- Use of tolerant and resistant varieties
- Crop rotation should be followed.
- Previous crop remains should be destroyed.
- Removal of crop residues
- Spray Mancozeb at 2kg/ha.

**Head rot - *Rhizopus* sp.**

**Symptoms**

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

![Symptoms](image)

**Pathogen**

Pathogen produces dark brown or black coloured, non-septate hyphae. It produces many aerial stolens and *rhizoids*, *Sproanga* are globose and black in colour with a central columella. The *sporangiospores* are aplanate, dark coloured and ovoid.
Favourable Conditions

- Prolonged rainy weather at flowering.
- Damages caused by insects and caterpillars.

Disease Cycle

The fungus survives as a saprophyte in host debris and other crop residues. The disease is spread by wind blown spores.

Management

- Treat the seeds with thiram or carbendazim at 2g/kg.
- Control the caterpillars feeding on the heads.
- Spray the head with Mancozeb at 2kg/ha during intermittent rainy season and repeat after 10 days, if the humid weather persists.

Powdery mildew - *Erysiphe cichoracearum*

Symptoms

The disease produces white powdery growth on the leaves. White to grey mildew on the upper surface of older leaves. As plant matures black pin head sized are visible in white mildew areas. The affected leaves more luster, curl, become chlorotic and die.
Favorable Conditions

- The disease is more under dry condition to the end of the winter months.

Management

- Complete field and crop sanitation.
- Early varieties should be preferred.
- Removal of infected plant debris.
- Application of karathane or calixin 1L/ha or wettable sulphur 2 kg/ha is found effective in reducing the disease incidences.

Basal rot - *Sclerotium rolfsii*

Symptoms

Initial symptoms of the disease appear 40 days sowing. The infected plants can be identified by their sickly appearance. Plants dry up due to the disease infestation. The lower portion of stem is covered with white or brownish white fungal colonies. In extreme cases the plants wilts and dies. Dark brown lesions appear on the base of the stem near ground level, leading to withering. Large numbers of sclerotia are seen.
Favourable Conditions

- Infection occurs in the crop in the month of July and August.
- The fungus survives through sclerotina in soil and plant debris.

Management

- Deep summer ploughing.
- Complete field and crop sanitation.
- Use of resistant or tolerant varieties.
- Collect and destroy plant debris.
- Apply *Trichoderma* on seed and soil to reduce wilt.
- Apply and incorporate fungus *Coniothyrium minitans* before sowing as it invades and destroy the pathogen in the soil.
- Seed treatment with *Pseudomonas fluorescens* or *P.putida* strains protect sunflower from *Sclerotinia* infection during seedling stage.
- Seed treatment with captan or thiram at the rate of 3 g/kg of seed.
- Drenching the base of the plant with chestnut compound 3 g per litre of water.
- Seed treatment with carbendazim at 0.2% followed by the addition of *Trichoderma harzianum* 10 g/kg soil and spraying Carbendazim at 0.2 % to 15 days old seedling.

Necrosis - *Tobacco streak virus* (TSV)

Symptoms
Characterised by the sudden necrosis of part of lamina followed by twisting of leaves and systemic mosaic. Necrosis of lamina of the lamina, petiole, stem floral calyx and corolla.

Black streak on stem

Necrosis of stems and petioles, terminal growth curls down and plants often lodge

Advanced symptoms lead to plant death.

Pathogen

Caused by *Tobacco streak virus* an *Ilarvirus* 25-28 nm, *triptite* genome encapsidated separately

Disease cycle

Virus spreads through transmission by *thrips Frankliniella schultzii*. Weed hosts serve as natural virus reservoirs. Long and continuous dry spell increases the disease incidence.
Management

- Removal of weed hosts
- Management of vector population
- Changing planting dates
12. Diseases of Sesamum

Root rot or stem rot or charcoal rot - *Macrophomina phaseolina* (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*.

![Symptoms](image)

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 shriveled and become black in colour. Minute *pycnidia* are also seen on the infected capsules and seeds. The rotten root as well as stem tissues contains a large number of minute black *sclerotia*. The sclerotia may also be present on the infected pods and seeds.

**Pathogen**

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

**Favourable Conditions**

- Day temperature of 30°C and above
- Prolonged drought followed by copious irrigation.

**Disease cycle**

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

**Management**

- Seed treatment with carbendazim + thiram (1:1) at 2g/kg seed.
- Treat the seeds with *Trichoderma viride* at 4g/kg.
- Apply farm yard manure or green leaf manure at 10t/ha or neem cake 150 kg/ha. Spot drench with Carbendazim at 1.0 g/litre.

**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. These are around 3-5 septate and conidia are borne in chain over short conidiophore.

**Favourable Conditions**

- Low temperature (20-25°C),
- High relative humidity
- Cloudy weather.

**Disease Cycle**

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 2kg/ha or Iprodion 1L/ha.
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.

[Image of leaf with spots]

**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.

**Disease Cycle**

The fungus is externally and internally seed-borne. The fungus also survives in plant debris. Primary infection may be from the seeds 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 2kg/ha.

Wilt - *Fusarium oxysporum f.sp. sesami*
Symptoms

The disease appears as yellowing, drooping and withering of leaves. The plants gradually wither, show wilting symptoms 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.

Disease Cycle

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

Management

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

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 shriveled 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 (25°C)
- High relative humidity (above 90 per cent)

Disease Cycle

The fungus can survive in the soil through 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 or metalaxyl @ 4g/kg.
- Avoid continuous cropping of sesame in the same field.
- Remove and destroy infected plant debris.
• Spray metalaxyl 1kg/ha.

**Powdery mildew - *Erysiphe cichoracearum* (Syn: *Oidium acanthospermi*)**

**Symptoms**

Initially greyish-white powdery growth appears on the upper surface of leaves. When several spots coalesce, the entire leaf surface may be covered with powdery coating. In severe cases, the infection may be seen on the flowers and young capsules, leading to premature shedding. The severally 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 Pathogen produces hyaline, septate mycelium which is extophytic and sends *haustoria* into the host epidermis. *Conidiophores* arise from the primary mycelium and are short and non septate bearing conidia in long chains. The conidia are ellipsoid or barrel-shaped, single celled and hyaline. The *cleistothecia* are dark, globose with the hyaline or pale brown myceloid appendages. The *asci* are ovate and each ascus produces 2-3 ascospores, which are thin walled, elliptical and pale brown in colour.

**Favourable Conditions**

- Dry humid weather.
- Low relative humidity.

**Disease Cycle**
The Pathogen 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 karathane 1L/ha repeat after 15 days.

**Bacterial leaf spot - Xanthomonas campestris pv. 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 color. Several spots coalesce together forming irregular brown patches and cause drying of leaves. The reddish brown lesions may also occur on petioles and stem.

![Symptoms](image)

**Pathogen**

The bacterium is a Gram negative rod with a monotrichous flagellum.

**Disease cycle**

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

**Management**

- Remove and burn infected plant debris.
Bacterial leaf spot - *Pseudomonas sesami*

**Symptoms**

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

**Pathogen**

The bacterium is gram negative aerobic rod with one or more polar flagella.

**Disease cycle**

The bacterium remains viable in the infected plant tissues. It is internally seedborne and secondary spread through rain splash and storms.

**Management**

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

Phyllody - *Phytoplasma*

**Symptoms**

The symptoms starts with vein clearing of leaves. The disease manifests itself mostly during flowering stage, when the floral parts are transformed into green leafy structures, which
grow profusely. The flower is rendered sterile. The veins of phylloid structure are thick and prominent. The plant is stunted with reduced internodes and abnormal branching.

![Symptoms](image)

**Pathogen**

It is caused by pleomorphic mycoplasma like bodies present in sieve tube of affected plants, now designated as a phytoplasmal disease.

**Disease cycle**

The pathogen has a wide host range and survives on alternate hosts like *Brassica campestris var. toria*, *B. rapa*, *Cicer arietinum*, *Crotalaria sp.*, *Trifolium sp.*, *Arachis hypogaea* which serve as source of inoculum. 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 sesame. Nymphs are incapable of transmitting the phytoplasma. Vector population is more during summer and less during winter months.

**Management**

- Remove all the reservoir and weed hosts.
- Avoid growing sesame near cotton, groundnut and grain legumes.
- Rogue out the infected plants periodically.
- Spray Monocrotophos or Dimethoate at 500ml/ha to control the jassids
Soil treatment with Thirnet 10G @ 10 kg/ha or Phorate 10 G @ 11 kg/ha at the time of sowing.

**Minor disease**

**Anthracnose** - *Colletotrichum sp.*

Dark brown lesions on leaf stem and capsules with black *acervuli* in the central portion.
13. Diseases of Cotton

Wilt - *Fusarium oxysporum f.sp. vasinfectum*

Symptoms

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

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

**Pathogen**

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

![Image of macroconidia and microconidia](image_url)

**Favourable Conditions**

- Soil temperature of 20-30°C
- Hot and dry periods followed by rains
- Heavy black soils with an alkaline reaction
- Increased doses of nitrogen and phosphatic fertilizers
- Wounds caused by nematode (*Meloidogyne incognita*) and grubs of **Ash weevil** (*Myllocerus pustulatus*).

**Disease cycle**

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

- Treat the acid delinted seeds with Carboxin or Carbendazim at 2 g/kg.
- Remove and burn the infected plant debris in the soil after deep summer ploughing during June-July.
- Apply increased doses of potash with a balanced dose of nitrogenous and phosphatic fertilizers.
- Apply heavy doses of farm yard manure or other organic manures. Follow mixed cropping with non-host plants.
- Grow disease resistant varieties of *G. hirsutum* and *G. barbadense* like Varalakshmi, Vijay Pratap, Jayadhar and Verum.
- Spot drench with Carbendazim 1g/litre.

Verticillium wilt - *Verticillium dahliae*

Symptoms

The symptoms are seen when the crop is in squares and bolls. Plants infected at early stages are severely stunted. The first symptoms can be seen as bronzing of veins. It is followed by interveinal chlorosis and yellowing of leaves. Finally the leaves begin to dry, giving a scorched appearance. At this stage, the characteristic diagnostic feature is the drying of the leaf margins and areas between veins, which gives a “*Tiger stripe*” or “*Tiger claw*” appearance.

The affected leaves fall off leaving the branches barren. Infected stem and roots, when split open, show a pinkish discolouration of the woody tissue which may taper off into longitudinal streaks in the upper parts and branches. The infected leaf also shows brown spots at the end of the petioles. The affected plants may bear a few smaller bolls with immature lint.
Pathogen

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

Favourable Conditions

- Low temperature of 15-20°C,
- Low lying and ill-drained soils,
- Heavy soils with alkaline reaction
- Heavy doses of nitrogenous fertilizers.

Disease Cycle

The fungus also infects the other hosts like brinjal, chilli, tobacco and bhendi. The fungus can survive in the infected plant debris and in soils as micro sclerotia upto 14 years. The seeds also carry the micro sclerotia and conidia in the fuzz. The primary spread is through the
micro sclerotia or conidia in the soil. The secondary spread is through the contact of diseased roots to healthy ones and through dissemination of infected plant parts through irrigation water and other implements.

**Management**

- Treat the delinted seeds with Carboxin or Carbendazim at 2 g/kg.
- Remove and destroy the infected plant debris after deep ploughing in summer months (June-July).
- Apply heavy doses of farmy and manure or compost at 100t/ha.
- Follow crop rotation by growing paddy or lucerne or chrysanthemum for 2-3 years.
- Spot drench with 0.05g/l benomyl or carbendazim 500mg/l.
- Grow disease resistant varieties like Sujatha, Suvin and CBS 156 and tolerant variety like MCU 5 WT.

**Root rot - *Rhizoctonia solani***

**Symptoms**

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

**Pathogen**

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

**Favourable conditions**

- Dry weather following heavy rains,
- High soil temperature (35-39˚C),
- Cultivation of favourable hosts like vegetables,
- Oil seeds and legumes preceding cotton
- Wounds caused by *ash weevil* grubs and nematodes.

**Disease cycle**

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

**Management**

- Treat the seeds with *Trichoderma viride* 4g/kg of seed.
- Spot drench with 0.1% Carbendazim.
Apply farm yard manure at 10t/ha or neem cake at 150 Kg/ha.

- Adjust the sowing time, early sowing (First Week of April) or late sowing (Last week of June) so that crop escapes the high soil temperature conditions.

- Adopt intercropping with sorghum or moth bean (Phaseolus aconitifolius) to lower the soil temperature.

**Anthracnose - Colletotrichum capsici**

**Symptoms**

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

![Image of Anthracnose symptoms](image)

**Pathogen**

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

**Favourable Conditions**

- Prolonged rainfall at the time of boll formation
- Close planting.
Disease Cycle

The pathogen survives as dormant mycelium in the seed or as conidia on the surface of seeds for about a year. The pathogen also perpetuates on the rotten bolls and other plant debris in the soil. The secondary spread is by air-borne conidia. The pathogen also survives in the weed hosts viz., *Aristolochia bractiata* and *Hibiscus diversifolius*.

**Management**

- Treat the delinted seeds with Carbendazim or Carboxin or Thiram or Captan at 2g/kg.
- Remove and burn the infected plant debris and bolls in the soil.
- Rogue out the weed hosts.
- Spray the crop at boll formation stage with Mancozeb 2kg or Copper oxychloride 2.5 kg or Carbendazim 500g/ha.

Grey or Areolate mildew - *Ramularia areola* (Sexual stage: *Mycosphaerella areola*)

**Symptoms**

The disease usually appears on the under surface of the bottom leaves when the crop is nearing maturity. Irregular to angular pale translucent lesions which measure 1-10 mm (usually 3-4 mm) develop on the lower surface, usually bound by vein lets. On the upper surface, the lesions appear as light green or yellow green specks.

A frosty or whitish grey powdery growth, consisting of conidiophores of the fungus, appears on the lower surface. When several spots coalesce, the entire leaf surface is covered by white to grey powdery growth. White or grey powdery growth may occur on the upper surface also. The infection spreads to upper leaves and entire plant may be affected. The affected leaves dry up from margin, cup inward; turn yellowish brown and fall of prematurely.
Pathogen

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

Favourable Conditions

- Wet humid conditions during winter cotton season,
- Intermittent rains during North-East monsoon season,
- Low temperature (20-30°C) during October-January,
- Close planting, excessive application of nitrogenous fertilizers,
- Very early sowing or very late sowing of cotton

Disease cycle

The pathogen survives during the summer in the infected crop residues. The perennial cotton plants and self-sown cotton plants also harbour the pathogen during summer months. The primary infection is through conidia from infected plant debris and secondary spread is through wind, rain splash, irrigation water and implements.

Management

- Remove and burn the infected crop residues.
- Rogue out the self-sown cotton plants during summer months.
- Avoid excessive application of nitrogenous fertilizers/manures.
- Adopt the correct spacing based on soil conditions and varieties.
- Spray the crop with Carbendazim at 500g/ha, repeat after a week.
- Grow the resistant varieties like Sujatha and Varalakshmi.

Boll rot - Fungal complex

It is a complex disease caused by several fungal pathogens viz., Fusarium moniliforme, Colletotrichum capsici, Aspergillus flavus, A. niger, Rhizopus nigricans, Nematospora nagpuri and Botryodiplodia sp.

Symptoms
Initially, the disease appears as small brown or black dots which later enlarge to cover the entire bolls. Infection spreads to inner tissues and rotting of seeds and lint occur. The bolls never burst open and fall off and prematurely. In some cases, the rotting may be external, causing rotting of the pericarp leaving the internal tissues free. On the affected bolls, a large number of fruiting bodies of fungi are observed depending upon the nature of the fungi involved.

![Image of diseased cotton boll]

**Favourable Conditions**

- Heavy rainfall during the square and boll formation stage,
- Wounds caused by the insects,
- Especially red cotton bug *Dysdercus cingulata*
- Close spacing and excessive nitrogen application.

**Disease Cycle**

The fungi survive in the infected bolls in the soil. The insects mainly help in the spread of the disease. The fungi make their entry only through wounds caused by the insects. The secondary spread of the disease is also through air-borne conidia.

**Management**

- Adopt optimum spacing.
- Apply the recommended doses of fertilizers.
- Spray *Copper oxychloride* 2.5kg along with an insecticide for bollworm from 45th day at 15 days interval.
- Two or three sprays are necessary.

**Leaf blight** - *Alternaria macrospora*
Symptoms

The disease may occur in all stages but more severe when plants are 45-60 days old. Small, plate to brown, irregular or round spots, measuring 0.5 to 6mm diameter, may appear on the leaves. Each spot has a central lesion surrounded by concentric rings. Several spots coalesce together to form blighted areas. The affected leaves become brittle and fall off. Sometimes stem lesions are also seen. In severe cases, the spots may appear on bracts and bolls.

Pathogen

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

Favourable Conditions

- High humidity.
• Intermittent rains.
• Moderate temperature of 25-28° C.

**Disease cycle**

The pathogen survives in the dead leaves as dormant mycelium. The pathogen primarily spreads through irrigation water. The secondary spread is mainly by airborne conidia.

**Management**

• Remove and destroy the infected plant residues.
• Spray Mancozeb 2 kg or Copper oxychloride at 2kg/ha at the intimation of the disease. Four to five sprays may be given at 15 days interval.

**Bacterial blight** - *Xanthomonas axonopodis pv. malvacearum*

**Symptoms**

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

**i) Seedling blight:**

Small, water-soaked, circular or irregular lesions develop on the cotyledons, later, the infection spreads to stem through petiole and cause withering and death of seedlings.

**ii) Angular leaf spot:**

Small, dark green, water soaked areas develop on lower surface of leaves, enlarge gradually and become angular when restricted by veins and veinlets and spots are visible on both the surface of leaves. As the lesions become older, they turn to reddish brown colour and infection spreads to veins and veinlets.

**iii) Vein blight or vein necrosis or black vein:**

The infection of veins cause blackening of the veins and veinlets, gives a typical ‘bloating’ appearance. On the lower surface of the leaf, bacterial ooze are formed as crusts or scales. The affected leaves become crinkled and twisted inward and show withering. The infection also spreads from veins to petiole and cause blotting leading to defoliation.

**iv) Black arm:**

On the stem and fruiting branches, dark brown to black lesions are formed, which may girdle the stem and branches to cause premature drooping off of the leaves, cracking of stem and
gummosis, resulting in breaking of the stem and hang typically as dry black twig to give a characteristic “black arm” symptom.

v) Square rot / Boll rot:

On the bolls, water soaked lesions appear and turn into dark black and sunken irregular spots. The infection slowly spreads to entire boll and shedding occurs. The infection on mature bolls lead to premature bursting. The bacterium spreads inside the boll and lint gets stained yellow because of bacterial ooze and looses its appearance and market value. The pathogen also infects the seed and causes reduction in size and viability of the seeds.

Angular leaf spot

Bacterial blight lesions on leaf and the blackleg symptom on the leaf petiole
Pathogen

The bacterium is a short rod with a single polar flagellum. It is Gram negative, non-spore forming and measures 1.0-1.2 X 0.7-0.9 µm.

Favorable Conditions

- Optimum soil temperature of 28°C,
- High atmospheric temperature of 30-40°C,
- Relative humidity of 85 per cent, early sowing,
- Delayed thinning,
- Poor tillage, late irrigation and
- Potassium deficiency in soil.
- Rain followed by bright sunshine during the months of October and November are highly favorable.

Disease Cycle

The bacterium survives on infected, dried plant debris in soil for several years. The bacterium is also seed-borne and remains in the form of slimy mass on the fuzz of seed coat. The bacterium also attacks other hosts like Thumbergia thespesioides, Eriodendron anfructuosum and Jatropha curcas. The primary infection starts mainly from the seed-borne bacterium. The secondary spread of the bacteria may be through wind, wind blown rain splash, irrigation water, insects and other implements.

Management

- Delint the cotton seeds with concentrated sulphuric acid at 100ml/kg of seed. Treat the delinted seeds with carboxin or oxycarboxin at 2 g/kg or soak the seeds in 1000 ppm Streptomycin sulphate overnight.
- Remove and destory the infected plant debris. Rogue out the volunteer cotton plants and weed hosts.
Follow crop rotation with non-host crops.

- Early thinning and early earthing up with potash.
- Grow resistant varieties like Sujatha, 1412 and CRH 71.
- Spray with Streptomycin sulphate +Tetracycline mixture 100g along with Copper oxychloride at 1.25 Kg/ha.

**Leaf Curl Disease** - *Cotton leaf curl virus*

**Symptoms**

Downward and upward curling of leaves and thickening of veins and enation on underside of leaves are the characteristic symptoms of the disease. In serve infection all the leaves are curled and growth retarded. Boll bearing capacity is reduced.
Pathogen

It is caused by *Cotton leaf curl virus* - a *begomovirus* of family *geminiviridae*. The virions are typical *geminate* particles, *ss circular DNA*, *bipartite genome* with DNA-A and DNA-B components.

Disease Cycle

The primary source is the *viruliferous whitefly* vector *Bemisia tabaci*. The alternate hosts and cultivated hosts serve as virus reservoirs throughout the year. Not transmitted by seed or contact.

Management

- Management of planting date to avoid peak vector population.
- Elimination of volunteer perennial cotton and alternate hosts including malvaceous hosts like wild okra
- Use of fungus *Paecilomyces farinosus* which parasitizes *B.tabaci*. It brings down vector population.
- Foliar application of neem leaf extract and 1% neem oil resulted in 80% reduction of virus transmission.
- Vector management by application of granular *systemic insecticides*.

Stenosis or Small leaf - *Phytoplsama*

**Symptoms**

The disease appears when the plants are two to three months old and affected plants are stunted. They put forth numerous extremely small leaves in cluster and the dormant buds are stimulated resulting in profuse vegetative growth. The leaves are disfigured and variously lobed. Flowers remain small with abortive ovary.

Large number of flower buds and young seeds. Root system is poorly developed and can be easily pulled out. Sometimes, the disease affects only the base of the plant, resulting in the formation of clump of short branches which bear small and deformed leaves. The mode of transmission of disease and the role of vector are unknown.
Management

- Rogue out the infected plants periodically.
- Cotton varieties developed from *Gossypium hirsutum* and *G. barbadense* are found to be resistant to the disease.

Minor diseases

**Leaf spot - *Cercospora gossypina***

Round or irregular grayish spots with dark brown or blackish borders appear on older leaves.

**Myrothecium leaf spot - *Myrothecium roridum***

Reddish spots of 0.5 mm- 1 cm diameter may appear near the margins of the leaves. The affected portions fall off leaving irregular shot holes in the leaves.

**Rust - *Phakopsora desmium***

Yellowish brown raised pustules appear on the lower surface of leaves with rusty spores. Several pustules join to give rusty appearance to entire leaf. The sori may also develop on bolls.

**Sooty mould - *Capnodium sp.***

Dark specks appear on the leaves and bolls, slowly spread and black powdery growth covers the entire leaf area and bolls.
14. Diseases of Red Gram

Wilt - *Fusarium udum*

**Symptoms**

The disease may appear from early stages of plant growth (4-6 week old plant) up to flowering and podding. The disease appears as gradual withering and drying of plants. Yellowing of leaves and blackening of stem 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 discoloration. 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 produce thick walled, spherical or oval, terminal or intercalary chlamydospores singly or in chains of 2 to 3.

**Favourable conditions**

- Soil temperature of 17-25°C.
- Continuous cultivation of red gram in the same field.

**Disease cycle**

The fungus survives in the infected stubbles in the field. The primary spread is by soil-borne chlamydospores and also by infected seed. 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 (10⁶ cfu/g).
- Avoid successive cultivation of red gram in the same field.
- Crop rotation with tobacco.
- Mixed cropping with sorghum in the field.
- Grow resistant cultivars like Sharad, Jawahar, Maruthi, Malviya Arhar-2, C-11, Pusa-9, Narendra Arhar-1 and Birsa Arhar-1

Diseases of Field Crops and Their Management

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 occurs 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. Large number of brown dots seen on the stem portion represents 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 110-130μm 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 28-35°C.

Disease cycle

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

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

**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 of 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 produces 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 rainfall.

Disease Cycle

The fungus survives in the soil through 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/ha or Wettable sulphur 2 kg/ha at the initiation of the disease and repeat after 15 days.

Stem blight - Phytophthora drechsleri fsp. cajani

Symptoms

Initially purple to dark brown necrotic lesions girdle the basal portion of the stem and later may occur an aerial parts. Initially lesions are small and smooth, later enlarging and slightly depressed. Infected tissues become soft and whole plant dies. In grown up plants, infection is mostly confined to basal portions of the stem. The infected bark becomes brown and the tissue softens causing the plant to collapse. 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 hyaline bearing 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
- High temperature (28-30°C).

Disease Cycle

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

Management

- Treat the seeds with Metalaxyl at 6 g/kg.
- Spray Metalaxyl at 500 g/ha.
- Adjust the sowing time so that crop growth should not coincide with heavy rainfall.

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.

Disease cycle

The fungus survives in the infected plant tissues. The disease is spread by airborne conidia.

Management

• Remove the infected plant debris and destroy.
• Spray Mancozeb 2 kg or Carbendazim 500 g/ha soon after the appearance of symptom and repeat after a fortnight.

Sterility Mosaic Disease (SMD) - Pigeonpea sterility mosaic virus (PPSMV)

Symptoms

The Symptoms are characterized by bushy and pale green appearance of plants. The excessive vegetative growth, stunting, prominent mosaic on leaves and reduction in leaf size. Complete or partial cessation of flowering leads to sterility. Depending on genotype three types of symptoms are recognized. They are

a. Severe mosaic and sterility
b. Mild mosaic and partial sterility
c. Chlorotic ringspot without any noticeable sterility.
Light and dark green mosaic pattern on leaves

Sterility mosaic infected plant (right side) without flowers and pods compared to normal plant (left side)

Pathogen

It is caused by *Pigeonpea sterility mosaic virus* (PPSMV). The virions are slender highly flexuous filamentous virus like particles (VLPS) of 3-10 nm diameter, a major virus specific proteins of 32kDa and 5-7 major RNA species of 0.8-6.8kb.

Disease cycle

It is not transmitted by infectious sap. It is transmitted by an eriophid mite, *Aceria cajani* in a semi persistent manner, mites retaining the virus 12-13 hours, eggs of mites do not transmit. The self grown redgram plants and perennial species act as source of virus inoculums.

Management

- Rogue out infected plants up to 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.

Minor diseases

Seedling blight - *Sclerotium rolfsii*

Small brown water soaked dots appear near collar region, expands to irregular necrotic spots leading to girdling of stem and death of seedling.

Brown blotch - *Colletotrichum capsici*

Purple brown discolouration occurs mainly on pods but also on petioles, leaf veins, stems and peduncles. Pods become distorted and have black fruiting bodies.

Anthracnose - *Colletotrichum lindemuthianum* (*Glomerella cingulata*)

Black lesions develop on stem which spreads to leaf petiole and leaves. Black sunken lesions also develop on pod.

Stem rot - *Pythium aphanidermatum*

Seedlings of 2-3 weeks old are severely attacked at collar region and death occurs immediately. Greyish green water soaked lesions develop on adult plants, leading to girdling of stem.

Leaf spot - *Alternaria alternata*

Water soaked, circular to irregular spots occur. The centre of the spot is straw coloured with raised reddish brown margins.

Halo blight - *Pseudomonas phaseolicola*

Small brown spots appear on leaves and develop a chlorotic halo. The spots extend and form dried brown zone. Brown elongated streaks appear on petioles, stem and pods.

There are two other virus diseases reported on pigeonpea, mosaic and yellow mosaic transmitted by aphids and whiteflies which are of sporadic occurrence only.
15. Diseases of Black gram

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.

![Symptoms](image)

**Pathogen**

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

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

Disease cycle

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

Management

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

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

Symptoms

The symptom can be observed in all aerial parts of the plants and at any stage of crop growth. The fungus produces dark brown 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 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 (15-20˚ C)
• Cool rainy days.

Disease cycle

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

Management

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

**Leaf spot - *Cercospora canescens***

**Symptoms**

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

**Pathogen**

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

**Favourable Conditions**

- Humid weather and dense plant population.

**Disease cycle**

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

**Management**

- Remove and burn infected plant debris.
- Spray Mancozeb at 2 kg/ha or Carbendazim at 500 g/ha.
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.

![Symptoms](image)

**Pathogen**

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

**Favourable Conditions**

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

**Mode of Spread and Survival**

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

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

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

Symptoms

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

![Symptoms](image)

Pathogen

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

Favourable conditions

- Day temperature of 30°C.
- Prolonged dry season followed by irrigation.
Disease cycle

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

Management

- Treat the seeds with carbendazim + thiram at 2 g/kg (1:1 ratio) or pellet the seeds with Trichoderma viride at 4 g/kg \((10^6\text{cfu/g})\) or Pseudomonas fluorescens @ \((10^6\text{cfu/g})\) of seed.
- Apply farm yard manure or green leaf manure \((Gliricidia maculata)\) at 10 t/ha or neemcake at 150 kg/ha.

Mungbean Yellow mosaic disease - *Mungbean yellow mosaic virus (MYMV)*

Symptoms

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

Pathogen

It is caused by *Mungbean yellow mosaic India virus (MYMIV)* in Northen and Central region and *Mungbean yellow mosaic virus (MYMV)* in western and southern regions. It is a
Begomovirus belonging to the family geminiviridae. Geminate virus particles, ssDNA, bipartite genome with two gemonic components DNA-A and DNA-B.

**Disease cycle**

Transmitted by whitefly, *Bemisia tabaci* under favourable conditions. Disease spreads by feeding of plants by viruliferous whiteflies. Summer sown crops are highly susceptible. Weed hosts viz., *Croton sparsiflorus*, *Acalypha indica*, *Eclipta alba* and other legume hosts serve as reservoir for inoculum.

**Management**

- Rogue out the diseased plants up to 40 days after sowing.
- Remove the weed hosts periodically.
- 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.
- Treat the seeds with *Thiomethoxam*-70WS or *Imidacloprid*-70WS @4g/kg
- Spray *Thiamethoxam*-25WG @ 100g or *Imidacloprid* 17.8% SL @ 100 ml in 500 lit of water.

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

**Symptoms**

Crinkling and curling of the tips of leaflets and increase in leaf area. Crinkling and rugosity in older leaves becomes severe and leaves thickened. Petioles as well as internodes are shortened. Infected plant gives a stunted and bushy appearance. Flowering is delayed, if inflorescence is formed, is malformed with small size flower buds and fails to open.

**Pathogen**

Casual organism of the disease is not yet ascertained.

**Disease cycle**

Presence of weed hosts like *Aristolochia bracteata* and *Digera arvensis*. Kharif season crop and continuous cropping of other legumes serve as source of inoculum. The virus is seed-
borne and primary infection occurs through infected seeds. Perhaps white fly, *Bemisia tabaci* helps in the secondary spread. The virus is also sap transmissible.

**Management**

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

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

**Symptoms**

Upward cupping and curling of leaves with vein clearing. Infected leaves turn brittle and sometimes show vein necrosis on the under surface of the leaves, extending to the petiole. Plants affected in the early stages of growth develop top necrosis and die. Plant may produce a few small and malformed pods.

**Pathogen**

It is caused by *Groundnut bud necrosis virus*

**Disease cycle**

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

**Management**

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

**Minor diseases**

**Ascochyta leaf spot - *Ascochyta phaseolorum***

Small irregular spot with grey to brown centre and yellow border. They rapidly enlarge to produce very large brown lesions with concentric markings.

**Bacterial blight - *Xanthomonas phaseoli***
Circular, reddish brown spots appear on leaves, enlarge to form irregular brown lesions. Water soaked, sunken spots with red border occur on pods.
16. Diseases of Green gram

Powdery mildew - *Erysiphe polygoni*

Symptoms

Powdery mildew is one of the widespread diseases of several legumes in green gram. White powdery patches appear on leaves and other green parts which later become dull colored. These patches gradually increase in size and become circular covering the lower surface also. When the infection is severe, both the surfaces of the leaves are completely covered by whitish powdery growth. Severely affected parts get shriveled and distorted. In severe infections, foliage becomes yellow causing premature defoliation. The disease also creates forced maturity of the infected plants which results in heavy yield losses.

Pathogen

The fungus is ectophytic, spreading on the surface of the leaf, sending haustoria into the epidermal cells. Conidiophores arise vertically from the leaf surface, bearing conidia in short chains. Conidia are hyaline, 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

- The pathogen has a wide host range and survives in oidial form on various hosts in off-season.
- Secondary spread is through air-borne oidia produced in the season

Disease Cycle

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

Management

- Use resistant varieties
• The seeds must be sown early in the month of June to avoid early incidence of the disease on the crop.
• Spray Carbendazim 500g or Wettable sulphur 1.5 kg or Tridemorph 500 ml/ha at the initiation of disease and repeat 15 days later.

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

**Symptoms**

The disease appears on all aerial parts and at any stage of plant growth. Circular, black, sunken spots with dark center and bright red orange margins on leaves and pods. In severe infections, the affected parts wither off. Seedlings get blighted due to infection soon after seed germination.

**Pathogen**

The Disease appears on 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**

• The disease is more severe in cool and wet seasons.

**Disease cycle**

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**

• Hot water treatment at 54º for 10 min.
• Use disease free seed.
• Follow crop rotation
• Remove and destroy infected plant debris in soil.
• Treat the seeds with Carbendazim at 2 g/kg.
• Spray Carbendazim 500g or Mancozeb 2kg/ha soon after the appearance of disease and repeat after 15 days.

Leaf spot - *Cercospora canescens*

**Symptoms**

This is an important disease of green gram and is usually occurs in a severe form, causing heavy losses in yield. Spots produced are small, numerous in numbers with pale brown centre and reddish brown margin. Similar spots also occur on branches and pods. Under favourable environmental conditions, severe leaf spotting and defoliation occurs at the time of flowering and pod formation.

**Pathogen**

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

**Favourable conditions**

• High humidity favours disease development.

**Disease cycle**

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

**Management**

• Cultivate resistant varieties.
• Intercrop the moong with tall growing cereals and millets.
• Follow clean cultivation.
• Use disease free seed.
• Maintain low crop population density and wide row planting.
• The crude extracts of cassava, garlic, and zinger are applied for controlling the disease effectively.
• Mulching reduces the disease incidence resulting in increase yield.
• Spray Mancozeb 2kg/ha or Carbendazim 500 g/ha.

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

The disease appears as circular reddish brown pustules which appear more commonly on the underside of the leaves, less abundant on pods and sparingly on stems. When leaves are severely infected, both the surfaces are fully covered by rust pustules. Shrivelng followed by defoliation resulting in yield losses.

Pathogen

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

Favourable Conditions

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

Disease Cycle

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

Management

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

Dry root rot - Rhizoctonia bataticola (Pycnial 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 in abundance. The fungus also produces dark brown, globose ostiolated pycnidia on the host tissues. The pycnidiospores are thin walled, hyaline, single celled and **elliptical**.

**Favourable conditions**

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

**Disease cycle**

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

**Management**

- Treat the seeds with Carbendazim + Thiram at 2 g/kg or pellet the seeds with *Trichoderma viride* at 4 g/kg or *Pseudomonas fluorescens* @ 10g/kg of seed.
- Apply farm yard manure or green leaf manure (*Gliricidia maculate*) at 10 t/ha or neem cake at 150 kg/ha.

**Yellow mosaic disease** - *Mungbean yellow mosaic virus* (MYMV)

**Symptoms**

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

**Pathogen**

It is caused by *Mungbean yellow mosaic India virus* (MYMIV) in Northen and Central region and *Mungbean yellow mosaic virus* (MYMV) in western and southern regions. It is a Begomovirus belonging to the family geminiviridae. Germinate virus particles, ssDNA, bipartite genome with two gemonic components DNA-A and DNA-B.
Disease cycle

Transmitted by whitefly, *Bemisia tabaci* under favourable conditions. Disease spreads by feeding of plants by viruliferous whiteflies. Summer sown crops are highly susceptible. Weed hosts viz., *Croton sparsiflorus, Acalypha indica, Eclipta alba* and other legume hosts serve as reservoir for inoculum.

Management

- Rogue out the diseased plants up to 40 days after sowing.
- Remove the weed hosts periodically.
- Increase the seed rate (25 kg/ha).
- Grow resistant green gram variety like Pant Moong-3, Pusa Vishal, Basanti, ML-5, ML-337, PDM-54 and Samrat.
- 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.
- Treat the seeds with Thiomethoxam-70WS or Imidacloprid-70WS @4g/kg
- Spray Thiamethoxam-25WG @ 100g or Imidaclorpid 17.8% SL @ 100 ml in 500 lit of water.

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

Symptoms

Crinkling and rugosity in older leaves becomes severe and leaves thickened. Crinkling and curling of the tips of leaflets are seen. Petioles as well as internodes are shortened. Infected plant gives a stunted and bushy appearance. Flowering is delayed, inflorescence, if formed, are malformed with small size flower buds and fail to open.

Pathogen

Casual organism of the disease is not yet ascertained work is in progress in different laboratories.

Disease Cycle

Presence of weed hosts like *Aristolochia bracteata* and *Digera arvensis*. Kharif season crop and continuous cropping of other legumes serve as source of inoculum. The virus is seed-
borne and primary infection occurs through infected seeds. Perhaps white fly, *Bemisia tabaci* helps in the secondary spread. The virus is also sap transmissible.

**Management**

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

**Leaf curl / Necrosis - *Groundnut bud necrosis virus***

**Symptoms**

Upward cupping and curling of leaves with vein clearing. 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. Plant may produce a few small and malformed pods.

**Pathogen**

Caused by groundnut bud necrosis virus

**Disease Cycle**

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

**Management**

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

**Minor diseases**

**Ascochyta leaf spot - *Ascochyta phaseolorum***

Small irregular spot with grey to brown centre and yellow border. They rapidly enlarge to produce very large brown lesions with concentric markings.

**Bacterial blight - *Xanthomonas phaseoli***
Circular, reddish brown spots appear on leaves, enlarge to form irregular brown lesions. Water soaked, sunken spots with red border occur on pods.
17. Diseases of Bengal gram

Ascochyta blight - *Ascochyta rabiei*

**Symptoms**

All above ground parts of the plant are infected. On leaf, the lesions are round or elongated, bearing irregularly depressed brown spot and surrounded by a brownish red margin. Similar spots may appear on the stem and pods. The spots on the stem and pods have pycnidia arranged in concentric circles as minute block dots. When the lesions girdle the stem, the portion above the point of attack rapidly dies. If the main stem is girdles at the collar region, the whole plant dies.
Pathogen

The fungus produces hyaline to brown and septate mycelium. Pycnidia are spherical to sub-globose with a prominent ostiole. Pycnidiospores are hyaline, oval to oblong, straight or slightly curved and single celled, occasionally bicelled.

Favourable conditions

• High rainfall during flowering.
• Temperature of 20-25°C.
• Relative humidity of 60%.

Disease cycle

The fungus survives in the infected plant debris as pycnidia. The pathogen is also externally and internally seed-borne. The primary spread is from seed-borne pycnidia and plant debris in the soil. The secondary spreads is mainly through air-borne pycnidiospores (conidia). Rain splash also helps in the spread of the disease.

Management

• Remove and destroy the infected plant debris in the field.
• Treat the seeds with Thiram 2g or Carbendazim 2 g or Thiram + Carbendazim (1:1 ratio) at 2 g/kg.
• Exposure of seed at 40-50°C reduced the survival of A. rabiei by about 40-70 per cent.
• Spray with Carbendazim at 500 g/ha or Chlorothalonil 1kg/ha.
• Follow crop rotation with cereals.

Rust - *Uromyces ciceris-arietini*

Symptoms

The infection appears as small oval, brown, powdery lesions on both the surface, especially more on lower surface or leaf. The lesions, which are uredosori, cover the entire leaf surface. Late in the season dark teliosori appear on the leaves. The rust pustules may appear on petioles, stems and pods. The pycnial and aecial stages are unknown.

Pathogen

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

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

Management

- Destory weed host.
- Spray Carbendazim 500 g/ha or Propiconazole 1L/ha.

Wilt - *Fusarium oxysporum f.sp. ciceris*

Symptoms

The disease occurs at two stages of crop growth, seedling stage and flowering stage stage. The main symptoms on seedlings are yellowing and drying of leaves, drooping of petioles and rachis, withering of plants. In the case of adult plants drooping of leaves is observed initially in upper part of plant, and soon observed in entire plant. Vascular browning is conspicuously seen on the stem and root portion.

Pathogen

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

- High soil temperature (above 25°C).
- High soil moisture.

Disease cycle

The disease is seed and soil borne. The primary infection is through chlamydospores in soil, which remain viable upto next crop season. The secondary spread is through irrigation water, cultural operations and implements.

Management

- Treat the seeds with Carbendazim or Thiram at 2 g/kg or Carbendazim 1 g+Thiram 1g/kg or treat the seeds with *Trichoderma viride* at 4 g/kg (10^6 cfu/g) *Pseudomonas fluorescens* @ 10g/kg (10^6 cfu/g) of seed.
- Apply heavy doses of organic manure or green manure.

Stunt disease - Virus

Symptoms

Affected plants are stunted and bushy with short internodes. The leaflets are smaller with yellow, orange or brown discoloration. Stem also shows brown discoloration. The plants dry prematurely. If survive, a very few small pods are formed. Phloem browning in the collar region is the most characteristic symptom of the stunt, leaving xylem normal.

Disease cycle

The virus is transmitted by *Aphis craccivora*. 
Management

- Rogue out the infected plants.
- Spray Monocrotophos at 500 ml/ha.

Collar rot - *Sclerotium rolfsii*

Symptoms

It comes in the early stages i.e up to six weeks from sowing. Drying plants whose foliage turns slightly yellow before death, scattered in the field is an indication of the disease. Seedlings become chlorotic. The joint of stem and root turns soft slightly contracts and begins to decay. Infected parts turn brown white. Black dots, like mustard in shape known as sclerotia are seen appearing on the white infected plant parts.

Favorable conditions

- High soil moisture, low soil pH and high temperature.
- The presence of undecomposed organic matter on the soil surface and high moisture at the time of sowing and at the seedling stage
- Disease incidence is higher when sown after rice or early sown crop.

Management

- Deep ploughing in summer.
- Avoid high moisture at the sowing time.
- Seedlings should be protected from excessive moisture.
- Destroy the crop residues of last crop and weeds before sowing and after harvest.
• All undecomposed matter should be removed from the field before land preparation.
• Treat the seeds with a mixture of Carbendazim + Thiram (1:1) @ 2g per kg of seed.

**Minor diseases**

**Foot rot** - *Oerculella padwickii*

Rotting is evident from collar region onwards. Internal brown discolouration appears above the rotten portion (only on bark portion).

**Stemrot** - *Sclerotinia sclerotiorum*

The disease appears mostly on stems rot of adult plants as water soaked lesion on upper parts of stem. The affected portion is covered with white cottony growth and black sclerotial bodies.

**Bacterial leaf blight** - *Xanthomonas campestris pv. cassiae*

Small water soaked lesions develop on leaves with chlorotic haloes which later turn to dark brown spots. Post emergence seedling rot is also common.

**Bean Common Mosaic - Virus**

Stunted, bushy appearance of plant with mosaic mottling. Vector: *Aphis gossypii* and *A. craccivora*.
18. Diseases of Soybean

Dry root rot - *Macrophomina phaseolina*

**Symptoms**

The disease symptom starts initially with yellowing and drooping of the leaves. The leaves later fall off and the plant dies within the 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.

![Symptoms](image_url)

**Pathogen**

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

**Favourable conditions**

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

**Disease cycle**

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

Management

- Treat the seeds with Carbendazim or Thiram at 2 g/kg or pellet the seeds with *Trichoderma viride* at 4 g/kg or *Pseudomonas fluorescens* @ 10g/kg of seed.
- Apply farm yard manure or green leaf manure (*Gliricidia maculata*) at 10 t/ha or neem cake at 150 kg/ha.

**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.
Pathogen

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 20-25°C and moist humid weather.

Disease cycle

The fungus survives in the infected stubbles in the field. The primary spread is through soilborne chlamydospores and infected seeds. 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 drenching with Carbendazim at 0.5 g/litre.

Leaf spot - Cercospora sojana

Symptoms

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

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

**Management**

- Use resistant varieties.
- Use healthy or certified seeds.
- Rotate soybean with cereals.
- Completely remove plant residue by clean ploughing the field soon after harvest.
- Destroy last years infected stubble.
- Seed treatment with Thiram + Carbendazium (1:1) @ 2g/kg seed.
- Spray Mancozeb @ 2g/L or Carbenzadium (500 mg/L).

**Mosai - Soybean mosaic virus** (SMV)

**Symptoms**

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

**Pathogen**

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

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

Favorable conditions

• Temperature around 18\(^\circ\) C
• Humid weather.

Management

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