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 magesium 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 shrunken 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.

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.

**Rosette** - *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.

**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-dirtting 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 rosette. 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.