Disease of Horticultural Crops

& Their Management

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Disease of Horticultural Crops & their Management

ICAR e-Course
For
B.Sc (Agriculture) and B.Tech (Agriculture)

INDEX

Lecture 01 - Diseases of Citrus

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

Symptoms

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

Pathogen

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

Favourable conditions

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

Mode of spread and survival

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

Management

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

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

Symptoms

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

Pathogen

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

Mode of Spread and Survival

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

Management

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

Canker : *Xanthomonas campestris* pv *citri*

Symptoms

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

Pathogen

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

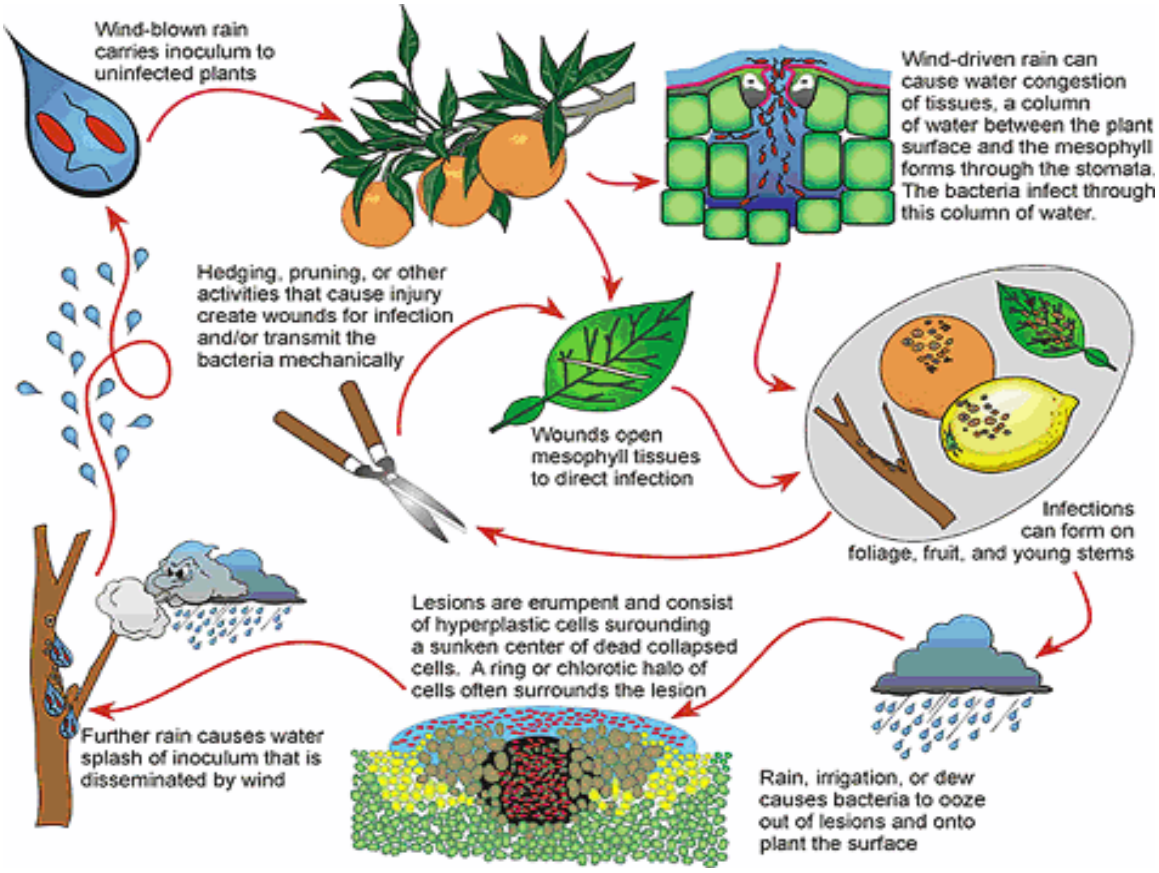
Favourable conditions

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

Mode of survival and spread

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

Disease Cycle



Management

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

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

Symptoms

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

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

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

Pathogen

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

Mode of spread

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

Management

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

Exocortis of scaly butt: *Viroid*

Symptoms

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

Pathogen

Viroid is free RTVA without protein coat.

Mode of Spread and Survival

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

Management

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

Greening: *Liberobactor asiaticum* (Phloem limited bacteria)

Symptoms

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

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

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

Pathogen

Rickettsia like organisam

Mode of spread

Infected budwood; psyllid vector-*Diaphorina citri*

Management

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

Lecture 02 - Diseases of Mango (2 Lectures)

Anthracnose: *Colletotrichum gloeosporioides*

Symptoms:

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

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

Pathogen

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

Mode of survival and spread

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

Favourable conditions

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

Disease cycle

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

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

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

Management

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

Powdery mildew: *Oidium mangiferae (Acrosporum mangiferae)*

Symptoms

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

Pathogen

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

Mode of survival and spread

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

Disease Cycle

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

Management

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

Mango malformation : *Fusarium moliliforme var. subglutinans*

Symptoms

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

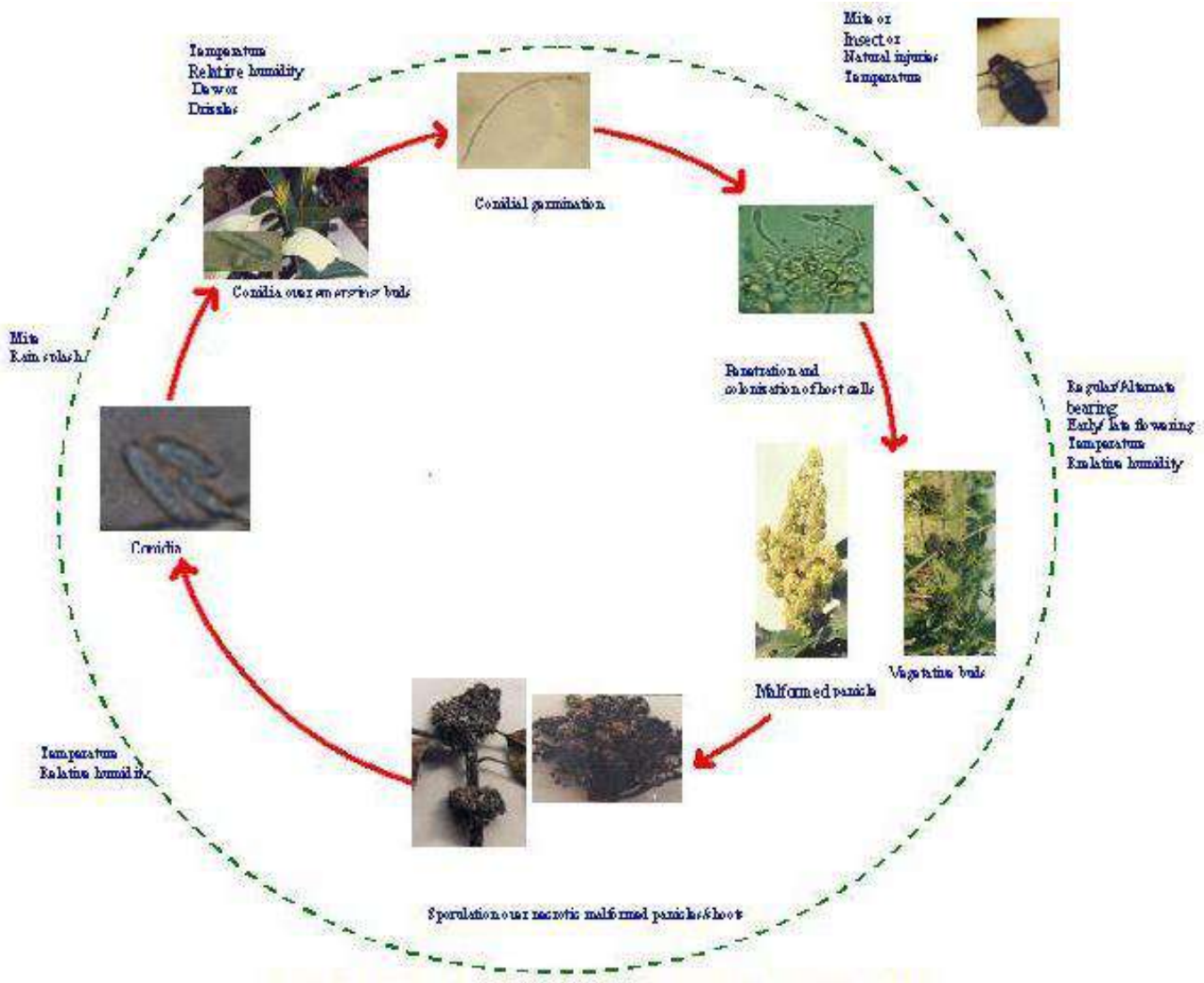
Pathogen

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

Mode of spread

Diseased propagatives materials.

Disease Cycle



Management

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

Stem end rot: *Diplodia natalensis*

Symptoms

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

Pathogen

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

Mode of spread and survival

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

Management

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

Red-rust: *Cephaleuros virescens*

Symptoms

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

Pathogen

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

Management

Bordeaux mixture (0.6%) or Copper oxychloride 0.25%

Grey Blight : *Pestalotia mangiferae*

Symptoms

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

Pathogen

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

Mode of survival and spread

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

Favourable conditions

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

Management

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

Sooty mould : *Capnodium mangiferae*

Symptoms

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

Favourable conditions

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

Management

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

Lecture 03 - Diseases of Banana (2 Lectures)

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

Economic Importance

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

Symptoms

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

Pathogen

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

Mode of spread and survival

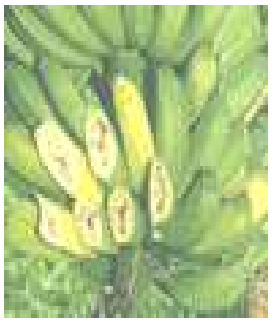
The pathogen is soil borne. It survives in soil as chlamydospores for longer periods. The primary spread of the disease is through infected rhizomes and secondary spread is through irrigation water. Continuous cultivation results in build up of inoculum.

Management

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

Moko disease : *Pseudomonas solanacearum / Burkholderia solanacearum*

Symptoms



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

Pathogen

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

Mode of spread and survival

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

Management

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

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

Symptoms

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

Management

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

Sigatoka disease : *Mycosphaerella musicola (Cercospora musae*)

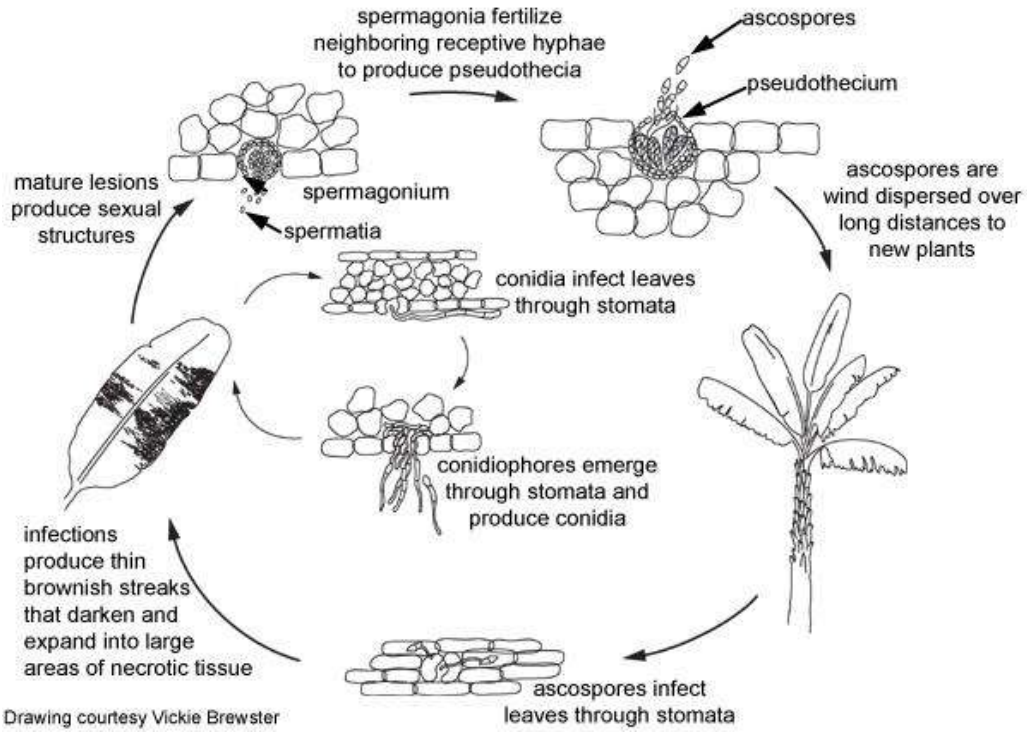
Symptoms

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

Pathogen

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

Disease Cycle



Management

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

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

Symptoms

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

Pathogen

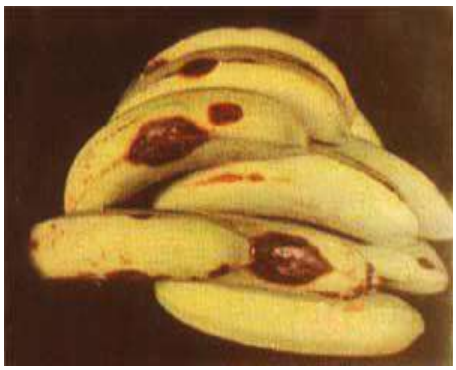
Conidiophores are usually solitary or in small groups. Conidia are hyaline, oblong to cylindrical. They are borne at the end of tapering phialides, aggregated into rounded, mucilaginous translucent heads.

Control:

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

Anthracnose*:* *Gloeosporium gloeosporioides*

Symptoms:



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

Pathogen

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

Mode of spread and survival

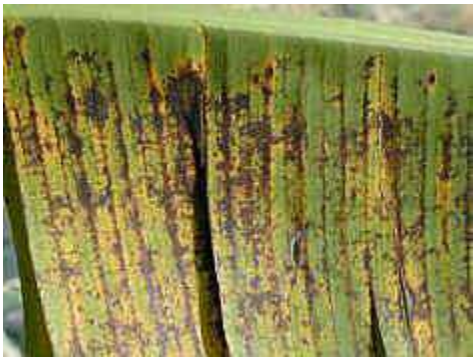
The spread of the disease is by air borne conidia and numerous insects which frequently visit banana flowers also spread the disease.

Management

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

Freckle or Black Spot: *Phyllostictina musarum*

Symptoms



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

Pathogen

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

Management

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

Banana bunchy top: *Banana bunchy top virus*

Economic Importance

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

Symptoms

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

Mode of spread

Secondary spread is through the aphid vector *Pentalonia nigronervosa*

Management

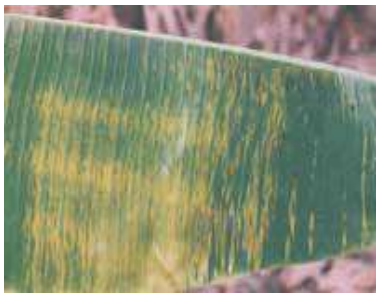
Select suckers from disease free areas. Control vector by spraying methyl demoton 1 ml/l.or Monocrotophos, 2 ml/l.or Phosphomidon 1 ml / lit. or Injection of Monocrotophos 1 ml / plant (1 ml diluted in 4 ml). Infected plants are destroyed using 4ml of 2, 4, D (50g in 400 ml of water).

Infectious chlorosis: *Cucumber mosaic virus*

Economic Importance

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

Symptoms



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

Management

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

Lecture 04 - Diseases of Grapes

Downy mildew: *Plasmopara viticola*

Symptoms

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

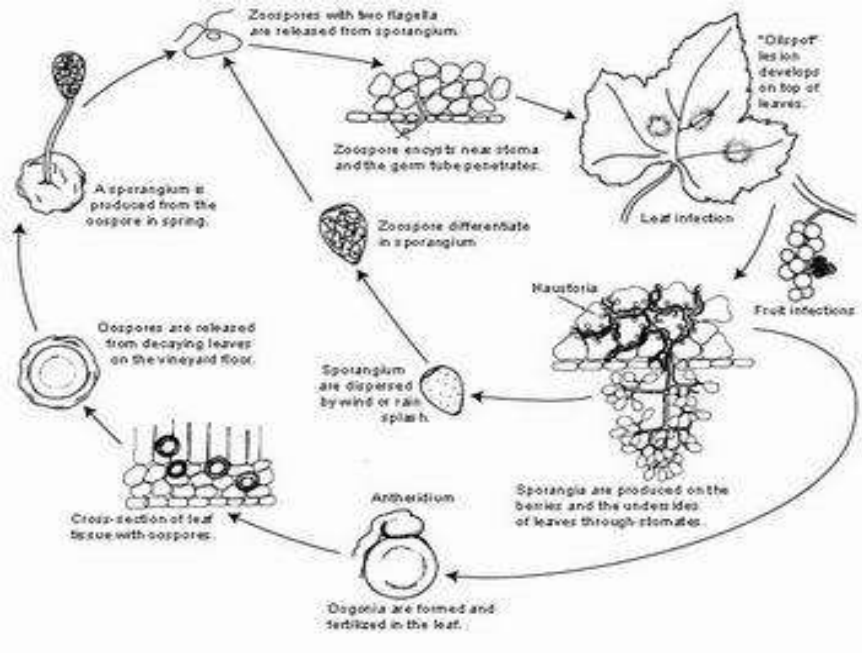
Pathogen

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

Mode of Spread and Survival

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

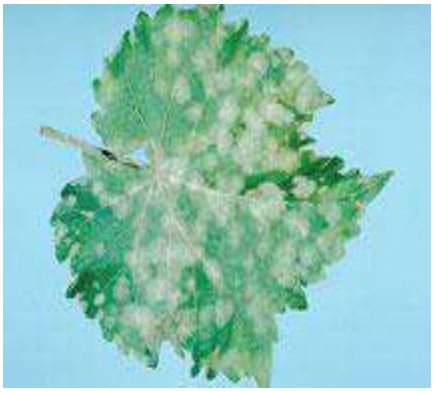
Disease Cycle



Management

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

Powdery mildew : *Uncinula necator*



Symptoms

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

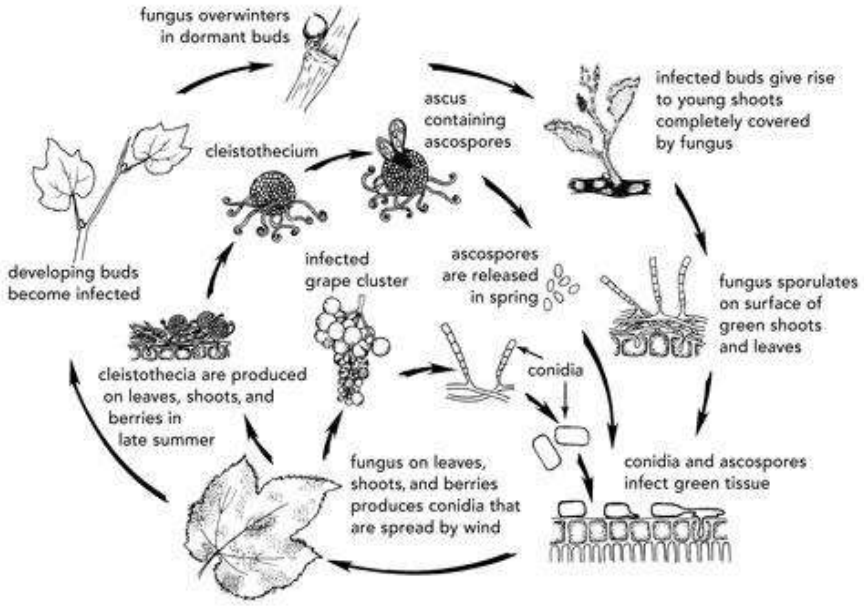
Pathogen

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

Mode of Spread and Survival

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

Disease Cycle



Management

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

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

Symptoms

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

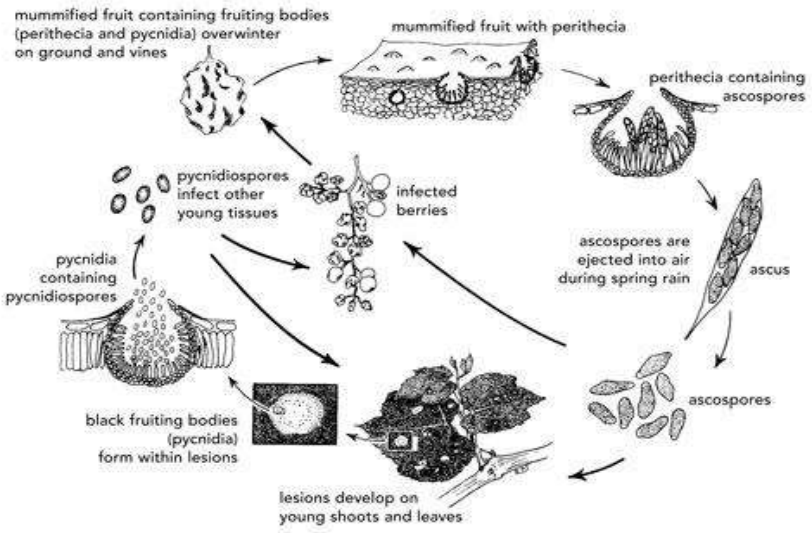
Pathogen

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

Mode of Spread and Survival

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

Disease Cycle



Management

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

Lecture 05 - Diseases of Pomegranate and Papaya

Pomegranate

Cercospora fruit Spot: *Cercospora* sp.

Symptom

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

Management

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

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

Symptom

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

Pathogen

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

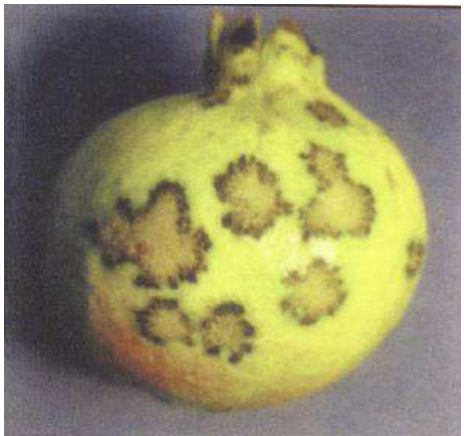
Mode of spread and survival

The pathogen spread through wind borne conidia.

Management

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

Alternaria fruit spot: *Alternaria alternata*



Symptom

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

Management

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

Fruit Rot (*Aspergillus foetidus):*

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

Management

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

Papaya

Stem rot / Foot rot – *Pythium aphanidermatum*

Symptoms

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

Pathogen

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

Management

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

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

Powdery mildew – *Oidium caricae*

Symptoms

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

Pathogen

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

Mode of spread and survival

The pathogen spread through wind borne conidia.

Management

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

Papaya ring spot – Papaya ring spot virus

Symptoms

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

Pathogen

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

Mode of spread

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

Management

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

Leaf curl – *Papaya leaf curl virus*

Symptoms

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

Mode of spread

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

Management

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

Anthracnose – *Colletotrichum gloeosporioides*

Symptom

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

Mode of spread

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

Management

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

Lecture 06 - Disease of Guava

Powdery mildew – *Oidium caricae*

Symptoms

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

Pathogen

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

Mode of spread and survival

The pathogen spread through wind borne conidia.

Management

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

Papaya ring spot – Papaya ring spot virus

Symptoms

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

Pathogen

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

Mode of spread

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

Management

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

Leaf curl – *Papaya leaf curl virus*

Symptoms

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

Mode of spread

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

Management

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

Anthracnose – *Colletotrichum gloeosporioides*

Symptom

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

Mode of spread

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

Management

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

Lecture 06 - Disease of Guava and Sapota

Guava

Anthracnose: *Colletotrichum gloeosporioides*

Symptoms

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

Pathogen

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

Mode of Spread and Survival

The conidia are spread by wind or rain.

Management

Spray Mancozeb 0.25%.

Guava rust: *Puccinia psidii*

Symptoms

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

Management

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

Lecture 07 - Disease of Apple (2 Lectures)

Scab –*Venturia inaequalis*

Symptoms

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

Pathogen

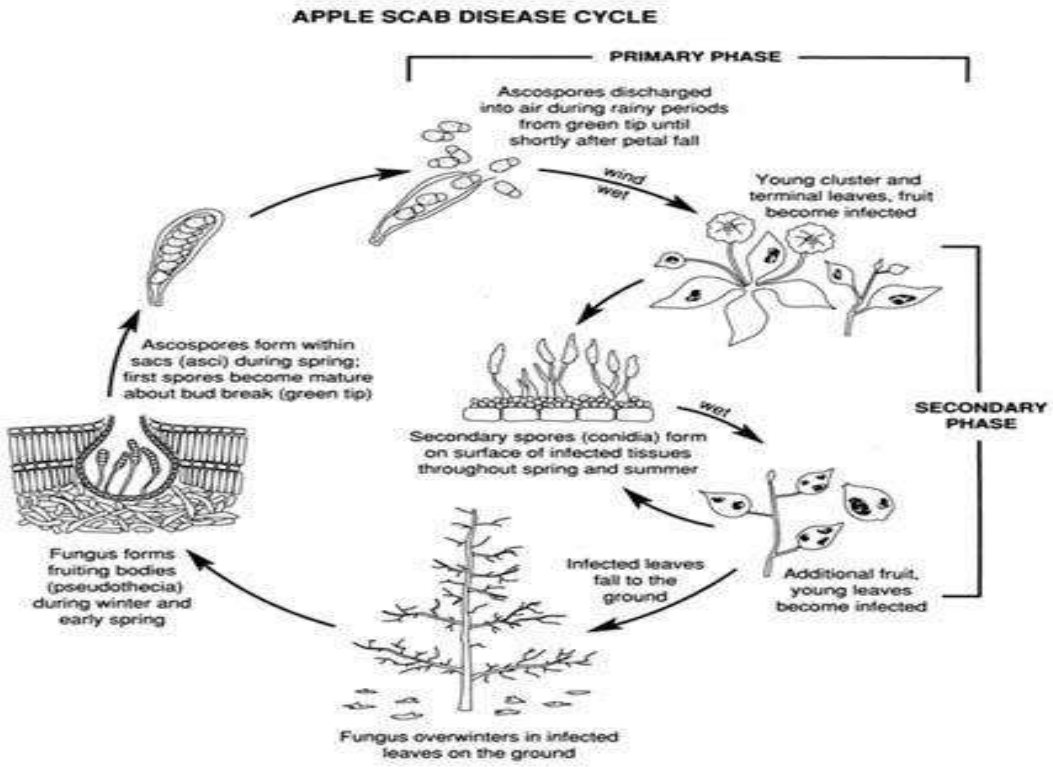
The mycelium is internal. Ascospores are two celled, greenish, grey or yellowish in color.

Mode of Spread and Survival

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

Disease cycle

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



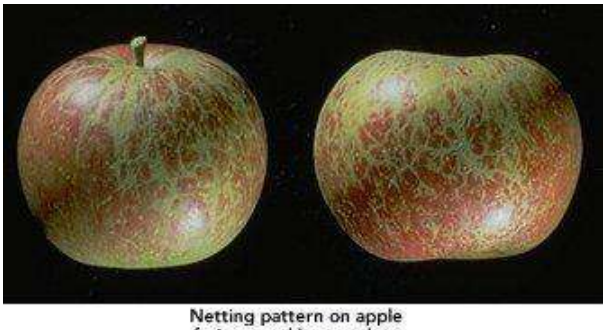
Clean cultivation, collection and destruction of fallen leaves and pruned materials in winter to prevent the sexual cycle. Spray Tridemorph 0.1% before flowering. Spray Mancozeb 0.25 % at bearing stage. Spray 5 % urea prior to leaf fall in autumn and 2 % before bud break to hasten the decomposition of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

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

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

Powdery mildew – *Podosphaera leucotricha*.

Symptom



Netting pattern on apple fruit caused by powdery mildew infection.

Malformation of apple leaves caused by powdery mildew.

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

Pathogen

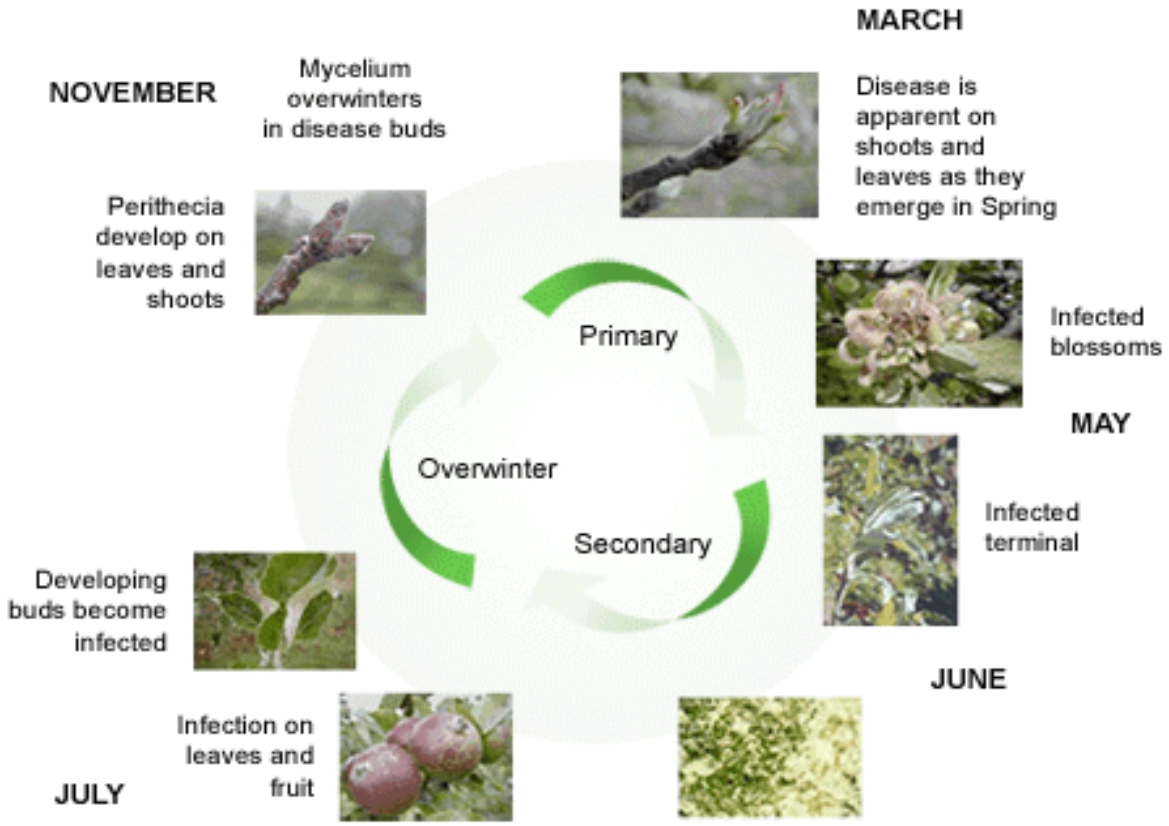
Powdery mildew is caused by, *Podosphaera leucotricha*, an ascomycetous heterothallic fungus. Conidia are ellipsoidal, truncate and hyaline. Perithecia are subglobose, are densely gregarious, and rarely scattered, and have apical and basal appendages. The asci in the perithecia are oblong to subglobose.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

Mode of Spread and Survival

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

Disease Cycle

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



Management:

Spray Dinocap 0.05% or Chinomethionate 0.1%

Fire blight- *Erwinia amylovora*

Symptom

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

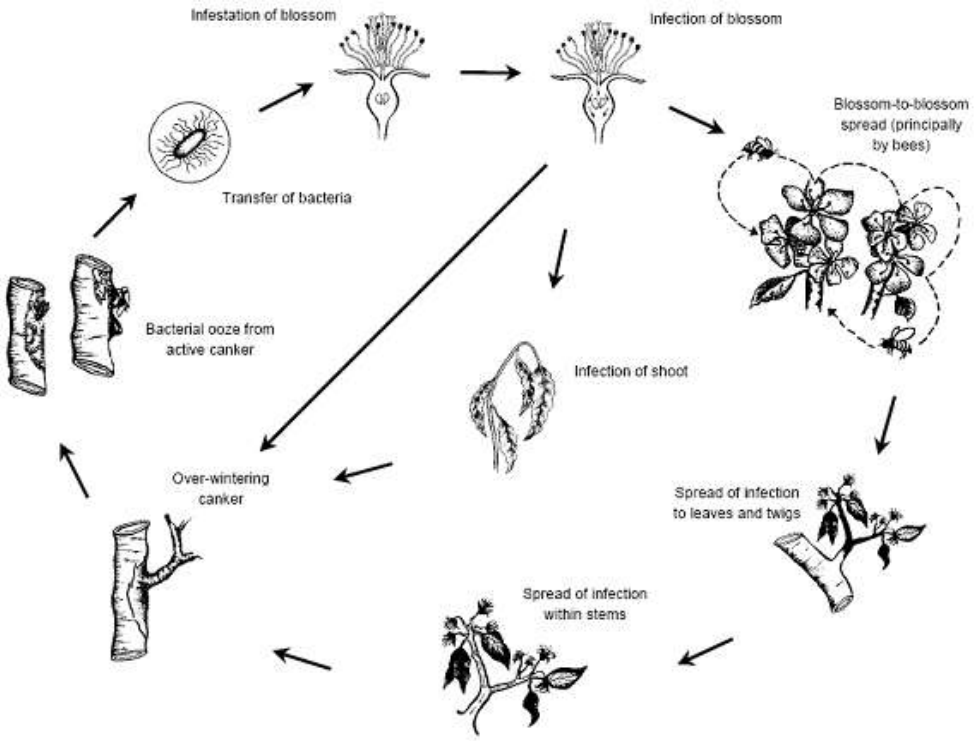
Pathogen

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

Mode of Spread and Survival

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

Disease Cycle

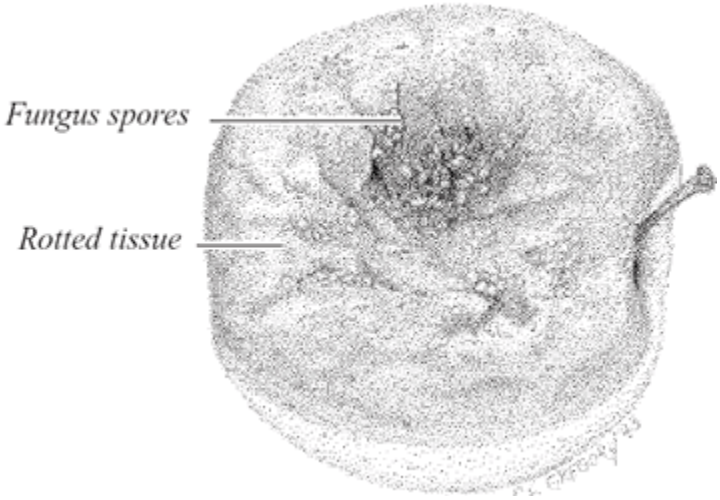


Management

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

Soft rot – *Penicillium expansum*

Symptom



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

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

Pathogen

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

Mode of Spread and Survival

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

Disease cycle

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

Management

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

Bitter rot – *Glomerella cingulata*

Symptom

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

Disease Cycle

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

Management

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

Lecture 08 - Diseases of Chilli

Damping off: *Pythium aphanidermatum*

Symptoms:

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

Pathogen

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

Mode of spread and survival

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

Management

Soil drenching with Copper oxychloride 0.25%

Fruit Rot and Die Back- *Colletotrichum capsici*

Symptoms:

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

Pathogen

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

Mode of spread and survival

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

Management

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

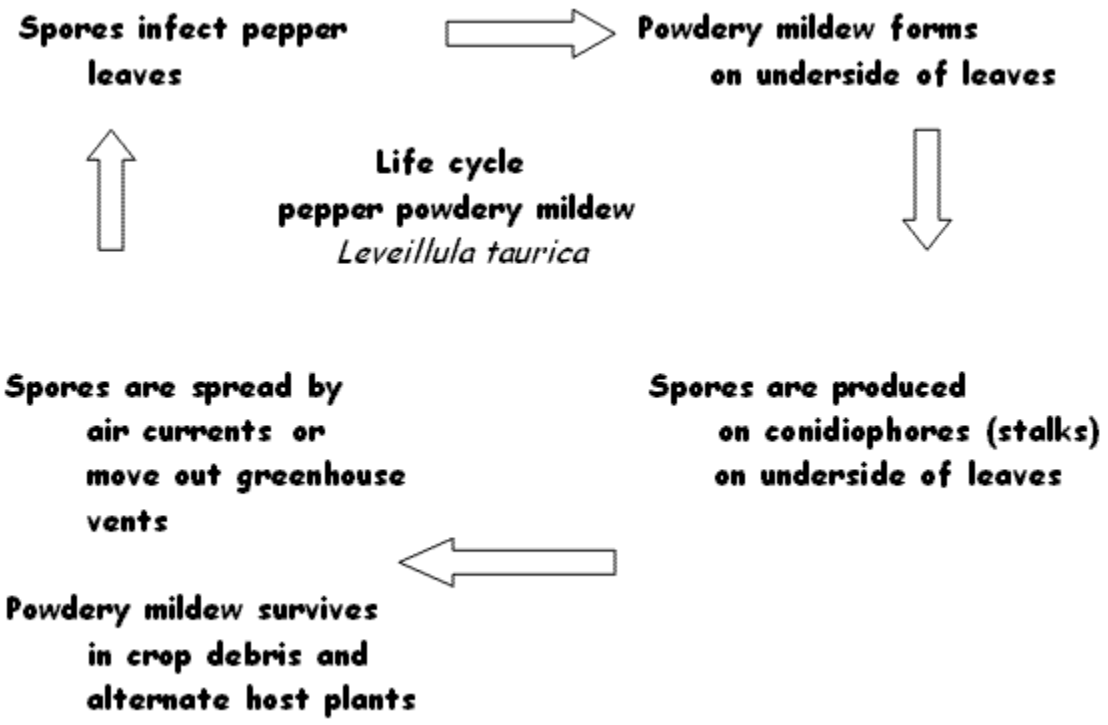
Powdery mildew: *Leveillula taurica*

Symptoms

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

Disease cycle

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



Management

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

Bacterial leaf spot: *Xanthomonas campestris pv. vesicatoria*

Symptoms

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

Mode of spread and survival

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

Management

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

Cercospora leaf spot :*Cercospora capsici*

Symptoms

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

Pathogen

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

Mode of spread and survival

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

Management

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

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

Symptoms

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

Pathogen

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

Management

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

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

Leaf curl

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

Mosaic Viruses

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

Management of viral diseases

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

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

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

Symptoms

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

Conditions for Disease Development

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

Management

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

Alternaria Rot- *Alternaria sp.*

Symptoms

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

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

Pathogen

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

Mode of spread and survival

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

Management

Pre storage dry heat

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

Lecture 09 - Diseases of Brinjal

Bacterial Wilt: *Pseudomonas solanacearum*

Symptoms

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

Pathogen

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

Mode of spread and survival

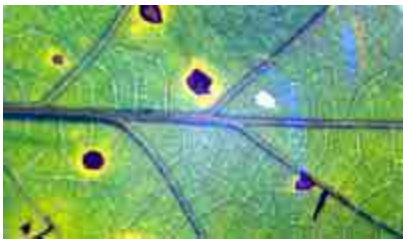
The bacterium infects banana, chillies, fennel, ginger, potato, radish, tomato etc., the bacterium though a non spore former is found to be alive and viable for more than 16 months under laboratory conditions. The pathogen is found to be alive in the infected plant debris for about 10 months. Presence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

Management

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

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

Symptoms



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

Pathogen

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

Mode of spread and survival

The disease is spread by air borne conidia.

Management

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

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

Symptoms

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

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

Pathogen

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

Mode of spread and survival

The disease is spread by wind borne conidia.

Management

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

Little Leaf of Brinjal

Economic Importance

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

Symptom

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

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

Pathogen

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

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

Mode of spread and survival

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

Management

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

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

has been recommended for vector control.

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

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

Symptom

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

Management

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

Tobacco mosaic virus (TMV)

Symptoms

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

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

Management

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

Collar rot :*Sclerotium rolfsii*

Symptoms

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

Management

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

Fruit rot :*Phomopsis vexans*

Symptoms

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

Pathogen

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

Mode of spread and survival

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

Management

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

Lecture 10 - Diseases of Bhendi

Bhendi

Cercospora Leaf Spots:*Cercospora malayensis,*

C. abelmoschi

Symptoms

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

Pathogen

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

Mode of spread and survival

The fungus survives in the diseased crop material.

Management

Spraying Mancozeb 0.25 % control the disease.

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

Symptoms

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

Pathogen

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

Mode of spread and survival

The fungus is soil borne.

Management

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

Powdery mildew: *Erysiphecichoracearum*

Symptoms

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

Pathogen

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

Management

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

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

Symptoms

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

Pathogen

The virus particles are 16 – 18nm in diameter.

Mode of spread

The virus is spread by whitefly.

Management

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

Phoma canker (*Phoma exigua)*

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

Lecture 11 - Diseases of Potato (2 Lectures)

Late blight of potato: *Phytopthora infestans*

Symptom:

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

Pathogen

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

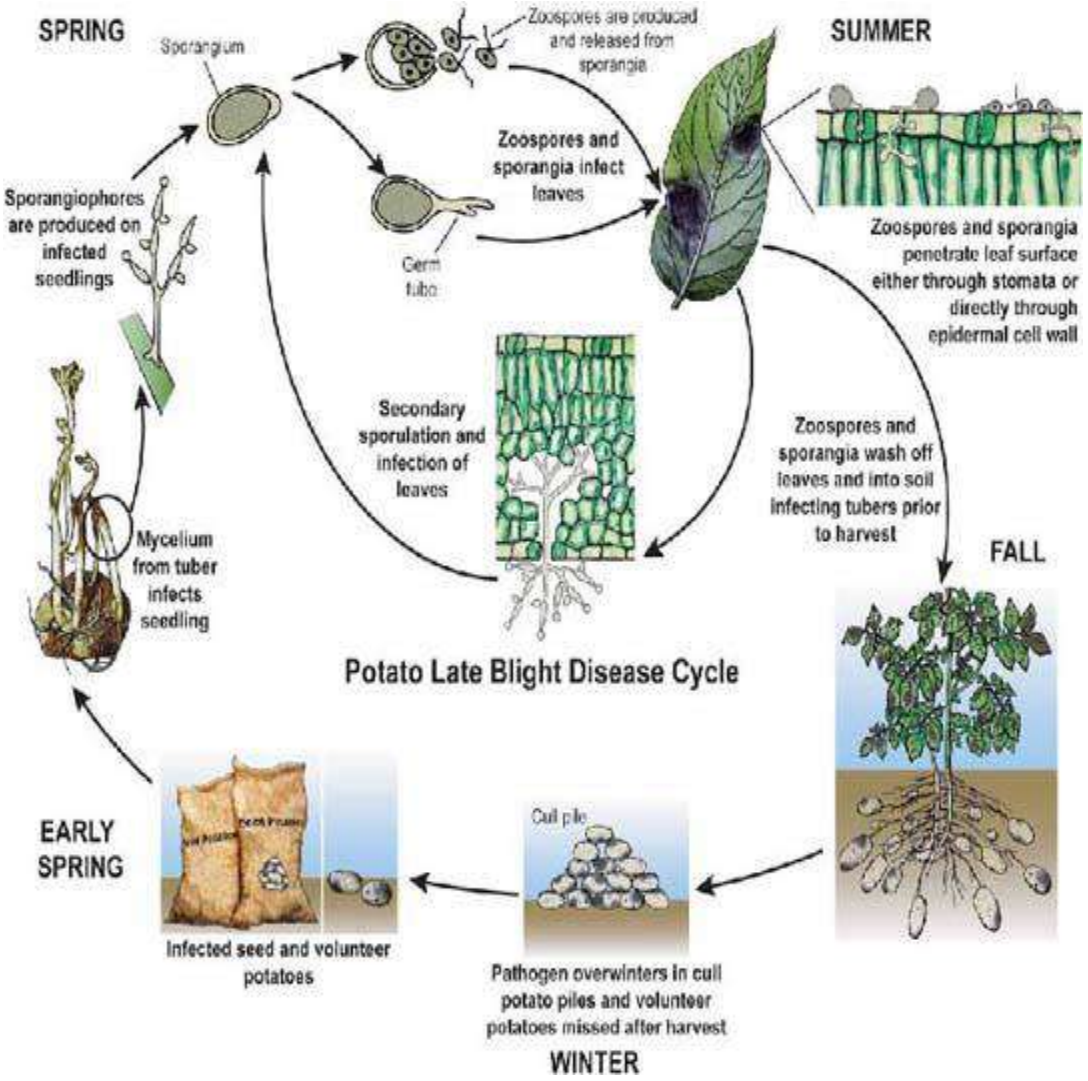
Mode of spread and survival

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

Favourable conditions

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

Rainfall at least 0.1mm, the following day.



Management

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

Early blight: *Alternaria solani*

Symptoms

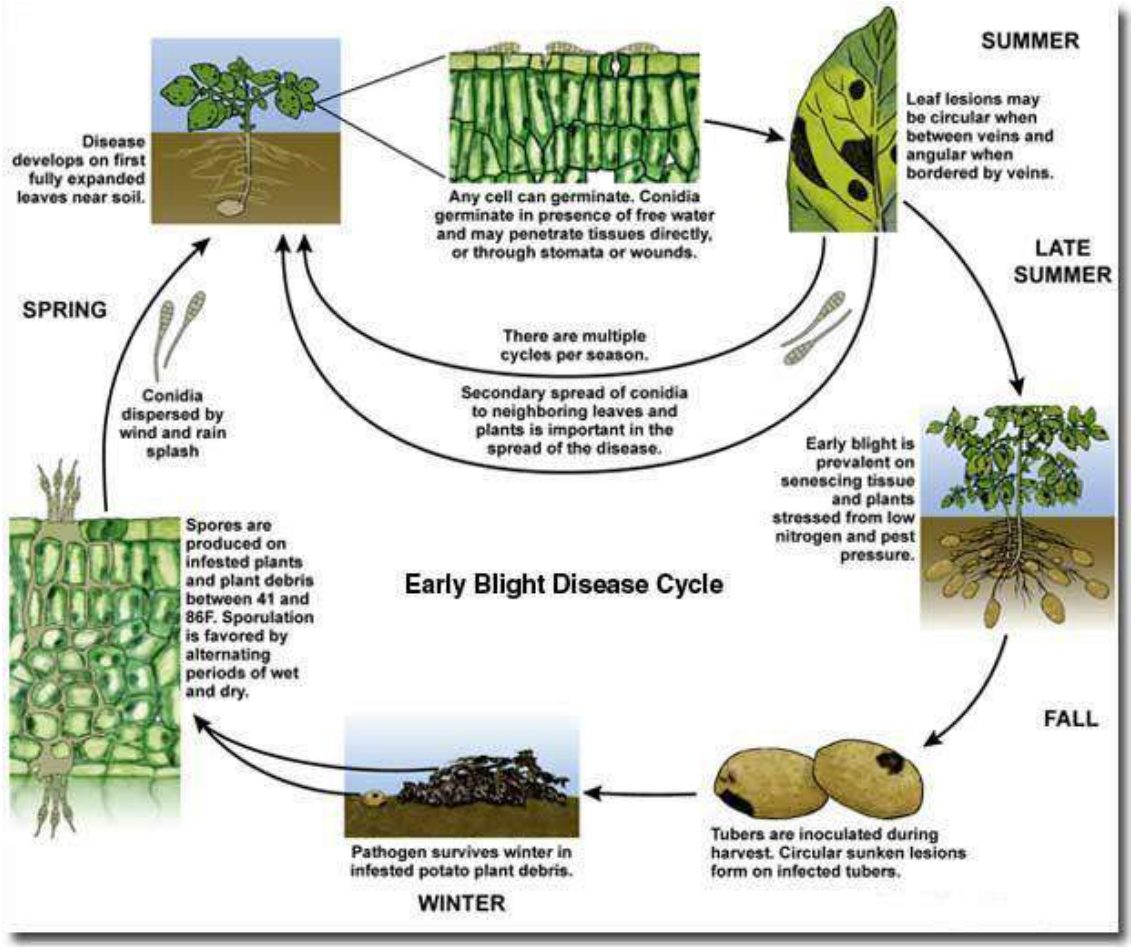
It is present in both hills & plains. Brown-black necrotic spot-angular, oval shape characterized by concentric rings .Several spot coalesce & spread all over the leaf. Shot holes on fruits.

Pathogen

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

Favourable condition

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



Mode of spread and survival

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

Management

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

Post-harvest tuber rots - *Sclerotium rolfsii*

Symptoms

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

Pathogen

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

Favorable condition

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

Mode of spread and survival

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

Management

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

Black scurf- *R. solani*

Symptoms

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

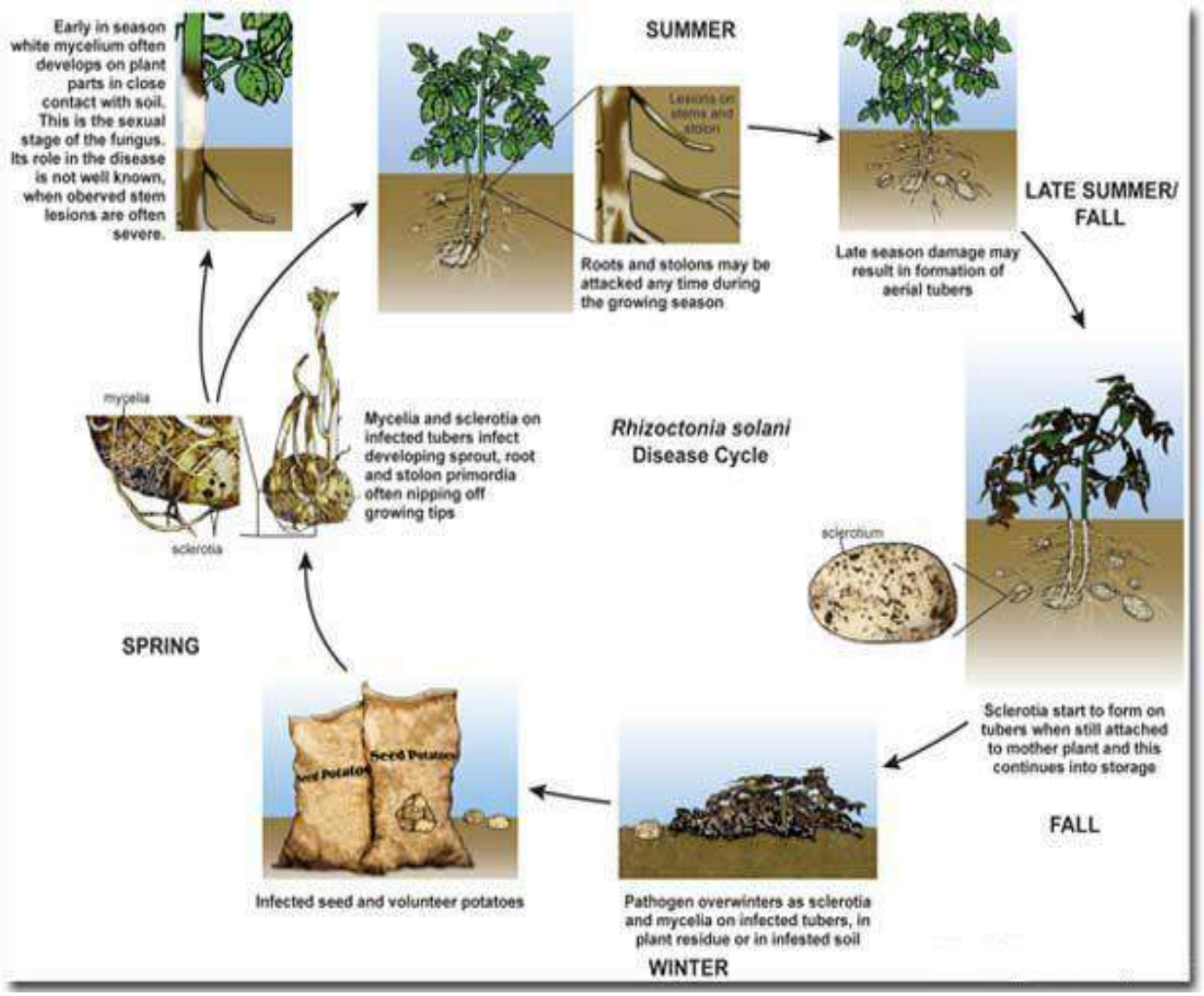
Pathogen

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

Mode of spread and survival

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

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

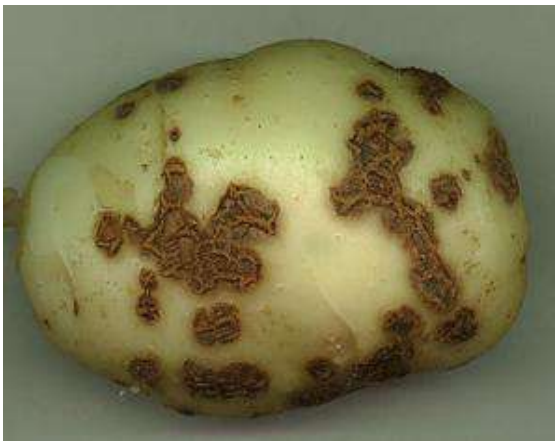


Management

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

Common scab or corkey scab – *Streptomyces scabies*

Symptoms



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

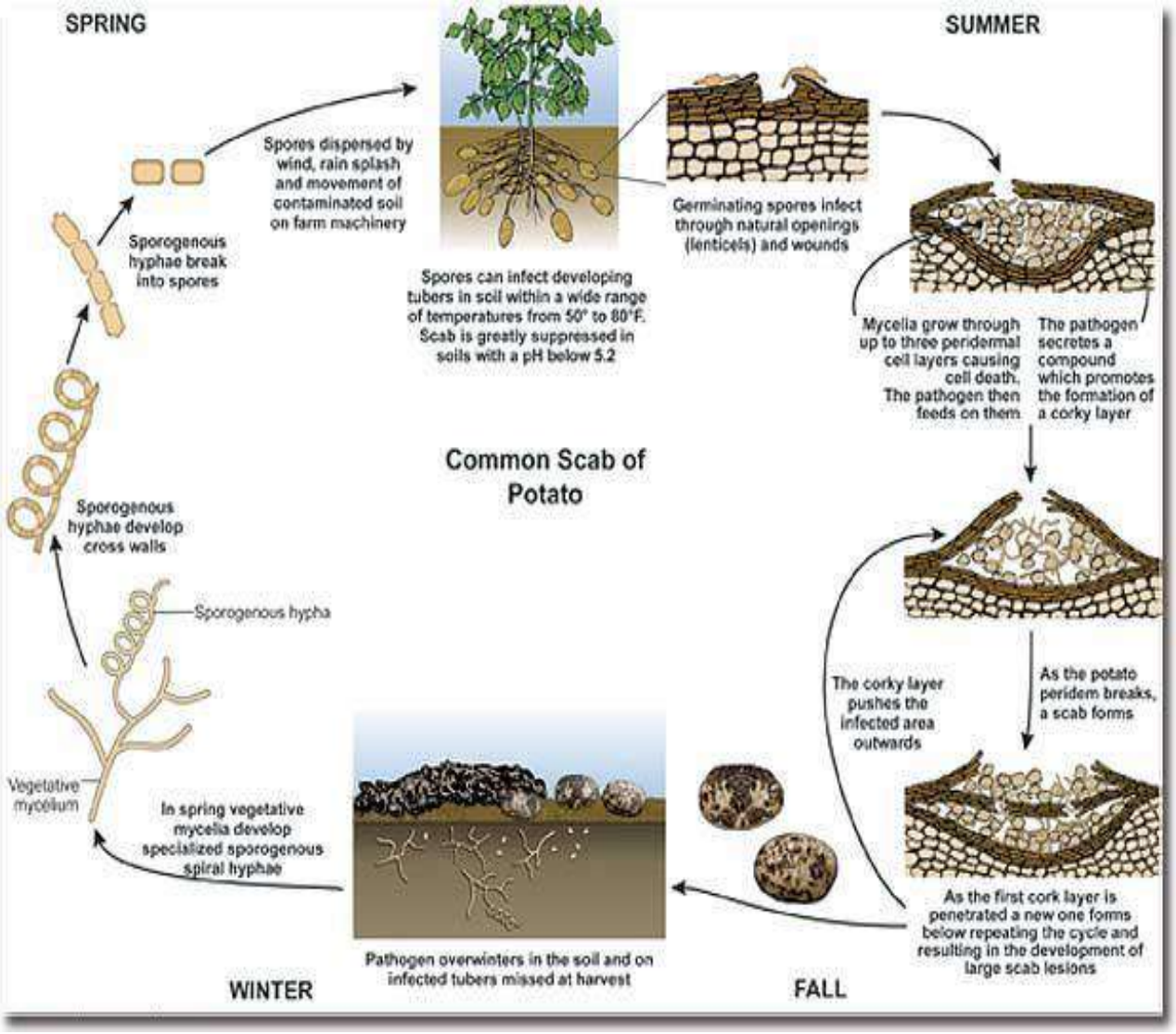
Pathogen

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

Mode of spread and survival

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

Disease Cycle



Management

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

Brown rot or Bangle blight -*Ralstonia solanacearum*

Symptoms

brown vascular bundle more severe infection

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

Pathogen

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

Favourable condition

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

Mode of spread and survival

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

Disease cycle

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

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

Soft rot- *Erwinia carotovora* subsp *caratovora*

Symptoms


Erwinia carotovora

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

Pathogen

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

Mode of spread and survival

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

Management

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

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

Lecture 12 - Diseases of Cucurbits (2 Lectures)

Cucumber and squash

Vascular Wilt: *Erwinia tracheiphila*

Symptoms

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

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

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

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

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

Pathogen

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

Mode of spread and survival

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

Management

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

Scab: *Cladosporium cucumerinum*

Symptoms

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

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

Pathogen

Conidia are oblong, dark, mostly aseptate.

Mode of spread and survival

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

Disease Cycle

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

Management

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

Musk melon and water melon

Gummy Stem Blight - *Mycosphaerella melonis*

Symptoms

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

Mode of spread and survival

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

Disease Cycle

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

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

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

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

Management

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

Bacterial Wilt - *Erwinia tracheiphila*

Symptoms

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

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

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

Pathogen

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

Mode of spread and survival

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

Management

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

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

Symptoms

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

Mode of spread and survival

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

Management

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

Anthracnose *Colletotrichum orbiculare (= C. lagenarium)*

Symptoms

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

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

Mode of spread and survival

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

Management

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

Sudden Wilt

Symptoms

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

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

Management

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

Powdery mildew - *Erysiphe cichoracearum*

Symptoms

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

Pathogen

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

Mode of spread and survival

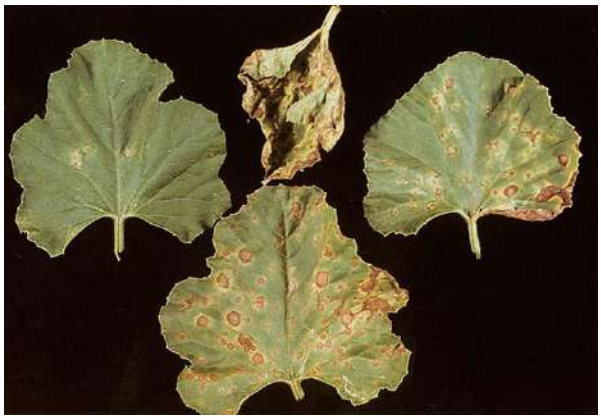
Perithecia developed on left over cucurbit crop in isolated areas serve as primary inoculum. Wild cucurbits harbour the conidial stage of the fungus and release conidia for primary infection to the spring or summer sown cucurbits. Conidia are spread by wind , thrips and other insects.

Management

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

Alternaria Blight - *Alternaria cucumerina*

Symptoms



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

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

Pathogen

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

Mode of spread and survival

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

Management

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

Downy mildew - *Pseudoperonospora cubensis*

Symptoms

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

Pathogen

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

Mode of spread and survival

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

Disease cycle

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

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

Management

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

Angular Leaf Spot - *Pseudomonas lachrymans*

Symptoms

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

Pathogen

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

Mode of spread and survival

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

Management

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

Gourds

Downy mildew: *Pseudoperonospora cubensis*

Symptoms

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

Pathogen

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

Mode of spread and survival

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

Management

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

Powdery mildew: *Erysiphe cichoracearum*

Symptoms

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

Pathogen

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

Mode of spread and survival

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

Management

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

Mosaic: PRSV/CMV

Symptoms:

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

Mode of spread and survival

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

Management

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

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

Lecture 13 - Diseases of Crucifers (2 Lectures)

Beet root

Leaf Spot: *Cercospora beticola*

Symptoms

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

Pathogen

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

Mode of spread and survival

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

Management

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

Downy Mildew: *Perenospora schachtti*

Symptoms

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

Pathogen

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

Management

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

Curly-top virus

Symptoms

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

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

Pathogen

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

Mode of spread and survival

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

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

Management

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

Beet Yellows: Virus

Symptoms

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

Pathogen

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

Mode of spread and survival

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

Management

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

Purple Leaf of Beet virus

Symptoms

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

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

Mode of spread

The virus is readily transmissible through sap.

Management

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

Radish

Alternaria Blight :*Alternaria raphani*

Symptoms

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

Pathogen

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

Mode of spread and survival

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

Management

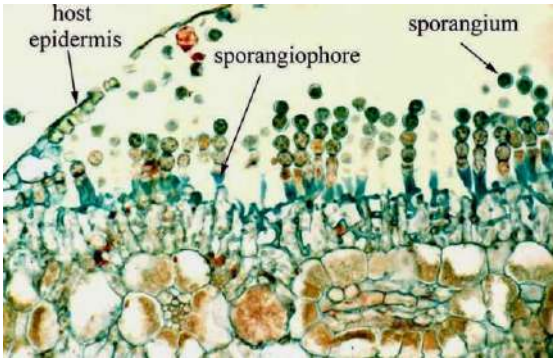
Spraying with Mancozeb 0.25 %

White Rust: *Albugo candida*

Symptoms

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

Pathogen



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

Mode of Spread and Survival

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

Management

Regular spraying with Mancozeb 0.25 % effectively controls the disease.

Cauliflower

Downy Mildew: *Peronospora parasitica*

Symptoms

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

Pathogen

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

Mode of Spread and Survival

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

Management

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

Wire stem: *Rhizoctonia solani*

Symptoms:

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

Pathogen

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

Management

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

Cabbage

Black leg: *Phoma lingam*

Symptoms

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

Pathogen

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

Mode of Spread and Survival

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

Management

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

Downy mildew: *Peronospora parasitica*

Symptoms

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

Pathogen

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

Mode of Spread and Survival

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

Management

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

Root rot: *Rhizoctonia solani*

Symptoms

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

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

Pathogen

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

Management

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

Black spot: *Alternaria* sp.

Symptoms

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

Pathogen

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

Mode of Spread and Survival

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

Management

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

Club root: *Plasmodiophora brassicae*

Symptoms

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

Pathogen

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

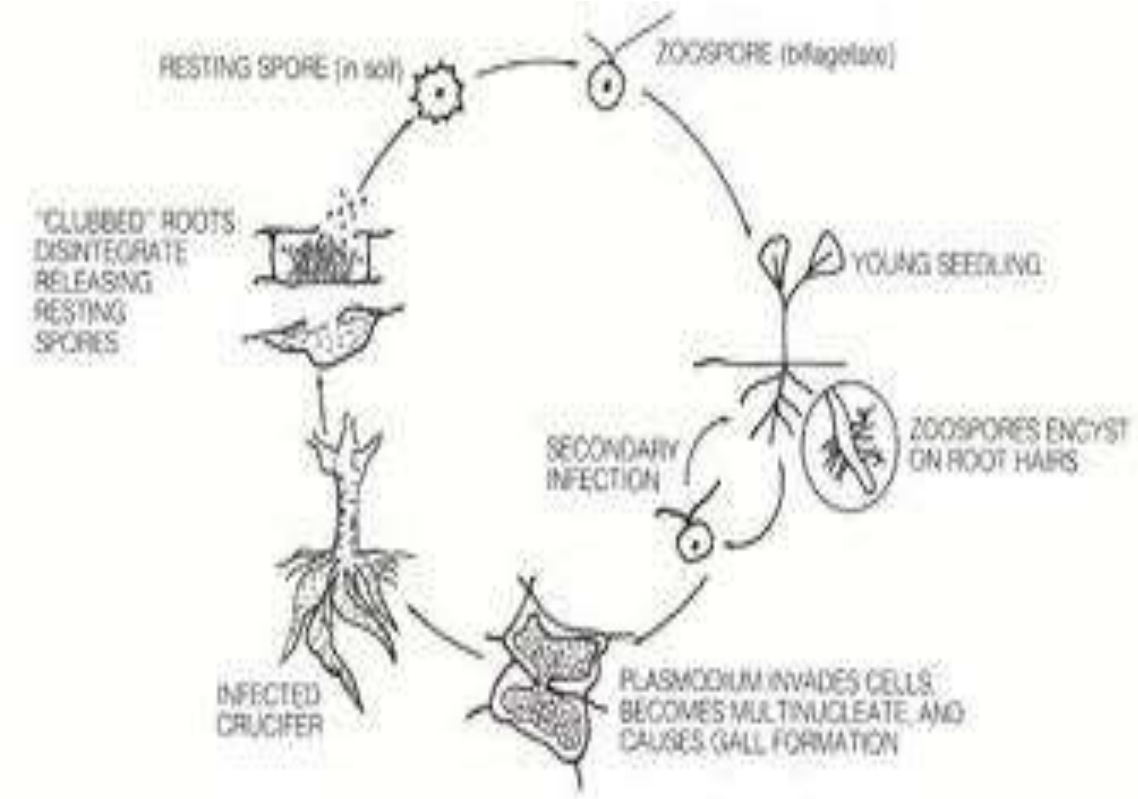
Mode of Spread and Survival

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

Disease Cycle

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

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



Management

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

Powdery mildew: *Erysiphe polygoni*

Symptoms

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

Pathogen

Conidiophores are septate. The cleistothecia are sharp and globose.

Mode of Spread and Survival

The disease spread through water and wind borne conidia.

Management

Spary inorganic sulphur 0.25% or Dinocap 0.05%.

Bacterial diseases

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

Symptoms

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

Pathogen

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

Mode of Spread and Survival

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

Management

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

Turnip

Alternaria Leaf Spot: *Alternaria* spp.

Symptoms

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

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

Pathogen

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

Mode of Spread and Survival

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

Management

Spraying with Mancozeb 0.25 %

Carrot

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

Symptoms

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

Pathogen

The bacterium is rod shaped and polar flagellum.

Mode of Spread and Survival

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

Disease Cycle

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

Management

Spraying early with Copper oxychloride 0.25 %.

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

Symptoms

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

Pathogen

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

Mode of Spread and Survival

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

Management

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

Cercospora leaf spot: *Cercospora carotae*

Symptom

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

Pathogen

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

Mode of Spread and Survival

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

Management

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

Sclerotinia Rot or White mold: *Sclerotinia sclerotiorum*

Symptom:

Mycelia growth and sclerotia (red arrow)

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

Management

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

Asparagus

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

Symptoms

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

Management

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

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

Purple Spot: *Stemphyllium vesicarium*

Symptoms

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

Management

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

Rust: *Puccinia asparagi*

Symptoms

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

Management

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

Lecture 14 - Diseases of Tomato

Damping off: *Pythium aphanidermatum*

Symptoms

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

Mode of spread and survival

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

Management

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

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

Symptom

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

Pathogen

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

Mode of spread and survival

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

Management

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

Early Blight : *Alternaria solani*

Symptoms

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

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

Pathogen

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

Mode of spread and survival

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

Management

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

Septoria Leaf Spot:*Septoria lycopersici*

Symptom

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

Pathogen

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

Mode of spread and survival

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

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

Management

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

Bacterial wilt: *Burkholderia solanacearum*

Symptom

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

Pathogen

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

Mode of spread and survival

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

Management

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

Bacterial Leaf Spot : *Xanthomonas campestris pv. vesicatoria*

Symptom

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

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

Pathogen

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

Mode of spread and survival

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

Management

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

Mosaic: *Tomato mosaic virus* (TMV)

Symptom

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

Pathogen

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

Mode of spread and survival

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

Management

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

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

Symptom

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

Pathogen

The virus particles are 80nm in diameter.

Mode of spread and survival

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

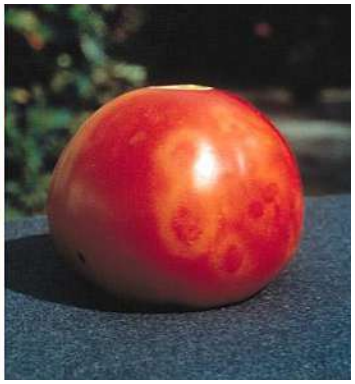
Management

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

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

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

Symptom

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

Pathogen

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

Mode of spread and survival

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

Management

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

Gray Mould: *Botrytis cinerea*

Symptoms

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

Pathogen

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

Mode of spread and survival

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

Management

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

Early Blight : *Alternaria solani*

Symptoms

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

Pathogen

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

Mode of spread and survival

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

Management

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

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

Symptoms

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

Phoma Rot: *Phoma destructive*

Symptoms

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

Pathogen

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

Mode of spread

The pathogen is seed borne.

Management

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

Lecture 15 - Diseases of Beans

Beans

Anthracnose: *Colletotrichum lindemuthianum*

Symptoms

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

Pathogen

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

Mode of spread and survival

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

Disease Cycle

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

Management

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

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

Symptoms

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

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

Management

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

Rust: *Uromyces appendiculaters*

Symptoms

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

Pathogen

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

Mode of spread and survival

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

Management

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

Bacterial Blight: *Xanthomonas campestris* pv *phaseoli*

Symptoms

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

Pathogen

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

Mode of spread and survival

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

Management

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

Mosaic Viruses

Symptoms

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

Pathogen

Cucumber mosaic virus

Mode of spread and survival

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

Management

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

Powdery Mildew: *Erysiphe polygonii*

Symptoms

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

Pathogen

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

Mode of spread and survival

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

Management

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

Cercospora Leaf Spot: *Cercospora* sp.

Symptoms

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

Management

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

Watery Soft Rot: *Sclerotinia sclerotiorum*

Symptoms

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

Management

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

Lecture 16 - Diseases of Onion & Garlic

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

Symptoms

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

Pathogen

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

Mode of spread and survival

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

Management

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

Downy mildew: *Peronospora destructor*

Symptoms

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

Pathogen

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

Mode of spread and survival

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

Disease Cycle

Dormant Period

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

Primary Spread

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

Secondary Spread

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

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

Management

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

Leaf Blight (Blast)*: Botrytis* spp.

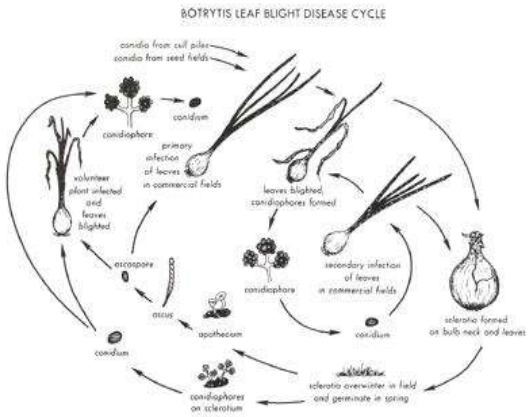
Symptoms

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

Pathogen

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

Disease cycle



Dormant Period

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

Primary Spread

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

Secondary Spread

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

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

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

Management

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

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

Symptoms

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

Pathogen

Pythium aphanidermatum

Epidemiology

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

Management

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

Smut: *Urocystis cepulae*

Symptoms

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

Pathogen

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

Mode of spread and survival

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

Management

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

White Rot: *Sclerotium cepivorum*

Symptoms

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

Mode of spread and survival

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

Disease Cycle

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

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

Management

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

Purple blotch: *Alternaria porri*

Symptoms

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

Pathogen

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

Management

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

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

Symptoms

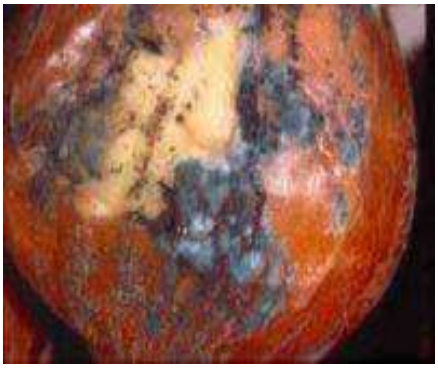
Symptoms usually appear after harvest, although infections originate in the field. Greatest epidemic development occurs when cool (50° to 75°F), moist weather prevails for some days before or during harvest. If the weather remains dry during harvest and curing, losses found in storage are usually small.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

Mode of spread and survival

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

Blue mould rot- *Penicillium* sp

Symptoms



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

Pathogen

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

Mode of spread and survival

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

Black mould- *Aspergillus niger*

Symptoms

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

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

Management

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

Lecture 17 - Diseases of Coconut and Oil palm

Coconut

Bud rot

Phytophthora palmivora

Symptoms

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

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

Pathogen

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

Favorable Conditions

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

Mode of Spread and Survival

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

Mangement

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

Basal Stem Rot (Thanjavur wilt / Bole rot)

Ganoderma lucidum

Symptoms

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

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

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

Pathogen

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

Favourable Conditions

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

Mode of Spread and Survival

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

Management

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

Stem bleeding

Theilaviopsis paradoxa

(*Ceratocystis paradoxa*)

Symptoms

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

Pathogen

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

Favourable Conditions

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

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

Mode of Spread and Survival

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

Management

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

Root wilt disease (Kerala wilt)

Phytoplasma

Symptoms

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

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

Pathogen

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

Favourable Conditons

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

Mode of Spread and Survival

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

Management

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

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

Grey leaf blight

Pestalotia palmarum

Symptoms

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

Pathogen

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

Favourable conditions

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

Mode of Spread and Survival

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

Management

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

Oil palm

Anthracnose: *Botryodiplodia palmarum*

Symptoms

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

Management

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

Lecture 18 - Diseases of Mulberry

Mulberry

Root rot- *Macrophomina phaseolina*

Symptoms

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

Management

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

Stem canker- *Lasiodiplodia (Botryodiplodia) theobromae*

Symptoms

Nursery

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

Grown-up plants

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

Management

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

Leaf rust - *Cerotelium fici*

Symptoms

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

Management

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

Leaf spot- *Cercospora moricola*

Symptoms

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

Management

Spraying carbendazim @ 500-625 g/ha

Powdery mildew - *Phyllactinia corylea*

Symptoms

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

Pathogen

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

Mode of spread and survival

The fungus spreads through conidia or ascospores.

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

Bacterial blight- *Pseudomonas mori*

Symptoms

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

Management

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

Root knot nematode *Meloidogyne incognita*

Symptoms

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

Management

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

Lecture 19 - Diseases of Betelvine

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

Symptoms

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

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

Pathogen

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

Favourable Conditions

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

Mode of Spread and Survival

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

Management

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

Sclerotium foot rot and wilt - *Sclerotium rolfsii*

Symptoms

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

Pathogen

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

Favourable Conditions

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

Mode of Spread and Survival

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

Management

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

Powdery mildew- *Oidium piperis*

Symptoms

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

Pathogen

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

Favourable Conditions

Dry humid weather during the months of May-July.

Mode of Spread and Survival

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

Management

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

Anthracnose- *Colletotrichum piperis*

Symptoms

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

Pathogen

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

Mode of Spread and Survival

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

Management

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

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

Symptoms

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

Pathogen

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

Favourable Conditions

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

Mode of Spread and Survival

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

Management

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

Lecture 20 - Diseases of Coffee

Coffee

Coffee leaf rust - *Hemileia vastatrix*

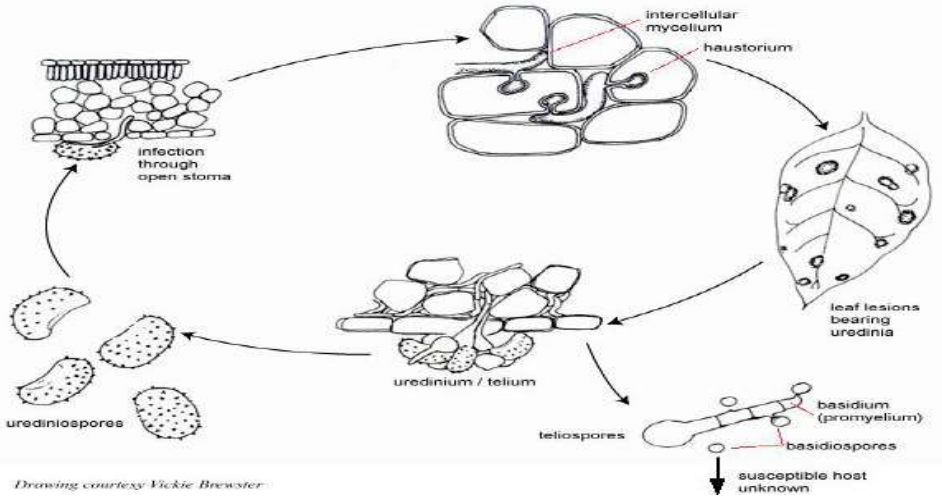
Symptoms

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

Pathogen

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

Disease Cycle



Mode of spread and survival

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

Management

Three applications of 0.5% Bordeaux mixture for susceptible varieties.

Black rot (*koleroga roxia*)

Economic Importance

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

Symptoms

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

Pathogen

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

Mode of spread and survival

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

Management

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

Berry blotch

Symptoms

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

Pathogen

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

Mode of spread and Survival

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

Management

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

Damping off / Collar rot – *Rhizocotonia solani*

Symptoms

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

Mode of spread and survival

The disease is soil borne

Management

Soil drenching with Copper oxychloride 0.25%.

Die back or Anthranose – *Collectorichum coffeanum*

Symptoms

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

Mode of spread and survival

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

Management

Spraying Mancozeb 0.25%

Lecture 21 - Diseases of Tea

Blister blight – *Exobasidium vexans*

Symptoms

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

Pathogen

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

Mode of spread and survival

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

Management

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

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

Black rot

Symptoms

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

Pathogen

Corticium invisum and *C. theae*

Mode of spread and Survival

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

Epidemiology

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

Management

Prune in December end, remove the prunings immediately, burn after drying. Collect all dead and dried leaves. Spray a copper fungide in third week of April.

Red rust: C*ephaleurus mycoidea*

Symptoms

Orange yellow, circular patches appear on upper surface of leaves. The spots become brown and dry up. When it affects the given stem it hardens prematurely.

Pathogen

C*ephaleurus mycoidea* also attacks *Tephrosia* sp. and *Desmodium gyroides* grown as green manure and shade.

Epidemiology

Rainy season is best suited for propagation of algae.

Management

Removal of infected portion and spraying with Copper oxychloride 0.25 %

Black root: *Rosellina areuata*

Symptoms

The fungus originate from the dead heaped leaves of 5 – 7.5 above the soil level. From there if spreads to roots region of tea bushes. When bark is removed star like growth of mycelium can be seen. At the surface of the soil the mycelium surrounds the stem and kills the bank for the length of 7.5 – 10.0 cm. A swollen ring of tissue is formed round the stem above the dead patch.

Pathogen

The fungus produces two kinds of fructification, a conidial stage and a perithecial stage. The conidia are borne on short bristle like stalks. The perithecia are black and spherical. They bear asci which in turn bear ascospores.

Mode of spread

The disease is spread by wind

Management:

Removal and destruction of infected plant. Clean cultivation with out fallen leaves

Dig a drench around the infected bush to provide sunlight in the drench which prevent the spread of mycelium.

Lecture 22 - Diseases of Rose

Black spot- *Diplocarbon rosae*

Economic Importance

Black spot of rose is a serious problem in chill and cold climate of temperate regions. The disease causes marked reduction in the size and number of flowers.

Symptoms

Black lesions with feathery margins surrounded by yellow tissue are found on the leaves. Infected leaves drop prematurely. Purple/red bumpy areas on first year canes may be evident. Plants may be weakened due to defoliation and reduced flower production may be observed.

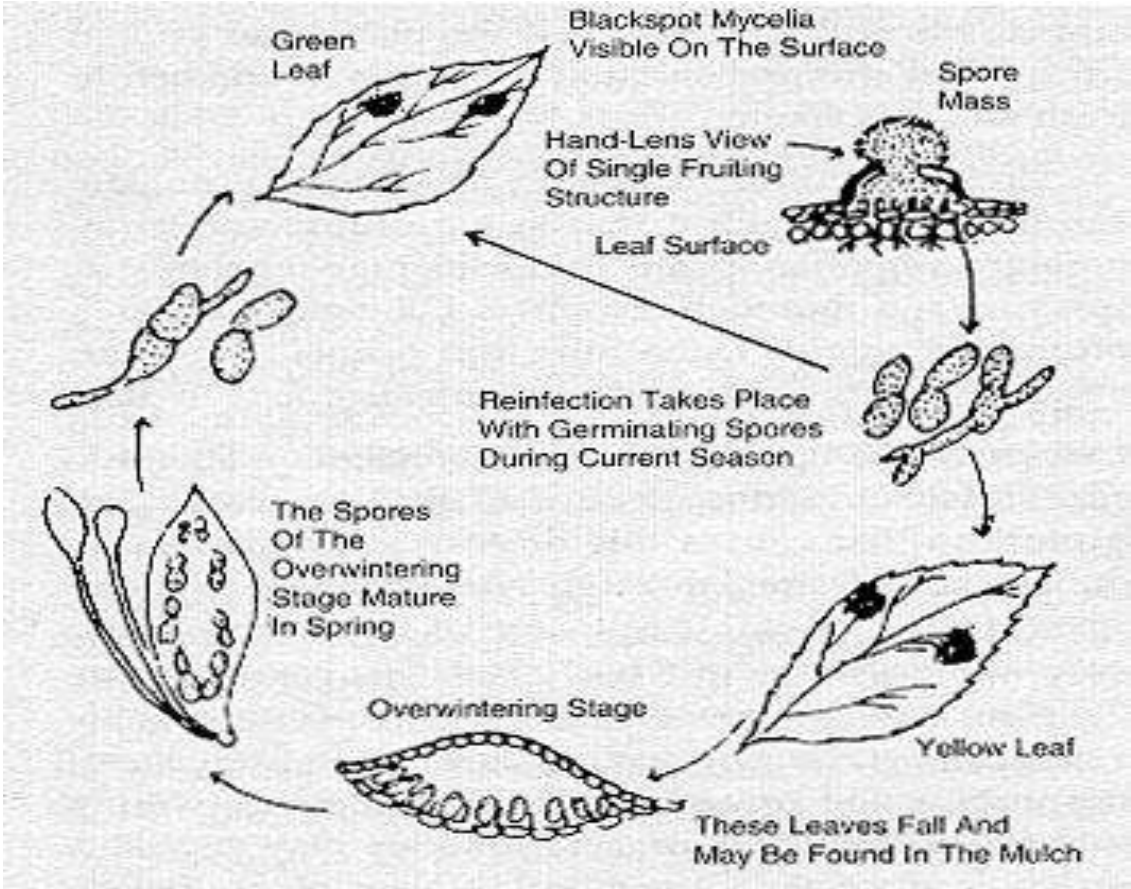
Pathogen

The vegetative body of the fungus consists of two parts *viz*., the subcuticular mycelium and the internal mycelium. The fungus produces acervuli on the central part of the tar spots as blister like projections. Asci are discoid, sub epidermal, erumpent and 84 to 224 micron meter in diameter. Stroma is thin. Conidiophores are hyaline short and cylindrical. Conidia are hyaline, two celled, fusiform or allantoid to obclavate, upper end round, base narrow, guttulate, 18 – 25 x 5 – 6 micron meter.

Mode of spread and survival

The fungal spores are spread primarily by splashing rain or water. Germination of the spores and infection occur when free water remains on the leaf surface for a period of 6 hours or longer. Leaf spots develop within 5 to 10 days.

Disease Cycle



Management

Cultural-Roses should be planted where the sun can quickly dry the night's dew. Space roses far enough apart for good air circulation Avoid overhead watering and keep foliage as dry as possible.Remove infected canes and burn diseased leaves. Spraying with Mancozeb (or) Chlorothalonil 0.2% (or) Benomyl 0.1% or a copper dust .

Powdery mildew – *Sphaerotheca pannosa*

Economic Importance

It is one of the widely distributed disease of rose. Powdery mildew is prevalent during Oct – Jan in south India and Dec- Feb in North India.

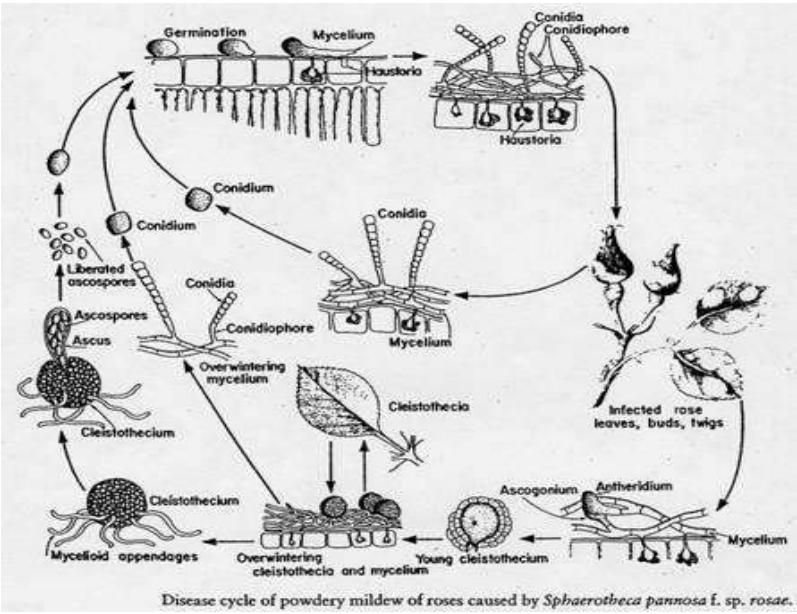
Symptoms

The symptom appears as grayish-white powdery substance on the surfaces of young leaves, shoots and buds. Infected leaves may be distorted, and some leaf drop may occur. Flower buds may fail to open, and those that do may produce poor-quality flowers. It can occur almost anytime during the growing season when temperatures are mild (70 - 80 °F) and the relative humidity is high at night and low during the day. It is most severe in shady areas and during cooler periods.

Pathogen

Mycelium is white , septate, ectophytic and sends globose haustoria into the epidermal cells of the host. Conidiophores are short and erect. Conidia are one celled, oblong, minutely verrucose with many large fat globules and 22.5 – 29.0 x 12.9 to 14.5 micron meter. Cleistothecia are formed towards the end of the season on the leaves, petals, stems and thorns. Cleistothecia are with simple myceloid appendages. Each ascus contains eight ascospores.

Disease Cycle



Disease cycle of powdery mildew of roses caused by *Sphaerotheca pannosa* f. sp. *rosae*.

Mode of spread and survival

The fungus over winters as mycelium in dormant buds and shoots which are not entirely killed. Either conidia or ascospores serve as primary inoculum. The secondary spread is through wind borne conidia.

Management

Collection and burning of fallen leaves. Spray with Wettable sulphur 0.3% (or) Dinocap 0.07% (or) Carbendazim 0.1% 2-3 sprays at 15 days interval is effective. Sulphur dust at 25 kg/ha. Use of sulphur at higher temperature conditions will be phytotoxic.

Die back – *Diplodia rosarum*

Economic Importance

In India it was first reported in 1961 from Delhi. Now it occurs in all the rose growing areas.

Symptoms

Drying of twigs from tip down wards. Blackening of the twigs. The disease spreads to root and causes complete killing of the plants.

Pathogen

The fungus produces round, black pycnidia which bear spores. The pycnidiospores are dark coloured and two celled. Perithecia are immersed in the host tissue and are surrounded by a pseudostroma. Ascospores are ellipsoidal or fusoid, hyaline, two celled with the septum in or near the middle.

Mode of spread and survival

The fungus persists in dead twigs and the stalks of the withered blooms.

Management

Pruning should be done so that lesions on the young shoots will be eliminated. Apply chaubatia pastic in the pruned area. Spray with COC 0.2% (or) Difolatan 0.2% (or) Chlorothalonil 0.2% (or) Mancozeb 0.2%

Rust – *Phragmidum mucronatum*

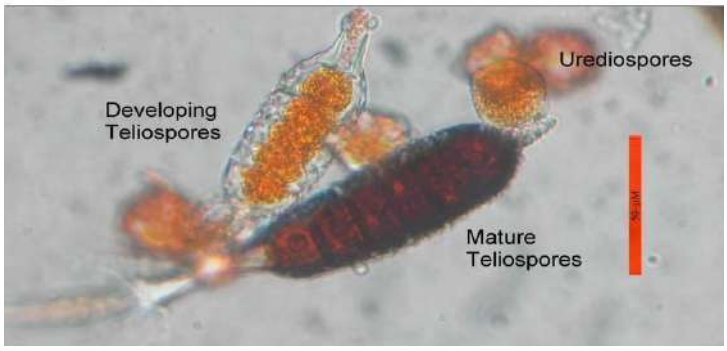
Economic Importance

Rose rust is restricted to higher altitudes. It occurs in Jammu and Kashmir, Himachal Pradesh, Punjab, Tamil Nadu and Uttar Pradesh. Outbreaks of rust disease was reported from Udaipur district of Rajasthan in the variety Chaiti Gulab.

Symptoms

Damage to lemon yellow pustules appear on lower surface of the leaves and stems. Then the color changes to blackish red. The affected leaves turn yellow deformed and fall prematurely. Die back symptom also appear due to weakening of the plant.

Pathogen



Phragmidum mucronatum on rosa sp. Aecidiospores are verrucose, orange yellow, 24 – 25 x 18- 21 micron meter.They are surrounded in the aecidium by club shapped paraphyses. Uredospores are ellipsoid or ovate, echinulate, orange yellow and 21 – 28 x 14 -20 micron meter. The uredospores are borne on short pedicels and are surrounded by paraphyses. Teleutospores are dark coloured, cylindrical, 6- 8 celled with a pointed papilla and 65 – 120 x 30 – 40 micron meter.

Management

Collection and burning of fallen leaves. Spray with Carboxin 0.1% or Wettable sulphur 0.25% or Captan 0.2%

Lecture 23 - Diseases of Chrysanthemum

Wilt -*Fusarium oxysporum* f.sp. *chrysanthemi*

Symptoms

Initial symptoms are in the form of yellowing and browning of leaves. Affected leaves die from the base of the plant upward. Infected plants are stunted and often fail to produce flower. Wilting may cause rotting of root or the base of the stem.

Mode of spread

The fungus is soil borne. The disease spreads through cuttings.

Management

Drenching the soil with or Carbendazim 0.1% is effective. Before planting dipping the rooted cuttings in a solution of Thiram @1.5g/litre of water. Since the disease spreads mostly through cuttings, it is important to use disease free planting material. Disease can further be minimized by following strict sanitation; periodical monitoring; crop rotation and roguing of infected plants.

Rust -*Puccinia chrysanthemi*

Symptoms

The disease symptoms are in the form of brown blister-like swellings, which appear on the undersides of leaves. These burst open releasing masses of brown, powdery spores. Severely infected plants become very weak and fail to bloom properly.

Management

Early removal of infected leaves/plants helps to prevent the further spread of the disease. Spraying the plants with Karathane @ 0.025% or Wettable Sulphur @ 0.3 % is effective in controlling the disease.

Septoria Leaf Spot -*Sepotria chrysanthemella*

Symptoms

Leaf spots occur during cool-wet periods of the rainy season. Since the pathogens are spread through rain splashes the lowermost leaves get infected first. Serious infection may result in premature withering of the leaves; the dead leaves hang to the stem for some time. When flowering starts the infection occurs on flower buds, which rot completely.

Pathogen

Pycnidia are numerous, amphigenous, sub epidermal, globose or lens shaped. Conidia are hyaline, filiform, straight or flexuous often curved or worm like.

Mode of spread and survival

Infected debris in the soil appeared to be primary source of infection or systemic infection carried through suckers. The fungus do not infect other members of the family compositae and is specific to chrysanthemum cultivars only.

Management

This disease can be controlled by spraying Carbendazim 0.1% six times at 15 days intervals from the end of July or spraying Benomyl (0.1%) followed by Captafol (0.2%) Destruction of disease debris and avoiding excessive irrigation is recommended.

Powdery Mildew -*Oidium chrysanthemi*

Symptoms

Infection is more severe in older plants under humid conditions. The growth of the fungus on the leaves appears as powdery coating. Infected leaves turn yellow and dry out. Infected plants remains stunted and fail to flower.

Management

Disease can be effectively controlled with Sulphur fungicides or Captan (0.2%). Good ventilation and proper spacing for free circulation of air is recommended.

Lecture 24 - Diseases of Jasmine and Crossandra

Jasmine

Cercospora leaf spot – *Cercospora jasminicola*

Economic Importance

In India, the disease was first reported in 1946. Now it's known to be widely distributed.

Symptoms

Circular to irregular reddish brown spots of 2-8 mm dia appear on the surface of the leaves. Later the spots become irregular covering larger areas of the leaves.

Pathogen

Stromata are pale to dark brown, globular, filling stomatal openings. Fascicles are mostly dense. Conidiophores are pale olivaceous brown, narrow, sparingly septate and straight or sinuous. It has bluntly rounded tip and are 2 to 4 x 5 to 25 micron meter. Conidia are pale to pale olivaceous obclavate cylindric, indistinctly septate and straight to mildly curved. Its base is obconically truncate and tip is subobtuse and 20 to 66 x 2 to 4 micron meter.

Mode of spread and Survival

It attacks all species of Jasminum. The disease spreads through wind borne conidia.

Management

Spraying with Mancozeb 0.25% (or) Carbendazim 0.1%

Alternaria leaf blight – *Alternaria jasmine, A. alternate*

Symptoms

In the leaves dark brown spots appear. On fumed condition the spots enlarges covering larges area causing blighting of leaves. Concentric rings can be seen the lesions. The disease also affects stem, petiole and flowers.

Mode of spread and Survival

The disease spreads through wind borne conidia.

Epidemiology

The disease attacks Jathi malli (*J. grandiflorum*) and mullai (*J. auriculatum*). The disease is severe during winter months (Oct-Dec). In certain areas the disease is noticed even upto February.

Management

Collection and removal of fallen leaves. Spray with Copper oxychloride 0.25% or Mancozeb 0.25%

Collar rot and Root rot – *Sclerotium rolfsii*

Symptoms

Plants at all stages are infected. First the older leaves become yellow followed by younger leaves and finally death of the plant. In the root black discoloration can be seen. On the infected tissues and stem surface white strands of mycelia and mustard like sclerotia are seen.

Management

Soil drenching with Copper oxychloride 0.25%. Heavy application of FYM with *Trichoderma viride*

Phyllody – *Phytoplasma*

Symptoms

Leaves become small malformed and bushy. In the place of flowers green leaf like malformed flowers are formed.

Mode of spread

The disease is transmitted by grafting and whitefly, *Dialeurodes kirkaldii*.

Management

Selection of cuttings from healthy plants. Spraying insecticide to control the vector.

Lecture 25 - Diseases of Crossandra

Wilt: *Fusarium solani*

Economic Importance

In India it was first reported from Tamil Nadu in 1976. The disease is formed in both air black and sandy loam soil and losses upto 80 % of plants has been reported.

Symptoms

Wilt is observed in patches. In the field the disease is observed one month after transplanting. Leaves of infected plants become pale and droop. Margin of the leaves show pinkish brown discoloration. The discoloration spreads to the midrib in a period of 7 to 10 days. Stem portion gets shriveled. Dark lesions are noticed on the roots extending upto collar region which result in sloughing off the cortical tissue.

Mode of spread and Survival

Chlamydospores survive in soil and they are spread by irrigation water.

Epidemiology

Incidence is more in the presence of root lesion nematode, *Pratylenchus delatrei* and *Helicotylenchus dihystera*.

Management

Affected plants should be pulled out and destroyed to reduce the disease. The nematode can be controlled by soil application of Phorate at the rate of 1 g/plant on 10th day of transplanting. Soil drenching with Carbendazim 0.1 per cent or Copper oxychloride 0.25 per cent on 30 days interval controls the disease. The treatment may be repeated after 3 to 4 weeks if needed.

Stem rot: *Rhizoctonia solani*

The pathogen also causes pre-emergence damping off, Brown to black lesions develop on stem just above soil level and result in girdling of the stem. The lesions extend to the upper part of the stem. The lesions extend to the upper part of the stem and result in collapse of seedlings. The roots are also rotted.

Management

Drenching with Fosesty1-A1 has been found effective in the control of the disease.

Leaf blight: *Colletotrichum crossandrae*

Symptoms

The symptoms of leaves consist of the development of brownish, depressed necrotic areas surrounded by reddish and slightly raised margins. Initially the spots appear as brownish specks but become darker as they expand. The lesions are more prominent on lower leaves and confined to the margins. Infected leaves roll up, shrivel and drop off, leaving a barren stem with a whorl of young leaves at the top.

Management

Spraying with benomyl 0.1% (or) Mancozeb 0.2% (or) Carbendazim 0.1%

Alternaria leaf spot: *Alternaria amaranthi* var. *crossandrae*

Symptoms

This disease was first reported from Tamil Nadu during 1972. Infected leaves show small, circular or irregular yellow spots on the upper surface. They soon enlarge turn brown and develop dark brown concentric rings. Infected leaves become yellow and drop off prematurely.

Management

Spraying with Benomyl 0.1% (or) Mancozeb 0.2% (or) Carbendazim 0.1%.

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

Author

TNAU,

Tamil Nadu

INDEX

1. Diseases of Rice

Fungal Diseases

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

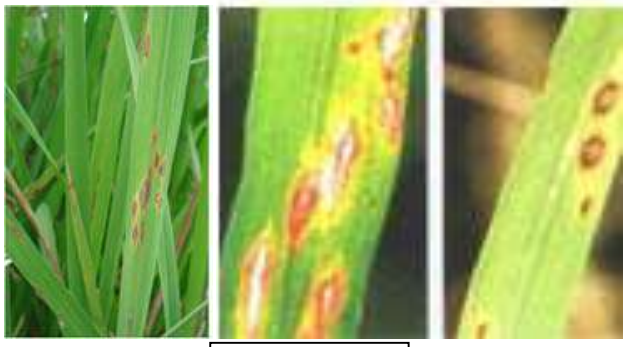
Symptoms

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.

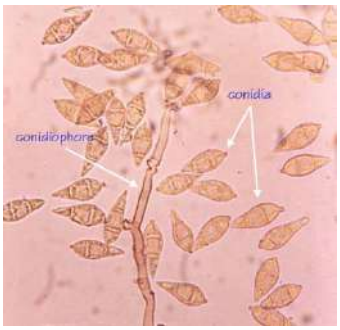


Leaf blast

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.



Conidia and Conidiophore of *P. grisea*

Favourable Conditions

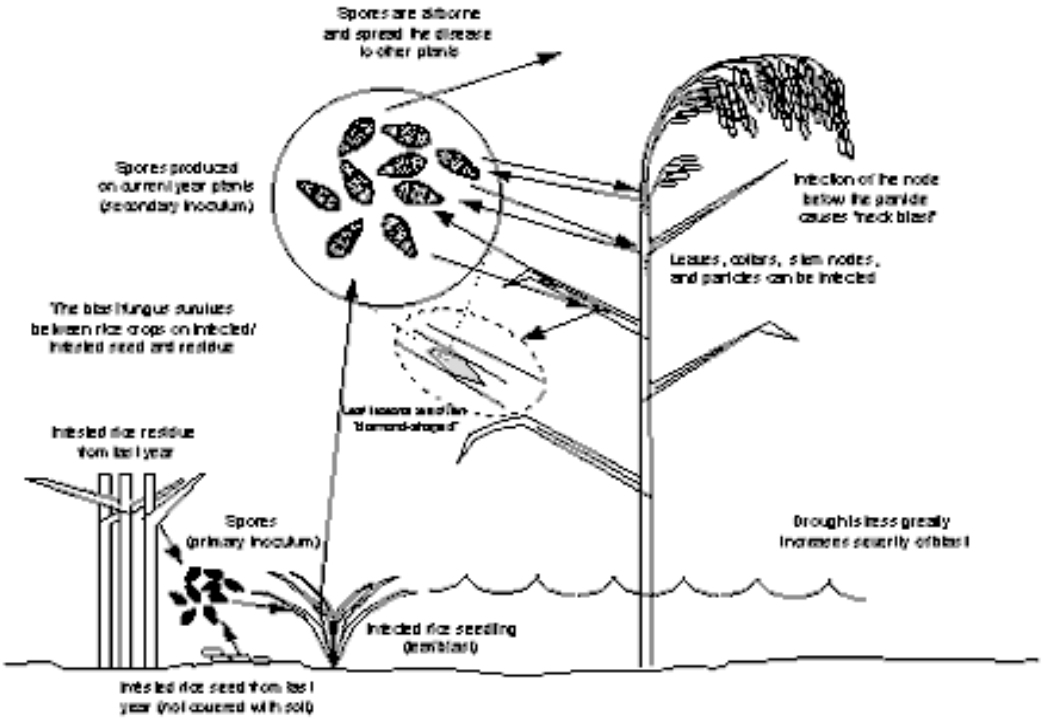
- Intermittent drizzles, cloudy weather, more of rainy days, longer duration of dew high relative humidity (93-99 per cent).
- Low night temperature (between 15-20˚C or less than 26˚C).
- Aavailability of collateral hosts and excess dose of nitrogen.

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.

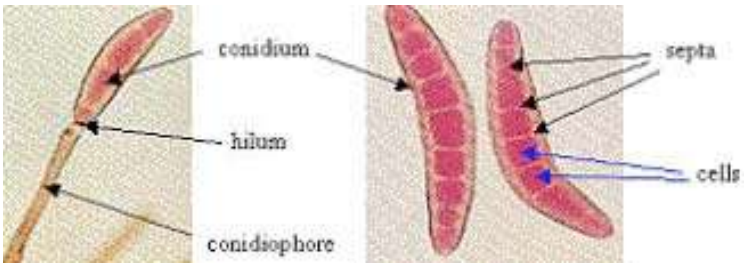
Leaf symptoms

Glume infection

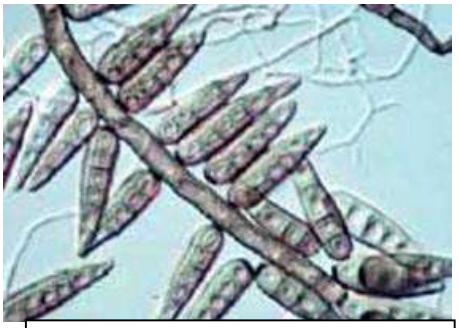
The spots become cylindrical or oval, dark brown with yellow halo later becoming circular. Several spots coalesce and the leaf dries up. The seedlings die and affected nurseries can be often recognised from a distance by scorched appearance. Dark brown or black spots also appear on glumes leading to 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 cylinderical, 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.



Coidia and Conidiophore

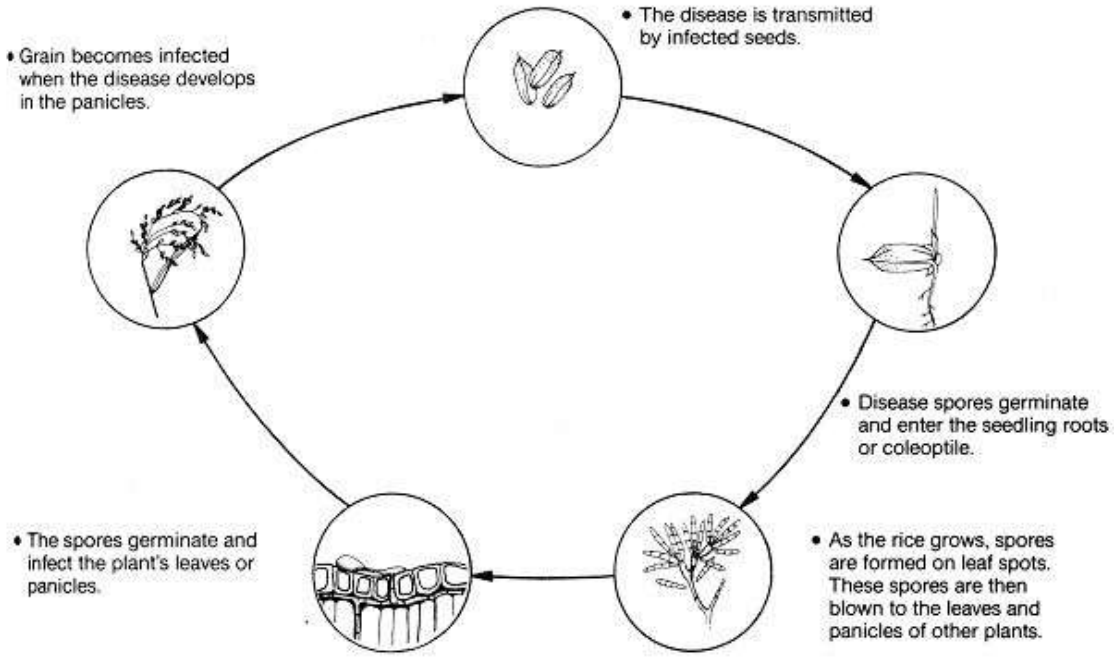
Favourable Conditions

- 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

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

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.

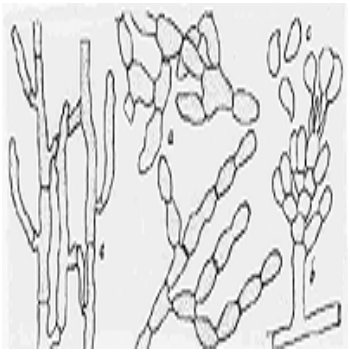
Lesions on the upper parts of plants extend rapidly coalesing with each other to cover entire tillers from the water line to the flag leaf. The presence of several large lesions on a leaf sheath usually causes death of the whole leaf, and in severe cases all the leaves of a plant may be blighted


Symptoms

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.

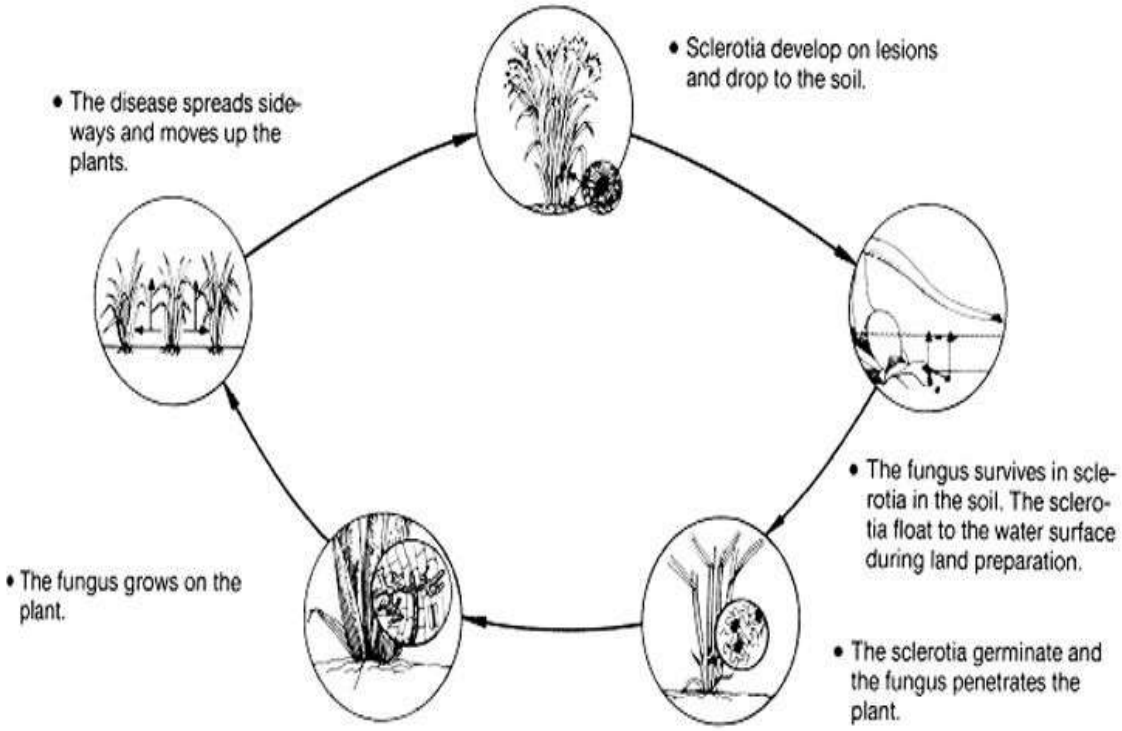



Favourable Conditions

- 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)*

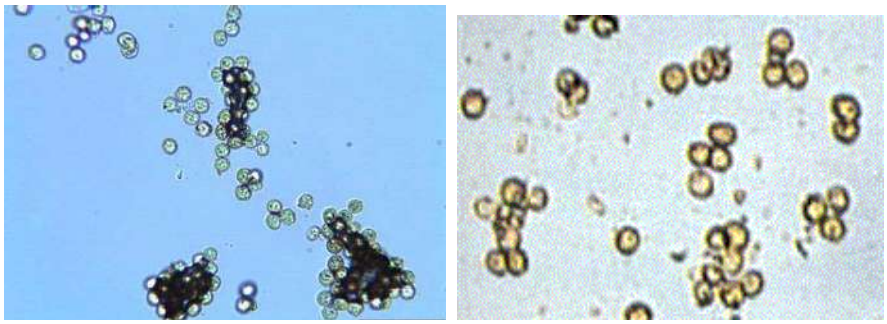
Symptoms

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

Symptoms

Pathogen

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



Spore balls

Disease Cycle

Grasses and wild rice species are alternate hosts. The main source of inoculum is air-borne spores. Ascospores produced from sclerotia act as primary source of infection while chalmydospores 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: *Balansia oryzae-sativa)*

Symptoms

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

Symptoms

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.

Symptoms

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.

Symptoms

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.

Symptoms

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*)

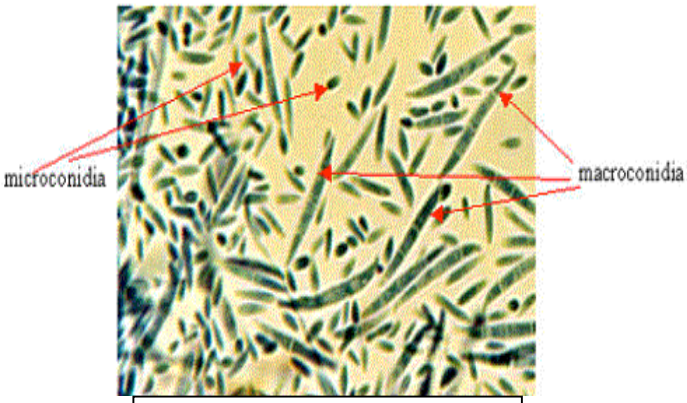
Symptoms

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

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.



Micro and macro conidia

Management

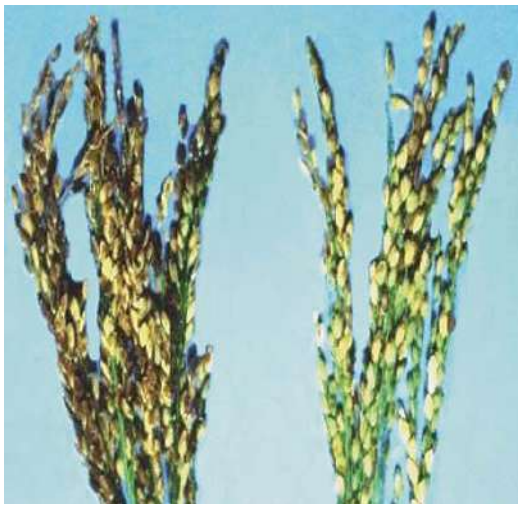
- The fungus is externally seed-borne.
- Treat the seeds with Thiram or Captan or Carbendazim at 2 g/kg.

Grain discolouration - *Drechslera oryzae*, *D. rostratum*, *D.tetramera*, *Curvularia lunata*, *Trichoconis padwickii*, *Sarocladium oryzae*, *Alternaria tenuis*, *Fusarium moniliforme*, *Cladosporium herbarum*, *Epicoccum purpurascens*, *Cephalosporium sp.*, *Phoma sp.*, *Nigrospora sp.*

Symptoms

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

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



Symptoms

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 Disesases

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.

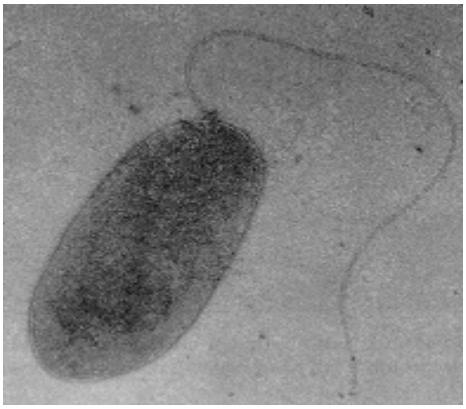
Kresek symptom

Leaf blight symptom

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.



Bacterium

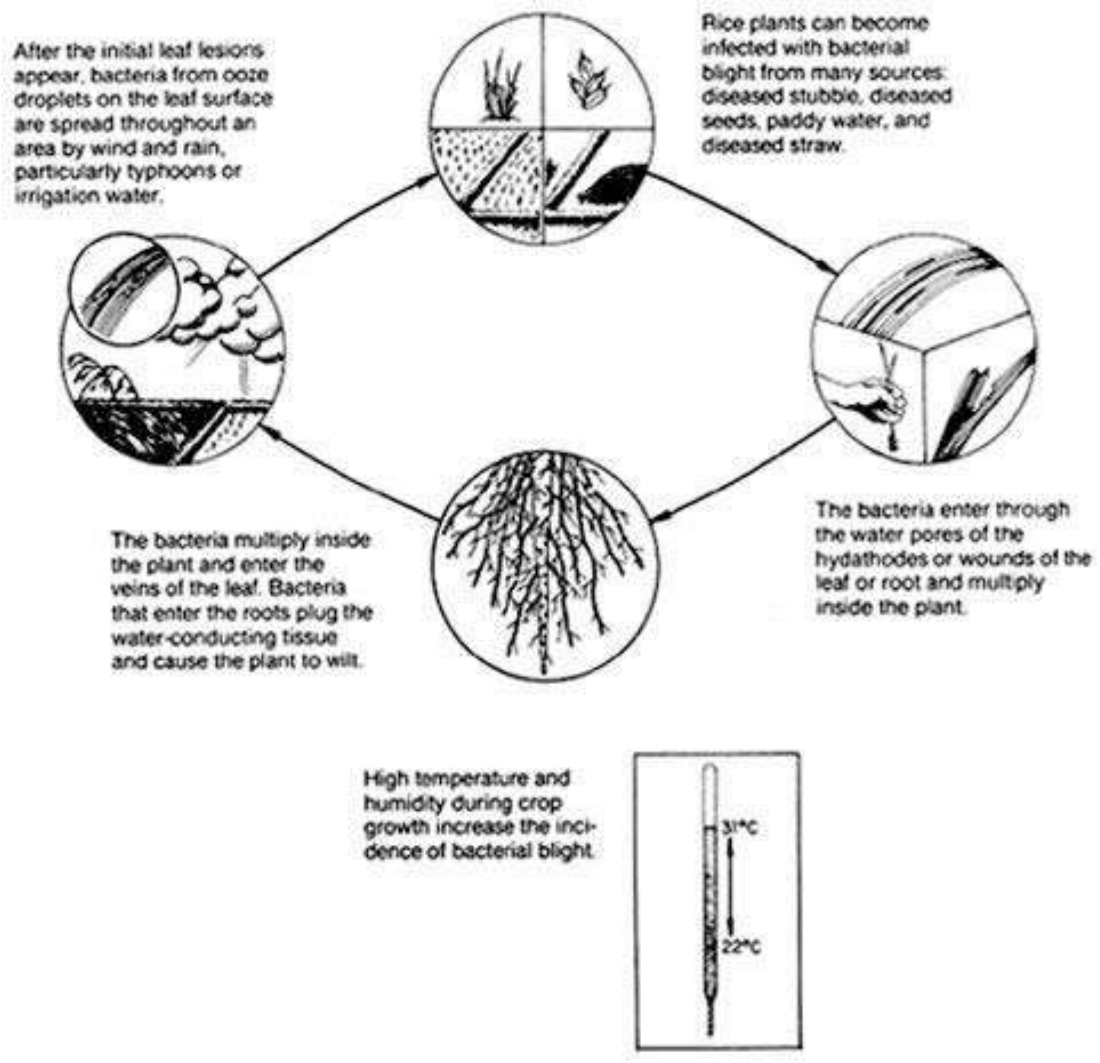
Favorable Conditions

- 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 najor*, *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.

Symptoms

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.

Symptoms

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*, *Recllia dorsalis* and *N. nigropictus* in a persistent manner. The transmission is transovarial through eggs. Gramineous weeds *Echinochloa crusgalli* and *Panicum miliaceaum* serve as source of inoculum.

Management

- Destory weed host that serve as source of inoculum
- Spray Phosphamidon or Fenthinon 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.

Symptoms

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.

Symptoms

Pathogen

- Caused by a phytoplasma (rice yellow dwarf phytoplasma 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

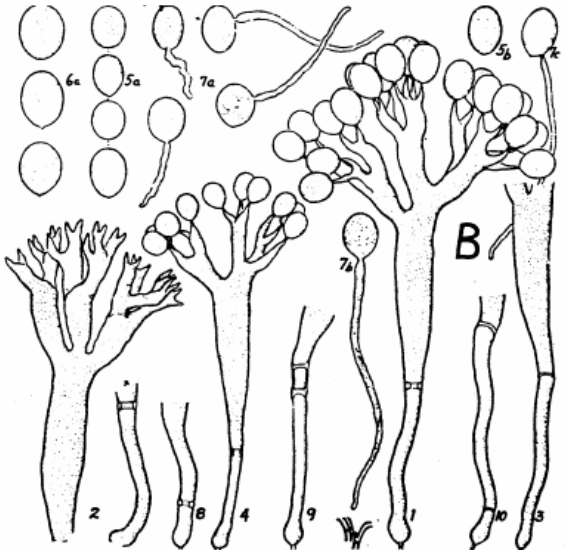
The fungus causes systemic downy mildew of sorghum. It invades the growing points of young plants, either through oospore or conidial infection. As the leaves unfold they exhibit green or yellow colouration. Abundant downy white growth is produced on the lower surface of the leaves, which consists of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

Symptoms

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. Sporangiophores 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.



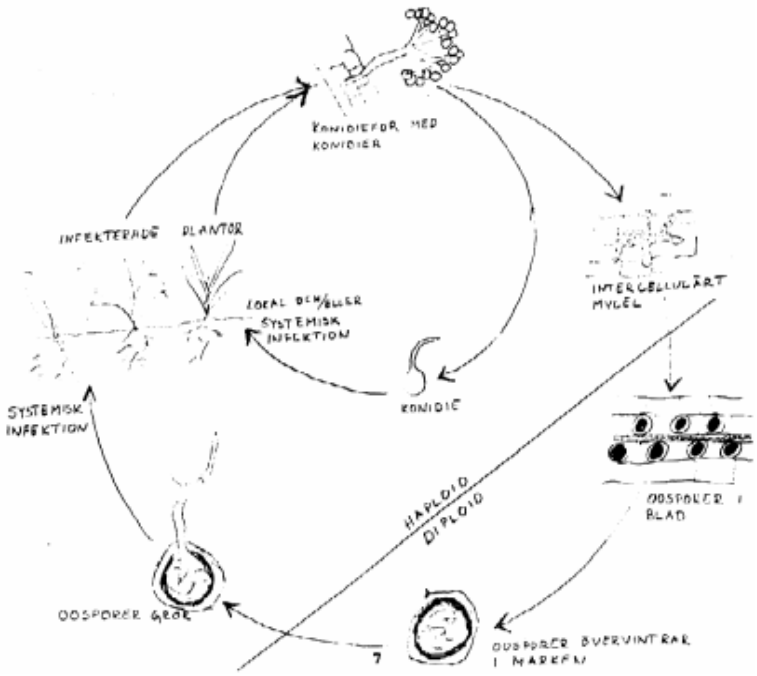
Sporangia and sporangiophores

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, *Heteropogen 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^{0}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.
- Grow moderately resistant varieties like Co25 and Co26.
- 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.

Symptoms

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.

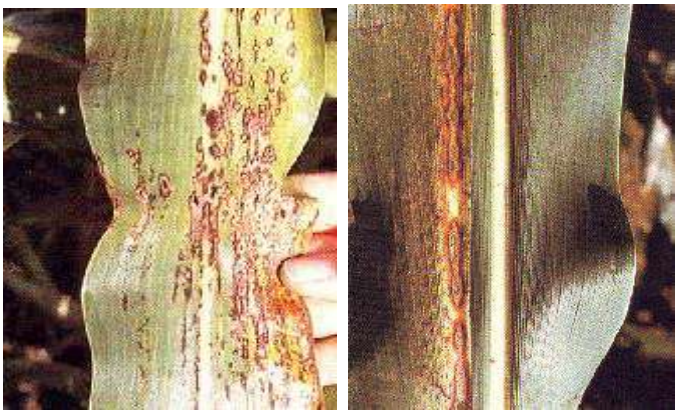
Management

- 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.



Symptoms

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

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 cyclindrical and are covered with a tough creamy skin (peridium) which often persists unbroken up to thrashing. Ratoon crops exhibit higher incidence of disease.

Symptoms

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.

Symptom

Long smut - *Tolyposporium ehrenbergii*

Symptoms

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

Symptoms

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.

Symptoms

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.

Symptom

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 occusr. 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

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.

Symptoms

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.

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.

Symptoms

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.


Symptoms

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*

Symptom

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.

Symptoms

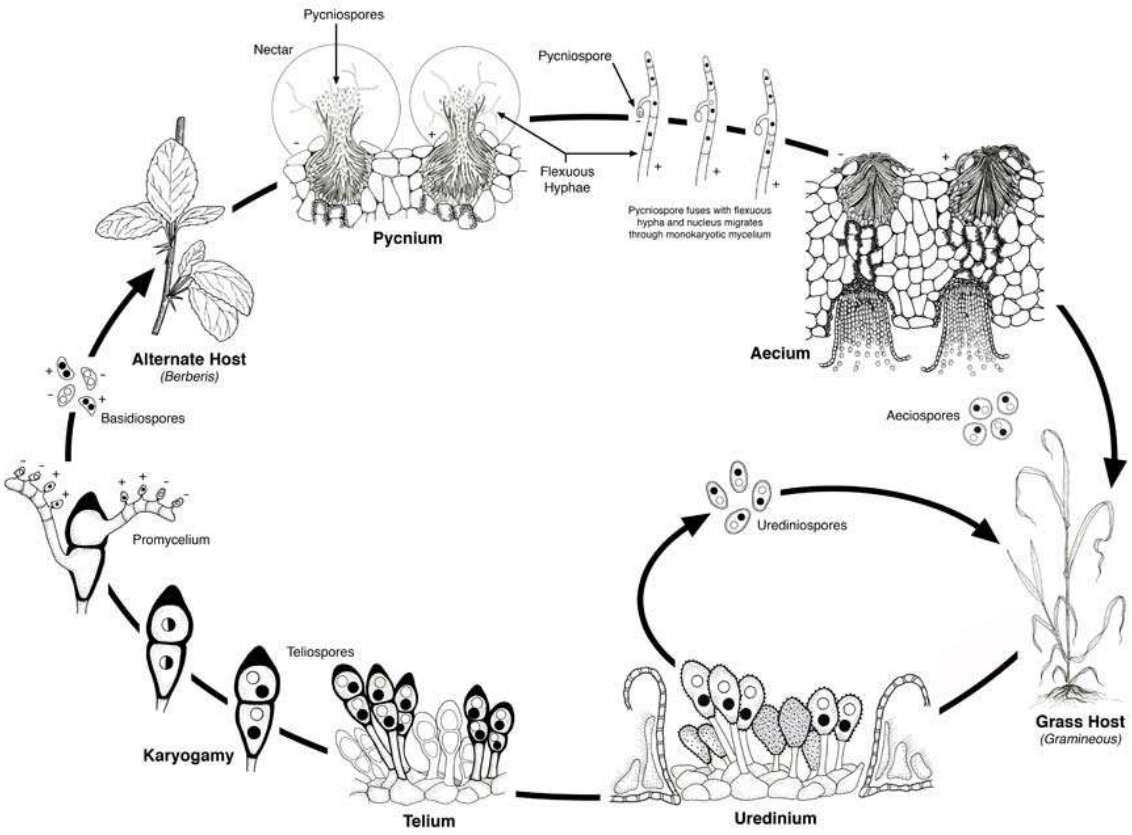
Pathogen

The uredospores of rust pathogen are almost round or oval in shape and bright orange in colour. The teliospores are bright organge 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^{o} 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.

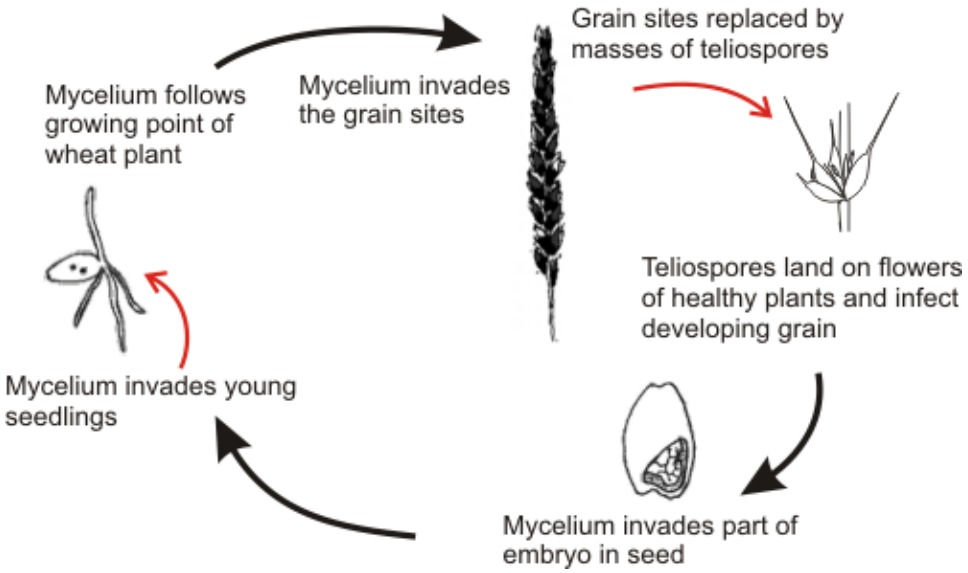
Symptoms

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*

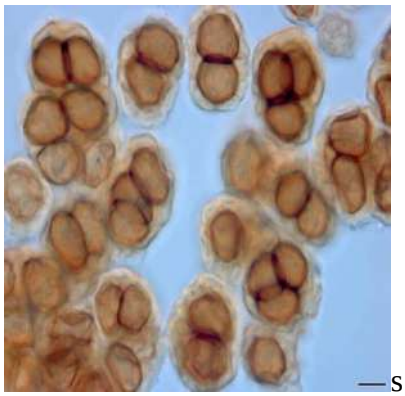
Symptoms

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.

Symptoms

Pathogen

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



Spore balls

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*

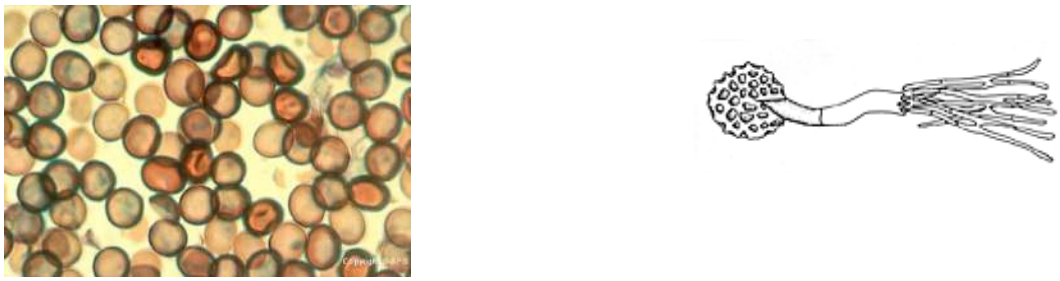
Symptoms

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.

Symptoms on earhead and grains

Pathogen

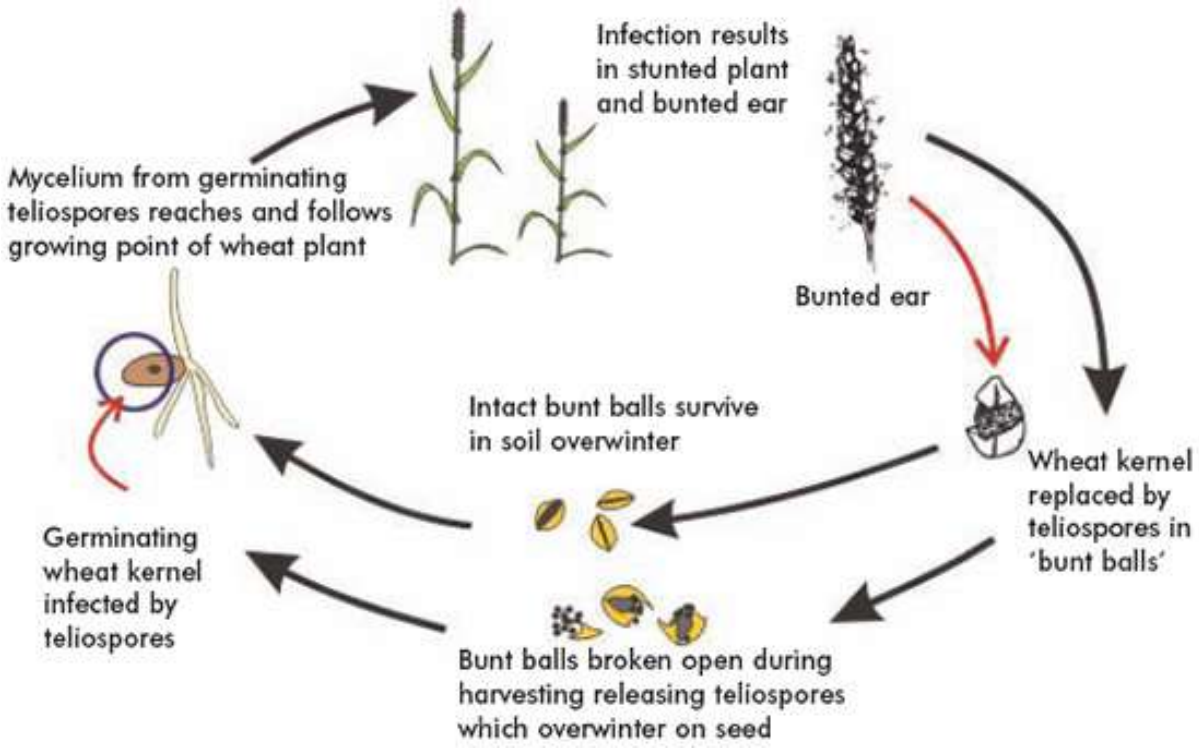
Reticulate, globose and rough walled. No resting period. Germinate to produce primary sporidia which unite to form 'H' shaped structure.



Spores and its germination

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

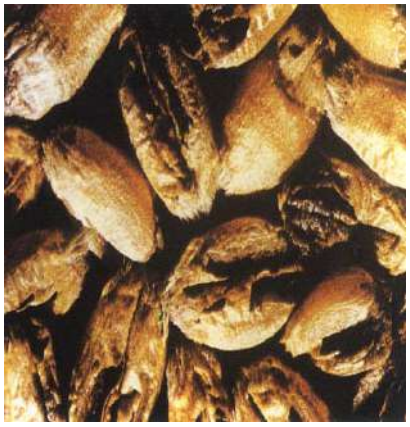
- Treat the seeds with carboxin or carbendazim at 2g/kg.
- Grow the crop during high temperature period.
- Adopt shallow sowing.
- Grow resistant varieties like Kalyan sona, S227, PV18, HD2021, HD4513 and HD4519.

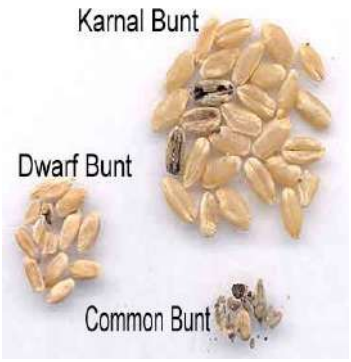
Karnal bunt - *Neovassia indica*

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.





Symptoms

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.

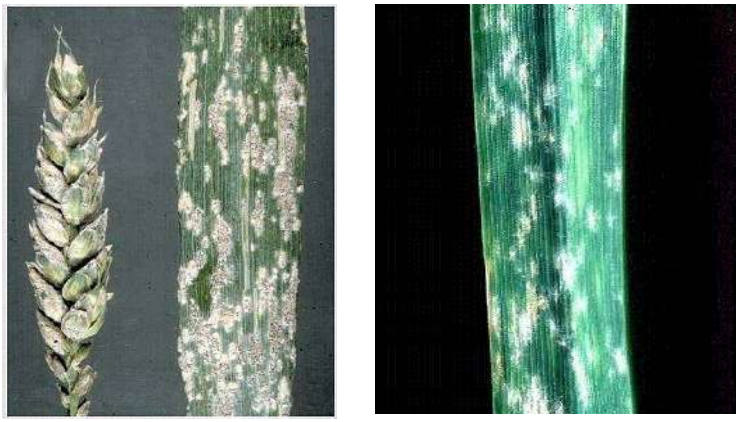
Management

- 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.

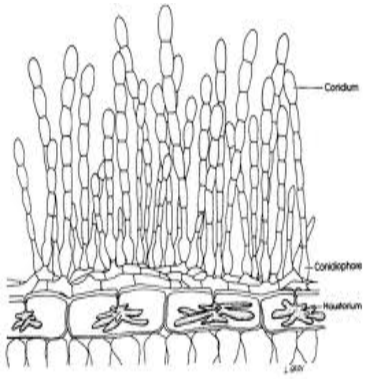


Symptoms

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.



Conidia and conidiophores

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.

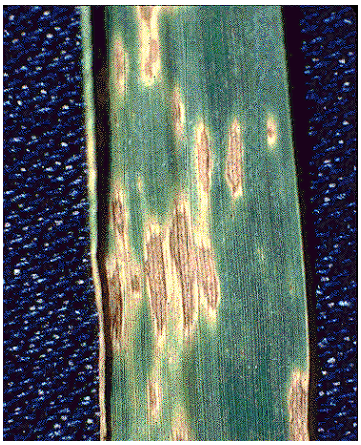
Management

- 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.*



Symptoms

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)

4. Diseases of Pearlmillet

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 sporangiophores 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.

Symptoms

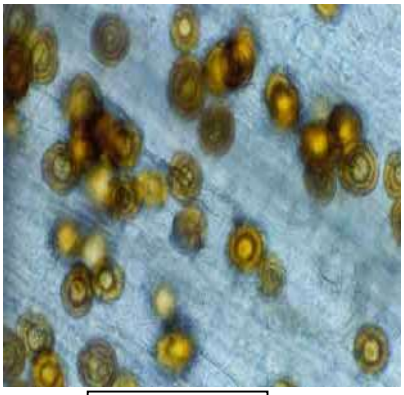
The inflorescence of infected plants gets completely or partially malformed with florets converted into leafy structures, giving the typical symptom of **green ear**.

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.

Green ear symptoms

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.



Oospores

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.

Symptoms

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.

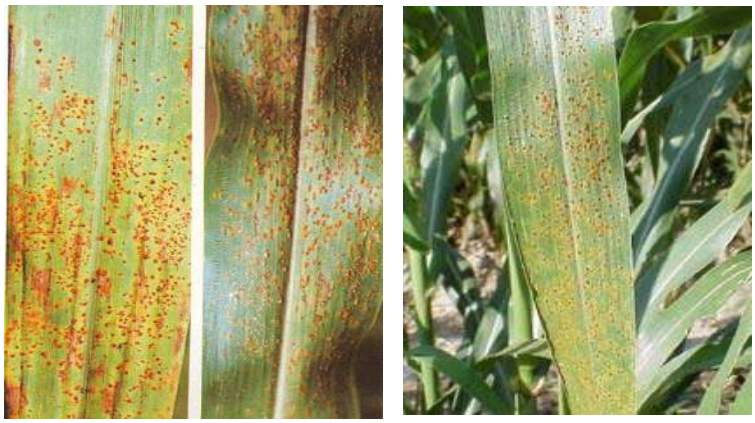
Management

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

Rust - *Puccinia pennisetti*

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.



Symptoms

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.

Symptoms

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*

Phlippine 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 auxillary buds on the stalk of tassel and the cobs is common **(Crazy top).**

Symptoms

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, COH1and COH2.

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

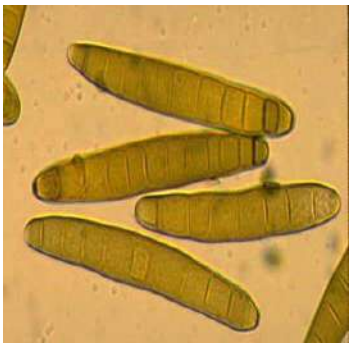
Symptoms

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 velvetty masses of conidia and conidiophores.

Symptoms

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.



Conidia

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.

Symptoms

Pathogen

Uredospores are globose or elliptical finely echinulate, yellowish brown with 4 germpores. 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.

Uredospores and teliospoes

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.

Symptoms

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

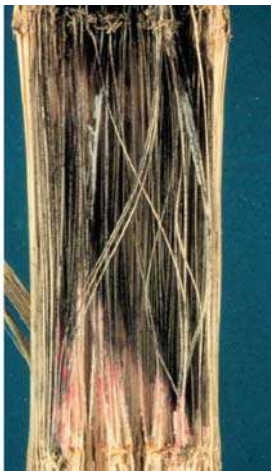
- Field sanitation.
- Crop rotation with pulses.
- Treat the seeds with Captan or Thiram at 4 g/kg.

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.



Symptoms

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, pearlmillet, fingermillet 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

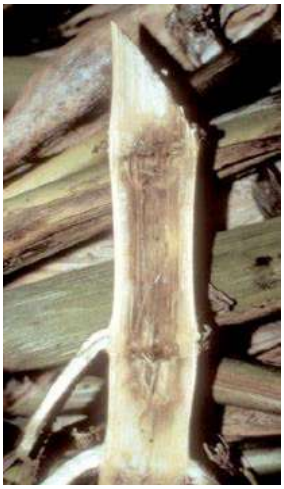
- Long crop rotation with crops that are not natural host of the fungus.
- Irrigate the crops at the time of earhead emergence to maturity.
- Treat the seeds with Carbendazim or Captan at 2 g/kg.
- Grow disease tolerant varieties viz., SN-65, SWS-8029, Diva and Zenit.

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



Symptoms

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

Symptoms

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.

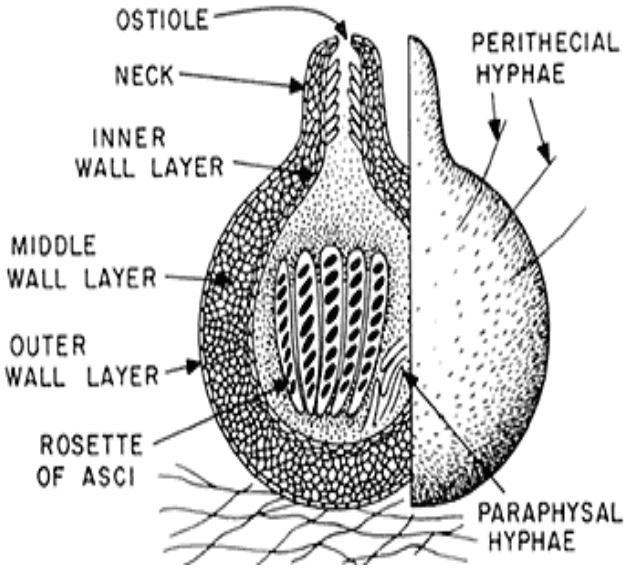
Symptoms

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

Pathogen

The fungus produces thin, hyaline, septate, profusely branched hyphae containing oil droplets. The fungus produces black, minute velvetty acervuli with long, rigid bristle-like, septate 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.
- Aviod 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.

Symptoms

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 destory 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.

Symptoms

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 perithecium 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 25-30^{o} C
- Prolonged rainfall after planting.

Disease Cycle

The fungus survives as conidia and chlamydospores in the soil and in the infected, burried 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.

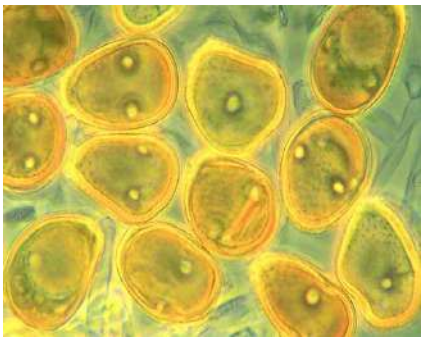
Rust - *Puccinia erianthi* *(*Syn: *P. melanocephala* and *P. kuehnii)*

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 severse 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.



Uredospores

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

Favourable Conditions

- Temperature of 30˚C.
- Rumidity 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 vasuclar 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, pearlmillet 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 pearlmillet near the sugarcane fields.

Red stripe - *Pseudomonas rubrilineans*

Symptoms

The disease first makes it 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 spreads 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.

Symptoms

Pathogen

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

Favourable Conditions

- Continuous ratooning and prolonged rainy weather with low temperature (25^0 C)

Disease cycle

The pathogen remains viable in the soil and infected plant residues. The bacterium also survives on sorghum, pearlmillet, maize, fingermillet 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.

Symptoms

Pathogen

Sugarcane mosaic potyvirus is a flexous 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.
- G row 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.

Symptoms

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 discolouration zone.

Leaf scald - ***Xanthomonas albilineans***

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.

Symptoms

Disease cycle

Pathogen is soil-borne, therefore primary inoculam 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 drenced 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

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.

Symptoms

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 Cpper 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).

Pre and post emergence

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 zoopores. The zoospores are kidney shaped and biflagellate. Oospores spherical, light to deep yellow or yellowish brown coloured, measuring 17-19µm in diameter.

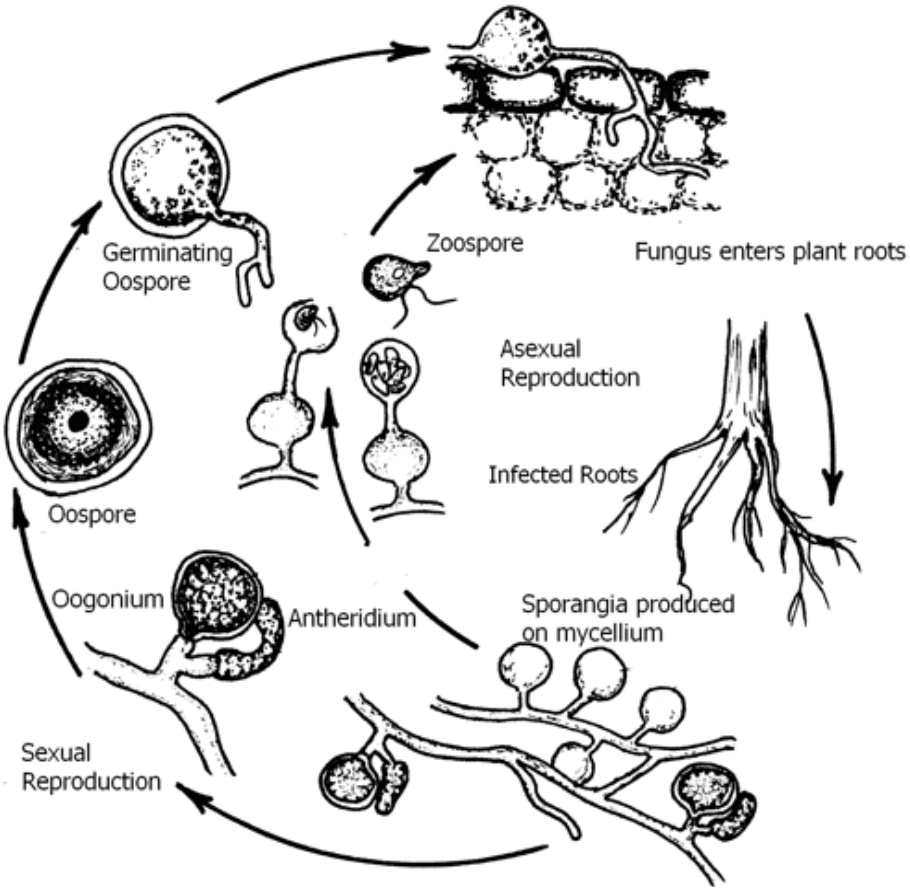
Favourable Conditions

- 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/2.5m^2).
- 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

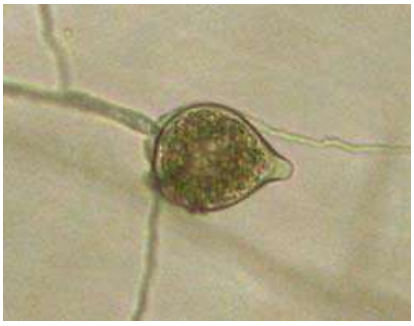
The pathogen may affect the crop at any stage of its growth. Even though all parts are affected, the disease infects chiefly the roots and base of the stem. Seedlings in the nursery show black discolor of the stem near the soil level and blackening of roots, leading the wet rot in humid condition and 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.

Symptoms

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 globoose and thick walled chlamydospores, measuring 27-42µm in diameter. Oospores are thick walled, globose, smooth and light yellow coloured, measuring 15-20µm in diameter.



Sporangia

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 Captafol or 0.2 per cent Copper oxychloride or 1 per cent Boreaux mixture and repeat after 10 days.

- Select healthy, disease free seedlings for transplanting.
- Remove and destroy the affected plants in the field.
- Spray Mancozeb 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.

Symptoms

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

Conidia and conidiophores

Favourable Conditions

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

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

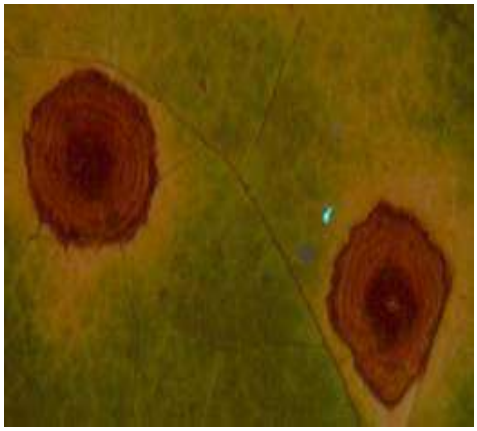
- Apply balanced ferilizers.
- Avoid overcrowding of plants.
- Remove and destroy the affected leaves.
- Plant early in the season so that crop escapes the cool temperature at maturity phase.
- Spray dinocap at 375 ml or Carbendazim at 500g/ha.

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.

Disaease 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)

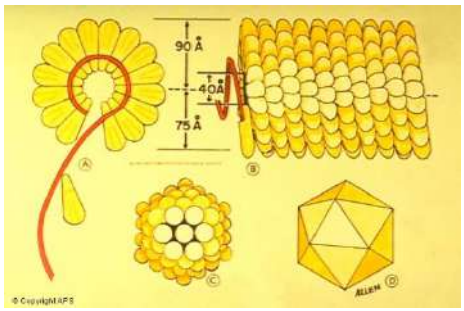
Symptoms

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.

Symptoms

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.

Pathogen ***C. arachidicola*** **(Sex** **Symptoms** *achidis*)

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 inclusters 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 phosporus 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 destory the infected plant debris.

- Eradicate the volunteer groundnut plants.
- Keep weeds under control.
- Treat the seeds with Carbendazim or Thiram at 2g/kg.
- Spray Carbendazim 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.

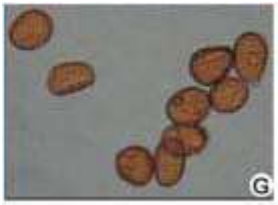
Symptoms

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 germpores. Teliospores are dark brown with two cells. Pycnial and aecial stages have not been recorded and there is no information available about the role of alternate host.

Teliospores



Uredospores

Favourable Conditions

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

Disease cycle

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

Management

- Avoid monoculturing of groundnut.
- Remove volunteer groundnut plants and reservoir hosts.
- Spray mancozeb 2 kg or Wettable Sulphur 3 kg or Tridemorph 500ml or Chlorothalonil 2 kg/ha.
- Grow moderatelyresistant 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.

Symptoms

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

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.

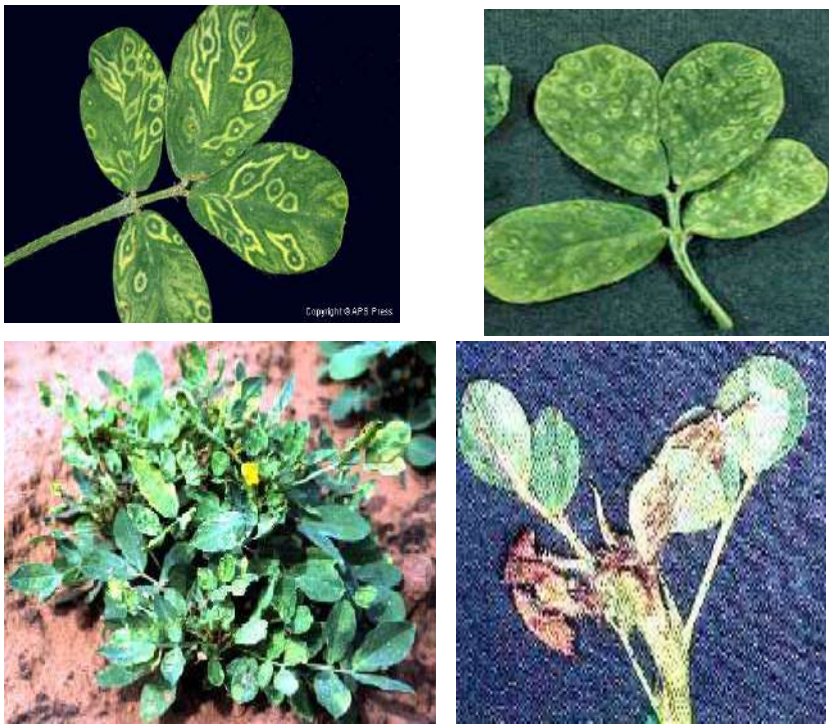
Management

- 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.



Symptoms

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 destory 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.

Symptoms

Management

- Cultural practices such as deep' covering or burial of organic matter before planting, non-dirting 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.

Symptoms

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.

Symptoms

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

Spot on older leaf

Leaf blight symptom

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.

Powdery mass covering entire leaf

Pathogen

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

Disease cycle

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

Management

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

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 un branched 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

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.

Affected plant showing drooping of leaves

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. *ricinicola*

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.

Symptoms

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)***

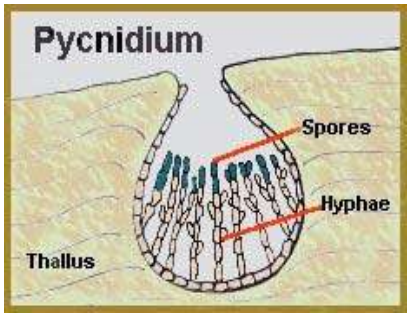
Symptoms

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.

Symptoms

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*

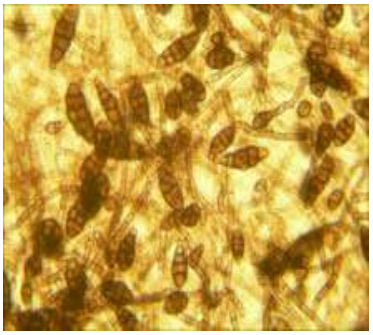
Symptoms

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.

Symptoms

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.

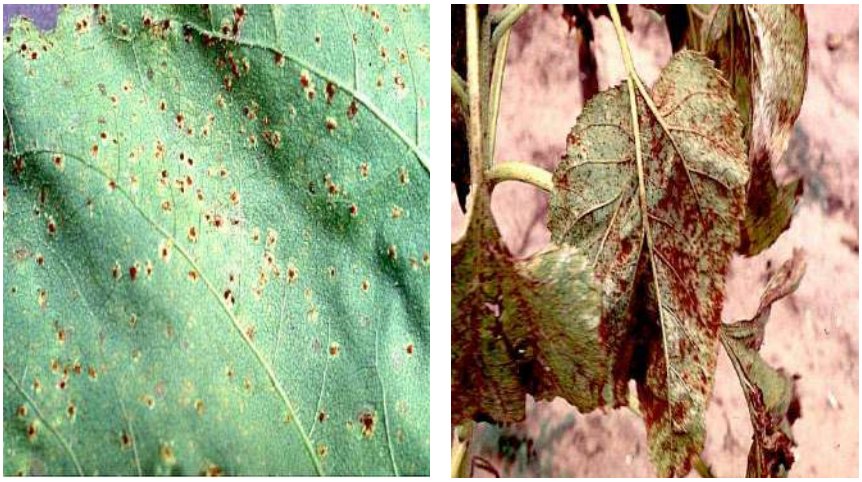
Management

- 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.



Symptoms

Pathogen

The uredospores are round or elliptical, dark cinnamon-brown in colour and minutely echinulated with 2 equatorial germpores. Teliospores are elliptical or oblong, two celled, smooth walled and cheshnut brown in colour with a long, colourless pedicel.

Uredospores and Teliospores

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.

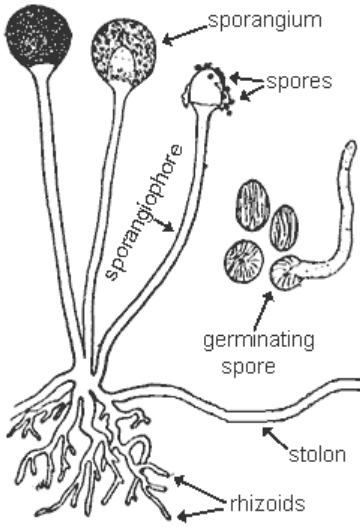
Symptoms

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

Pathogen

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



Structure of *Rhizopus*

Favourable Conditions

- Prolonged rainy weather at flowering.
- Ddamages 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 leavesWhite 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.

Symptoms

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.


Symptoms

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, tripartite genome encapzidated 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 veetor 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

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.

Symptoms

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.

Symptoms

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.

Symptoms

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 sesamum in the same field.
- Remove and destrosy 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.

Symptoms

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

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.

- Spray Streptomycin sulphate or oxytetracycline hydrochloride or strephocyclin at 100g/ha.

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 resticted by veins and veinlets. The colour of spot may be dark brown with shiny oozes of bacterial masses.

Symptoms

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

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 sesamum 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.

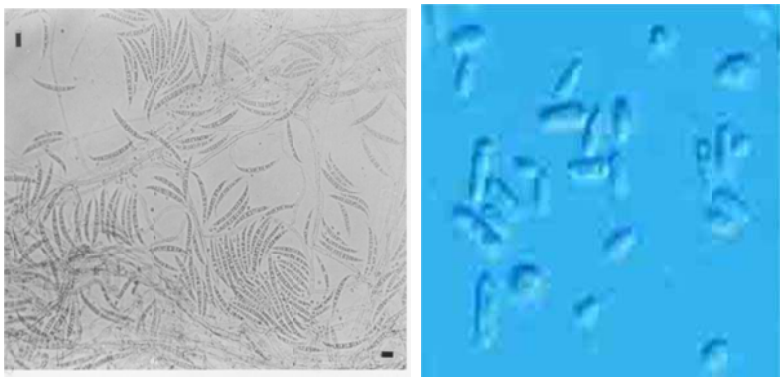
Symptoms start from the older leaves at the base, followed by younger ones towards the top, finally involving the branches and the whole plant. The defoliation or wilting may be complete leaving the stem alone standing in the field. Sometimes partial wilting occurs; where in only one portion of the plant is affected, the other remaining free. The taproot is usually stunted with less abundant laterals.

oms

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 tappering 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.



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

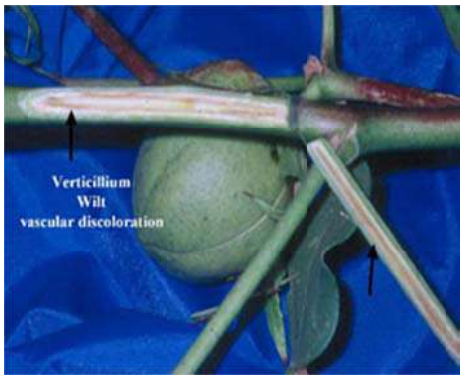
- Treat the acid delinted seeds with Carboxin or Carbendazim at 2 g/kg.
- Remove and burn the infected plant debris in the soil after deep summer ploughing during June-July.
- Apply increased doses of potash with a balanced dose of nitrogenous and phosphatic fertilizers.
- Apply heavy doses of farm yard 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 appearence. 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.



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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 condiophores. 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.

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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 shreds and even extends above ground level. In badly affected plants the woody portions may become black and brittle. A large number of dark brown sclerotia are seen on the wood or on the shredded bark.

Pathogen

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

oms

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., *Aristolachia 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 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.

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Pathogen

The pathogen produces endophytic, septate mycelium. Conidiophores are short, hyalineand 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 flattend 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.

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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*

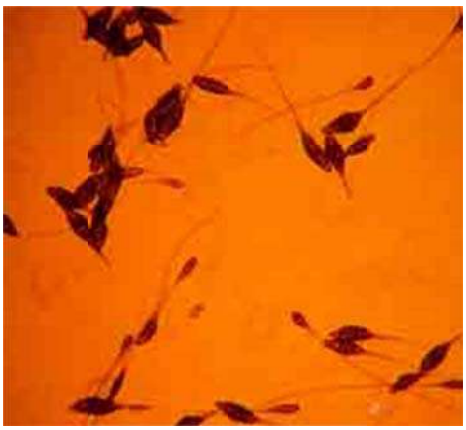
Symptoms

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.

ıptoms

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 ‘blighting’ appearance. On the lower surface of the leaf, bacterial oozes are formed as crusts or scales. The affected leaves become crinkled and twisted inward and show withering. The infection also spreads from veins to petiole and cause blighting leading to defoliation.

iv) Black arm:

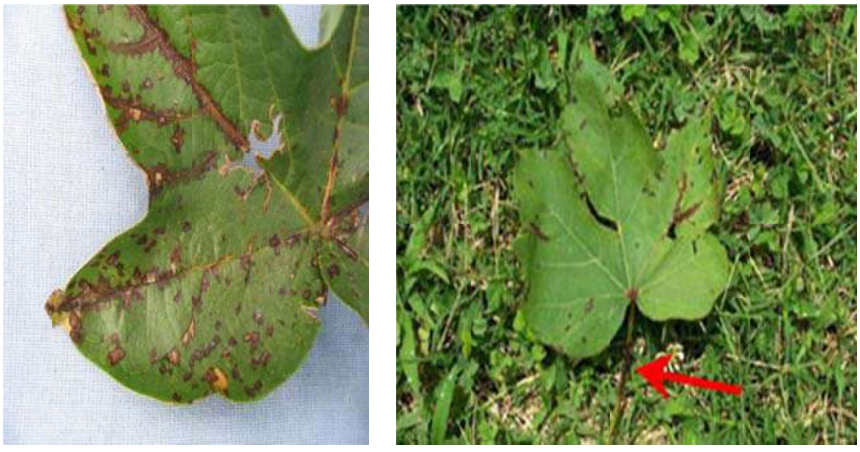
On the stem and fruiting branches, dark brown to black lesions are formed, which may girdle the stem and branches to cause premature drooping off of the leaves, cracking of stem and

gummosis, resulting in breaking of the stem 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

Boll rot

Pathogen

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

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 curcus*. 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 +Ttetracycline 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 appearence 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 poduce 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 redgram 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 (10^6cfu/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

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.

Symptoms

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 (10^6cfu/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.



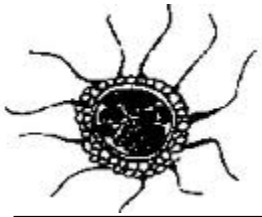
Symptoms

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.

Conidia and conidiophores



Cleistothecium

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.

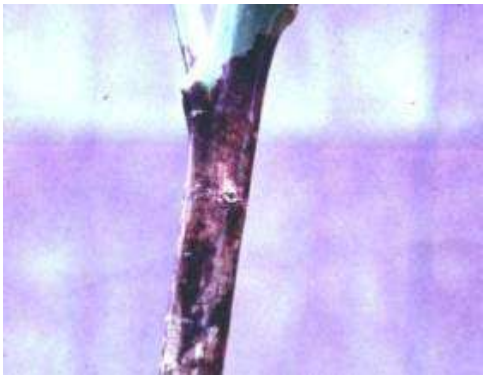
Management

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.



Symptoms

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.

Symptoms

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 eriophyid mite, *Aceria cajani* in a semi persistant 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 - *Colletrtrichum 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 appearon 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

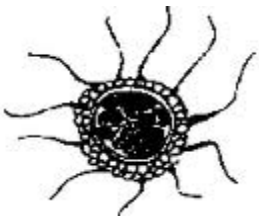
Small, irregular powdery spots appear on the upper surface of the leaves, sometimes on both the surfaces. The disease becomes severe during flowering and pod development stage. The white powdery spots completely cover the leaves, petioles, stem and even the pods. The plant assumes greyish white appearance;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

Symptoms

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.

Conidia and conidiophores



Cleistothecium

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.

Symptoms

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.

Symptoms

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

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 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.

Symptoms

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 (10^6cfu/g) or *Pseudonomas fluorescens* @ (10^6cfu/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.

Symptoms

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 part 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 sever 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. Shriveling 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* (Pycnidial stage: *Macrophomina phaseolina*)

Symptoms

The disease symptom starts initially with yellowing and drooping of the leaves. The leaves later fall off and the plant dies with in week. Dark brown lesions are seen on the stem at ground level and bark shows shredding symptom. The affected plants can be easily pulled out

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

Pathogen

The fungus produces dark brown, septate mycelium with constrictions at hyphal branches. Minute, dark, round sclerotia 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 *Pseudonomas 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 Imidacloprid 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 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.

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.

Symptoms

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 pycnidiopores (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.

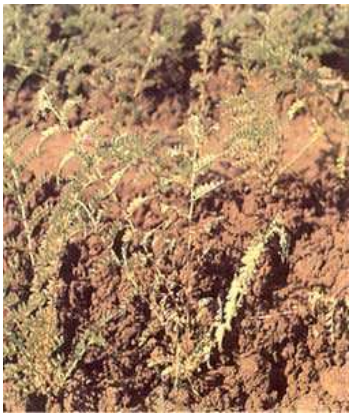
Management

- 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



Symptoms

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 5septate, 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 (10^6cfu/g) *Pseudonomas fluorescens* @ 10g/kg (10^6cfu/g) of seed.
- Apply heavy doses of organic manure or green manure.
- Grow resistant cultures like ICCC 42, H82-2, Avrodhi, Alok Samrat, Pusa-212, JG- 322, GPF-2, Haryanachana-1 and Kabuli chickpea like Pusa-1073 and Pusa-2024.

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.

Symptoms

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.

Symptoms

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 pluoghing 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 - *Operculella padwickii*

Rotting is evident from collar region onwards. Internal brown discolouration appears above the rotton 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 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.

Symptoms

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 *Pseudonomas 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.

Symptoms

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.

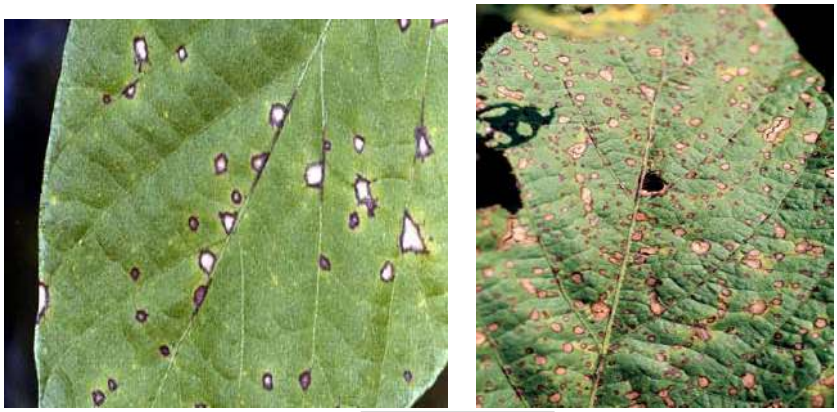
Management

- 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.



Symptoms

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.

Symptoms

Pathogen

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

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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^{o} 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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