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)
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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.

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**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. Ascospores 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 (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 budding 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 prominent 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/litre 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 prematurely 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. The other 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**

*Cephalciuros 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. They are formed singly on the vegetative filaments. When the sporangia are ripe, the contents 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 spor. 

**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.
Panama disease: *Fusarium oxysporum* f. *spubense*

**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. Chlamydosporas - 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 with Carbofuran granules. Corm injection of 3 ml of 2% Carbenduzim injected in the corm by making a hole to a depth of 10 cm with 45° angle on 5th and 7th 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 elliptical 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 hyaline, 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 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 olivaceous 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. \textit{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 – \textit{Oidium caricae}**

**Symptoms**

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

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

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**Mode of spread**

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

**Management**

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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 conidiophore 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.

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

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

*Powdery mildew – Podosphaera leucotricha.*
Symptom

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**


**Soft rot – Penicillium expansum**

**Symptom**

Young spots start 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.
Damping off: *Pythium aphanidermatum*

**Symptoms:**

Seedlings killed before emergence. Water soaking and shrivelling of stem. Factors favouring infection: Moist soils poor drainage 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. Partially 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 and 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 orthophosphate 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 with 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.

<table>
<thead>
<tr>
<th>Insecticide</th>
<th>Concentration</th>
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</thead>
<tbody>
<tr>
<td>Methyldemeton 25 EC</td>
<td>2 ml / litre</td>
</tr>
<tr>
<td>Dimethoate 30 EC</td>
<td>2 ml / litre</td>
</tr>
<tr>
<td>Malathion 50 EC</td>
<td>2 ml / litre</td>
</tr>
</tbody>
</table>

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 seedlings 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 cerein @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 rotation 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, sometimes 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: Erysiphe cichoracearum**

**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**

Spray 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.
Late blight of potato: *Phytophthora 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 airborne 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 30-35°C. 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 sclerititia 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 incidence. 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

![Image of infected potato tuber]

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 organism 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.
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 rotation 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**

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 35°C, 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**

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 at least 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 at least 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 oxysporum* 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 melons 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 pathogen 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**

![Image of Downy Mildew Symptoms](image_url)

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 sporangioshores 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**

![Image of powdery mildew symptoms](image)

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 schachti*

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 of 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 particles 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, while 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 chlamydosporces. 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. Ascocaeips 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 broadly 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 chlamydomspores. 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 m² 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/10m² 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**

Spray 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**

![Image of carrot with symptoms](Erwinia Carotovora)

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 on 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:** *Stemphyllum vesicarium*

**Symptoms**

![](image)

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 than 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**

![](image)
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.
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 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 veinlet 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 up to 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**

![Bacterial Wilt Symptoms](image)

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, grollut 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**

![Mosaic symptom](image)

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.
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 alive in 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 appendiculatetis***

**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 appendiculatetis*. 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 humidity 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 chlamydomospores 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 permanent 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. Sclerotia 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 enough 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.

**Management**

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-spires. 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. Microcondidia (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 survives 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 *Phytoplasma* which is frequently identified in the phloem tissues of infected trees.

**Favourable Conditions**

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 in 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 P2O5 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 severer 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**

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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 epidermis a 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 fungicide in third week of April.

**Red rust:** *Cephalurus 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**

*Cephalurus 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 it 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 - Diplocarpon 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
**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 – Phragmidium 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 shaped 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 - *Septoria chrysanthemella***

**Symptoms**

![Image of Septoria Leaf Spot]

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**

![Image of Powdery Mildew]
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 Fosetyl-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%.
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