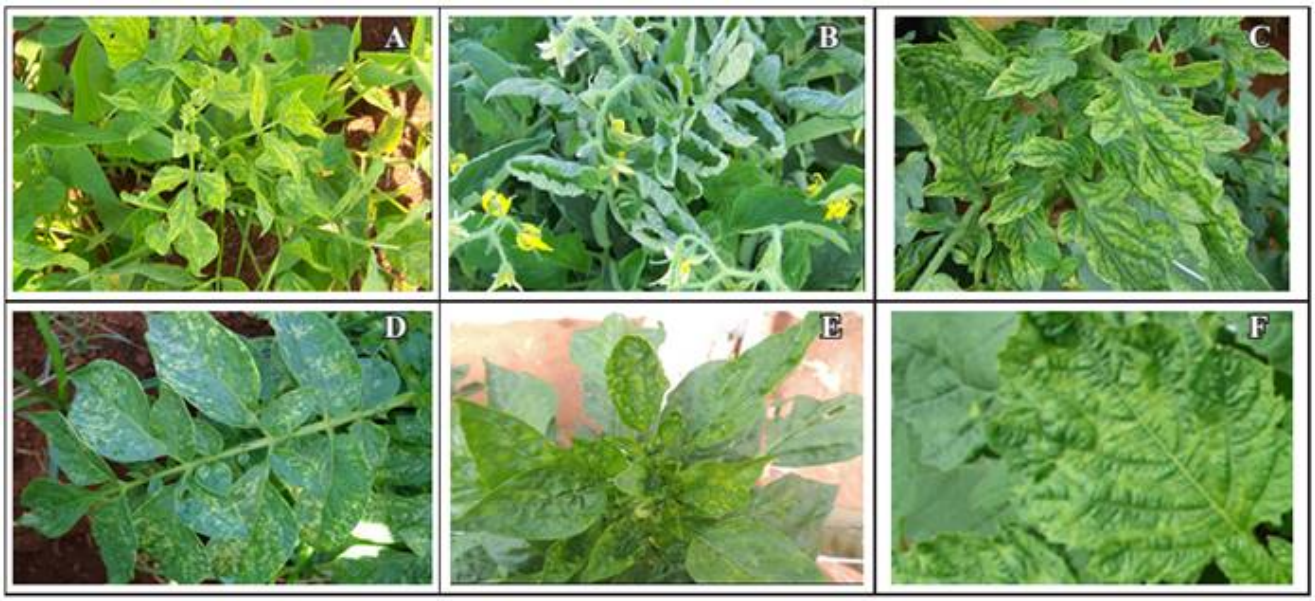
Diseases of Vegetable, Ornamental and Spice Crops (HPI 202) 3 (2+1)



ICAR E-Courses

4. Diseases of Vegetable, Ornamental and Spice Crops (HPI 202) 3 (2+1)

Etiology, symptoms, mode of spread, epidemiology and integrated management of diseases of the following vegetables, ornamental and spice crops: tomato, brinjal, chilli, bhindi, cabbage, cauliflower, radish, knol-khol, pea, beans, beet root, onion, garlic, fenugreek, ginger, potato, turmeric, pepper, cumin, cardamom, nutmeg, coriander, clove, cinnamon, jasmine, rose, crossandra, tuberose, geranium. Important post-harvest diseases of vegetables and ornamental crops and their management.

***Practical**: Observations of symptoms, causal organisms and host parasitic relationship of important diseases, examination of cultures of important pathogens of vegetables, ornamental and spice crops.*

Lecture-1

NURSERY DISEASES OF TOMATO AND OTHER VEGETABLES

Tomato (*Solanum lycopersicon* L.) is an important vegetable crop grown globally and consumed as salad, ketchups, sauce, soup, pickles etc. It is also cooked along with other vegetables to provide taste and is the basic need of the Indian kitchen. Ripened fruits are good source of ascorbic acid and minerals. The crop is generally grown during winter months (Oct.- April) in the plains of India while in the hills it is mostly grown during summer and rainy season, thus fresh fruits are available round the year in the market. Prevalence of high humidity and warm temperature not only favours the luxuriant growth of the crop but also favours the development of various fungal, bacterial and viral diseases. Under favourable environmental conditions, epiphytotics of certain diseases have often reduced the seedling stand considerably in certain years. Some of the important fungal, bacterial and viral disease problems of nurseries are described in this lecture.

Aim: To Know the different diseases occurring in nursery beds and infecting tomato and other vegetables and their management

A) FUNGAL DISEASES

1. Damping Off

- It is an important disease specially in tomato and also other vegetable crops like bell pepper, chillies, brinjal, cabbage, cauliflower, broccoli etc. for which nursery is raised for transplanting.
- The disease is fairly common in poorly managed nursery beds, incited often by the seed and soil borne pathogens.
- It is responsible not only for the poor seed germination and stand of seedlings but also for carry over of the pathogens to the field where transplanting is done.

Symptoms

The disease manifests in two phases

i) pre-emergence damping-off and ii) post-emergence damping-off.

i) ***Pre-emergence damping-off*:**

- Failure of seedling emergence from the soil either due to seed rots or killing of young seedlings before their emergence from the soil.
- Resulting in patchy appearance of seedlings stands in the nursery in early stages (Plate-1a).

Plate-1a Symptoms of Pre-emergence damping-off showing patchy appearance

ii) ***Post emergence damping-off*****:**

- This phase is characterized by toppling over of infected seedlings at any time after their emergence from the soil.
- The infected tissue initially appears to be water- soaked and soft.
- Subsequently, the stem at the infection points get constricted resulting in toppling over and mortality of the seedlings (Plate-1b).

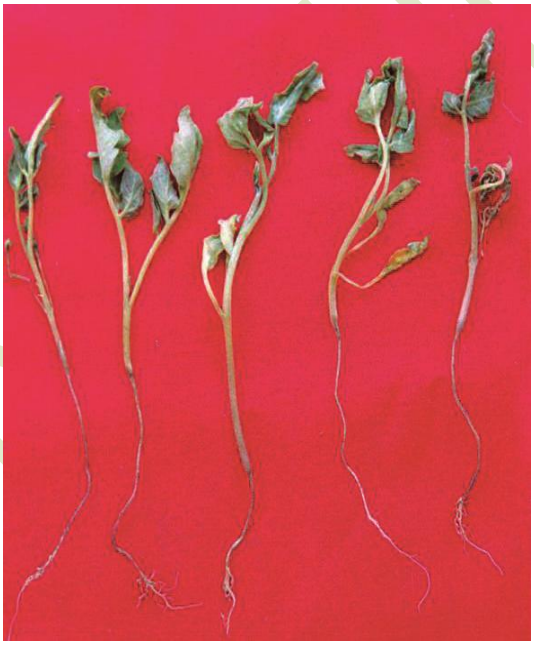


Plate 1b. Seedling showing symptoms of post-emergence damping-off

Pathogen (s):

- Species of *Pythium, Phytophthora, Fusarium* and *Rhizoctonia solani* Kuhn are the most commonly associated pathogens.

- Among these fungi, the species of *Pythium* viz., *P. aphanidermatum* (Edson) Fitzp. and *P. butleri* Subram. are more common than others.
- The mycelium of *Pythium* is intracellular, consists of slender, coenocytic and much branched hyphae.
- Asexual reproduction is mostly by means of zoospores, that which are differentiated in a vesicle, kidney shaped and biflagellate, which later become encysted and germinate by forming germtube.
- In sexual reproduction, one male nucleus from antheridium passes into the oogonium through conjugation tube which then forms a thick wall spores known as oospore.

Disease cycle and epidemiology

- Most of the fungi responsible for this disease are both seed and soil borne (Fig.1.).
- The species of *Pythium* survives from one season to the other as mycelium in plant debris or oospores in the soil.

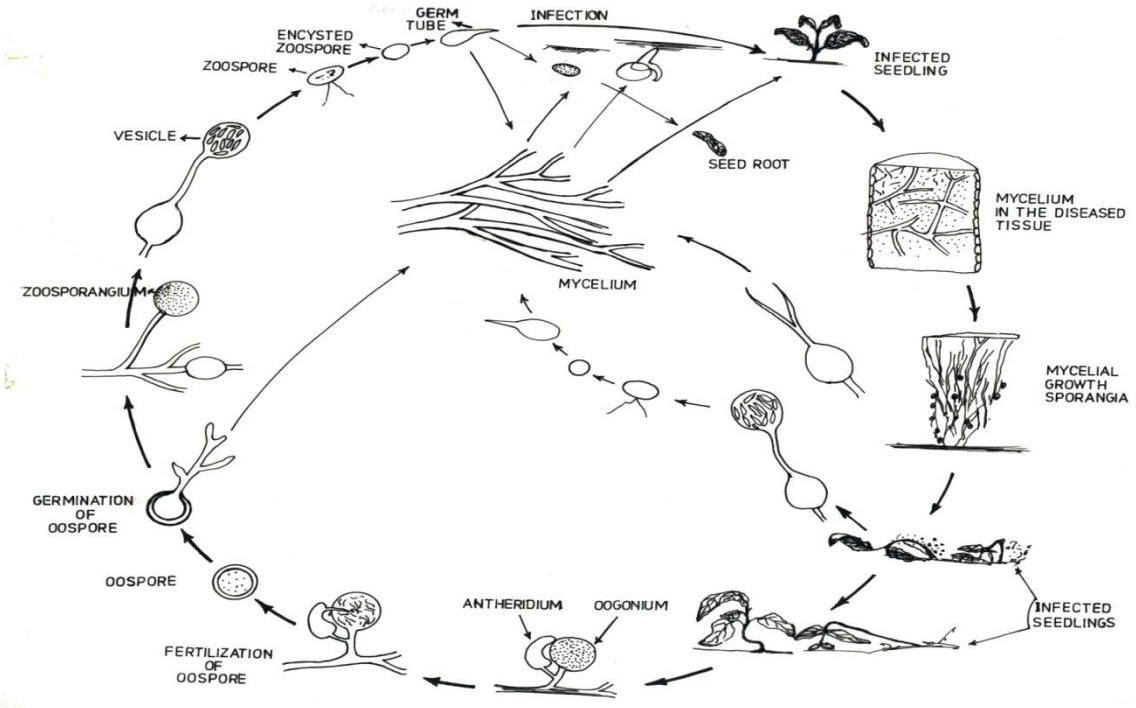


Fig. 1. Disease cycle of damping-off of seedlings (*Pythium aphanidermatum*)

- *Rhizoctonia solani* survives in soil indefinitely in the form of sclerotia. Hyphae grow from these sclerotia and infect seedlings in the nursery beds. Species of *Pythium* attack the plants when they are in juvenile stage.
- Soil moisture near saturation stimulates mycelial growth as well as asexual reproduction.
- High soil moisture, pH 6.0 and heavy soils favour disease development.

- Pre-emergence damping-off is maximum at 20-25° C while post emergence at 30-40°C.
- The disease is further aggravated in ill-aerated soils with poor drainage having thick stand of the seedlings.

Management

- Change the nursery site every year.
- Either solarize the soil of the bed with transparent polyethylene (25 µm) sheet for 40-45 days during summer months or treat the soil with Formalin (5%) at least 20 days before sowing or apply bioagents like *Trichoderma harzianum* or *T. viride* (40 g/m^2).
- Treat the seed with captan (0.3%).
- After seedling emergence from the soil, drench the bed with the mixture of mancozeb (0.25%) and carbendazim (0.1%) and repeat at 7-10 days interval.
- Give light but frequent irrigations.

B) BACTERIAL DISEASES

- Several bacterial pathogens infest the foliage of nursery plants after their emergence from the soil surface.
- Sometimes bacterial spot (*Xanthomonas campestris* pv. *Vesicatoria* (ex. Doidge) Vauterin *et al.* and canker (*Clavibacter michiganensis* pv. *michiganensis* (Smith) Davis *et al.*) in tomato and bell pepper and chili etc. and black rot (*Xanthomonas campestris* pv. *campestris* (Pammel) Dowson) in crucifers appear in nurseries and multiply rapidly.

Pathogen characters, disease cycle and epidemiology has been described in lecture No. 3 and 9.

Management

- Use of healthy seed.
- Dip the seed in Streptocycline (100 ppm) solution for 30 minutes.
- With the initiation of the disease spray the nursery beds with Streptocycline (100 ppm).

C) VIRAL DISEASES

- Several diseases like leaf curl and mosaics are of viral nature appear in the nurseries of some vegetables and carried from nursery to the fields.
- These viruses are transmitted through their insect vectors like aphids and white fly.
- To protect the nursery from these diseases, it is better to raise the nursery under a net house (40 mesh size) so that the nursery is not exposed to the viruliferous vectors.
- Insecticides Metasystox or Rogor (0.1%) should be applied at regular intervals to keep the vectors under check

- Some insecticide dust like Malathion dust should be applied around the borders of the bed to restrict the entry of ants and other soil borne insects in the nursery.

Raising virus free vegetable seedlings:

- Leaf curl is a white fly transmitted viral disease, infestation starts from seedling stage and continue till harvest of the crop.
- The disease is specially seen in the tomato and sometime in chilli too and causes great loss of the crop.
- The leaves of affected plants show curling, mottling, rolling puckering etc. It can be controlled by the following ways:

Management

- Treat the soil of the nursery by carbofuran 3-5 g/sqm^2.
- Seed treatment with Imidachloprid @ 2.5 g/kg seed.
- Cover the seed bed after seed sowing by Agronet by making a tunnel like structure.
- Spray the nursery beds 15 days after seed germination at 7 days interval with Metasytox @ 1.5 ml/litre of water. Last spray is done 2 days before transplanting.
- Remove the infected plants if any in the field and buried in with soil or burn.

D) **NEMATODE DISEASE(S)**

- Invariably nurseries of tomato, brinjal and cucurbits raised in untreated soils are infested with nematodes like *Meloidogyne* spp. (Plate-2)
- Infested seedling if transplanted in field carry the nematodes to the fields where the problem does not exist earlier which may result in severe crop yield losses in later stages.
- Either treat nurseries with Formalin (5%) at least 20 days before sowing or with Carbofuran (5 g/m^2) at the time of nursery bed preparation.

Plate-2.Symptoms of root knot on tomato seedlings

Lecture-2

FUNGAL DISEASES OF TOMATO

Tomato (*Solanum lycopersicon* L.) is an important vegetable crop grown globally and consumed as salad, ketchups, sauce, soup, pickles etc. It is also cooked alongwith other vegetables to provide taste and is the basic need of the Indian kitchen. Ripened fruits are good source of ascorbic acid and minerals. The crop is generally grown during winter months (Oct.- April) in the plains of India while in the hills it is mostly grown during summer and rainy season, thus fresh fruits are available round the year in the market. Prevalence of high humidity and warm temperature not only favours the luxuriant growth of the crop but also favours the development of various fungal, bacterial and viral diseases. Under favourable environmental conditions, epiphytotics of certain diseases have often reduced the yield considerably in certain years. Some of the important fungal, bacterial and viral disease problems are described in this lecture.

Aim: To know about different fungal diseases of Tomato and their management.

1. Late blight

Symptoms

- On leaves, disease appears as pale, green, irregular spots on the tips and margins, which in moist weather enlarge rapidly with central tissue turning necrotic and dark brown or black.
- On the lower surface of the leaves, a white downy growth of the fungus appears around the dead areas, which is more prevalent in morning hours when climate is moist (Plate-1a).
- Brown streaks also develop along the stems.
- On tomato fruits, dark, olivaceous greasy spots are formed, which gradually cover the entire fruit surface (Plate-1b).
- Finally rotting of leaves and fruit takes place.

Plate-1a On leaves **Plate-1b. On fruits**

Plate-1. Symptoms of late blight of tomato

Pathogen:

- ✓ The disease is caused by *Phytophthora infestans* (Mont.) de Bary.
- ✓ The pathogen, disease cycle and epidemiology is described in detail under potato diseases.
- ✓ In India, *P. infestans* infecting tomato, 3 races namely 1.3.4.7.11, 3.11 and 4.7 have been identified on the basis of differentials reaction tested on R_0 to R_{11} on potato differentials.
- ✓ Race 1.3.4.7.11 has been observed to be occurring both on potato and tomato.

Management

- ➢ Follow crop rotation and avoid solanaceous crops like potato in rotation.
- ➢ Improve drainage of the field and keep field free from weeds.
- ➢ Collect and destroy the infected fruits regularly.
- ➢ Before the initiation of the disease, apply one protective spray of mancozeb (0.25%) followed by metalaxyl + mancozeb (0.25%) at critical stage of disease appearance and repeat the sprays at 7-10 days interval.

2. Buckeye rot

Symptoms:

- ➢ Immature fruits (green colour) irrespective of their development stages are susceptible.
- ➢ Water soaked light brown discoloured spots appear which increase readily showing concentric dark brown rings slightly resembling the markings as a buckeye (Plate-2a).

- The lesions rapidly enlarge and with in 3-4 days, whole of the fruit surface turns dark brown and feels soft to touch.
- In warm and humid weather, white flocculent superficial growth of the fungus consisting of sporangia and sporangiophpores also develops on the diseased fruits.
- Later, these fruits may drop off from the plant (Plate-2b).

Plate 2a. Infected fruits **Plate-2b. Infected fruits fallen on the ground**

Plate-2. Symptoms of Buckeye rot of tomato

Pathogen:

- The disease is caused by *Phytophthora nicotianae* Breda de Hann. var. *parasitica* (Dastur) Waterhouse.
- The mycelium of the pathogen is hyaline and coenocytic with branching typically at right angles. The sporangiophores arise from hyphal threads and produce sporangia.
- The sporangia are broadly ovoid to globose in shape having one hemispherical papilla at the tip. Chlamydospores are smooth, globose, and slightly yellowish with thick brown walls, produced abundantly in culture and germinate by producing zoospores or germ tubes.
- Antheridia are amphigynous, spherical or oval and oogonia are rough, thick walled and yellowish brown in colour.
- Oospores are aplerotic, 18 to 20 μm in diameter with 2 μm thick wall.

Disease cycle and epidemiology

- ✓ The fungus overwinters in the soil in the form of oospores or chlamydospores and can remain active in soil for at least one year without the support of a susceptible host (Fig.1.).
- ✓ With the onset of monsoon rains, in the presence of high soil moisture and moderate temperatures (20-25°C), the chlamydospores and oospores start germinating by producing mycelium and sporangia.
- ✓ The sporangia in turn produce biflagellate zoospores, which are splashed by rain to the fruits.
- ✓ The symptoms develop on fruits after 3-4 day of infection.
- ✓ Infected fruit become mummified and fall down on the ground.
- ✓ The sporangia produced on infected fruits, liberate zoospores which are again splashed by rain and cause secondary infection.

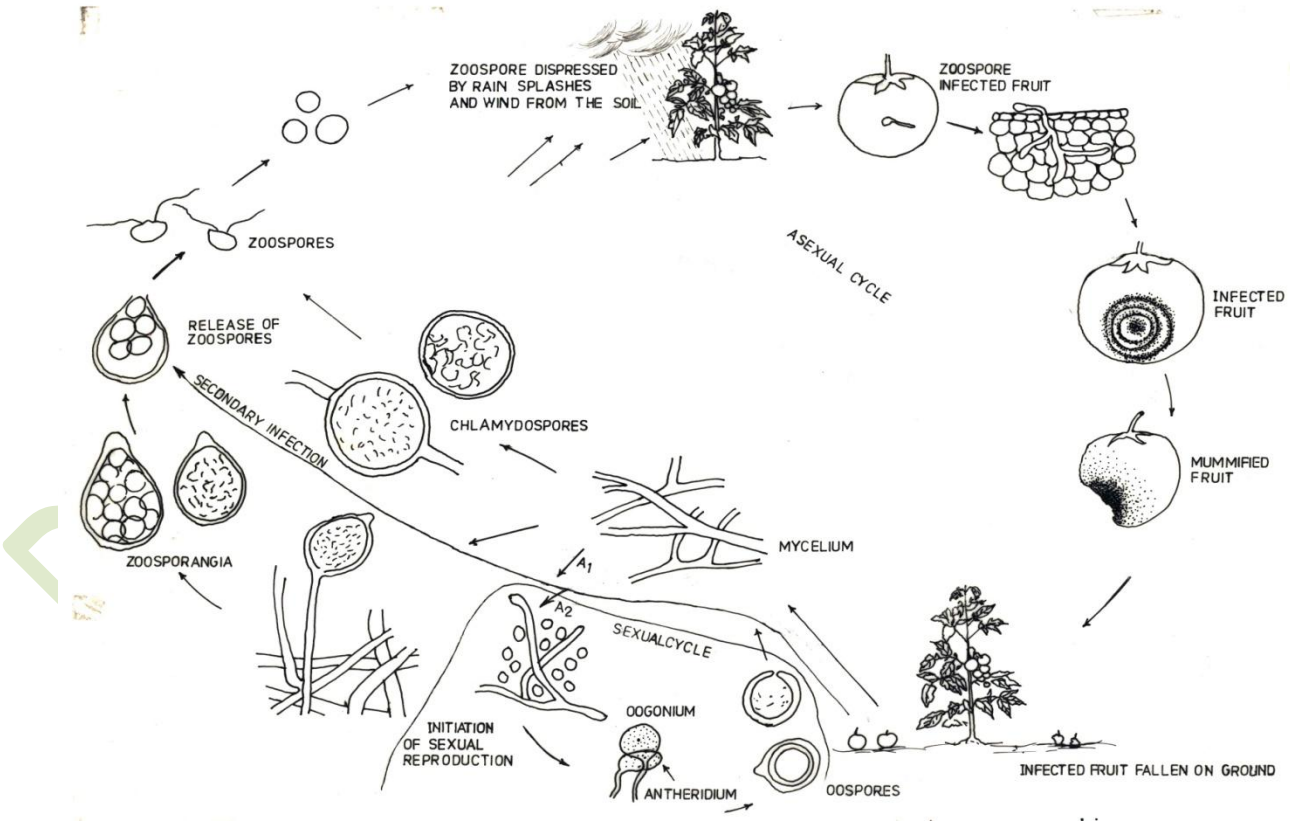


Fig. 1. Disease cycle of buckeye rot (*Phytophthora nicotianae* var. *parasitica*) of tomato

- ✓ Maximum fruit infection under field conditions occurs at a temperature range of 20-25°C, RH > 80 per cent and high rainfall conditions.
- ✓ Higher doses of N resulted in higher fruit rot while higher levels of P resulted in more yield of healthy fruits and less fruit rot.

- Based on weekly temperature and cumulative rainfall, short term forecasting of the disease can be done. June 20 is considered as the zero date.
- The disease is not expected to occur at temperatures at or below 20°C, though at temperatures of 22.5°C or above even a slight rainfall (10 mm) will result in disease appearance, which is expected to appear after 4 days of infection.

Management

- Stake the plants erect and remove foliage and fruit up to a height of 15-20 cm to avoid moist and stagnant air conditions.
- Collect and destroy the affected fruits regularly.
- Apply pine needle/grass mulch on the field floor to create a barrier between the host and soil borne inoculum.
- With the onset of monsoon rains, spray the crop with metalaxyl + mancozeb (0.25%) followed by sprays of either mancozeb (0.25%) or copper oxychloride (0.3%) or Bordeaux mixture (4:4:50) and repeat at 7-10 days interval.

3. ALTERNARIA LEAF SPOTS

The disease is caused by three different species of *Alternaria.*

Symptoms

***Alternaria solani*:**

- Dark brown spots with concentric rings develop on the leaves, which give target board effect, the most characteristic symptom of the disease (Plate-3a).
- In humid weather, the affected areas coalesce and form dark brown patches. In severe attacks, affected leaves shrivel and fall down prematurely resulting in early defoliation.

Plate-3a. Symptoms of *Alternaria solani* on tomato

***Alternaria alternata*:**

- Spots are small, circular scattered, dark brown spots (Plate-3b).
- Older spots are surrounded by yellow halo.
- The affected leaves dry prematurely.

Plate-3b. Symptoms of *Alternaria alternata* on tomato

***A. alternata* f.sp. *lycopersici*:**

- ✓ Spots are small, angular, scattered and light brown in colour (Plate-3c).
- ✓ Spots are not surrounded by yellow halo.
- ✓ The symptoms also appear on stems and branches as light to dark brown spots(Plate 3d).

✓  Plate 3 c

Plate-3 C,d. Symptoms of *A. alternata* f.sp. *lycopersici* on tomato leaf and stem

Pathogen(s)

***Alternaria solani* (Ell. and Mart) Jones and Grout:**

- ➢ The mycelium consists of septate, branched, light brown hyphae, which become darker with age. Conidiophores emerge through the stomata from the dead centers of the spot.
- ➢ Conidia are beaked, muriform, dark and borne singly or in chains of two.
- ➢ Five to ten transverse septa and a few longitudinal septa are present in each conidium.

***A. alternate* (Fr.) Keissler:**

- The conidia occurring on leaflet lesions under field conditions are 1-6 septate.
- The size of beak range from 3.0 - 10.0 µm with an average length of 7.9 µm.

***A. alternata* f.sp. *lycopersici* Grogan Kimble and Misaghi:**

- The conidia occurring on leaflet and stem lesions under field conditions are 1-5 septate measuring 17.0 - 50.0 x 10.0 - 13.0 µm (av. 34.40 x 12.30 µm) and beak length ranged from 3.0 - 7.0 µm with an average of 6.13 µm.

Disease cycle and epidemiology

- *Alternaria* species survive in diseased plants debris and can persist for one to two years.
- Seed borne nature of *A. solani* has also been reported.
- Primary infection of lower leaves first takes place through conidia formed on crop debris in soil. Secondary spread of the disease occurs through conidia developed on primary spots.
- These conidia are blown by wind, water and insects to the neighbouring leaves\ plants.
- The optimum temperature for infection of *A. solani* is 28 to 30^{O}C while for *A. alternata f.* sp. *lycopersici* and *A. alternata*, it ranges between 25-30 and 20-25^{O}C, respectively.
- Maximum dispersal of conidia occurs in advanced stages of disease development and in between 9 am and 12 noon.

Management

- Collect and destroy the infected plant debris. Follow at least two years crop rotation.
- Select healthy seed and treat it with captan (0.3%).
- Remove the foliage particularly in indeterminate type of cvs/hybrids up to 15-20 cm to avoid moist and stagnant air conditions.
- Spray the crop with chlorothalonil (0.2%) or mancozeb (0.25%) and repeat at 10 to 14 days interval.

4. SEPTORIA LEAF SPOT

Symptoms:

- ✓ Water soaked spots appear on the lower surface of leaf.
- ✓ Later these spots become more or less circular in outline and show definite brown coloured margins with grey centre (Plate-4).

- ✓ A few black glistering pinhead sized pycnidia may be seen in the center of spots.
- ✓ On stem, disease appears as small, slightly elongated, dark spots containing numerous black pycnidia.
- ✓ Black circular spots may also be seen on young as well as on mature fruits.

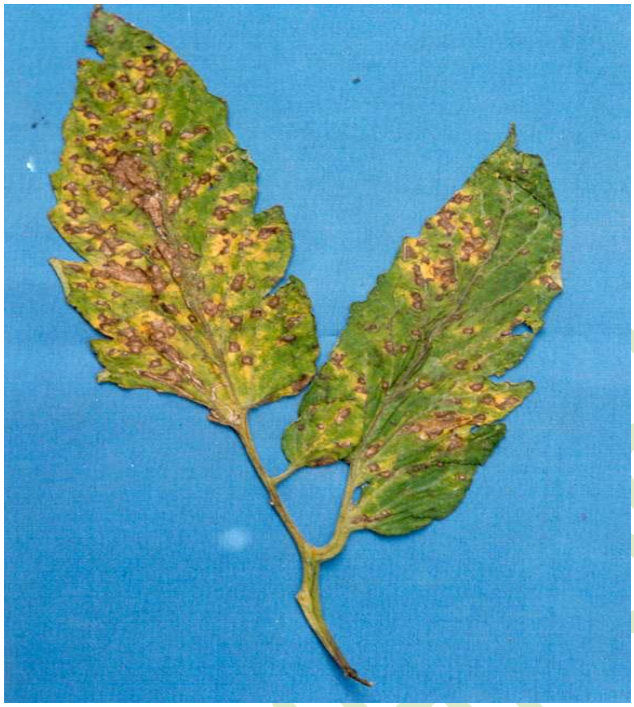


Plate-4 Symptoms of Septoria leaf spot

Pathogen:

- ➢ The disease is incited by *Septoria lycopersici* Speg.
- ➢ On the host, young mycelium is hyaline, thin walled and sparingly septate.
- ➢ Pycnidia are subglobose, composed of 2-3 layers of brown cells.
- ➢ Pycnidiospores are filiform, slightly curved, hyaline and septate with pointed or rounded ends. No sexual stage has been reported.
- ➢ Two physiologic races have been identified.

Disease cycle and epidemiology:

- ➢ The pathogen overwinters in infected plant debris in the form of mycelium or conidia or in debris of solanaceous weed hosts, such as horse nettle (*Solanum carolinense* L.) (Fig. 2.).
- ➢ The conidia are splashed by rain to the lower leaves of the plants.
- ➢ The spores produced in these pycnidia continue the secondary cycle of this disease.
- ➢ Temperature of 20-25^{O}C with 75-92 per cent relative humidity is congenial for disease development.

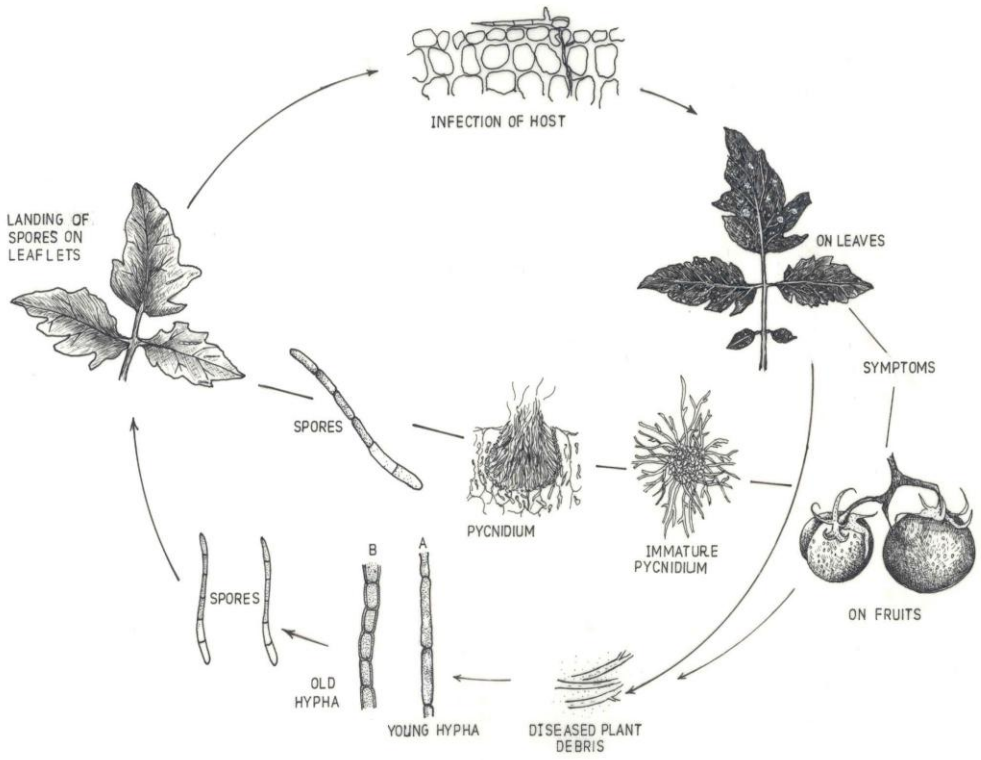


Fig. 2. Disease cycle of Septoria leaf spot (*Septoria lycopersici*)

Management:

- Destruct infected plant debris and use of clean seed.
- One to two years crop rotation of non solanaceous crop, trimming of lower leaves and staking of plants in case of indeterminate varieties/ hybrids have been found useful.
- With the initiation of the disease, spray the crop with carbendazim (0.1%) or mancozeb (0.25%) and repeat at 10 to 14 days interval.

5. FUSARIUM WILT

Symptoms:

- The disease first appears in the field as yellowing of the lower leaves most commonly at the time of flowering stage.
- The affected leaves die prematurely and the symptoms continue to appear on successively younger leaves.
- One or more branches may be affected while the others remain symptom less.
- Browning of the vascular bundles follows rather promptly, and is best seen by cutting of stem or petiole and examining a cross section.

Pathogen:

- The disease is caused by the fungus *Fusarium oxysporum* Schlechtend f.sp. *lycopersici* (Sacc.) Snyder and Hans.

- Mycelium is septate and hyaline at first and becoming cream coloured with age, however, some isolates produce blue or red pigment.
- Micro conidia are ellipsoidal, straight to curved, one to two celled and hyaline.
- Macro conidia are hyaline, 3-5 septate, falcate having gradually pointed and curved ends and appear on sporodochia.
- Chlamydospores, both rough and smooth walled, characterized by thick walls are terminal or intercalary.
- They are often solitary but occasionally form in pairs or chains.
- Three physiological races viz., race 1, 2 and 3 have been reported world over.

Disease cycle and epidemiology:

- The pathogen is soil borne in nature and overwinters in the infected plant debris and in the soil as mycelium and spore forms especially as chlamydospores.
- It spreads over small distances by means of water and contaminated farm equipments, and over long distances, primarily in infected transplants or in the soil carried with them.
- Usually, once an area becomes infested with *Fusarium*, it remains there indefinitely.
- The optimum soil temperature for disease development varies from 25-31 ^{o}C.
- Hot dry weather favour wilt development.
- The disease is more severe in acidic soils (5.6 to 6.5 pH) than in alkaline ones.
- Presence of root knot nematodes and monoculture enhance the disease further.
- Low nitrogen and high potassium levels in soil predisposes the plant to this disease.

Management

- Follow crop rotation of non solanaceous crops and use of healthy seed.
- Avoid movement of water from infected to healthy plants.
- Combined application of inorganic fertilizers and organic manures is considered effective in reducing disease incidence.
- Application of phosphate fertilizers and nitrate fertilizers help in reducing the wilt incidence whereas ammonium fertilizers increase disease development.
- Treat the seed with carbendazim (0.2%).
- Drench the affected plants with carbendazim/ benomyl (0.1%).
- *Pseudomonas* isolates from rhizosphere of different plants can also reduce infection.

Other important fungal diseases of tomato:

- Powdery mildew (*Erysiphe cichoracearum* DC and *Leveillula taurica* (Lev.) Arnaud),
- Phoma rot (*Phoma destructiva* Plowr. and *P. lycopersici* Cooke),

- Verticillium wilt (*Verticillium albo atrum* Reinke),
- Root rot and collar rots (*Fusarium solani* (Mart.) Sacc.,
- *Sclerotium rolfsii* Sacc., *Rhizoctonia solani* Kuhn)

Lecture-3

BACTERIAL AND VIRAL DISEASES OF TOMATO

Tomato (*Solanum lycopersicon* L.) is an important vegetable crop grown globally and consumed as salad, ketchups, sauce, soup, pickles etc. It is also cooked along with other vegetables to provide taste and is the basic need of the Indian kitchen. Ripened fruits are good source of ascorbic acid and minerals. The crop is generally grown during winter months (Oct.- April) in the plains of India while in the hills it is mostly grown during summer and rainy season, thus fresh fruits are available round the year in the market. Prevalence of high humidity and warm temperature not only favours the luxuriant growth of the crop but also favours the development of various bacterial and viral diseases. Under favourable environmental conditions, epiphytotics of certain diseases have often reduced the yields considerably in certain years. Some of the important bacterial and viral disease problems of tomato are described in this lecture.

Aim: To know the bacterial and viral diseases of tomato and their management.

A) BACTERIAL DISEASES

1. Bacterial wilt

- Bacterial wilt is one of the most devastating disease in solanaceous plants.
- It is more serious in warm temperate, subtropical and tropical regions of the world.
- The disease is also called southern bacterial wilt, solanaceous wilt, southern bacterial blight and by many other common names in countries wherever it occurs.

Symptoms

- Characteristic symptoms of bacterial wilt on most of the hosts are sudden wilting and death of infected plants.
- Petioles of the older leaves of the infected plants droop down without yellowing.
- Leaves show epinasty symptoms accompanied by yellowing and stunting of whole plant.
- There is a typical browning of the vascular tissues of roots and stems (Plate-1.).
- Bacterial polysaccharides mechanically block the vascular system, which checks the translocation of water and minerals resulting in wilting of plants.
- Excessive development of adventitious roots may also take place in tomato.
- Cross sectioned part of the affected root or stem yield whitish milky bacterial ooze.

Plate-1. Symptoms of bacterial wilt of tomato

Pathogen:

- The disease is caused by bacterium *Ralstonia solanacearum* (Smith) Yabuuchi *et al.*, 1995).
- The bacterium is gram negative, rod shaped, frequently occurs in pairs, motile with 1 to 4 polar flagella and measures 0.5-0.7 x 1.5 –2.5 µm.
- It is aerobic and catalase and oxidase-positive and forms nitrites from nitrates.
- Five races (race1, 2, 3, 4, 5) and four biovars (biovars I, II, III and IV) are known to occur in different parts of the world.
- In India race 1 and 3 and biovar II, III and IV are prevalent.

Disease cycle and epidemiology:

- ✓ The pathogen is both soil and seed borne in nature and overwinters in infected plant parts, in potato tubers, in wild host plants and solanaceous weeds and can survive at least for 2 years in the absence of any host.
- ✓ Injured and decaying infected tissues release bacteria in the soil which enter plants through wounds occurring in roots due to cultural practices, nematodes and insects.
- ✓ Relatively high soil moisture and soil temperature favour the disease.

Management

- Control of bacterial wilt in infested soil is very difficult as all the commercial cultivars are susceptible and no chemical control is available.
- Follow long crop rotation with non-solanaceous crops.
- Avoid the movement of water from infected plant to healthy plants.
- Shifting of date of transplanting to avoid period of high temperature, heavy rainfall or both.

- Green manuring or biofumigation with *Brassica* spp. may reduce the bacterial inoculum in soil.
- Bacterial antagonists such as *Pseudomonas fluorescens*, *P. glumae*, *P. cepacia* and *Bacillus* spp. have also been known to reduce disease incidence.
- Application of bleaching powder (15 kg/ ha) has also been found effective against this disease.
- The disease can also be controlled effectively if dazomet application is combined with soil solarization.
- Seedling dip in Streptocycline (100 ppm) for 30 minutes is also effective to some extent.
- Use resistant cvs./hybrids for cultivation.

2) BACTERIAL CANKER

Symptoms:

- Leaves show wilting symptoms.
- On stem, brown streaks and canker develop.
- Small brown, scabby lesions surrounded by white halo appear on the fruits which resembles to bird's eye.

Plate-2a Canker on stem

Table-2b Canker on fruits

Plate-2 Symptoms of bacterial canker on tomato

Pathogen:

- Disease is caused by *Clavibacter michiganensis* sub sp. *michiganensis* (Smith) Davis *et al.*
- The bacterium is aerobic, Gram + ve, coryneform rod with cell growing either singly or in pairs.
- Pigmented mutants develop due to possession of a range of carotenoid compounds.
- The organism is characterized by oxidation of carbohydrates, being nonlipolytic, it can only liquify gelatin slowly, the ability to hydolyse starch is weak or absent and amino acids like biotin, nicotinic acid and thiamine are required for growth.

Disease cycle and epidemiology:

- The bacterium perennates in infected seed, plant debris and weed hosts.
- Optimum temperature for disease development is 28^{o} C.
- The disease is more severe in wet weather.

Management

- ✓ Collect and destroy all infected plant debris.
- ✓ Follow crop rotation and tomato should not be grown in infested field for at least 3 years.
- ✓ Use disease free seed and treat them by dipping in the Streptocycline (100 ppm) solution for 1 h.
- ✓ Spray the crop with Streptocycline (100 ppm) followed by copper oxychloride (0.30%) or Bourdeaux mixture (4:4:50) and repeat at 7-10 days interval.

Other important bacterial disease:

Bacterial spot (*Xanthomonas campestris* pv. *vesicatoria* (ex. Doidge) Vauterin *et al.*)

B) VIRAL DISEASES

3. Leaf curl

Symptoms:

- o The major symptoms include chlorosis of leaflets and reduction in their size accompanied by curling inwards.
- o Significant reduction of nodes and internodal lengths occurs giving the plant a bushy appearance.
- o In advanced stages of infection severe stunting and partial to complete sterility occurs. Infected plants bear few or no fruits.

Pathogen:

- The disease is caused by Tobacco yellow leaf curl virus (TYLCV) which belongs to the Gemini Virus group.
- The particles are geminate, non-enveloped, 18 nm in diameter and genome consists of single stranded DNA.

Disease cycle and epidemiology:

- ➢ The virus is transmitted through grafting but not through sap, contacts between plants or seeds. The sole agency of its transmission is the white fly *Bemisia tabaci* that transmits the virus in a persistent manner.

- The virus has a wide host range including cultivated species, weed hosts and ornamentals.

4. TOMATO MOSAIC

Symptoms:

- Mosaic is characterized by presence of dark and light green patches on the leaflets which may get distorted and puckered (Plate -3).
- Malformation and reduction in leaf size may also occur. In case of early infection the plants are reduced in size and remain stunted.

Plate 3: Symptoms of Tomato mosaic virus

Pathogen:

- Tomato mosaic virus (ToMV) is a member of *Tobamo virus* group.
- ToMV is an RNA-containing virus with straight rod particles about 300 x 18 nm.

Disease cycle and Epidemiology:

- Virus transmission in the field either occurs through contact between plants and field implements.
- The other modes of transmission are through sap and grafting but there is no vector involved.
- The virus is externally seed borne. The infection occurs during transplanting and the virus has a wide host range.

5. Spotted wilt

Symptoms:

- Plants show bronzing, curling, necrotic streaks and spots on leaves (Plate-4a).
- Dark brown streaks also appear on leaf petioles, stems and growing tips.
- The plants remain small and stunted.
- The ripe fruit show pale or red or yellow areas on the skin (Plate-4b).
- Sometimes affected plants are killed by severe necrosis.

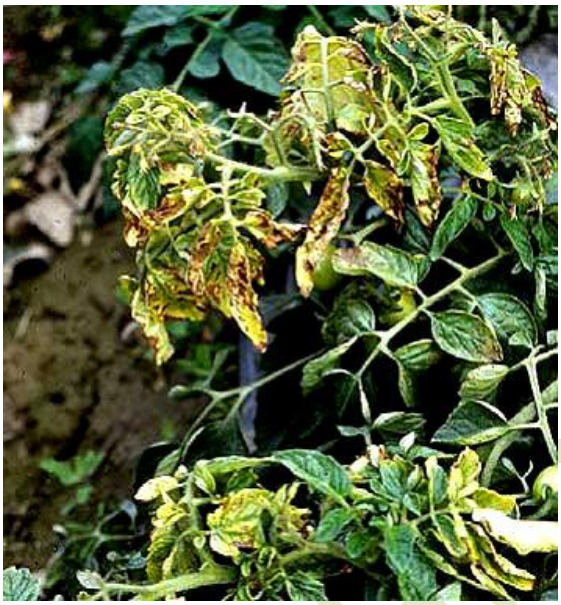


Plate-4a. Symptoms of tomato spotted wilt virus on plant

Plate-4b. Symptoms of tomato spotted wilt virus on fruits

Pathogen:

- The disease is caused by Tomato Spotted Wilt Virus (TSWV), Tospo virus.
- The virus particles are isometric, enveloped, and 85 nm in diameter.
- The genome consists of single stranded RNA.

Disease cycle and epidemiology:

- The virus is transmitted by thrips in persistent manner.

- The virus is acquired only by intensive feeding of the larvae and transmitted by adults.
- The virus has wide host range including bell pepper, lettuce, pea, tobacco, potato and large number of ornamental plant species.

Management:

- The viral diseases of tomato can be managed effectively through use of virus free seed (ToMV).
- Remove alternate hosts (TLCV, TSWV).
- Use insecticides against the vectors and cultivation of resistant varieties / hybrids.
- Seedling bed should be in isolation from ornamental plants and susceptible crops and the surrounding areas should be kept free from weeds.
- Fine mesh netting may be useful for excluding thrips and other insect vectors.
- Aqueous extracts of *neem* (3-5ml/litre water) are known to possess strong antiviral substances against ToMV.

Lecture-4

DISEASES OF BRINJAL

Brinjal (*Solanum melongena* L.) also known as egg-plant, aubergine, Guinea squash, is one of the important vegetable crops grown almost worldwide. It is native to India, where it has been cultivated since remote antiquity for its fleshy fruits. Eggplant fruits were a common food in China as long as 600 BC, when it was called Malayan purple melon. Now, it is extensively grown in eastern and southern Asia, including India, USA and other countries. The raw vegetables contains only 15 calories/ 100 g but its caloric value rises sharply when it is fried. Africans following folk medicine have long used brinjals to treat epilepsy and convulsions. In Southeast Asia, it is still used to treat measles and stomach cancer. During cultivation, crop is affected by several diseases of fungal, bacterial and phytoplasma nature, which inflict heavy losses in its production. A detailed account of various diseases affecting this crop and their management are described in this lecture.

Aim: To know about different diseases infecting brinjal and their management.

A) FUNGAL DISEASES

1.) Phomopsis blight

Symptoms:

The plants are attacked at all stages of growth.

- In nursery bed, it causes damping off which results from the infection of the seedlings at the collar portion just above the soil line. After transplanting, circular grey spots with light coloured centers appear on the leaves (Plate 1a).
- In later stages, the lighter portion is studded with numerous black pycnidia.
- Affected leaves turn yellow and fall down pre-maturely.
- At the base of the stem, the fungus causes characteristic constrictions leading to canker development and toppling of the plants.
- On fruits, the disease manifests as pale sunken spots, which later enlarge and cover the entire fruit surface. A large number of dot like pycnidia also develop on such spots (Plate-1b). If infection of fruits takes place through calyx, the whole fruit becomes mummified due to dry rot.

1a. On fruits **1b. On leaves**

Plate-1. Symptoms of Phomopsis blight on brinjal

Pathogen:

- The fungus responsible for this disease is *Phomopsis vexans* (Sacc. and Syd.) Harter. *Diaporthe vexans* Gratz is the perfect stage.
- The mycelium is hyaline and septate.
- The conidiophores (phialides) in the pycnidium are hyaline, simple or branched, sometimes septate and arise from the innermost layer of cells lining the pycnidial cavity.
- The pathogen is reported to produce two types of conidia viz., alpha and beta in its pycnidium.
- Formation of conidia in pycnidia of *P. vexans* is temperature dependent. At low temperature (10-16^{o}C), the pathogen produces beta conidia and at high temp. (25-28^{o} C), the alpha conidia.
- These two forms of conidia get inter-converted when subjected to specific temperature and alpha and beta are two forms of the same conidium.
- *P. vexans* produces only one type of conidia in its pycnidia during summer months. Conidia are hyaline, one celled and sub cylindrical which gradually changed into beta form.
- Beta form of conidia, the stylospores is filiform, curved, hyaline and septate.
- These spores normally do not germinate. But inoculation of host with beta conidia caused interveinal necrosis.

- Perithecia are observed only in cultures which are usually in clustures, beaked, carbonaceous, sinuate, and with irregular ostiole.
- Asci are clavate, sessile and contain eight ascospores.
- Ascospores are hyaline, narrowly ellipsoid to bluntly fusoid, one-celled, septate and constricted at the septum.

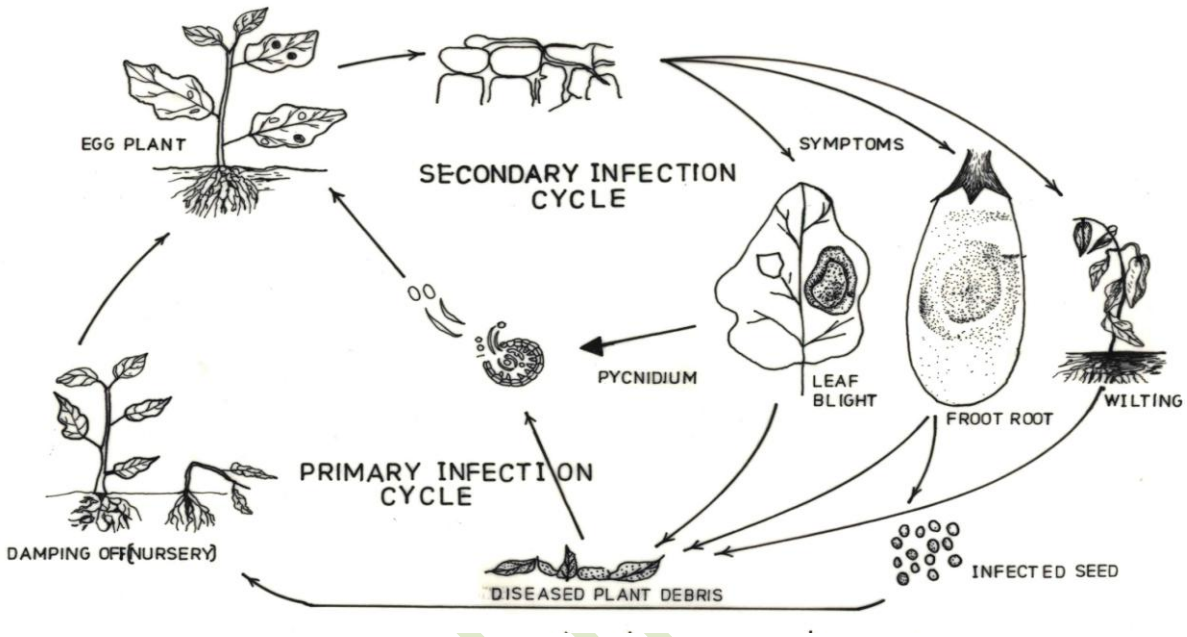


Fig. 1. Disease cycle of Phomopsis blight (*Phomopsis vexans*) of brinjal

Disease cycle and epidemiology:

- The pathogen is seed borne and also survives in plant debris both as mycelium and pycnidia (Fig.1.).
- Seed infections directly lead to diseased seedlings.
- The pycnidiospores are disseminated through rain splashes, irrigation water, agricultural implements and insects.
- High relative humidity coupled with high temperatures is favourable for disease development.
- Pathogen grows best at a temperature of 21 – 32.5°C with a optimum temperature of 28 – 29°C. Storage rot is maximum at a temperature of 25°C.
- Maximum disease development takes place at about 26^{o}C under wet weather conditions.

Management:

- Cultural practices like collection and destruction of the diseased plant debris, crop rotation and use of disease free seeds are recommended to reduce the initial inoculum.

- Treat the seed with carbendazim (0.2%) or thiophenate methyl (0.2%). With the initiation of the disease, spray the crop with carbendazim (0.1%) or combination of mancozeb (0.25%) and carbendazim (0.05%) or copper oxychloride (0.3%) and repeat at 10 to 14 days interval.

2. Sclerotinia wilt

The disease is more important in tarai area (foot hills of H.P and Uttrakhand) of the country where it causes considerable yield losses.

Symptoms:

- Circular to elongate water-soaked lesions closer to the inflorescence appear on the branches followed by watery soft rot.
- At the point of infection, a dry, discoloured spot develops.
- As a result of tissue necrosis, the portion of the plant beyond the point of infection wilts.
- If the infection is at the base of the main stem, the entire plant wilts and if only few branches are attacked than partial wilting of the plant may take place.
- In the advance stages under cool humid conditions, the mycelium emerges out and creamy coloured compact sclerotial initials as well as matured black sclerotia of varying sizes are evident on above ground parts.
- Fruits are also attacked. The rotting of the flesh takes place and in rotting tissue large number of sclerotia are seen.

Pathogen:

- The disease is caused by *Sclerotinia sclerotiorum* (Lib.) de Bary.
- Mycelium in culture as well as on host surface is hyaline, cottony, branched, consisting of closely septate hyphae, and filled with dense granular protoplasm.
- Fungus germinate by two means i.e. myceliogenic and carpogenic (forming apothecia). Primary infection occurs through ascospore whereas secondary infection takes place through mycelium.
- Microconidia (spermatia) are produced on short lateral branches of the vegetative mycelium in chains.
- When food supply is exhausted and the vegetative growth ceases, the hyphae with granular protoplasm collect in small dense masses and form sclerotia.
- These sclerotia are first white in colour and then turn black.
- The sclerotia also germinate by producing stalked apothecia, which range from one to five per sclerotium when proper humidity and light conditions are provided to overwintered sclerotia.
- The best apothecial development takes place at 7-11°C and their production ceases at 16-28°C with low soil moisture.
- Apothecia are brown in colour and are round or lobate type.
- Asci are cylindrical measuring 108-153 x 4.5- 10 μm in size and each ascus contains eight ascospores, which are released in clouds.
- Fungus produces oxalic acid which kills cells in advance resulting characteristic hollow stem.

Disease cycle and epidemiology:

- The fungus survives in soil in the form of sclerotia.
- In the presence of proper humidity and light conditions, sclerotia germinate by forming apothecium which in turn forms asci and ascospores.
- The spores, upon escaping from the ascus, lodge on a susceptible host, and a new infection may originate.
- Mycelium from sclerotia is also capable of infecting eggplant.
- The pathogen can infect the susceptible host over a wide range of temperatures i.e. from 0 to 25 ^{o}C with an optimum at 15 to 20 ^{o}C.
- The fungus can tolerate wide pH range but is best adapted to an acidic substrate. Application of nitrogenous fertilizers enhances this disease.

Management:

- Collect and destroy the infected plant debris.
- Follow crop rotation with cereals i.e. paddy or maize.
- With the initiation of the disease, spray the crop with carbendazim (0.1%) or thiophanate methyl (0.1%) or combination of mancozeb (0.25%) and carbendazim (0.05%) and repeat at 10 to 14 days interval.
- Fungi like *Trichoderma harzianum* and *T. viride* (40 g/ m^2 at the time of field preparation) have also been reported as antagonistic to this fungus.

3. Cercospora leaf spot

Several leaf spot diseases affect this crop but the leaf spots caused by *Cercospora* species are important which under high humidity conditions cause considerable yield losses.

Symptoms

***Cercospora melongenae*:**

- The symptoms of the disease appear as circular to irregular leaf spots, which are usually large and brown to grayish brown in colour.
- Later spots coalesce together and affected leaves fall down prematurely and sometimes fruits may rot.

***Cercospora egenulae* :**

- Early symptoms start as minute water-soaked specks, which gradually enlarge to become circular to angular leaf spots of 2-6 mm diameter.
- Early infection noticed on the lower leaves which gradually spreads upwards with high humidity and rainfall.
- These spots often coalesce to form bigger spots having a papery pale yellow necrotic region in the center.
- The necrotic region in the center disintegrates bearing a shot hole.
- Infected leaves turn yellow, droop and fall prematurely resulting in quick defoliation of the entire plant.

Cercospora solanigena:

Leaf spots amphigenous circular to irregular later coalescing, necrotic, dark brown in center and light brown in margin, 0.5-2 mm wide.

Pathogen(s)*:*

The disease is caused by several species of *Cercospora* like *C. melongenae* Wells., *C. solanigena* Bartiya, Dubey and Singh and *C. egenulae* (Syd.) Chupp and Doidge.

C. melongenae:

- Conidiophores are simple, dark septate and 30-60 x 4-6 µm in size.
- Conidia are clavate, tapering to a blunt distal tip, sub-hyaline, 3-12 septate and 38- 119 x 4-8 µm in size.

***C. egenulae* :**

- Fruiting both epi- and hypophyllous, stromata distinct dark brown, globose to irregular, fascicles dense to olivacious brown, paler and narrower towards the tip, a septate, not branched, notgeniculate, undulate with rounded apex and 2.5-4.5 x 3 - 31µm in diameter.
- Conidia hyaline to subhyaline, shorter ones distinctly cylindric and very long ones obclavate or almost acicular, straight or nearly so, 1-5 septate, base subtruncate to rounded, tip obtuse, 3-6 x 20-85 µm in diameter.

***C. solanigena*:**

- Mycelium internal, hyphae branched, septate, dark olivaceous. Stromata well developed, pseudoparenchymatous, dark, olivaceous, 10-30 µm in diameter.
- Conidiophores arising in fascicles (1-4) from stromata, macronematous to fasciculate, erect, straight to flexuous, unbranched, smooth, 1-6 transversely septate, geniculate, light olivaceous to dark brown, 16-100 x 3-5 µm in size.
- Conidiogenous cells integrated, terminal, polyblastic, cicatrized, scars conspicuously thickened paler in colour.
- Conidia acropleurogenous, holoblastic, dry, solitary, unbranched, 1-5 transversely septate, smooth walled, hyaline, acicular, straight to curved, base truncate, apex sub acute to sub obtuse with thickened hilum.

Disease cycle and epidemiology*:*

- The fungus survives from one season to another in infected plant debris in soil and also in infected seeds wherever fruit rot occurs.
- Warm days and cool nights are ideal for the infection.
- The disease is favoured by high humidity and heavy persistent dews.
- Water droplets must be present for spore germination and germ-tube penetration.
- Moist wind, irrigation water and insects help in local transmission of the pathogen.

Management:

- Cultural practices like destruction of crop debris, crop rotation, use of disease free seeds and wider plant spacing should be followed to reduce the primary inoculum of the pathogen in field.
- With the initiation of the disease spray the crop with zineb (0.25%), carbendazim (0.1%) or thiophanate methyl (0.1%) and repeat at 10 to 14 days interval.

B) BACTERIAL DISEASES

4. Bacterial wilt

Bacterial wilt is a limiting factor in successful cultivation of eggplant crop particularly in tropical, subtropical and some warm temperate climates. In hills, the incidence of this disease was high at intermediate elevations i.e. between 800 to 1400 meter m.a.s.l. and decreased with the increase in altitude. Symptoms, pathogen, disease cycle, epidemiology and management are same as described under tomato.

5. Little leaf

This is an important disease particularly in Southeast Asian countries where the activity of the vector is more.

Symptoms:

- The disease is characterized by reduction in leaf size which has shortened petioles and leaf lamina (Plate-2).
- There is production of more number of branches and all this results in stunted and bushy appearance of the plants.
- The flowers are not produced but in case these are produced, that remain green.

Pathogen:

- The disease is caused by Phytoplasma having ovoid or spherical body.
- These measure 40-300 nm in dia. and lack a rigid cell wall.

- The Phytoplasma are present in phloem sieve tubes.

- 

Plate-2. Symptoms of little leaf of brinjal

Disease cycle and epidemiology:

- Phytoplasma perennates on weed hosts like *Datura* spp., *Vinica rosea* and several others.
- The disease is transmitted by grafting and through leaf hopper *Hishimonas phycitis* and *Empoasca devastans* but later is less efficient.

Management:

- Destruction of weed hosts from in and around the field and control of vectors through application of insecticides is advocated.
- Dipping of seedlings in tetracycline (0.05%) before transplanting followed by soil application of Phorate (1kg/ha) also keeps the disease under check.

Lecture-5

BELL PEPPER AND CHILLIES

Bell pepper and Chilli (*Capsicum annuum* L.) popularly known as Shimla Mirch" and 'Lal Mirch", respectively, are important solanaceous crops grown throughout the world. The fruits are a good source of vitamin A and ascorbic acid. These are native of Mexico with secondary centre of origin in Guatemala and Portuguese explorer introduced it to Asia. In India, these are grown in most of the states of the country. During cultivation these crops are affected by various diseases which reduce the potential yields drastically. In this lecture, a detailed account of the diseases has been given.

Aim: To know about different diseases infecting bell pepper and chilli crop and their management.

A) FUNGAL DISEASES

1. PHYTOPHTHORA LEAF BLIGHT AND FRUIT ROT

It is an important disease in those areas where fruiting coincides with the onset of monsoon rains. Due to favourable climatic conditions the disease is so severe that sometimes it causes complete defoliation of the plants. In India, disease was first time reported in1968 as *Phytophthora nicotianae var. nicotianae.*

Symptoms:

- Symptoms of the disease appear as water-soaked bleached spots on any portion of the leaf (plate 1a) resulting in premature leaf fall.
- Small water soaked spots also appear on the fruits and the flesh below the skin become soft and usually there is a distinct line of demarcation between the invaded tissue and healthy (Plate 1b).
- Whitish mould appears on the rotten fruits under humid conditions and completely rotten fruits may fall down on the ground.
- Symptoms also appear on collar region of adult plants as water-soaked areas with whitish growth of mycelium engirdling the collar region and the point of contact of the soil line.
- The rot often progresses downwards to the roots in the affected plants and there is sudden drooping of leaves giving the appearance of sudden wilt.

Plate 1a Symptoms of leaf blight

Plate 1b Symptoms of fruit rot

Pathogen (s):

- Two species of *Phytophthora* namely *P. capsici* Leon and *P. nicotianae* (Breda de Hann) var. *nicotianae* Waterhouse have been found associated with the disease.
- In *P. capsici* mycelium is hyaline, branched and non-septate but few septa are found in case of old hyphae.
- The sporangia are hyaline, ovoid to pyriform or sometimes round to lemon shaped, non-pedicillate with a predominant, hemispherical papilla at the apex.
- The oospores are circular to spherical which are found to germinate either by germ-tubes or by stalked or sessile germ sporangia.
- In *P nicotianae* var. *nicotianae*, the mycelium is hyaline branched and non-septate with branches at right angles.
- The sporangia are hyaline, non-pedicellate, globose to subglobose with a prominent beak like papilla at the apex (Plate 2a).
- The sporangia germinate by production of 15 to 20 zoospores.
- Oospores are formed abundantly on aerial and submerged mycelium in culture.
- The oospores are thick walled golden brown in colour (Plate 2b).

Plate 2a Sporangia of *P. nicotianae* var. *nicotianae*

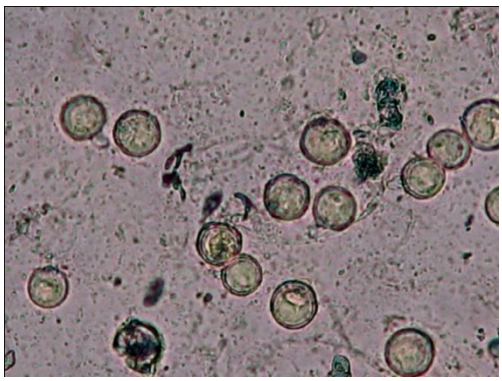


Plate 2b Oospore of *P. nicotianae* var. *nicotianae*

Disease cycle and epidemiology:

- Both pathogens survive in the form of oospores in the soil as well as in infected seed (Fig.1.).
- Fungus germinates only by means of zoospore and not by means of germ tube as in buck-eye rot pathogen of tomato.
- Presence of abundant rainfall, high RH and warm weather are essential for initiation of this disease.
- Heavy rains help in sporangial formation and their germination by zoospores which are splashed with spattering rains.
- After infection, sporangia are produced on the fruit surface and are carried by wind to the adjoining fruits and foliage.
- Temperature ranging from 22 -25^{o} C along with high humidity (>80%) favour disease development.

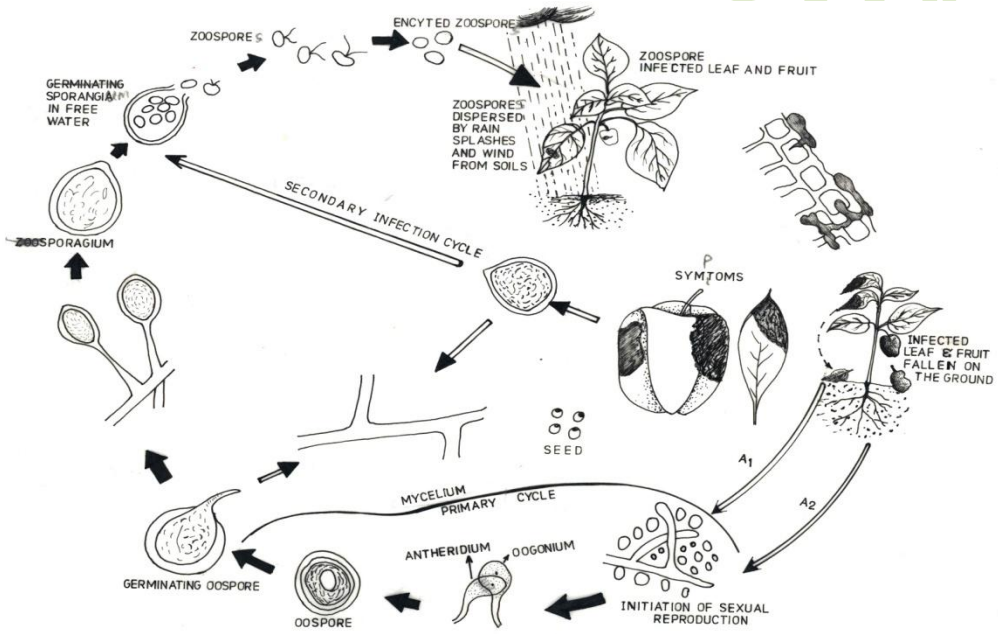


Fig. 1. Disease cycle of leaf blight and fruit rot (*P. nicotianae var. nicotianae*)

Management:

- Collect and destroy the infected leaves and fruits regularly.
- Drainage of the field should be proper to avoid water stagnation.
- Apply pine needles/ grass mulch on the field floor before the onset of monsoon rains.
- Spray the crop with metalaxyl + mancozeb (0.25%) with the onset of monsoon rains followed by sprays of either mancozeb (0.25%) or copper oxychloride (0.3%) or Bordeaux mixture (4:4: 50) at 7 to 10 days interval.

2. ANTHRACNOSE AND RIPE ROT

Symptoms: The symptoms of the disease appear in two phases i.e. die-back and anthracnose and ripe fruit rot.

a) Die-back :

- Symptoms appear as necrosis of tender twigs from the tip downwards.
- The entire plant or branch may wither away.
- The twigs become straw coloured in advanced stages of the disease.
- Large numbers of black dots (acervuli of the fungus) are seen scattered all over the necrotic parts of the plants.

b) Anthracnose and ripe fruit rot:

- The disease is noticed on fully mature green fruits as well as on red ripe fruits.
- The symptoms are characterized by the appearance of small, circular, yellowish to pinkish, sunken spots on the skin of the fruits which spread in the direction of long axis (Plate-3).
- As the fruit matures, these spots become brownish to black and severely infected fruits look straw coloured and bear numerous dots like acervuli in concentric rings.
- The seeds produced in such fruits are discoloured and covered with mycelial mat.

Plate-3. Symptoms of anthracnose and ripe fruit rot of bell pepper

Pathogen:

- The disease is caused by *Colletotrichum capsici* (Syd.) Butler and Bisby. (Tel: *Glomerella cingulata* (Stoneman) Splaud and Schrenk.
- The mycelium of *C. capsici* is septate, intercellular as well as intra-cellular and aerial mycelium appears light to dark grey in colour.
- Acervuli are round and elongated in shape (Plate-4). Setae are scattered, brown, 1-5 septate, rigid and swollen at base and acute at apex.
- Conidiophores are short, hyaline to faintly brown, cylindrical, septate or aseptate.

- Conidia are falcate, fusiform with acute apices and narrow truncate base and they are uni one celled, hyaline and uninucleate.

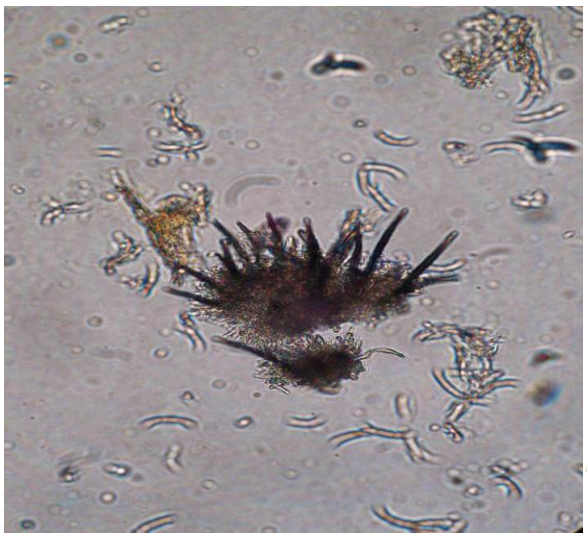


Plate-4. Acervulus of *Colletotrichum capsici*

Disease cycle and epidemiology:

- The pathogen survives both in infected plant debris and in the infected seed.
- The fungus can survive in plant debris in the soil for at least nine months which serve as source of primary infection, whereas secondary infection takes place through wind borne conidia.
- A temperature of 26^{o} C and presence of free moisture or RH 100 per cent is optimum for disease development and progress.

Management:

- Collect and burn the infected plant debris.
- Remove and destroy solanaceous weed hosts from in and around the field.
- Use seed from healthy fruits.
- Treat the seeds with captan (0.3%).
- Spray the crop with carbendazim (0.1%) or thiophanate methyl (0.1%) or combination of mancozeb (0.25%) and carbendazim (0.05%) and repeat at 10 to 14 days interval.

3. Cercospora leaf spot:

Symptoms:

- On leaves, symptoms appear as circular spots with brown borders and light coloured, faded or grayish central part which resemble the frogeye (Plate-5), so the disease is also called as frogeye leaf spot.
- When many spots appear on the leaves, such leaves turn yellow and fall down prematurely.
- Symptoms also appear on the stems and petioles as elongated or irregular spots.
- Symptoms are also observed on fruits.

Plate-5 Symptoms of Cercospora leaf spot of bell pepper

Pathogen:

- The fungus responsible for this disease is *Cercospora capsici* Heald & Wolf.
- The fungus is characterized by dark brown stromata, which produce fasciculate, straight or slightly curved, continuous, simple yellowish brown conidiophores, producing polymorphous, cylindrical, fusoid 1to 3 septate, acrogeneous yellow to brown conidia broad and tapering towards the apex.

Disease cycle and epidemiology:

- The fungus mainly perennates through the infected plant debris in the form of mycelium or stromata.
- In areas where fruit infection occurs, the fungus can also overwinter in the form of infected seed.
- The conidia develop on the overwintering stromata which serve as the source of primary infection.
- These conidia are disseminated by wind and rain splashes to the leaf surface where they germinate and cause infection.
- Disease development is favoured by high humidity (> 95%) and temperature (20-25^{o} C).

Management:

- Collect and burn the infected plant debris. Follow crop rotation and maintain proper drainage in the field.
- Use healthy seeds and treat the seeds with captan (0.3%).

- Spray the crop with carbendazim (0.1%) or thiophanate methyl (0.1%) or combination of mancozeb (0.25%) and carbendazim (0.05%) or difenoconazole (0.03%) and repeat at 10 to 14 days interval.

4. Powdery mildew:

It is one of the important disease problems of this crop particularly in protected cultivation conditions.

Symptoms*:*

- On the under surface of the leaves white to grey-coloured spots appear (Plate-6) while their corresponding upper surface exhibit yellow lesions with brown necrotic centers. Affected leaves curl upwards.
- Premature senescence of the leaves results in defoliation.
- Severe infection may cause die-back of the twigs or branches and stunting of plants followed by fruit drop.

Plate-6 Symptoms of powdery mildew on the under surface of the leaves

Pathogen*:*

- The fungus *Leveillula taurica* (Lev.) Arnaud (anamorph = *Oidiopsis sicula scalia* Syn. *Oidiopsis taurica* (Lev.) Arnaud) is responsible for this disease.
- It is an obligate parasite and ectendophytic mycelium.
- Conidiophores are long and multi-branched.
- Conidia are dimorphic (pyriform and cylindrical) and conidia are borne singly or in short chains and their size vary according to the isolate.

Disease cycle and epidemiology:

- The pathogen has wide host range including both cultivated and wild hosts which ensures the survival of the pathogen from one season to another.
- Temperatures of 25-30^{o} C favour germination of conidia.

- High relative humidity during night than day time and temperatures < 30^{o} C are favourable for the disease.
- Low relative humidity i.e. < 50 per cent favours disease spread.

Management:

- Cultural practices like wider plant spacing, sprinkler irrigation and misting in protected structures along with increased air circulation are effective in keeping the disease under check.
- With the initiation of disease spray the crop with systemic fungicides like hexaconazole (0.05%) or carbendazim (0.1%) or difenoconazole (0.03%) and repeat at 10 to 14 days interval.

B) BACTERIAL DISEASES

6. Bacterial spot

The disease affects bell pepper/ chillies and tomato. Bacterial spot of capsicum was first reported from Florida, USA by Sherbakoff (1918), but in India, disease was observed for the first time at Poona during 1948.

Symptoms*:*

- On leaves, initially the lesions are water-soaked, circular or irregular that becomes necrotic with brown centers and thin chlorotic borders (Plate-7a).
- These lesions are generally sunken on the upper surface of the leaves and slightly raised on the bottom.
- In favourable environmental conditions, these spots coalesce and give a blighted appearance and such leaves turn yellow and fall down prematurely.
- Lesions on the stem are narrow, elongated and raised.
- Lesions on fruit initially begin as green spots, which enlarge and later become brown in colour (Plate-7b).
- These spots are raised with a cracked, roughened wart like appearance.
- During periods of high humidity, fruit around the lesions may start rotting.

a) On leaves **b) On fruits**

Plate 7. Symptoms of bacterial blight

Pathogen:

- The bacterium responsible for this disease is *Xanthomonas campestris* pv. *vesicatoria* (ex Doidge) Vauterin *et al*.
- The bacterium is motile, strictly aerobic, Gram -ve, rod shaped with round ends, non-spore forming and possesses a single pollar flagellum (Monotrichous).
- Bacteria occur singly but occasionally in pairs and rarely in short chains.
- Its colonies on nutrient agar or on specific medium yeast dextrose agar (YDA) are lemon yellow, slightly convex, wet and shining.

Disease cycle and epidemiology:

- The bacterium persists in infected seeds and plant debris.
- Moderate temperatures along with high precipitation and relative humidity favour disease development.
- Infection of the plants can take place at a wide range of temperature i.e. 15 to 35 ^{o} C.
- The optimum temperature lies between 22 to 34^{o} C and maximum disease develops in between July and September.
- The bacterium is disseminated within a field by wind driven rain droplets, clipping of transplants and aerosols.

Management:

- Since the disease development requires high precipitation and high relative humidity, fields should be well drained, free of low-lying areas to minimize water-logged conditions.
- The field should be rotated with non-solanaceous crops and bell pepper/ chillies should not be rotated with tomato.
- Use of disease free seeds also reduce the immediate availability of primary inoculum and ensures disease free transplants.
- Sprinkler or overhead irrigation of the field should be limited to keep the disease under check.
- Treat the seeds by dipping in Streptocycline (100 ppm) for 30 minutes. Spray the crop with Streptocycline (100 ppm) with the initiation of the disease.

7. Bacterial wilt

It is an important disease of this crop. Symptoms, pathogen, disease cycle and epidemiology and management are same as described in tomato.

C) VIRAL DISEASES

8. Mosaic

Symptoms:

- Different types of mosaic pattern are produced depending upon the type of virus or virus (s) involved.
- Potato virus Y and its strains generally induce vein and stem necrosis.
- Pepper veinal mottle virus causes mosaic, mottling, distortion and filiform of the leaves leading to bushy appearance of the infected plants (Plate- 8).
- Cucumber mosaic virus produces mosaic, mottling symptoms, reduction in leaf lamina and filiformy.

Plate-8. Symptoms of pepper veinal mottle virus

Pathogen:

- Different viruses like Potato virus Y (PVY) and its strains, cucumber mosaic virus (CMV), tobacco mosaic Virus (TMV), tobacco etch virus, pepper vein banding virus, pepper mottle virus and pepper sever mosaic virus besides chilli mosaic virus are involved in producing mosaic symptoms in this crop.
- PVY virions are filamentous flexuous rods, non-enveloped and consist of single stranded RNA.
- CMV virions are isometric, non enveloped, 29 nm in dia. having single stranded RNA.
- TMV virions are rod shaped non-enveloped having ss RNA.
- Pepper mottle virus is filamentous flexuous having ss RNA.
- Pepper severe mosaic virus is also filamentous flexuous of 761 x 13 nm size consisting of ss RNA.

Disease cycle and epidemiology:

- All associated virus (s) are sap transmissible.
- Except TMV, others are aphid transmissible.
- Predominant aphid vectors are- *Myzus persicae*, *Aphis gossypii* and *A. craccivora* which transmit the virus (s) in non-persistent manner.
- The abundance activity of vectors and the prevailing environmental conditions affect the virus multiplication and spread.

9. Leaf curl

It is an important disease of this crop and cause significant yield reductions if the vector is present in the area.

Symptoms:

- The symptom of the disease appears as curling of the leaves followed by reduction in their size which later turn pale yellow in colour (Plate-9).
- The older leaves become leathery and brittle.
- The affected plants become stunted.
- Fruits formation in susceptible cultivars is rudimentary and distorted.

Plate-9 Symptoms of tobacco leaf curl virus

Pathogen*:*

- The disease is caused by tobacco leaf curl virus (TLCV), which belongs to Bigeminivirus group.
- The virus consists of geminate isometric particles measuring 18 to 20 nm in diameter.

Disease cycle and epidemiology:

- The virus survives from one season to another on various annual and perennial hosts and transmitted to pepper by the vector white fly named *Bemisia tabaci*.

- The epidemic of the disease depends on the availability of the vector, sources of inoculum, vector activity and their population and prevailing environmental conditions.
- During wet weather, the spread of the disease becomes slow due to reduced activity of vector.

Management:

- Pepper varieties like Punjab Lal, Perennial and Guhati Black are resistant to mosaic viruses belonging to Poty and Cucumovirus groups.
- Planting of maize as barrier crop is also helpful in reducing the mosaic incidence.
- Combined use of yellow traps and insecticidal sprays reduces the population of vectors.

Minor disease of importance are:

i. Stem rot (*Sclerotium rolfsii* Sacc.),
ii. Grey mould (*Botrytis cinerea* Pers. ex Fr.),
iii. Choenophora blight or bud rot *(Choenophora cucurbitarum* (Berk. & Rav.) Thaxter),
iv. Fusarium wilt (*Fusarium oxysporum* Schlecht. ex Fr. f. sp. *capsici*)
v. Soft rot (*Erwinia caratovora* sub. sp. *carotovora* (Jones) Bergey *et al*.).

Lecture-6

DISEASES OF BHINDI/LADYFINGER/OKRA

Bhindi (*Abelmoschus esculentus* (L.) Moench) also known as 'okra' or 'lady's finger' is one of the important vegetables grown throughout the country both under open and protected structures. During cultivation, the crop is severely infected with various diseases, which not only reduce the quantity but also affect the quality of the fruits. This lecture deals with the fungal and viral diseases affecting this crop and their management.

Aim: To know about different diseases infecting bhindi /okra and their management.

A) FUNGAL DISEASES

1. FUSARIUM WILT

The disease can appear on any stage of plant growth and the crops planted during summer months suffer more than the crops planted during February-March.

Symptoms:

- The characteristic symptoms of the disease appear as yellowing and stunting of the plants followed by wilting and rolling of the leaves (Plate-1) and finally the plant dies.
- Before the appearance of typical wilting symptoms, the leaves hang down during daytime and recover again in the night but ultimately they wilt and die.
- Vascular bundles of the affected plants appear as dark streaks and the whole stem is blackened in case of severe infection.

Plate-1. Symptoms of Fusarium wilt of okra

Pathogen:

- The disease is caused by *Fusarium oxysporum* f.sp. *vasinfectum* (Atkinson) Snyder and Hansen.
- The mycelium is hyaline and intracellular in the host.

- Macroconidia are mostly 3 septate and microconidia are 0 to 1 septate.
- Macroconidia are fusiform, falcate, curved and formed on sporodochia and pionnotes. In mass these conidia appear buff to salmon orange in colour.
- The microconidia are 5-12 x 2-3.5 µm while macroconidia are 40-50 x 3-4.5 µm in size.
- Both intercalary and terminal chlamydospores are formed which are broadly ovate.

Disease cycle and epidemiology:

- The fungus survives from one season to other in the form of chlamydospores and in infected seeds.
- In contact with the host roots, the chlamydospores or conidia germinate and penetrate.
- The pathogen proliferates extensively in the cortical tissues, extending up and down.
- After some growth in root cortex, the pathogen reaches the xylem vessels where it multiplies very rapidly and result in browning of xylem vessels and clogged at further places.
- The optimum temperature for growth of the fungus is 25^{o}C and for disease development ranges between 22 – 28^{o} C.
- Moisture is not that much important for infection.

Management:

- Cultural practices like long crop rotations, exposing the soil to the sun during summer months by deep ploughing, soil solarization and destruction of diseased roots are some of the practices which can reduce the disease.
- Some cvs. of okra like Pusa Sawani and Pusa Makhmali have been reported resistant to this disease.
- Use healthy seed and treat with carbendazim (0.2%).

2. CERCOSPORA LEAF SPOT

Symptoms

Three different species of *Cercospora* are associated with this disease. The symptoms of each species are described below:

Cercospora abelmoschi:

- The fungus produces indefinite leaf spots but grows as a sooty mould on the lower surface of the leaves (Plate-2).
- Severely affected leaves roll, wilt and fall down to the ground.

Plate-2. Symptoms of Cercospora leaf spot on okra

Cercospora hibiscina: The fungus produces dark olivaceous patches of mould on the lower surface of the leaves.

Cercospora malayensis: The spots caused by this species produce definite leaf spots with grey centers and red to purple borders.

Pathogen(s):

- The disease is caused by three species of *Cercospora* like *C. abelmoschi* Ell. & Ev., *C. hibiscina* Ell. & Ev. and *C. malayensis* Stevens.
- These species differ in their size of conidiophores and conidia.
- In *C. abelmoschi*, the conidiophores are long, brown and bear pale olivaceous, slightly tapered conidia.
- The conidiophores of *C. hibiscina* are extremely long sometimes up to 1000 µm in size, narrow and bear conidia that are sometimes hyaline and appreciably more narrow then those of *C. abelmoschi*.
- The conidiophores of *C. malayensis* are borne in clusters of 5-20 and bear conidia, which are colourless, narrow, long, and tapering from the blunt base to the sharp tip.

Disease cycle and epidemiology:

- The fungus overwinters in the infected plant debris as conidia or stromata in soil and also on the wild species of *Abelmoschus*.
- The conidia in favourable weather conditions, germinate and cause infection of the host through stomata.
- The spores produced on the primary spots are blown by wind and cause secondary infections.
- The three species cause infection in moderate temperatures (25-29^{o} C) and high humidity.

Management:

- Cultural practices like collection and destruction of infected plant debris, crop rotation and destruction of wild hosts from in and around the field should be followed to keep the disease under check.
- With the initiation of the disease, spray the crop with fungicides like carbendazim (0.1%) or benomyl (0.1%) or mancozeb (0.25%) or chlorothalonil (0.2%) and repeat at 10 to 14 days interval.

3. Powdery mildew

Powdery mildew is an important disease of this crop and under favourable weather condition causes significant yield reductions particularly if the infection takes place at in early stages of plant growth.

Symptoms:

- Symptoms first appear as minute discoloured patches with thin fine meshwork of white mycelium arising at many places on the upper surface of lower leaves (Plate-3).
- These white patches soon join together to form larger white greyish powdery coating discernible on the severely affected leaves and in later stages the affected leaves turn yellow and finally drop.

Plate-3. Symptoms of powdery mildew of okra

Pathogen:

- The disease is caused by *Erysiphe cichoracearum* DC.
- The conidia are single celled, hyaline, barrel-shaped and in long chains.
- The conidial dimensions vary with the physiologic race and the host.
- Cleistothecia are globose, dark with hyaline to dark brown and mycelioid appendages.

- They contain 8 to 18 asci and the asci are pedicellate, ovate to broadly ovate or ellipsoid.
- The number of ascospores per ascus is usually two, rarely three. The ascospores are one celled and hyaline oval to sub-cylindrical.

Disease cycle and epidemiology:

- The primary infection of the leaves is caused by wind blown ascospores (wherever cleistothecia are present) or by conidia formed on the earlier sown crop/ indoor cultivation in the neighbouring areas.
- In the process of infection, conidia germinate and after penetration, colonization of the entire leaf takes place.
- The conidia formed abundantly on primary infections are blown by wind and air currents and cause secondary infections and the cycle is repeated.
- The pathogen requires 60-80 per cent relative humidity for the development of the disease and dry conditions for the growth and sporulation of the fungus.

Management

With the initiation of the disease spray the crop with fungicides like wettable sulphur (0.25%) or dinocap (0.05%) or carbendazim (0.05%) or hexaconazole (0.05%) or difenoconazole (0.03%) and repeat at 10 to 14 days interval.

Other Important Diseases

Root rot by *Macrophomina* and *Rhizoctonia solani*

B) VIRAL DISEASES

4. Yellow vein mosaic (YVM)

Symptoms:

- The characteristic symptoms include vein yellowing and thickening of leaves forming a net work of veins and veinlets in the infected leaves (Plate-4).
- Initially the leaves exhibit only yellow coloured veins but under severe infection, the leaves become completely chlorotic and turn yellow.
- The chlorophyll content of the leaves is reduced. Infected plants produce very few small sized leaves, deformed and give a stunted look.
- Affected plants produce very few small sized pale fruits.

Plate-4. Symptoms of yellow vein mosaic in okra

Virus

- The disease is caused by Yellow Vein Mosaic Virus (YVMV) and belongs to the Bigeminivirus.
- The size of virus is 18 x 30 nm.
- Virus particles are spherical, isometric measuring 28-30 nm.

Transmission

- Virus is not sap transmissible but under artificial conditions, it can be transmitted by grafting.
- In nature, virus is transmitted by white fly *Bemisia tabaci*.

Disease cycle and epidemiology:

- The virus is not sap transmissible but under artificial conditions it can be transmitted by grafting. In nature, the virus is transmitted by insect vector, white fly (*Bemisia tabaci* Genn.) in a persistent manner.
- Both wild and cultivated plants serve as the source of inoculum and the incidence of the disease in a particular area depends upon the prevalence of wild or cultivated host plants, population buildup of the vectors and environmental conditions.
- Dry hot weather with little or no rainfall was conducive for disease development and also for the multiplication of the vector population

Management:

- Various cultural practices like destruction of wild hosts from in and around the field, avoidance of mixed cropping of pumpkin , adjustment of date of sowing to avoid the

period of maximum population of the whiteflies and regular removal of affected plants upto 55 days help in reducing the incidence of the disease.

- Application of yellow coloured polythene mulch significantly delays the appearance of the disease.
- Use resistant cultivars like Punjab Padmini, Punjab-8, Prabhani Kranti and Hissar Unnat.
- Four sprays of Metasystox (demeton-S-methyl) at 15, 30, 45 and 60 days after sowing have been reported effective in controlling the insect vectors and keeping the disease under check.

Lecture - 7

FUNGAL DISEASES OF CABBAGE, CAULIFLOWER, RADISH AND KNOL-KHOL -I

Cruciferous vegetables are important Kharif vegetable crops, which are grown both for table and seed purposes. This vegetable group constitutes crops like cauliflower, cabbage, radish, turnip and knol-khol. These crops are grown throughout the country and are attacked by number of diseases which not only reduce the quantity but also quality of the produce. The diseases, which are of common occurrence, are described in detail in this lecture.

Aim: To know about different fungal diseases infecting cabbage, cauliflower, radish and knol-khol and their management – Part I.

A) FUNGAL DISEASES (PART – I)

1. CLUB ROOT

This disease is also known as finger and toe disease and was first discovered by Woronin in Russia. This is a very serious disease of cauliflower, cabbage, broccoli and Chinese cabbage and in India it is common only in Darjeeling.

Symptoms:

- The disease initiates from the roots and first symptom on the above ground plant parts appear as epinasty on hot sunny days followed by yellowing of leaves and reduction in the vigour of the plants.
- The underground roots of such plants are hypertrophied forming clubs of different shape and sizes depending upon the infection sites.
- The malformation generally occurs in primary, secondary and tertiary roots and sometimes extends to hypocotyls.
- Infected plants wilt in direct sunlight.
- The clubs are easily invaded by the secondary saprophytic organisms, causing rotting of the roots.

Pathogen:

- The fungus responsible for this disease is *Plasmodiophora brassicae* Wor.
- The incitant is biotroph, endocellular and plasmodial fungus.
- The life cycle involves production of two different plasmodial phases i.e. sporangial plasmodium which gives rise to thin walled sporangia and sporogenic plasmodium which produces thick walled, hyaline and spherical resting spores.
- The planospores are anteriorly biflagellated and uninucleated.
- Plasmodia and secondary sporangia may be seen in root hairs and epidermal cells.

- The resting sporangia are spherical, uninucleate and minutely spiny.

Disease cycle and epidemiology:

- The fungus can survive in soil for several years in the form of resting spores which are disseminated by water and other cultural operations.
- Seedlings raised in infested soil also help in spreading of the pathogen to uninfested fields.
- The fungus can also perennates on wild cruciferous plants.
- The pathogen enters the host through the root hairs and injured roots.
- After entry, the invaded host cells enlarge and the fungus multiplies to develop into plasmodium.
- As a result of cell division, the fungal spores are distributed to the newly formed cells which give the club shaped appearance to the root tissues.
- Such infected roots decay and release the spores in the soil.
- Soil temperature ranging from 9-30^{o} C, moisture and pH play an important role in disease development and its progress.
- Cool and wet climate along with acidic soils (pH 5.0- 6.3) are most favourable for development of the disease.

Management:

- Destruction of diseased debris and wild hosts from in and around the field helps in reduction of primary inoculum.
- Amend the infected soil with lime to increase the soil pH to 7.2 and immediate irrigation is must.
- Use disease free seedlings for planting in healthy areas.
- Soil solarization has also been found effective in reducing the inoculum.
- Use resistant cultivars if available.
- Soil drenching or dipping of the seedlings in benomyl (0.05%) or carbendazim (0.05%) solution for 15-20 minutes before transplanting helps in reducing the disease incidence.
- Besides these, combination of calcium cyanamide with nitrogen and boron completely inhibits clubbing and root hair invasion by suppressing the resting spore germination.
- Biocontrol agents like *Pseudomonas fluorescens* and *Streptomyces graminofaciens* helps in reducing size and growth of club as well as root infection.

2. DOWNY MILDEW

It is widespread in those regions of the world, which have cool and wet climate.

Symptoms:

- The first symptoms of the disease are evident as small brown spots on the lower surfaces of the leaves.
- The spots are covered gradually with the downy growth consisting of conidiophores and conidia of the fungus (Plate-1a).
- Upper surface of the leaves corresponding to spots turns light yellowish in colour (Plate-1b).
- Young leaves having a number of such spots turn yellow and drop off early whereas older leaves usually persist.
- The deformities are also seen on inflorescence where flowering stalk thickens and becomes elongated and curved.
- Black sunken spots are also produced on cauliflower and broccoli curds as well as on cabbage heads.

Plate-1a. Symptoms of downy mildew on seedlings

Plate-1b. Symptoms of downy mildew on leaves

Pathogen:

- The fungus responsible for this disease is *Peronospora parasitica* (Pers.) Fr. which is an obligate parasite.
- The mycelium of the fungus is strictly intercellular with large, finger-shaped intracellular haustoria which become clavate and branched and nearly fill the cell cavity.
- The sporangiophores are produced during darkness, which are divided into primary and secondary branches (dichotomously branched), which ultimately bifurcate to form sterigmata which bear the single sporangium at the tip.
- The sporangia are broadly oval, ellipsoidal and hyaline and they fall off mainly by hygroscopic twisting of the sporangiophores in response to change in atmospheric humidity.
- The oospores are globose measuring 26 to 43 µm in diameter.
- They are enclosed in crest like folds and appear pale yellow in colour and germinate by germ tube.

Disease cycle and epidemiology

- The downy mildew pathogen perennates as oospores in senescence host tissues and *Albugo candida* induced malformed inflorescence (Fig.1.).
- It also survives as conidia on leaves and inflorescence and as latent systemic mycelium in seeds or infected plant debris.
- Infections are favoured by low temperatures and high atmospheric humidity following rain or dew.
- The penetration is usually direct but occasionally also occurs through stomata.

- Primary infection occurs due to soil borne oospores while conidia released from conidiophores found on the cotyledons or hypocotyls favour secondary spread.
- Water droplets also help the pathogen dispersal over short distances.
- The optimum temperature for conidial germination is 8 – 12 ^{o} C while penetration of the host by infection hyphae and formation of the haustoria occurred most rapidly at 16^{o} and 20-24^{o}C, respectively.

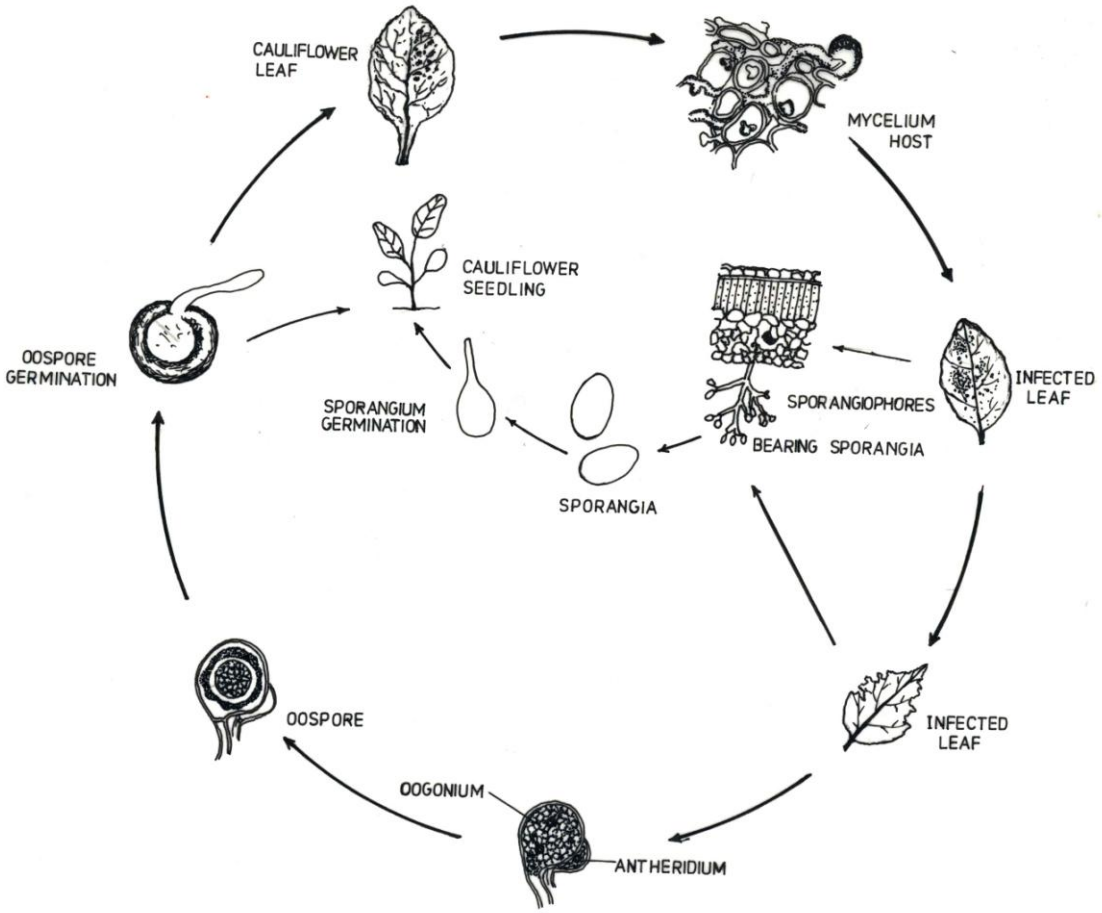


Fig. 1. Disease cycle of downy mildew of crucifers

Management:

- Collect and destroy the infected plant debris and perennial weed hosts.
- Crop rotation with non cruciferous plants.
- Use disease free seeds. Heat treatment of seeds at 43 – 50^{o} C for 20 minutes and treating them with Apron metalaxyl + mancozeb (0.3%) is also effective.
- Spray the crop with metalaxyl + mancozeb (0.25%) or mancozeb (0.25%) or copper oxychloride (0.3%) and repeat at 10 to 14 days interval.

3. ALTERNARIA LEAF SPOTS

Alternaria diseases are the most common and serious menace to the cultivation of crucifer vegetables world over. Infection on young leaves, stems and siliquae generally results in heavy yield losses as well as quality of seed.

Symptoms:

- The leaf spots incited by *A. brassicicola* appear as minute dark brown to black spots, which may enlarge by forming concentric rings (Plate-2a) and each spot is surrounded by a yellow halo of chlorotic tissues.
- In humid weather, the fungus appears as a bluish growth in the centre of the spots.
- The spots caused by *A. brassicae* are similar to those described above but they tend to remain smaller i.e < 1 cm and light brown in colour.
- The spots caused by *A . raphani* are slightly yellowish.
- Browning of cauliflower/ broccoli heads occur which generally begin at the margin of the individual flower or flower cluster.
- Plants grown for seeds show dark necrotic elongated lesions on the main axis, inflorescence, branches and on the siliquae.
- Infected seeds remain small in size, shrunken, discoloured or covered with fungal growth and have low viability.

Plate-2a. Symptoms of Alternaria leaf spot of crucifers

- Turnip and rutabaga foliage and roots may also become infected in field but root symptoms develop after being placed in storage.
- The leaf spots are nearly circular, often zonate and are of various shades of brown to black. Radish plants kept for seed purpose are severely affected by *A. raphani*.
- On leaf, the spots are raised, spherical to elliptical and 0.5-1 cm in diameter, black sporulation may also appear on the lesions in the humid weather, the center soon dries and may drop down.

Pathogen (s): Three species of *Alternaria* are responsible for this disease: *A. brassicicola* (Schw.) Wiltshire, *A. brassicae* (Berk.) Sacc. and *A. raphani* Groves & Skolko.

A. brassicae:

- Attack most of the crucifer vegetables and produce conidia of bigger size with larger beak.
- Mycelium is immersed, hyphae branched, septate, hyaline.
- Conidiophores are simple, erect, bearing one to several small but distinct conidial scars.
- Conidia are solitary or occasionally in chains of up to 4, obclavate, rostrate with 16-19 transverse septa and 0-8 longitudinal or oblique septa, pale or greyish olive, the beak about 1/3 to 1/2 the length of the conidium.

A. brassicicola*:*

- Produces smaller conidia with shorter beak.
- Mycelium immersed, hyphae branched septate, hyaline at first, later brown or olivaceous brown.
- Conidiophores arising singly or in groups of 2-12 or more, emerging through stomata, usually simple and erect Conidia mostly in chains of up to 20 or more, nearly cylindrical, usually tapering slightly towards the apex or obclavate, the basal cell rounded, the beak usually almost non-existent, mostly less than 6, transverse septa, often constricted at the septa, pale to dark olivaceous brown, smooth or becoming slightly warted with age.

Plate-2b. Conidia of *A. brassicicola*

***A. raphani*:**

- Usually attacks radish
- Conidiophores are simple or occasionally branched, septate and olivaceous brown in colour.
- Conidia are formed commonly in chains of 2-3, straight or slightly curved, obclavate or ellipsoidal, generally with a short beak, mid to dark golden brown or olivaceous

brown in colour, smooth or sometimes minutely verruculose, with 3-7 transverse and often a number of longitudinal or oblique septa.

Disease cycle and epidemiology:

- The pathogen perennates on infected plant debris, in or on infected seeds and cruciferous weeds.
- Moderate temperature (21- 27^{o} C), high relative humidity (95-100 % at least for 18h) and splashing rains favour the disease development and spread.
- The optimum temperature for sporulation is 18-24 and 20-30^{O}C for *A. brassicae* and *A. brassicicola*, respectively, at which both fungi produce spores in 12-14 h.
- At temperature >24^{O}C, sporulation of *A. brassicae* is inhibited.
- *A. raphani* infection progresses rapidly at 22-26^{O}C.

Management:

- Collect and destroy the infected plant debris. Use disease free seeds and treat them with captan (0.3%).
- Spray the crop with mancozeb (0.25%) or copper oxychloride (0.3%) and repeat at 10 to 14 days interval.

Lecture – 8

FUNGAL DISEASES OF CABBAGE, CAULIFLOWER, RADISH AND KNOL-KHOL -II

Cruciferous vegetables are important Kharif vegetable crops, which are grown both for table and seed purposes. This vegetable group constitutes crops like cauliflower, cabbage, radish, turnip and knol-khol. These crops are grown throughout the country and are attacked by number of diseases which not only reduce the quantity but also quality of the produce. The diseases, which are of common occurrence, are described in detail in this lecture.

Aim: To know about different fungal diseases infecting cabbage, cauliflower, radish and knol-khol and their management – Part II.

A) FUNGAL DISEASES (PART – II)

4. STALK ROT

This is an important disease of cauliflower seed crop (late varieties). Once this disease gets introduced in a field, it is very difficult to raise a healthy crop in the same field without taking appropriate control measures.

Symptoms:

- The symptoms of the disease start appearing with the earthing up of plants.
- The infection starts from the lower most leaf petiole touching the ground.
- The infected leaves lose their turgor during day time and droop down to the ground, but regain turgidity during night or early morning.
- The yellowing starts from tips of the older leaves downwards which shed, prematurely.
- In most of the cases, mid-rib and petioles at a point touching the soil, show small, discrete to large irregular dark brown to black necrotic lesions (Plate-3a).
- The lesions are covered with fluffy growth of extrametrical fungus under cool and humid weather.
- Rotting from petioles advances to the stalk where dark brown to black spots are produced which girdling whole of the stem at ground level.
- The stalk rot progresses towards the curd and occasionally whole of the pith portion up to the forks of curd branching get completely rotten (Plate-3b).

- The pith and the curd also rot, giving way to large cavities lined inside with fluffy mycelium and numerous sclerotia of the causal fungus.
- Under cool humid conditions, the mycelium emerges out and can be seen sticking to affected portion of the plant.
- With the progress of the disease, curds are also affected and show brown to dark brown rotting which may start from any portion of the curd, but generally from the center.
- The affected tissue becomes soft and mushy bearing numerous sclerotia.
- Inflorescence is also affected during the months of April-May in hilly areas.
- If there are plenty of rains during bolting and seed setting, the fungus progresses fast and engulfs whole of the branches and inflorescence where mycelium can be seen hanging out with sclerotia sticking to it.
- However, if the weather is dry, the mycelial growth is restricted only up to branches. affected branches become dry and bear shriveled seeds.

Plate-3a- On petioles **Plate-3b. On stalk with sclerotia**

Plate-3. Symptoms of Stalk rot of cauliflower

Pathogen:

- The disease is caused by *Sclerotinia sclerotiorum* (Lib.) de Bary.
- Mycelium in culture as well as on host surface is white, cottony, branched, consisting of closely septate hyphae.
- Micro-conidia are produced on short lateral branches of the vegetative mycelium in chains.
- When the food supply is exhausted and the vegetative growth ceases, the hyphae with granular protoplasm collect in small dense masses and form sclerotia.
- These sclerotia are first white in colour, then turn black and are round to irregular in shape.

Disease cycle and epidemiology:

- The fungus overwinters in the form of sclerotia in soil as well as in the diseased plant debris (Fig.2).
- In the presence of proper humidity and light conditions, sclerotia germinate by forming apothecia, which in turn form asci and ascospores.
- If the spores, upon escaping from the ascus, lodge on a susceptible host, a new infection may originate.
- Mycelium from sclerotia is also capable of infecting cauliflower.
- During cool and moist weather fluffy mycelium appears on the host surface and when food is exhausted, the mycelium coagulates and starts forming sclerotia.
- The fungus can infect the host at a temperature ranging from 0 to 25 ^{O}C with an optimum at 15 to 20 ^{O}C and high humidity (95-100%) also favours the disease development.
- Application of nitrogen aggravates the disease.

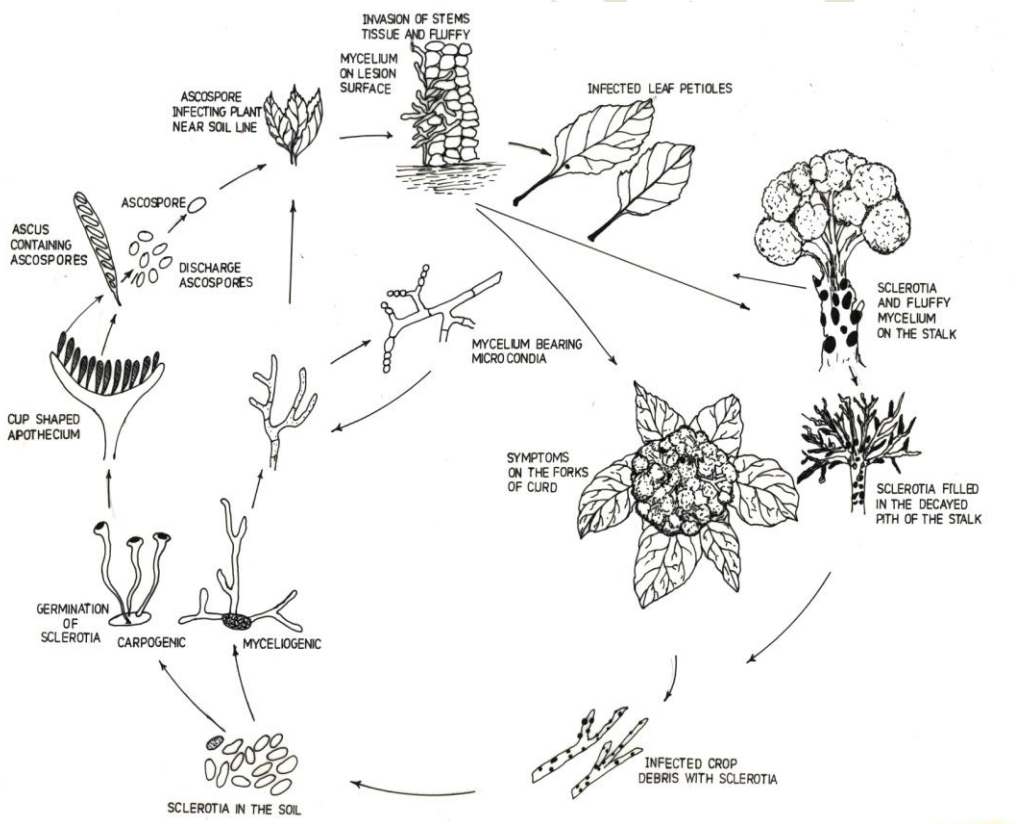


Fig. 2. Disease cycle of stalk rot (*Sclerotinia sclerotiorum*) of cauliflower

Management:

- Follow long crop rotations with paddy and maize.
- Remove infected leaves at weekly intervals.

- Soil amendment with oil cakes like sunflower and mustard and mulching with pine needles and sunflower inflorescence also reduces the disease incidence.
- Some antagonistic fungi like *Trichoderma harzianum, T. viride, Gliocladium virens* and *Coniothyrium minitans* have been found promising in managing this disease.
- These fungi either inhibit the development of new sclerotia or destroy the developed ones by colonizing them.
- In chemical control, sprays of fungicides like carbendazim (0.1%) or thiophenate methyl (0.1%) or mancozeb (0.25%) or combination of mancozeb (0.25%) and carbendazim (0.05%) effective in reducing the disease.
- Initiate sprays immediately after earthing up and repeat at fortnightly intervals.

5. WHITE RUST OR BLISTER

It is the most common disease of radish and other crucifers and cause considerable losses when it occurs along with downy mildew.

Symptoms:

- Two types of infection *i. e*. local or leaf phase and the systemic or stag head phase occur in different crucifers.
- Local infection results in isolated pustules on leaves and stems which show characteristic raised, glossy white blisters 1 to 2 mm in diameter.
- Systemic infection stimulates hypertrophy and hyperplasia resulting in enlarged and variously distorted organs (Plate-4).
- The inflorescence may become enormously thickened, fleshy and greenish leading to sterility.
- The thickened inflorescence is generally known as stag head.
- Blisters after rupturing the host epidermis release white powdery mass comprising of sporangia.

Plate-4. Symptoms of white rust of crucifers

Pathogen:

- The disease is cuased by *Albugo candida* (Pers. Ex Chev.) Kuntze.
- It is an obligate parasite reproducing both sexually and asexually.
- Mycelium is intercellular and non-septate.
- Numerous short conidiophores arise from the mycelium, which are arranged in rows beneath the host epidermal layer press on the epidermis to cause the pustule/ blister.
- Inside the pustules, sporangia are produced in chains arising from the sporangiophores at the base of the cavity.
- The sporangia are hyaline, spherical, thick walled, hyaline and interconnected by a pad of gelatinous disc like tissue.
- In the presence of moisture, these pads swell and disintegrate, freeing the sporangia in the chains and they germinate either by germtube or by producing zoospores.
- The oospore is spherical with a thick irregular wall, deep yellow to dark brown in colour.

Disease cycle and epidemiology:

- The fungus survives either as oospores in the infected plant debris or mixed with seeds or as perennial mycelium in some weed hosts (Fig.3).
- These oospores may germinate in the ensuing crop season to produce zoospores, which serve as primary inoculum.

- Secondary spread is through sporangia produced on the spots caused by primary inoculum.
- Moisture is essential for germination of sporangia through zoospores.
- The optimum temperature for germination of sporangia by zoospores is 20^{o} C.

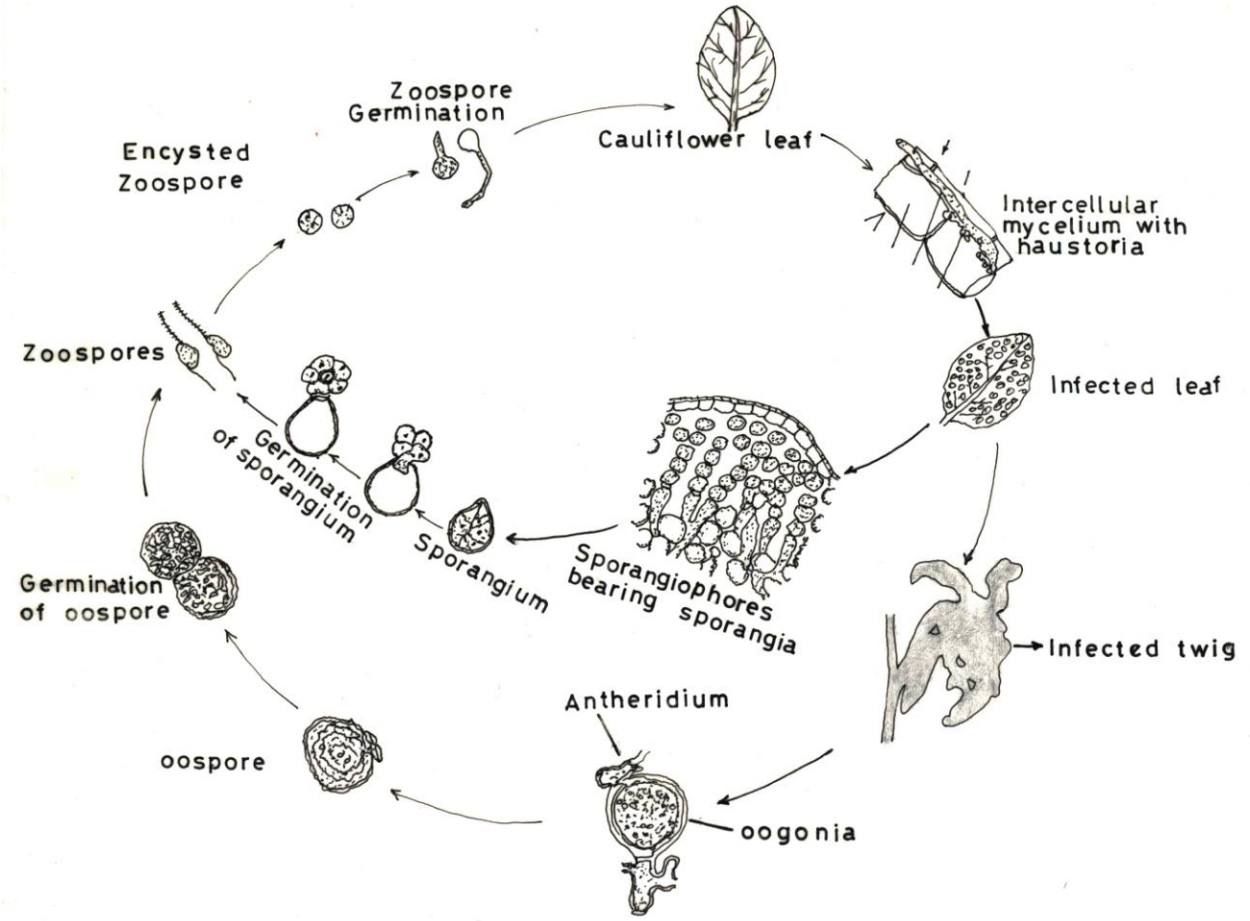


Fig. 3. Disease cycle of white rust of crucifers

Management:

- Practices like collection and burning of diseased plant material in order to prevent oospore formation, reduction of humid conditions around the plants by maintaining proper spacing, crucifer weed free cultivation, application of fertilizers like phosphorus, potassium and avoidance of excess application of nitrogen keep the disease under check.
- With the initiation of the disease, spray the crop with metalaxyl + mancozeb (0.25%) followed by sprays of Bordeaux mixture (4:4:50) or mancozeb (0.25%) or copper oxychloride (0.30%) and repeat at 10 to 14 days interval.

Lecture – 9

BACTERIAL AND VIRAL DISEASES OF CABBAGE, CAULIFLOWER, RADISH AND KNOL-KHOL

Cruciferous vegetables are important Kharif vegetable crops, which are grown both for table and seed purposes. This vegetable group constitutes crops like cauliflower, cabbage, radish, turnip and knol-khol. These crops are grown throughout the country and are attacked by number of diseases which not only reduce the quantity but also quality of the produce. The diseases, which are of common occurrence, are described in detail in this lecture.

Aim: To know about different bacterial and viral diseases infecting cabbage, cauliflower, radish and knol-khol and their management.

A) BACTERIAL DISEASES

1. BLACK ROT

Symptoms:

- Initial symptoms of the disease appear as chlorotic lesions along the margins of leaves which progress in the direction of midrib forming "V"-shaped lesions (Plate-5).
- The veins and veinlets in the chlorotic area turn black and with the passage of time, the blackening of veins advances to the stem and from there to other leaves and roots.
- The stem and stalk of infected leaves show blackening of vascular tissues.
- Due to the systemic infection, black spots appear on flower stalk and siliques.
- The heads of cabbage and curds of cauliflower are also invaded and become discoloured. The roots of radish and turnip are also invaded from leaves which show discolouration and internal breakdown.
- The infection of this pathogen may be followed by attacks of soft rot bacteria or *Sclerotinia sclerotiorum.*

Plate-5. "V" Shape lesions of black rot of crucifers

Pathogen:

- The disease is caused by *Xanthomonas campestris* pv. *campestris* (Pammel) Dowson.
- It is a small, rod shaped, aerobic, gram negative, non-spore forming bacterium.
- The bacterium has a single polar flagellum (monotrichos) and it is catalase positive, hydrogen sulphide positive, oxidase negative and does not produce nitrate or indole.
- It produces a yellowish extracellular polysaccharide (EPS) called Xanthan on media containing glucose.
- *X. c.* pv. *campestris* hydrolyses starch, a characteristic that is used for easy recognition of the bacterium on Schaad's selective medium.
- Its growth is inhibited or retarded in acidic range of pH.

Disease cycle and epidemiology:

- The pathogen survives in infected seeds, diseased plant debris and on cruciferous weeds (Fig.4.).
- When the infected seed germinates, the seed coat that comes out along with the cotyledons serves as the source of primary inoculum.
- The bacterium enters cotyledons through the marginal stomata and passes into true leaves through hydathodes.
- On entry, the bacteria move to the xylem vessels where they multiply rapidly and move up and down in the plant.
- When the disease inoculum is present in the field, secondary spread is facilitated by splashing of contaminated soil by irrigation water and/ or rains, blowing of fallen leaves and occasionally by insects.
- The optimum temperature for the disease development is about 26.5 to 30^{o}C, minimum being 5^{o}C and maximum 36^{o}C.
- Heavy rains have been reported to be responsible for the fast spread of the pathogen and the disease through splashes.

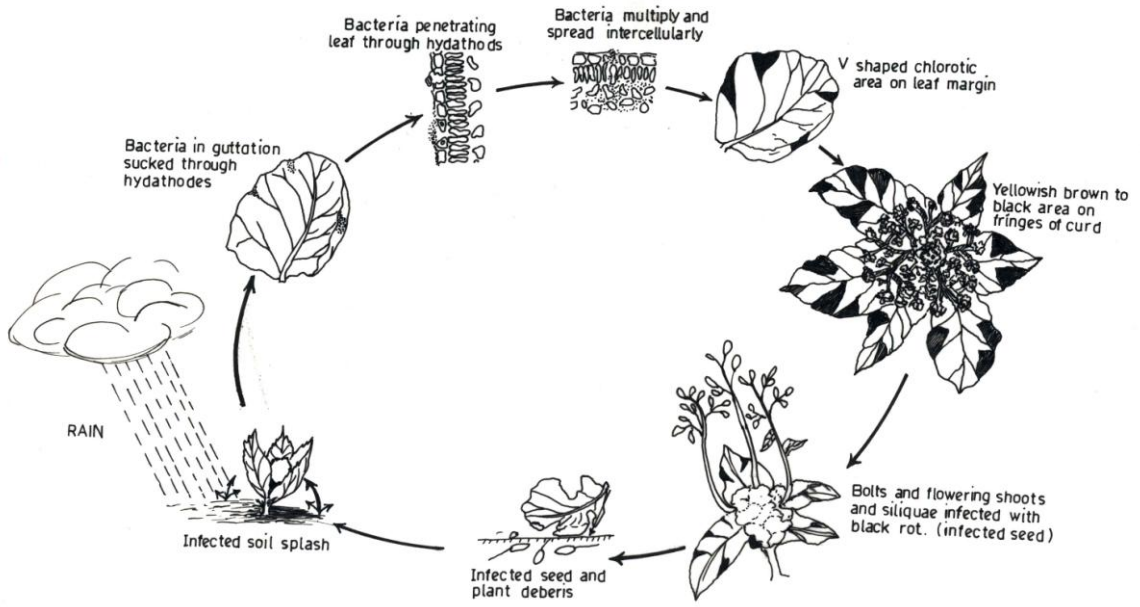


Fig. 4. Disease cycle of black rot (*X. campestris* pv. *campestris*) of crucifers

Management:

- Follow crop rotation with non-cruciferous crops for at least two years.
- Use disease free seeds and treat them either with hot water (52^{o} C) for 30 minutes followed by same duration dip in Streptocycline solution (100 ppm) or by dipping the seeds in Streptocycline (100 ppm) solution for 30 minutes to eradicate the pathogen.
- In disease prone areas, apply grass or pine needle mulch on the field floor.
- With the initiation of the disease, give fortnightly sprays of combination of Streptocycline (100 ppm) and copper oxyxhloride(0.3%) or mancozeb (0.25%).

B) VIRAL DISEASES

1. CAULIFLOWER MOSAIC

Symptoms:

- Vein clearing or banding symptoms are produced on the leaves ultimately leading to mosaic pattern (Plate-6).
- Only the young leaves are affected initially in which light green patches appear.
- Early infection may cause reduction in plant growth leading to stunted look.

Plate-6. Symptoms of cauliflower mosaic

Pathogen:

- The disease is caused by Cauliflower Mosaic Virus which belongs to caulimovirus (CaMV) group.
- Virions are isometric, 50 nm in dia. and non-enveloped and the genome consists of unipartite double stranded DNA.

Disease cycle and epidemiology:

- The virus is transmitted by mechanical inoculation as well as by different species of aphids particularly *Brevicoryne brassicae* and *Myzus persicae* in semi-persistent manner.
- Besides cabbage and cauliflower, the virus has been reported to infect other *Brassica* spp. under natural conditions.

Management:

Control of aphid vectors through the use of insecticides like Metasystox (0.1%) at an appropriate stage is helpful in reducing the spread of the disease.

Other disease of minor importance are:

i. **Yellows :** *Fusarium oxysporum* Schlech: Fr f.sp. *conglutinans* (Wollenw.) Snyder & Han
ii. **Wire stem** : *Rhizoctonia solani* Kuhn
iii. **Ring spot :** *Mycosphaerella brassicicola* (Fries ex Duby) Lindau
iv. **Powdery mildew :***Erysiphe polygoni* DC.
v. **Leaf spots :** *Cercospora brassicicola* Hennings; *C. cruciferarum* Ellis and Everhart
vi. **Soft rot or Curd rot :** *Erwinia caratovora* subsp. *carotovora* (Jones) Bergey *et al*.

Lecture - 10

DISEASES OF PEA

Pea (*Pisum sativum* L.) belonging to Leguminosae family includes both ornamental and edible forms –the garden pea and field pea. It is believed to be native of Europe and West Asia, while its wild prototype probably came from Ethiopia. It is one of the most important legume crops and is grown over a greater part of the world. During cultivation, the crop is severely affected by various diseases which not only reduce the yields but also affect the quality of the produce. The present lecture deals with the fungal, bacterial and viral diseases affecting this crop including their management.

Aim: To know about different diseases infecting pea and their management.

A) FUNGAL DISEASES

1. POWDERY MILDEW

Powdery mildew is one of the most common and serious diseases of pea which is prevalent throughout the world. In India this disease was first time reported by Butler (1918) from Dehradun.

Symptoms:

- Initial symptoms appear as minute discoloured patches with thin fine meshwork of white mycelium arising at many places on the upper surface of lower leaves (Plate-1) and these white patches soon join together to form larger whitish floury areas.
- In severe cases of disease development, both upper and lower surfaces of the leaf are infected.
- The conidia and condidiophores are produced in such profusion that the foliage looks as if dusted with flour and the whole crop in the field appeared white from a distance.
- With the progress of the disease, similar symptoms are also noticed on tendrils, petioles and stems.
- Pods are infected in all the developmental stages, but immature pods are more prone to rapid infection.
- The affected pods show white floury patches consisting of white powdery mass and these patches then turn light brown and finally dark brown in colour and later become necrotic.
- Such infected pods are either unmarketable or fetch minimum price in the open market.

Plate-1. Symptoms of pea powdery mildew

Pathogen:

- The disease is caused by *Erysiphe pisi* DC.
- The fungus is ectoparasitic, spreading on the surface of the host and sending haustoria into the epidermal cells to draw out nourishment.
- The fungus hyphae are hyaline and septate and conidiophores arise from it vertically bearing conidia either singly or in short chains (Plate-2).
- Conidia are ellipsoid to ovoid, mature conidia fall off and get dispersed by wind.
- Later in the season, cleistothecia (sexual stage) appear as dark coloured, round, minute bodies scattered in the mycelial web.
- The peridium is composed of distinct polygonal cells and is provided with a number of myceloid appendages.
- Each cleistothecium contains usually 2-8 asci and these asci originate from a single point in the fruiting body, appearing in a fanlike manner.
- They are ovate, nearly sessile and 46-72 x 30-45 µ in size.
- The asci contain 2-8 ascospores, which are elliptical, hyaline and unicellular.

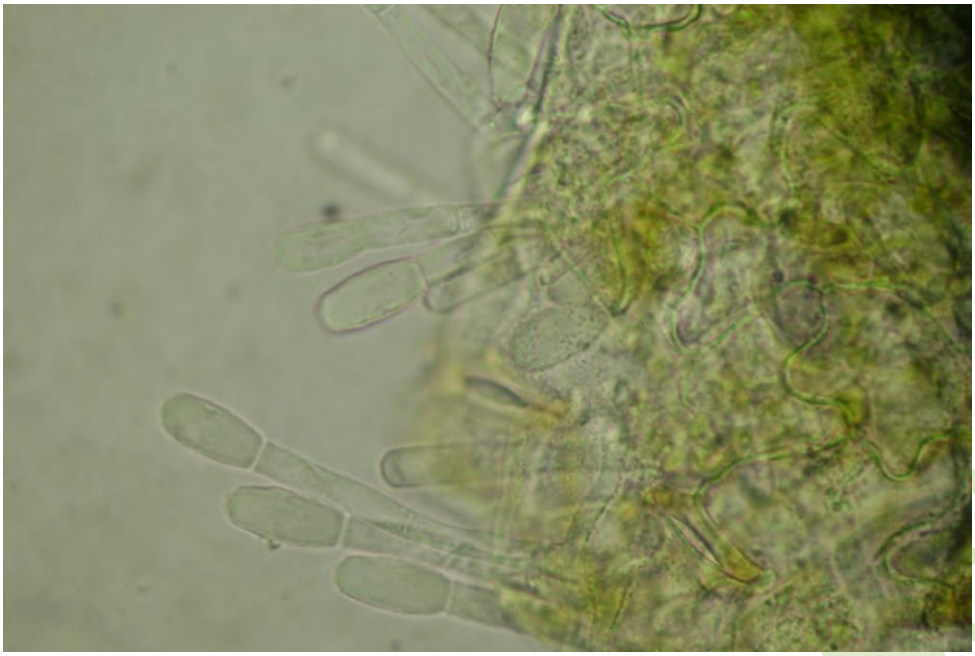


Plate-2. Conidiophore and conidia of *Erysiphe pisi*.

Disease cycle and epidemiology:

- In areas, where, cleistothecia are formed, ascospores play an important role as a source of primary inoculums (Fig.1).
- However, in other areas, the pathogen overwinters in conidial stage on different collateral hosts of the pathogen like *Pisum arvense, Lupinus, Medicago, Lathyrus aphaca, L. culinaris, Medicago hispida, Melilotus inica*, *Vicia sativa, V. hirsute* and *Aeschynomene indica*.
- There are reports that the pathogen survives in seed as dormant mycelium but the concept of seed borne nature of the disease could never be confirmed.
- Ascospores or conidia germinate by producing a germ tube which produces an appresorium on its tip and continues growing ectophytically.
- Each conidia of the fungus have 52 per cent moisture and germinate easily under dry conditions, so they are called as "dry weather fungus".
- Conidiophores bearing conidia are produced by the growing mycelium.
- Conidia are passively dispersed and cause secondary spread of the disease.
- The disease is more prevalent in dry weather and moderate temperatures.
- A temperature range of 10-30^{O}C is favourable for conidial germination with optimum being at 20^{O}C.
- A fairly dry soil and heavy application of nitrogenous fertilizers increase the disease incidence.

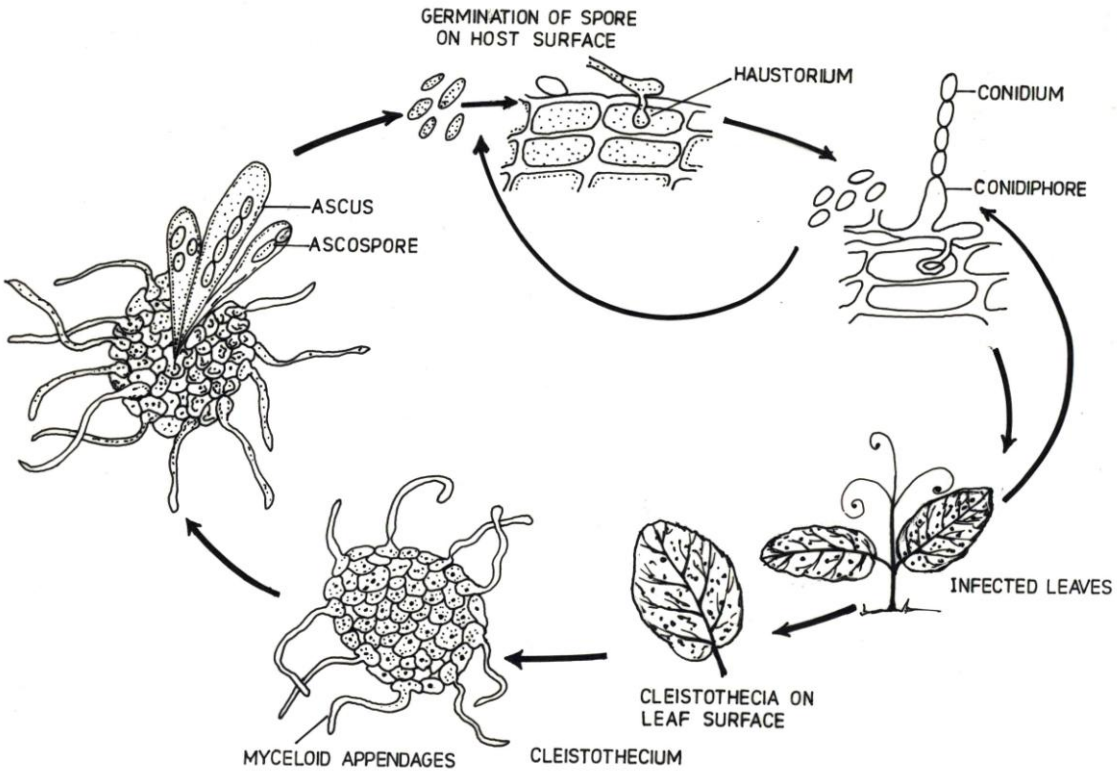


Fig. 1. Disease cycle of pea powdery mildew

Management:

- Collect and destroy the plant debris.
- Remove all the collateral hosts from in and around the field.
- Early sown (Late September to early October) crops escape the disease.
- A large number of cvs. /lines possessing resistance against this disease have been reported which can be used for sowing or in breeding programme for developing resistant varieties (Solan nirog, Rachna etc.).
- Use of biocontrol agent like *Ampelomyces quisqualis* serve as a best alternative to combat with this pathogen.
- With the initiation of flowering, spray the crop with wettable sulphur (0.20 %) dinocap (0.05 %) or carbendazim (0.05%) or hexaconazole (0.05%) or triadimefon (0.1%) or fenarimol (0.04%) or difenoconazole (0.03%) and repeat at 10-14 days interval.

2. FUSARIUM WILT

This is an important disease prevalent in those areas where pea is cultivated in autumn. In northern plains of India, wilt is a serious problem in the early sown (September planted) pea crop.

Symptoms: Symptoms of the diseases appear as true wilt and near wilt which are described below:

True wilt:

- Initially, the lower leaves look pale yellow and droop downwards (Plate-3).
- With the progress of the disease, similar symptoms appear on the upper leaves also and the affected plants give a stunted look.
- Slowly the upper plant parts loose their turgidity and the whole plants topple down.
- Stems are shriveled and some of the root lets are also injured.
- In diseased plants, yellow to orange brown discolouration of vascular system of upper tap root extending up to the stem is evident.

Plate 3: True wilt

Near wilt:

- Symptoms are also similar to those described above except for their slow development.
- In this case, the vascular discolouration of the roots is typically brick red and usually extends up to the growing tip.
- Cortical decay is also observed in lower parts of the stem and upper tap root.

Pathogen:

- The disease is caused by *Fusarium oxysporum* Schlech. ex. Fr. f.sp. *pisi* (Hall) Snyd. & Hansen (race 1= wilt; race 2= near wilt).
- The hypha of the fungus is septate, delicate white to peach coloured, usually with a purple tinge.
- Micro-conidia are borne on simple phialides arising laterally on hyphae or from short less branched conidiophores and these are oval ellipsoid to cylindrical or curved.
- Macroconidia are borne on branched conidiophores or on the surface of sporodochia and these are thin walled, 3-5 septate, fusoid-subulate and pointed at both ends.
- Chlamydospores are both terminal and intercalary.

Disease cycle and epidemiology:

- The pathogen is soil borne and can survive in the soil for several years.
- The fungus causes infection of the fibrous roots or epicotyl region, grows both inter and intracellularly in the cortex and ultimately concentrates in the xylem vessels.
- After death of the plant, the fungus continues to grow and sporulate on the stem cortex, resulting in the production of soil borne inoculum.
- The disease progresses fast at 21^{o} C while near wilt requires 24-28^{o} C.
- True wilt is not serious in very wet soils while wet soils favour near wilt.
- Neutral to alkaline soils favour true wilt while the acidic soils retard it.

Management:

- Collect and destroy the infected plant debris.
- Follow long crop rotation in the infested fields.
- Use healthy seed and treat the seed with carbendazim (0.2%).
- Use resistant sources, if available. *Trichoderma* spp. were found to be antagonistic to this pathogen but their utility in disease management has not yet been ascertained.

3. RUST

Pea rust is an important disease, which occurs quite frequently in pea growing areas in North and North western parts of India. Two rusts have been reported on peas caused by *Uromyces pisi* (Pers.) de Bary and *Uromyces viciae-fabae* (Schroet) of which the latter is of worldwide occurrence.

Symptoms:

- The symptoms appear on all above ground parts of the plant.
- Infection is evident first as minute, raised pustules, which later become distinct, yellowish, circular sori (plate-4a).
- The yellow spots have aecia and persist for longer time.
- The uredo pustules develop on both surfaces of the leaves as well as on other parts and give light brown powdery appearance to the plants in case of severe infections.
- Late in the season, teleuto pustules also develop on the leaves but most commonly on stems and petioles (Plate-4b). They are dark brown or almost black in colour.

Plate-4a. Aecial stage

Plate -4b Telial stage

Plate-4. Symptoms of pea rust

Pathogen:

- The disease is caused by *Uromyces viciae fabae* (Grev.) Fuckel.
- On peas, the pycnia occur in small groups associated with the aecia.
- The aecia are cupulate and 0.3 to 0.4 mm in diameter.
- The peridium is short and whitish.
- The aeciospores are round to angular or elliptical with hyaline wall and the wall of these spores is verucose.
- The urediniospoes are round to ovate, light brown, echinulate, with 3-4 equatorial germ pores.
- The telia occur in the same sorus as the uredia are dark brown to black.
- The teliospores are subglobose, ovate or elliptic with rounded or flattened apex, which is considerably thickened and appears papillate, smooth and are brown in colour.

Disease cycle and epidemiology:

- It is an autoecious rust with all its spore stages on the same host.
- The rust is seed borne in broad beans but in peas and lentil, it is mainly survive as teliospores in crop debris.

- In India, the rust appears to survive on weed hosts belonging to *Lathyrus, Vicia* etc. and the spores are wind blown to the main crop.
- The teliospores on germination produce a promycelium on which sporidia are borne.
- The sporidia germinate and germ tube infects the host, produce pycnia and finally aecial cups are produced.
- Uredia occur late in the season and are not of much importance in secondary spread.
- Later in the season, teliospores are produced which help in overwintering of the pathogen.
- Prevalence of low temperature (17-22^{o} C) coupled with high humidity, dew or frequent light rain showers are favourable for initiation of the disease by aeciospores.
- Rust severity and pustules/plant increase progressively with an increase in the duration of leaf wetness up to 24 h, at optimum temperature of 20^{O}C.

Management:

- Collect and destroy infected plant debris.
- Follow long crop rotations avoiding broad beans, *Vicia* and *Lathyrus* in rotation.
- In disease prone, areas sow the crop early.
- With the initiation of the disease, spray the crop with mancozeb (0.25%) or hexaconazole (0.1%) or triadimefon (0.1%) or difenoconazole (0.05%) and repeat at 10 to 14 days interval depending on the severity of the disease.

4. WHITE ROT

Symptoms:

- The symptoms of the disease appear as water-soaked lesions, which may develop on any part of the plant, but occurs mainly on the stems or branches (Plate-5).
- At the point of infection, a dry, discoloured spot develops.
- Under cool and humid conditions, the mycelium emerges and can be seen sticking to affected portions.
- Pods are also infected and flesh start rotting and in the rotting tissues, a large number of sclerotia of the fungus can be seen.
- The diseased tissues become whitish and may be shredded.
- The seeds also become shriveled and discoloured. Sclerotia appear on affected pods also.

(Courtesy A.S. Kapoor)

Plate-5. Symptoms of white rot of pea

Pathogen:

- The disease is caused by *Sclerotinia sclerotiorum* (Lib.) de Bary. The pathogen has been described in detail in the lecture of cauliflower diseases.

Disease cycle and epidemiology:

- The pathogen mainly perpetuates through sclerotia for many years in soil.
- It is also same as in cauliflower diseases lecture.
- Sclerotia are primary source of infection and the fungus is polyphagus in nature.
- Optimum temperature for initiation of the disease is 15.5 – 21^{o} C and free moisture for 42-75 h is essential for establishment of infection and lesion expression.

Management:

- Collect and destroy the infected plant debris.
- Follow crop rotation with paddy or maize.
- Maintain wider row spacing.
- Use resistant sources if available.
- Apply *Trichoderma harzianum* formulation (120 kg/ ha) at the time of sowing.
- Apply foliar sprays of carbendazim (0.1%) at flowering stage and repeat at 10-14 days interval.

5. ASCOCHYTA BLIGHT OR FOOT ROT

Three morphologically distinct species of *Ascochyta* are reported to be associated with this disease which causes different types of symptoms including leaf and pod spot, blight and fruit rot.

Symptoms:

***Ascochyta* or *Mycosphaerella* blight:**

- This disease is characterized by brown to purplish, irregular areas on the foliage which later become circular and somewhat zonated.
- The small, brown to purplish irregular spots, which appear on the pods, enlarge to irregular purplish area or large area could become blotched with the coalescing of lesions (Plate-6).
- Developing seeds may be deformed, remain small in size and are sometimes stained.
- Black to purplish streaks appears on stem.
- In lesions, pycnidia appear in characteristic ring pattern.

(Courtsey: A.S. Kapoor)

Plate-6. Symptoms of Ascochyta or *Mycosphaerella* blight of pea

***Ascochyta* foot rot:**

- The symptoms are similar to those described for blight but in this case foot rot lesions predominate (plate-7).
- The dark brown lesions on the stem start soon after germination below the soil line and gradually extend down to roots and up the stem.

Plate-7. Symptoms of Ascochyta foot rot

***Ascochyta* leaf and pod spot:**

- Symptoms of this disease appear as definite, circular, sunken, tan to brown spots on leaves and pods while elongated on stems and petioles (Plate-8).
- A dark brown margin usually surrounds the tan spot and pycnidia may form on these spots also.
- Underlying seeds in the pods may also be affected and remain shriveled.

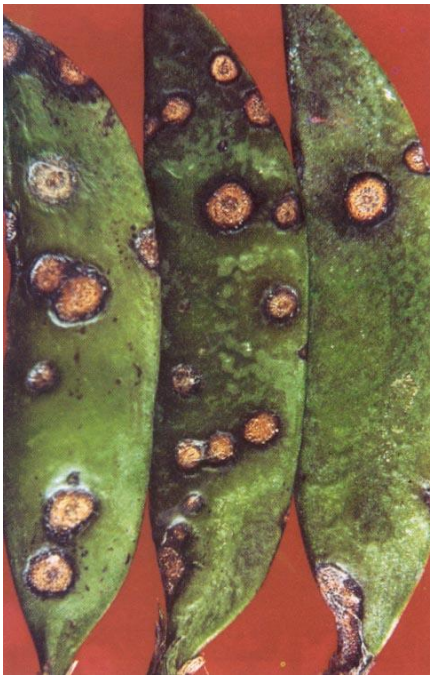


Plate-8. Symptoms of Ascochyta pod spot

Pathogen (s): Different *Ascochyta* species causes following diseases:

Blight	*Ascochyta pinodes* (Berk & Blox) Vestgrn. (perfect stage *Mycosphaerella pinodes* (Berk & Blox.) Vestergr),
Leaf and pod spot	*Ascochyta pisi* Lib.
Foot rot	*Phoma medicaginis* var. *pinodella* (Jones) Boerema (syn. *Ascochyta pinodella* Jones.).

Ascochyta pinodes:

- Perithecia are globose having beaked ostioles and contain bitunicate, cylindrical, clavate asci.
- Each ascus contains eight ascospores which are bicelled and hyaline.
- The fungus colonies are light to dark gray in colour and often have concentric rings.
- The conida have large number of globules.
- The spore ooze collectively appears buff coloured.

***Ascochyta pinodella*:**

- It produces single sometimes bicelled hyaline spores in pycnidia.
- This species is not known to produce perfect stage.
- In culture, fungus produces numerous chlamydospores singly or in chains.
- Colonies are dark gray and turn black at maturity.

***Ascochyta pisi*:**

- Conidia are longer and thin with slight constriction at septation.
- Colonies are light in colour and exude spore mass which is carrot red in colour.

Disease cycle and epidemiology:

- Different *Ascochyta* species perpetuates either in the seed or in diseased crop debris.
- In crop debris, these species survive as perithecia, sclerotia or chlamydospores.
- The pathogens may also survive through collateral hosts which are *Lathyrus* sp. and *Vicia* spp., for *A. pisi*; *Phaseolus* and *Lathyrus* for *A. pinodes* and *Vicia, Trifolium* and other legumes for *Phoma medicaginis* var. *pinodella*).
- Cool weather coupled with presence of sufficient moisture on the leaves is the most favourable conditions for disease development.
- The disease attains serious proportions during prolonged rainy periods.
- Atmospheric humidity >90 per cent helps in increasing the disease incidence while no infection occurs below 80 per cent RH.

Management:

- Use disease free seeds.
- Disease free seeds can only be produced if the crop is raised in low rainfall areas.
- The soil borne inoculum can be reduced by following long crop rotations and by destruction of the crop refuse by burning either in the field or after threshing.
- Use resistant sources, if available.
- Seed dressing with microbial cultures *of Trichoderma koningii and Gliocladium roseum* (4 g/kg seed) was also found effective in protecting the crop against *A. pisi*.
- Treat the seed with fungicides namely carbendazim (0.2%), benomyl (0.1%), thiabendazole (0.1%) + thiram (0.3%) and captan (0.3%).
- With the initiation of the disease spray the crop with fungicides like chlorothalonil (0.2%), carbendazim (0.05%) or combination of both and repeat at 10 days interval.

B) BACTERIAL DISEASES

6. BACTERIAL BLIGHT

It is an important disease of pea in those areas where cool and moist climate is prevalent during the growing season.

Symptoms:

- All aerial parts of the plants are infected by the disease.

- Symptoms of leaflets and stipules appear as small water soaked lesions usually developing into large irregular areas (Table-9).
- Lesions vary in colour, initially being dark green and water soaked, but generally becoming water soaked at the edge and a lighter shade of brown at centre.
- Under conditions of high moisture, chocolate brown linear streaks are observed on the stem and petiole.
- Later on, the whole stem turns chocolate brown and is shriveled leading to the death of the plants.
- Due to infection, immature pods become chocolate brown, thin, twisted and shriveled, lesions are bigger on older pods.
- Seeds developing in the pods are also discoloured and shriveled.

Table-9. Symptoms of bacterial blight of pea

Pathogen:

- The disease is caused by bacterium *Pseudomonas syringae* pv. *syringae* van Hall and *P. syringae* pv. *pisi* (Sackett) Young *et al*.
- The bacterium is gram -ve, non-spore forming and motile rods with one or more polar flagella.

Disease cycle and epidemiology:

- The pathogen overwinters in the infected seed.
- The seed carries the bacterium both externally and internally.

- It colonizes the intercellular and intracellular spaces of the seed coat but does not penetrate the embryo or cotyledons.
- Even a very low level of seed infection can cause economic loss, since the disease can spread fast from primary infection foci.
- The pathogen is carried in irrigation water, splashed by rain or blown in wet winds to other plants and field infection usually occurs through stomata and wounds.
- Cool, moist weather favours the disease while warm and dry weather retards it.
- The extent of disease spread depends upon the frequency of rainy periods.
- If soil is very wet at the time of emergence, it also favours blight transmission from the infected seed to resulting plant.

Management:

- Collect and destroy the infected plant debris.
- Follow at least three years crop rotation with non leguminous crops.
- Use disease free seed.
- Give pre-sowing seed dip treatment in Streptocycline (150 ppm) solution for 90 minutes.
- With the initiation of the disease, spray the crop with Streptocycline (100 ppm) and repeat at 7 days interval.

C) VIRAL DISEASES

7. PEA SEED BORNE MOSAIC

Symptoms:

- The symptoms of the disease include chlorotic pattern on the leaves followed by narrowing of leaves coupled with downward rolling and apical malformation.
- This is followed by vein clearing and production of malformed flowers.
- The size of pods is reduced which get distorted depending upon disease intensity.
- Such pods produce infected seeds which are shriveled.

Pathogen:

- The disease is caused by Pea Seed Borne Mosaic Virus which belongs to poty virus (PSbMV) group.
- The virus particles are filamentous and flexuous rods 770 x 12 nm in size and non-enveloped.
- The genome consists of single stranded RNA.

Disease cycle and epidemiology:

- The virus is transmitted mechanically, or by seed or by aphid vectors either semi-persistently or non-persistently.
- The natural vectors are *Acyrthosiphon pisum, Aphis craccivora* and *A. fabae*.
- The host range of the virus is wide but there are only three significant hosts like pea, lentil and broad bean with regard to economic importance, dissemination through seed.

Management:

- Use resistant pea cultivars.
- Use reflective mulches to reduce the incidence of this virus.
- Since seed borne viruses create a within field inoculum source, spray the crop with insecticides like Malathion (0.1%) or Metasystox (0.1%).

Other diseases of importance are:

i. **Downy mildew** : *Peronospora pisi* Syd.

ii. **Root rot** : *Aphanomyces euteiches* Drechs., *Fusarium solani* (Mart) Sacc. f. sp. *pisi* (Jones) Snyd. and Hans.

iii. **Leaf spot :** *Cercospora lathyrina* Ell & Ever., *C. pisa sativae* Stev., *Septoria pisi* West

iv. **Anthracnose :** *Colletotrichum pisi* Pat.

v. **Seed and seedling blight :** *Pythium* spp.

Lecture -11

DISEASES OF BEANS

French bean (*Phaseolus vulgaris* L.) is an important leguminous crop grown throughout the world for green pods and dry pulse. It has gained popularity due to its quality proteins and nutritional balance besides certain medicinal properties. Successful cultivation of this crop is hindered due to the attack of various diseases of fungal, bacterial and viral nature. Under favourable environmental conditions, epiphytotics of these diseases have often reduced the yields considerably. The diseases of common occurrence have been described in detail in this lecture.

Aim: To know about different diseases infecting beans and their management.

1. ROOT ROT AND WEB BLIGHT

Disease is of economic importance especially when crop is grown during rainy season and is more prevalent on bush type than pole type bean.

Symptoms: The symptoms of the disease appear in following two phases**:**

Root rot:

- Symptoms appear on roots and stem above and below the surface of the soil as reddish brown, sunken cankers (Plate-1).
- The lesions enlarge rapidly and girdle the stem at the collar region, extending longitudinally downward to the roots leading to partial or complete rotting of the root system.

Plate-1: Symptoms of root rot of French bean

Web blight:

- The symptoms on leaves appear as small, circular, water soaked spots which later become tan to brown in colour (Plate-2a) and these spots coalesce to form larger areas on the leaf blade.
- The pods are attacked at all stages of their growth and dark brown, more or less circular, slightly zonate and definitely sunken spots appear on pods which in moist weather are covered with hyphae and sclerotia of the fungus (Plate 2b).
- Tan brown to reddish brown discoloration is observed on infected seed.

Plate 2a: On leaves

Plate2b: On pods

Plate-2: Symptoms of web blight on French bean

Pathogen:

- The disease is caused by *Rhizoctonia solani* Kuhn.
- The basidial stage of the fungus is *Thanatephorus cucumeris* (Frank) Donk but occurs very rarely.
- The fungus consists of septate mycelium, the mycelium is branched near the distal septum of the mother hyphal cells at right or acute angles and the branches are constricted at or near the point of origin of the septum.
- In culture, sclerotia develop within 5-6 days.

Disease cycle and epidemiology:

- The pathogen perpetuates either through infected seed or in soil as sclerotia (Fig. 1.).
- The infected seeds after germination either rot in the soil itself or give rise to plants which later get damped off.
- The rain splashes spread the soil borne inoculum to foliage that leads to web blight symptoms.
- High rainfall and soil moisture coupled with high relative humidity and soil temperature of 23 to 25^{O}C favour disease development.

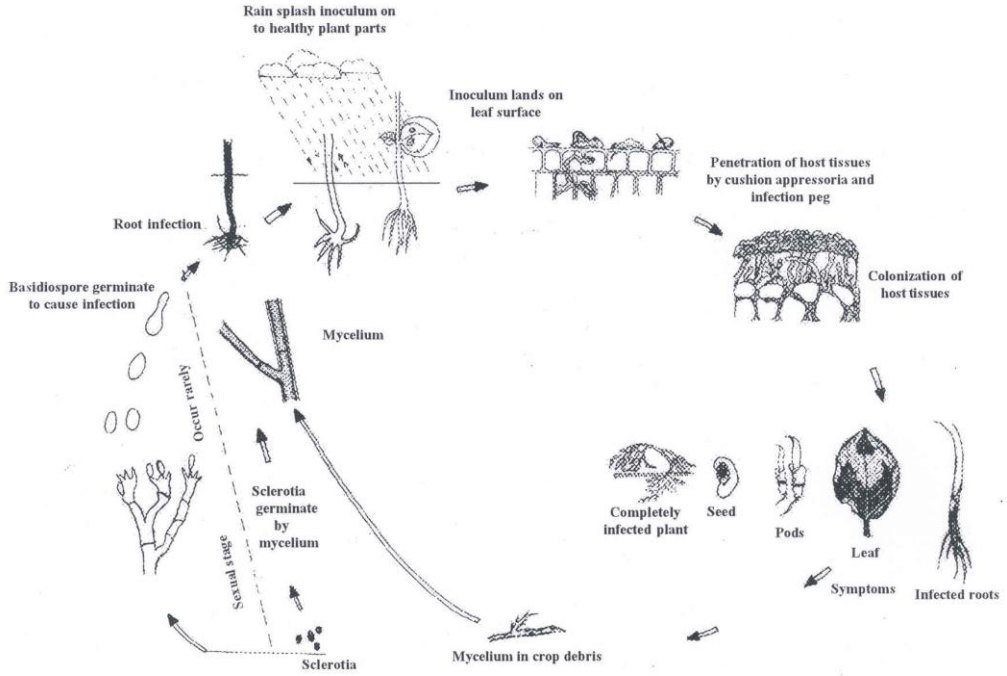


Fig. 1. Disease cycle of root rot and web blight of French bean

Management:

- Collect and destroy the infected plant debris.
- Follow long crop rotation.
- Solarize field soil at least for 40-45 days during summer months.
- Amend the soil with neem or mustard cake (250g/m^2).
- Use healthy seed.
- Dry seed treatment with carbendazim (0.2%).
- Before the onset of monsoon rains, apply pine needle/ grass mulch on the field floor to avoid splashing of pathogen inoculum to foliage.
- Spray the crop with carbendazim (0.1%) or mancozeb (0.25%) or combination of mancozeb (0.25%) and carbendazim (0.05%) or tebuconazole (0.06%) and repeat at 10-14 days interval for foliar control of the disease.

2. ANTHRACNOSE

The disease is more severe in temperate and subtropical mountaneous regions of the world with cool and wet climate.

Symptoms:

- Most striking symptoms of the disease appear on immature pods as brown and sunken spots with lighter or grey central area (Plate-3). The central portion of the spots shows pinkish masses of fungal spores, especially in wet weather.
- Later, the sides of these spots appear raised.

- On leaves, the infection appear on the under leaf surface as blackened dead portions of the veins which may extend to limited adjoining areas.
- In severely infected pods, brown to light chocolate coloured sunken cankers may also develop on seed coat.

Plate-3. Symptoms of bean anthracnose

Pathogen:

- The disease is caused by *Colletotrichum lindemuthianum* (Sacc. and Magn.) Briosi and Cav.
- The mycelium is branched, septate and hyaline at first becoming dark with age.
- The acervulus contains a layer of 3 to 50 conidiophores depending on the size of lesion (Plate-4).
- Conidia are borne successively and acrogenously on short conidiophores under favourable conditions until pinkish spore masses appear on the surface and setae are produced sparingly.
- Conidia are hyaline, cylindrical having rounded ends or somewhat pointed at one end and often bear a clear vacuole like body near the centre.
- The teliomorph of the pathogen has been identified as *Glomerella cingulata* (Ston.) Splaud & Schrenk (=*G. lindemuthiana* Shear).
- Perithecia contain hyaline and filiform periphyses and asci.
- Each ascus contains eight allantoid ascospores, which are ejected from the tip of ascus.

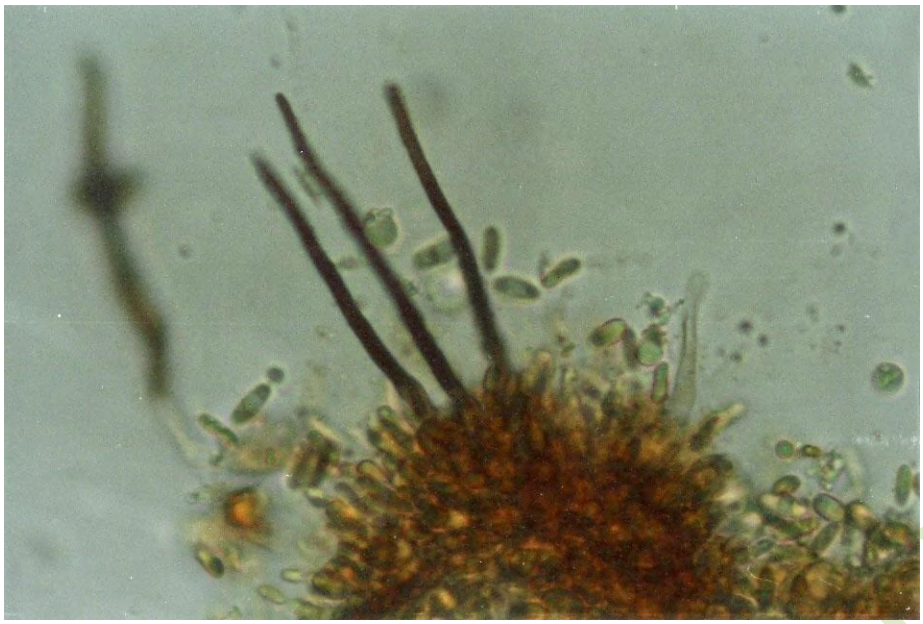


Plate-4. Acervulus of *Colletotrichum lindemuthianum*

Disease cycle and epidemiology:

- The pathogen perpetuate in infected seeds and in infected plant debris (Fig.2).
- When infected seed germinates, lesions appearing on cotyledons which serve as the source of secondary inoculum producing the spores as water borne.
- Primary leaves and the hypocotyl are foci of secondary infections.
- The teliomorph of the fungus is rarely found in nature.
- It develops most abundantly in cool, wet weather and largely disappears under hot and dry conditions.
- A relative humidity of 92 per cent and above is essential for infection, the optimum being close to 100 per cent.
- The fungus requires about 10 mm of rain to establish initial infection.
- The optimum temperature for disease development ranges from 18 to 27^{o}C with maximum intensity at 21^{o}C and is markedly reduced at 13^{o}C.
- The movement of insects, animals and man may spread conidia particularly when foliage is moist.

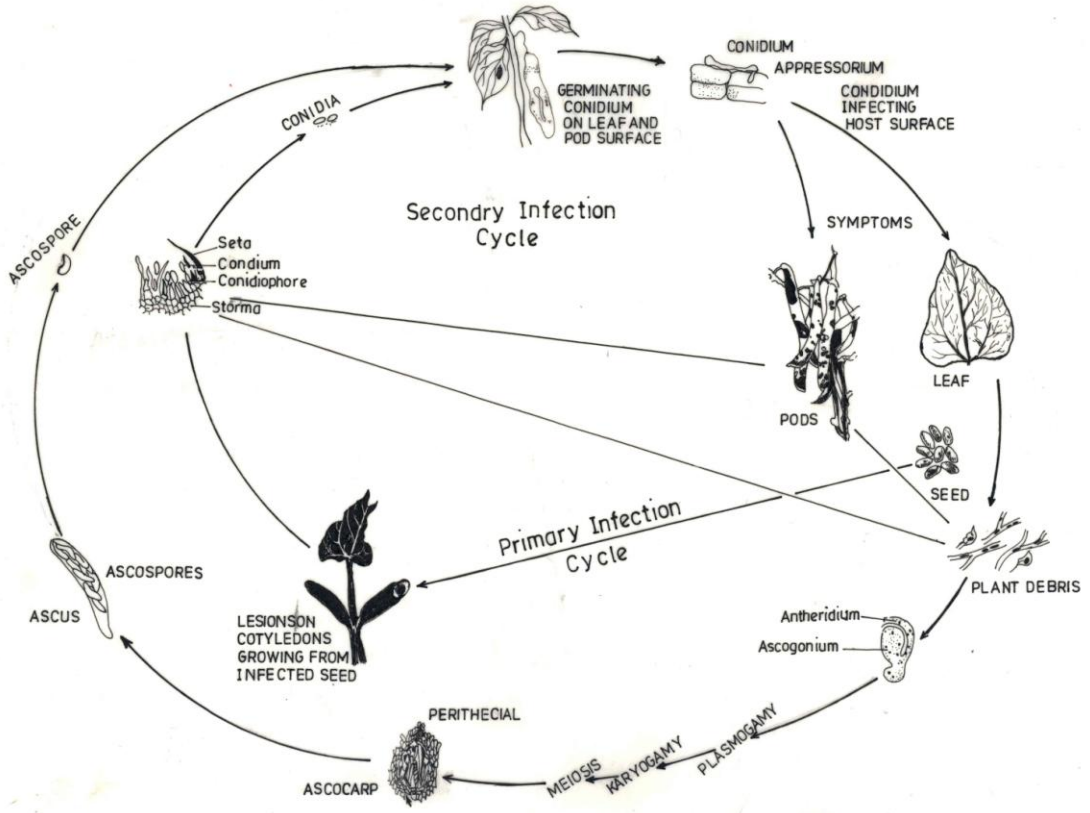


Fig.2. Disease cycle of bean anthracnose

Management:

- Follow 2-3 years of crop rotation with non-leguminous crops.
- Use disease free seed and treat them with carbendazim or benomyl (0.2%).
- Resistant cutivars/ lines like KRC 1, KRC 17, EC 42960 and EC 57080 have been identified as resistant to this pathogen.
- In field, the spread of the disease can be limited by application of fungicides like mancozeb (0.25%) or combination of mancozeb (0.25%) and carbendazim (0.05%) or tricyclazole (0.03%) and repeat at 10 -14 days interval.

3. ANGULAR LEAF SPOT

The disease was first reported by Saccardo (1878) from Italy. In India disease was first noticed from Nilgiri hills by Srinivasan whereas from H.P. by Sohi *et al*. (1963).

Symptoms:

- The symptoms appear as circular spots on cotyledonary leaves while, 3-5 angled spots appear in between veins and veinlets on true leaves which are dark grayish in colour on upper surface while light gray on the lower surface (Plate-5a).
- With the passage of time, spots change to reddish brown and finally to dark brown colour and on close observation, the spots reveal the presence of coremia bearing large number of spores.
- In severe infections, leaves show upward curling and defoliate prematurely.

- The fungus also attacks the pods causing superficial, smooth, usually circular spots with reddish brown center and ashy black borders but with time deeper tissues are also involved (Plate 5b).
- Elongated dark brown lesions also appear on stem and petioles.
- Under severe conditions of infections, these spots coalesce causing complete defoliation.
- Yellowish brown discolouration is also observed on infected seeds located underneath the pod lesions.

Plate-5a. On leaves **Plate-5b On pods**

Plate-5. Symptoms of angular leaf spot of French bean

Pathogen:

- The fungus responsible for this disease is *Phaeoisariopsis griseola* (Sacc.) Ferr. (Syn. *Isariopsis griseola*).
- The fungus produces a stromatic structure in the substomatal cavity.
- Columnar hyphae producing conidiophores are laterally joined together in the form of synnemata or coremia, which are darker at the base and lighter towards the apex and either curved, flexuous or septate (Plate-6).
- Conidia are light grey to hyaline, cylindrical to spindle shaped and straight to slightly curved, 1-3 (rarely 4-6) septate which are borne singly on the tips of conidiophore.
- No sexual stage of the fungus has yet been observed.

Plate-6. Synnemata or coremia of *Phaeoisariopsis griseola*

Disease cycle and epidemiology:

- The pathogen perpetuate both on seed and in infected plant debris (Fig.-3).
- Infected seed germinates to give rise to infected plantlets bearing coremia of the fungus on the undersurface of the cotyledonary leaves.
- The conidia released from the coremia disseminate either by rain splashes or with wind blown soil particles.
- The conidia germinate to form germ-tube, which enters through stomata and advances intercellularly in the mesophyll and palisade parenchyma.
- Within 7-10 days of infection, the fungus produces necrotic lesions, develops stromata in the substomatal cavity, which may sporulate during 24 to 48 h of continuous moisture.
- The conidia continue the cycle by acting as secondary inoculum.
- Infection from pods also goes to the developing seed.
- Optimum temperature for growth and sporulation, spore germination and development of the disease are 21-24°C, 18-24°C and 24°C, respectively while, optimum RH for sporulation, spore germination and symptoms development is >90 per cent.

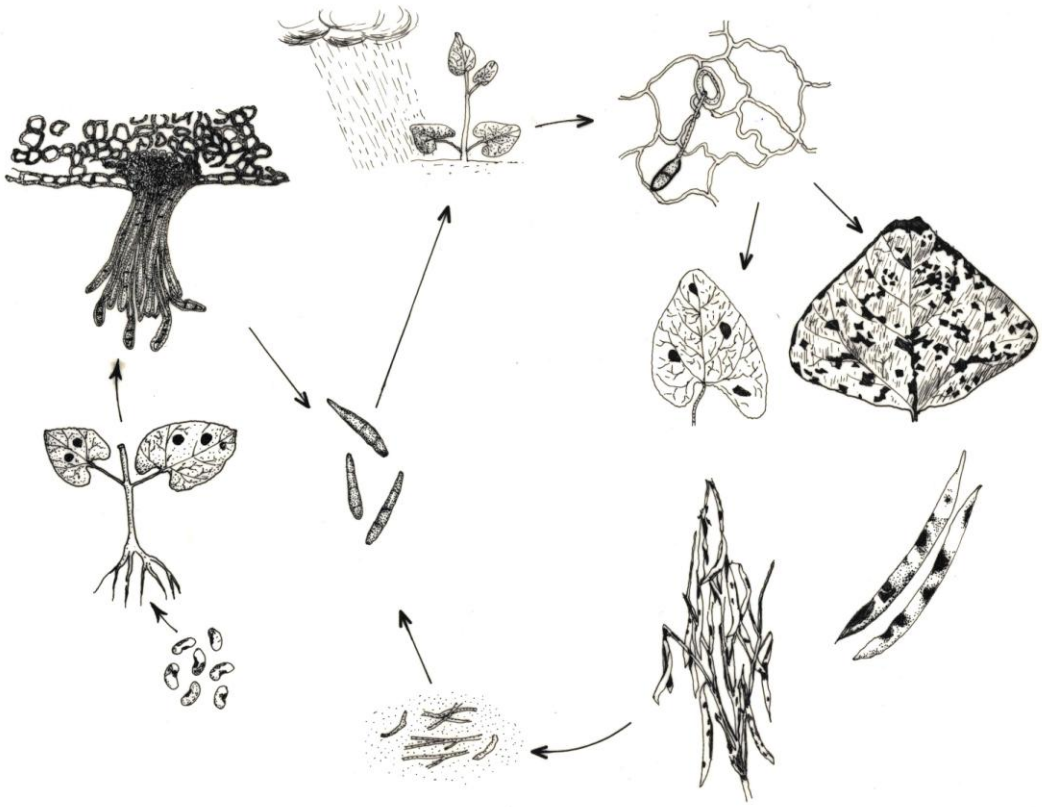


Fig. 3. Disease cycle of the Angular leaf spot

Management:

- Collect and destroy the infected plant debris.
- Follow at least 2-3 years crop rotation with non-leguminous crops.
- Use healthy seed and treat them with carbendazim (0.2%).
- With the initiation of the disease, spray the crop with mancozeb (0.25%) or combination of mancozeb (0.25%) and carbendazim/ benomyl/ thiophanate methyl (0.05%) or hexaconazole (0.05%) and repeat at 10-14 days interval.

4. FLOURY LEAF SPOT

Symptoms:

- The disease appears in the form of white tufty growth of the fungus on the lower leaf surface appearing as sprinkled with coarse flour, hence the disease is named as floury leaf spot (Plate-6).
- The corresponding upper side of the leaf turns yellow, then brown and finally dry up. Young leaves are less susceptible.

Plate-6. Symptoms of Floury leaf spot of french bean

Pathogen:

- The pathogen responsible for this disease is *Mycovellosiella phaseoli* (Drummond) Deighton (Syn.: *Ramularia phaseolina* Petrak).
- It is an obligate parasite.
- On the under surface of host, colourless conidiophores emerge in tufts from the stomata and interwine in clusters around the leaf hairs.
- Erect, more or less divergent, somewhat tapering towards the tip, simple or irregularly dichotomous, a few short, erect, single conidiophores were also found scattered over the leaf surface.
- Conidia are variable in shape and size (5.24-21.4 x 3.5- 5.3 µm) mostly ellipsoid or ovoid, tips acute or obtuse, base papillate at the point of attachment, stright hyaline, commonly aseptate, rarely one septate.

Disease cycle and epidemiology:

- The fungus is not seed borne and perpetuate in infected plant debris as conidia from one season to another.
- Overwintering conidia may be splashed by rain to lower leaves, which in the presence of free water germinate on host surface and cause infection.
- Conidia produced on primary spots are blown by wind and cause secondary infections.

- The disease is more common in altitudes of 1500 to 2000 m with heavy rainfall. The optimum temperature for conidial germination and germ tube elongation is 20^{o}C.

Management:

- Collect and destroy the infected plant debris.
- Follow at least three years of crop rotation with non-leguminous crops.
- Spray the crop with carbendazim/ benomyl (0.1%) and repeat at 10-14 days interval.

5. CHARCOAL ROT OR DRY ROOT ROT

The disease is also known as ashy stem blight, root rot, ashy grey stem and *Macrophomina* rot.

Symptoms:

- The initial symptom is yellowing of leaves, which soon dry up.
- The plant may wilt within a week after the appearance of symptoms.
- When the stem is examined closely, dark lesions may be seen on the bark at the ground level.
- On uprooting infected plants, dry rot symptoms may be seen on the basal stem and the main roots.
- The tissues are weakened and break off easily.
- Black sclerotial bodies are formed on the diseased tissue a few days after infection.

Pathogen:

- The disease is caused by *Macrophomina phaseolina* (Tassi) Goid.
- The sclerotial stage of the fungus is known as *Rhizoctonia bataticola* (Taub.) Butler.
- The mycelium is superficial or immersed, hyaline to brown, branched, septate and often tree-like in form.
- The pycnidia are at first buried beneath the lead-grey epidermis in which many sclerotial bodies are generally mixed.
- The conidiophores are more or less straight, sometimes crooked with a truncate tip.
- The conidia are one celled, more or less fusiform, straight or slightly curved.
- One end of the conidium is often pointed and the other is blunt.
- Sclerotia are readily produced, both on the plants in the field and in cultures.
- These are jet black, smooth, hard round to oblong or irregular in shape.

Disease cycle and epidemiology:

- The pathogen perpetuates as sclerotia in the soil and in infected seed.
- Survival is longer in dead host tissues than in free conditions in the soil.
- The fungus is a poor competitor in soil but readily colonizes plant debris.

- Population of the fungus increases when susceptible hosts are continuously grown in the field.
- When infected seed is sown in the field, it also introduces the fungus in the field.
- The sclerotia germinate and the mycelium cause primary infections.
- Environmental factors contributing to soil moisture stress/ water deficit predispose the plant to infection. Soil temperature of 28^{o}C or more favours disease development.

Management:

- Collect and destroy the infected plant debris.
- Follow long crop rotations with non-leguminous crops.
- Solarization of infected soils during summer months can also help in reduction of soil borne inoculum.
- Use disease free seed and treat them with carbendazim or benomyl (0.2%).
- Application of biocontrol agents like *Trichoderma viride* and *Aspergillus niger* have also been found promising but need further confirmation.

6. RUST

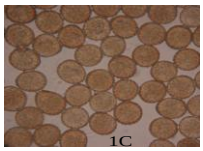
Symptoms:

- On leaves, the disease may appear on both the surfaces, but is more common on the abaxial surface (Plate-7).
- Pustules of the disease usually appear first as small and slightly raised spots that are almost white in colour.
- These pustules enlarge by forming reddish brown sori, up to 2 mm in diameter, containing the urediniospores.
- After coalescing, they may occupy larger areas.
- A ring of secondary sori may develop around the original infection on susceptible cultivars. Telial stage is linear, dark brown to black colour.
- The affected leaves may turn yellow and dry or fall prematurely.

Plate-7. Symptoms of bean rust

Pathogen:

- The disease is caused by *Uromyces phaseoli typica* Arth. and *U. appendiculalus* (Pers.) Unger. Being autoecious rust, all its spore stages are produced on bean plant but pycnial and aecial stages are not commonly encountered in nature.
- The urediniospores are brownish, globoid or ellipsoid, one celled and echinulate.
- Spore wall is golden brown 1-1.5 µm thick having two equatorial pores (Plate-8).
- Teliospores are one celled, pedicellate, globoid or broadly ellipsoid.
- They may be smooth or possessing a few verucose marks on the wall.
- The teliospores wall is chestnut brown in colour with a hyaline papilla over the pore.



1c = uridiniospores of *Uromyces appendiculatus*

Plate-8. Urediniospores of *Uromyces appendiculatus*

Disease cycle and epidemiology:

- In areas where the crop is grown continuously and collateral hosts are also present, inoculum in the form of urediniospores is available round the year.
- The life cycle of the fungus is completed on the host itself; however, in cooler areas where bean is taken as single crop in the year, teleutospores surviving in crop debris serve as source of perpetuation.
- Areas having high humidity and temperature upto 24^{o} C are most suitable for disease development.
- Urediniospores germinate optimally at 15-24^{o} C while for teliospores germination, 10-15^{o} C is optimum.

Management:

- Collect and destroy the infected plant debris.
- Follow long crop rotations.
- With the initiation of the disease, spray the crop with mancozeb (0.25%) or hexaconazole (0.1%) or difenoconazole (0.05%) or combination of mancozeb (0.25%) and hexaconazole (0.05%) and repeat at 10 days interval.

A) BACTERIAL DISEASES

7. COMMON BLIGHT

Symptoms:

- The symptoms appear as characteristic water soaked spots on the under side of the leaves that enlarge and coalesce together to form large blighted areas.
- Infected areas often become flaccid and turn brown giving it a sunscald appearance.
- If infection occurs through the vascular tissues, reddish discolouration of veins and midribs is observed.
- In advanced stages, tissues become necrotic and defoliation may also take place.
- Lesions on stems appear as sunken water soaked areas, which gradually enlarge to form reddish streaks extending longitudinally in the stem.
- Infection on pods may occur at any place as small water soaked spots, which are surrounded by distinct zones of narrow reddish brown or brick red band of tissue.
- On seeds, discoloration appears especially near the hilum region.

Plate-9. Symptoms of bean common blight

Pathogen:

- The disease is caused by *Xanthomonas axanopodis* pv. *phaseoli* (Smith) Dye.
- The bacterium is a motile, non-capsulated, non-spore forming, gram -ve, rod shaped, measuring 1.9 x 0.8 µm in size with a single polar flagellum.
- The pathogen forms characteristic yellow coloured colonies due to a water insoluble eacotenoid and a mucoid growth on nutrient glucose agar medium.

Disease cycle and epidemiology:

- The bacterium perpetuate in infected seed, crop debris and weed hosts of which contaminated seed has been observed to be the most efficient mode of survival and dispersal over long distances.
- Upon germination, the bacterium from infected seed infects the developing cotyledons and can then enter the vascular system of the plant.
- Alternatively the bacterium can be splashed to the plants from the plant debris.
- Once inside the plant, the bacteria invade and cause breakdown of the middle lamella.

- The bacterium is a warm temperature pathogen and causes greatest damage to the plants at 28-32^{o} C.
- High humidity and high rainfall increases the disease spread.
- Secondary spread occurs by wind blown rain (rain splashes), contact between leaves, irrigation water and insects like white flies and leaf miners.

Management:

- Collect and burn the infected plant debris.
- Follow at least three years crop rotation.
- Use healthy seed and treat them with hot water at 50^{O}C for ten minutes followed by diping in Streptocycline (100 ppm) solution.
- With the initiation of the disease spray the crop with copper oxychloride + zineb or copper oxychloride + mancozeb and repeat at 10 days interval.

C) VIRAL DISEASE

8. BEAN COMMON MOSAIC

Symptoms:

- The symptoms of the disease appear as mixture of light yellow and green areas on the leaves (Plate-10).
- This is coupled with production of narrower leaves which roll upward, mottle, pucker and become malformed.
- Early infection leads to yellow coloured dwarf plants and such plants usually fail to set pods or develop undersized pods.
- The pods on severely-infected plants are usually undersized and contain fewer ovules than those produced on normal plants.
- They are occasionally covered with small, dark green spots and often mature very late.
- The seed may be aborted, smaller or malformed.

Plate-10 Symptoms of bean common mosaic

Pathogen:

- The disease is caused by Bean Common Mosaic Virus (BCMV) which belongs to poty virus group.
- The virus particles are filamentous flexuous rods measuring 700-800 nm in length and 12-15 nm in diameter.
- The genome consists of unipartite single stranded DNA.

Disease cycle and epidemiology:

- The virus is seed borne in a range of legumes.
- The virus is also sap and graft transmissible.
- The virus can be transmitted in the non-persistent manner by several aphid species which do not normally colonize beans but transmit the virus as winged migrants, especially *Acyrthiosiphon pisum, Aphis fabae* and *Myzus persicae*.

Management:

- Use healthy and certified seed helps in checking the spread of the disease.
- Early planting when the incidence of aphid vectors is low gives better yields.
- Intercropping of maize with bean also reduces the incidence of this disease.

Lecture-12

DISEASES OF BEETROOT

Beet (*Beta vulgaris* L.) includes red beet, sugarbeet, fodder beet and swiss chard. During cultivation several diseases of fungal and viral nature attack these crops, which not only reduce quality of the produce but also the quantity.

Aim: To know about different diseases infecting beetroot and their management.

A) FUNGAL DISEASES

1. CERCOSPORA LEAF SPOT

Symptoms:

- The characteristic symptoms include, discrete circular lesions, 3-5 mm in diameter, with a necrotic center and reddish to dark brown margin (Plate-1) and in case of severe infection, the petioles are also infected.
- The spots are scattered at first but in case of severe attack they coalesce and cover the entire leaf blade and affect the quality and yield of seeds.
- In seed crop, all the above ground parts including seed clusters are affected.

Plate-1. Symptoms of Cercospora leaf spot of sugarbeet

Pathogen:

- The disease is caused by *Cercospora beticola* Sacc.
- The mycelium of the fungus is septate, dark coloured and intracellular.
- Small sclerotial masses are formed on the host tissue from which dark coloured conidiophores arise in clusters.
- The conidia are borne on the tip of the conidiophores and they are hyaline, elongated, filiform, multi-septate, broadly rounded at the point of attachment to the conidiophore and tapering slightly toward the opposite end.

Disease cycle and epidemiology:

- The fungus perpetuates in infected plant debris as mycelium or on the seed.
- The infected seeds result in infected seedlings.

- In the spring, overwintering mycelium in plant refuse starts producing conidia which are disseminated by wind currents, rain splashes and insects to the leaves.
- High relative humidity is an essential pre-requisite to sporulation.
- The conidial germination is best in the presence of free waters and moderate temperatures i.e. in between 15-32^{o} C.

Management:

- Collect and destroy the infected plant debris.
- Follow long crop rotations to prevent accumulation of soil borne inoculum.
- The resistance to *Cercospora* leaf spot has been found to be correlated with the 3-hydroxytyramine content of the leaves.
- Variety Desperzpoly RC has been reported as resistant to this disease besides giving highest tuber yield as well as sucrose content.
- Spray the crop with carbendazim or benomyl or thiophanate methyl (0.1%) and repeat at 20 days interval.

2. POWDERY MILDEW

Symptoms:

- The disease initially appears on the lower leaves and gradually spreads towards the top.
- The formation of first white, later grey-tan mildew areas on both the sides of the leaf characterize the disease (Plate-2).
- In general, infection is more on the upper surface of the leaf.
- In advanced stages of the disease development, mildew patches enlarge and coalesce and the leaf appears as if dusted with wheat flour.
- Severely affected leaves turn yellow and ultimately dry up.
- In favourable climatic conditions, cleistothecia develop as small dark round structures on the infected surface of the leaf.

Plate-2. Symptoms of powdery mildew of sugarbeet

Pathogen:

- The disease is caused by *Erysiphe betae* (Vanha) Weltzien.
- Mycelium is superficial and persistent.
- Conidiophores unbranched and erect.
- Conidia ripen singly, hyaline, ovoid and 30-50 x 15-20 µm in size.
- Cleistothecia are globose, dark brown/ black, with 4-8 asci per cleistothecium.
- There are mostly 2-3 ascospores/ ascus.

Disease cycle and epidemiology:

- Wherever cleistothecia of the fungus are formed, these serve as the possible mode of perpetuation.
- In normal situation, the pathogen survives from one season to other in conidial state either on the perennial weeds or on other related crops grown in the area.

Management:

- The disease can be managed by spraying wettable sulphur (0.2%), benomyl or carbendazim or thiophanate– methyl (0.1%), or hexaconazole (0.05%) or difenoconazole (0.03%) as the disease starts appearing.

3. SCLEROTIUM ROOT ROT

Symptoms:

- The disease is characterized by sudden rotting of mature roots below the soil surface.
- The affected root is usually covered with white mycelium that contains numerous brown sclerotia which is the most conspicuous sign of this disease.
- The fungal growth and sclerotia can be seen also in the soil around such roots.
- Later, when enough damage has been done to the roots, the leaves show yellowing and wilting and such plants can be easily pooled out.

Pathogen:

- The pathogen responsible for this disease is *Sclerotium rolfsii* Sacc.
- The hyphae are hyaline, thin walled, sparsely septate when young.
- The broader hyphae show clamp connections, which are absent in thin hyphae.
- The number of nuclei/ cell is highly variable.
- Mostly there are 2 nuclei in cells of secondary and tertiary branches.
- Sclerotial initials are formed from hyphal strands that consist of 3-12 hyphae lying parallel.
- Mature sclerotia are dark brown but variation from lighter brown to darker colour may be found.

- They are small, about the size of radish seed, hard and usually round.

  The basidial stage grows as a spreading white hymenium on the host surface or on the surface of the culture medium.
- Although the hymenia may be pure white in culture, on the host they may be grey, yellow or buff coloured.
- The basidia are obovoid. Each basidium bears 2-4 parallel or divergent sterigma, which are 2.5-4 µm long.
- Basidiospores are unicellular, elliptical to obclavate, sometimes rounded or pyriform, smooth walled, hyaline and epiculate at the base.

Disease cycle and epidemiology:

- The fungus is soil borne in nature and can survive as saprophyte on crop debris.
- The fungus also produces sclerotia that are left in the field and germinate under favourable weather conditions and cause infections.
- High temperature and humidity favour the disease development.
- In the plains of India, sugar beet is usually grown on ridges and this practice is likely to stimulate disease incidence because the lower leaves become covered with soil resulting in a 'bridge' of dead tissue which furnishes an ideal medium for initiating pathogenesis.

Management:

- Cultural practices like destruction of infected plant debris, crop rotation, deep summer ploughing, flooding, solarization during summer months etc. can be helpful in reducing the initial inoculum load in the field.
- Reduction in the incidence of root rot has also been recorded through the use of nitrogenous fertilizers including calcium nitrate, cyanamide and anhydrous ammonia, urea, ammonia sulphate and calcium ammonium.
- Drenching of ridge soil with carboxin and chloroneb at 2 and 15 kg/ 3000 l water/ha, respectively, significantly reduced root rot incidence and increased yields.
- Application of *Trichoderma harzianum* ($40g/m^2$) inoculum was found effective in reducing the disease caused by *S. rolfsii* in sugarbeet.

B) VIRAL DISEASES

4. RHIZOMANIA

Symptoms:

- Leaves may show flabbiness, wilting and mild yellowing on the infected sugarbeet plant in the field.

- The most characteristic symptoms found in beet leaves are a pattern of yellow areas along the veins.
- However, the virus mostly remains confined to roots and rarely moves to the foliage.
- Symptoms on roots are characterized by root stunting and a proliferation of lateral rootlets on the main tap root, giving it bearded appearance.

Pathogen:

- The virus responsible for this disease is Beet Necrosis Yellow Vein Virus (BNYVV) which is a probable member of the furovirus (fungus borne rod shaped virus) group.
- Ultrathin sections of BNYVV- infected tissue show the virus particles scattered in the cytoplasm and in angled layer aggregates.
- The virus is not seen in large aggregates but shows an erratic distribution in the mesophyll cells of systemic plants.

Disease cycle and epidemiology:

- The fungus *Polymyxa betae* has been considered to be the vector of BNYVV and was also shown to transmit soil borne virus.
- This fungus has worldwide distribution.
- The abnormal proliferation of the root lets, characteristic of rhizomania was found in the presence of non-viruliferous *P. betae* but zoospores transmit rhizomania symptoms to whole sugar beet plants, in the absence of detectable virus in the infected plants, indicating that BNYVV is not requisite to the etiology of the disease.
- The infectivity of BNYVV was retained with *P. betae* in air and dry soil for at least 15 years.
- BNYVV was detected by ELISA in extracts of resting spore clusters of a viruliferous *P. betae* isolate.

Management:

- Varieties Laetitia and Nagano have been reported to possess resistance to this disease. Soil disinfection with dicloropropene was recommended for the control of fungal vector *P. betae*.
- Preplant application of fumigants like dichloropropene, tolone 11 (1,3-dichloropropene), Vorled (1,3-dichloropropene + methyl isothiocyanate), Vorlex 201 (1,3- dichloropropene + chloropicrin + methyl isothiocyanate) and Pichlor 60 reduced disease incidence and significantly increased yields. Soil inoculation with *T. harzianum* reduced infection by vector, *P. betae*.

Lecture-13

DISEASES OF ONION AND GARLIC

Onion (*Allium cepa* L.) and Garlic (*Allium sativum* L.) are two most important bulbous vegetable crops cultivated in different parts of the country. These crops are susceptible to several diseases which of fungal, bacterial and viral nature, cause substantial production losses. Symptoms, pathogens, disease cycle and epidemiology and management strategies of major diseases are described in this lecture.

Aim: To know about different diseases infecting onion and garlic and their management - Part I.

1. DOWNY MILDEW

This disease was first time reported in Britain (1841), then in USA (1844), whereas in India it was first recorded during 1974-75 from Kashmir valley and appeared destructive in the year 1975-76. Yield losses upto 60-70 per cent have been reported.

Symptoms: Two types i.e. local and systemic infection occur.

- Systemic infection occurs when the plants are raised from diseased bulbs or infected seedlings are used for planting.
- Plants raised from such bulbs remain stunted, become distorted and light green in colour (Plate – 1a).
- In humid weather conditions, sporulation developed on the leaves and covered them with felty whitish to grayish fungal growth.
- Local infection is caused by air borne zoopsores which produce oval to cylindrical spots (Plate–1b).
- In humid weather, the fungus develops as white to purplish downy growth on these spots.
- Affected leaves become pale green, fold over, later collapse and in such cases undersize bulbs are produced.
- On seed stalks, circular to elongate lesions are produced and infected stalks break over with the weight of the seed umbel, thereby causing the seed to shrivel.

Plate – 1a. On bulbs **Plate – 1b On leaves**

Plate – 1. Symptoms of downy mildew

Pathogen:

- The disease is caused by *Peronospora destructor* (Berk.) Casp.
- The mycelium of the fungus is non-septate, intercellular with filamentous haustoria.
- The sporangiophores are aseptate, hyaline swollen at the base and 3 to 4 times dichotomously branched.
- The sterigmata were acute to subacute and bear pyriform to fusiform sporangia.
- Oospores develop late in the season and germinate by germ tube.

Disease cycle and epidemiology:

- The main source of primary infection is mycelium in infected onion bulbs and such bulbs when used for seed production give rise to diseased plant.
- The fungus may also perpetuate as oospore.
- The disease spread by wind borne, and short lived spores.
- The onion seedlings from disease prone areas also play significant role in introduction of disease in new localities.
- The sporulation and infection by *P. destructor* usually takes place overnight while dissemination of the newly formed conidia occurs during morning hours.
- The temperature i.e 13^{o} C and > 95 per cent relative humidity are optimum for the development of the disease.
- Presence of dew or raindrops on the leaf surface further enhances the chances of infection.
- Closely spaced and densely planted crops develope more disease compared with widely spaced ones while an increase in level of nitrogen fertilization increased disease severity proportionately and potassium decreased it.

- A preliminary model, ONIMIL was developed to forecast primary infection, which is able to determine for each day the probability of *P. destructor* establishing an infection on onion and its infectivity level.
- MILIONCAST, a model was developed based on the data from the controlled environment studies to predict the rate of sporulation in relation to temperature and relative humidity.

Management:

- Collect and burn the infected plant debris.
- Use healthy seed/ planting material for propagation.
- Onion lines like IC 48045, IC-32149, IC-33617, IC-49371 and DOP-11 have been reported resistant to this disease.
- Spray the crop with metalaxyl + mancozeb (0.25%) immediately with the initiation of the disease followed by sprays of mancozeb (0.25%) or copper oxychloride (0.3%) and repeat at 10-14 days interval.

2. PURPLE BLOTCH

This disease is common in India, both in seed as well as in bulb crop and mostly aggravated by *Botrytis* infection and thrips attack.

Symptoms:

- The characteristic symptoms of the disease appear as small, water-soaked lesions on the leaves or seed stalks that quickly develop white centres.
- These lesions enlarge, coalese become zonated and brown to purple under favourable conditions and extend both upward and downward (Plate -2).
- In moist weather, the surface of the spot is covered with the brown or almost black sporulation of the fungus.
- Usually the affected leaf or stem falls down and dies within 3 or 4 weeks under favourable environmental conditions.
- Similar lesions may form on seed stalks; as a result, seeds either do not develop or are shriveled.
- In garlic, the disease appears on leaves with the similar symptoms.

Plate – 2. Symptoms of purple blotch

Pathogen:

- The fungus responsible for this disease is *Alternaria porri* (Ellis) Cif.
- Conidiophores arise singly or in groups and are straight or flexuous, often geniculate, septate, pale to mid brown in colour.
- Conidia are muriform and are usually solitary, straight or curved, obclavate and taper to a beak that is commonly about the same length or slightly larger than the body of the conidium.
- Each conidium has 8 to 12 transverse and zero to several longitudinal or oblique septa, the beak is flexuous, pale and tapering and each cell of the conidium is capable of germination by germ tube.
- Existence of chlamydospores in the fungus is also reported.
- Sexual stage of the fungus is unknown.

Disease cycle and epidemiology:

- The fungus survives from one season to other in infected plant debris as dormant mycelium.
- The fungus can also survive in diseased onion leaf and seed stalk debris for 12 months buried at 5 and 7.5 cm depth.
- Wherever the chlamydospores of the fungus are formed they can also serve as source of perination.
- Persistent dew for extended periods or high relative humidity (>85%) are essential for infection and sporulation.
- The fungus can grow over a wide range of temperature (6-34^{o} C) with optimum at 25^{o}C.

Management:

- Collect and destroy the infected plant debris. Follow crop rotation with unrelated crops.
- Cvs./ lines viz. Pusa Red, IIHR-56-1, IC 48059, IC 48179, IC 39887, IC 48025, ALR and Ro-1 have been reported as resistant to this disease.
- Treat the seed with captan (0.3%) or thiram (0.3%).
- With the initiation of the disease, spray the crop with mancozeb (0.25%) or copper oxychloride (0.3%) or hexaconazole (0.05%) and repeat at 10-14 days interval.

3. STEMPHYLIUM BLIGHT

Symptoms:

- The first symptoms of the disease appear on the radical leaves as small, yellow to pale orange flecks or streaks in the middle of leaf, which soon become elongated, spindle shaped to ovate spots, surrounded by characteristic pinkish margin (Plate – 3).
- These spots turn grey at the centre and then become brown to dark olive brown with the development of conidiophores and conidia of the pathogen. Similar symptoms appear on the inflorescence stalks.
- In garlic crop, the symptoms of disease appear on the inner side of the leaf only.

Plate – 3. Symptoms of Stemphylium blight on leaves

Pathogen:

- The disease is caused by *Stemphylium vesicarium* (Wallr.) E. Simmons (Tel. : *Pleospora allii* (Rabenh) Ces. & de Not).
- Conidiophores are straight to variously curved, 1-4 septa, simple or occasionally one branched, cylindrical but enlarging apically to the site of the conidium production, light yellow brown to medium golden brown in the swollen apex.
- Conidia are oblong or broadly oval and sometimes in equilateral one to six transverse septa and one to three longitudinal septa and constricted at the major transverse septa.

- Conidia are light to medium golden brown to olive brown in colour and each has a conspicuous basal scar like zone.
- Perithecia mature within 3 to 6 months.
- Asci are cylindrical to clavate in shape. Ascospores are ellipsoidal and the upper half is narrowly tapered.

Disease cycle and epidemiology:

- The fungus perpetuates in the infected plant debris.
- Moderate temperature and humidity coupled with precipitation favour the disease initiation and development.

Management:

- Cultural practices like collection and destruction of plant debris, crop rotation and summer ploughings are recommended for the control of this disease.
- Sprays of mancozeb (0.25%) alongwith sticker Triton or Sandovit have been found to be very effective for the control of this disease.
- Initiate sprays in the first week of February and repeat at 10-14 days interval.

4. FUSARIUM BASAL ROT

Symptoms:

- The main symptoms of the disease are wilting and rapid dying back of leaves from the tips as the plant approach maturity.
- Infected garlic plant shows reddish or reddish purple discolouration on stems and bulbs early in the season with some discolouration on bulb sheath at harvest, the bulbs become soft and when cut a watering decay is noticed (Plate – 4)
- The damping-off symptoms of seedlings are also noticed.

Pathogen:

- The disease is caused by *Fusarium oxysporum* Schlechtend ex Fr. f.sp. *cepae* (Hans.) Snyd. & Hans. and *F. solani* (Mart.) App. & Wollenw.
- Both species of *Fusarium* produces microconidia, macroconidia and chlamydospores.
- Macroconidia are uniformly curved and have 3-4 septa.

Plate – 4. Symptoms of *Fusarium* basal rot

Disease cycle and epidemiology:

- Both pathogens perpetuate in soil through chlamydospores.
- The pathogen (s) may have been disseminated widely by infected onion sets and garlic cloves.
- The disease generally appears when soil temperature is 25-28^{o} C along with high soil moisture.

Management:

- Follow long crop rotation because both pathogens are persistent soil inhabitants.
- Cultivar like IIHR Yellow, breeding line SI.29 and Hybrid 1 have some level of resistance to this disease.
- Dipping of seedlings before transplanting in the suspension of carbendazim (0.1%) is recommended.
- Pre-harvest sprays of carbendazim (0.1%) also reduce the post harvest decay in storage.

5. WHITE ROT

Symptoms:

- The fungus invades roots and the basal part of the bulb scales.
- The first symptoms of the disease appear as yellowing and wilting of the leaves followed by a total collapse of the tops.
- If the diseased plants are pulled gently, they will come up readily because of the rotting of centre roots and affected parts are covered with a thick white mycelial mat bearing numerous small sclerotia.

Plate – 5. Symptoms of white rot

Pathogen:

- The disease is incited by *Sclerotium cepivorum* Berk.
- The fungus produces sclerotia which are uniformally round measuring 0.35 to 0.50 mm in size.
- Sclerotia germinate only once.

Disease cycle and epidemiology:

- The fungus is mainly disseminated by bulbs which have come from infected soil.
- The fungus persists in the soil and remains viable indefinitely.
- Disease develops rapidly in the soil having 40 per cent water holding capacity.
- Sclerotial germination occurs at temperatures ranging from 9 to 24^{o} C with optimum being 14 to 18^{o} C with low moisture.

Management:

- Collect and destroy the infected plant debris.
- Removal of infected plants during season reduces the sclerotial population and also avoids incorporation of the same in the soil.
- Soil solarization during summer months also reduces the incidence of this disease.
- Incorporation of *Trichoderma harzianum* in soil after solarization also controls *S. cepivorum* in soil effectively(40 g/m^{2}).

- Several fungicides have been advocated but Iprodione was found most effective in containing this disease.

6. ONION SMUT

Symptoms:

- The first symptoms of the disease appear on the cotyledons as dark, thickened areas on the surface which involve one to several millimeter of the surface.
- The seedlings often die before emergence (Plate -6).
- Large lesions can cause leaves to curve downward (Plate -6).
- Mature lesions exposed, black, powdery spore masses (teliospores).
- Infection progresses inward from leaf to leaf, and infected plants become stunted and may die within 3 to 4 weeks after emergence.
- If the plant survives, the disease becomes systemic and the plant remains in vegetative phase for the whole growing season.
- Bulbs are also covered with blackish lesions.

Plate – 6. Symptoms of onion smut

Pathogen:

- The disease is caused by *Urocystis cepulae* Frost.
- The sori of fungus looks dark coloured spore masses.
- Chlamydospores or smut teliospores are single celled, spherical or ellipsoid, reddish brown, smooth, thick walled, surrounded by a layer of small, slightly coloured, sterile cells and germinate by means of short promycelium while still held in ball.

Disease cycle and epidemiology:

- The pathogen is soil borne and perpetuates in the form of chlamydospores.
- If the host is available, the spores germinate immediately otherwise they remain dormant indefinitely.
- Optimum temperature for spore germination and growth is 13 to 22^{o} C.

Management:

- Use healthy seed and treat them with thiram or captan (0.3%).
- Before sowing, the nursery bed should be treated with Formaldehyde (5%).
- Avoid the raising of nursery at same location every year.
- No resistance source has been reported in onion but does occur in other *Allium* spp. which can be used in breeding programme.

7. ONION BLAST

Symptoms:

- The symptoms of the disease appear as white specks with necrotic centres surrounded by a light green halo and lesions may be either isolated and few in number or numerous (Plate -7).
- They may expand slightly with age and take on an elliptical shape and the halo may disappear.
- Many lesions remain restricted in size but under prolonged moist conditions, the fungus develops rapidly and causes blighting of leaves.
- Such leaves are killed prematurely resulting in reduction of bulb yield.

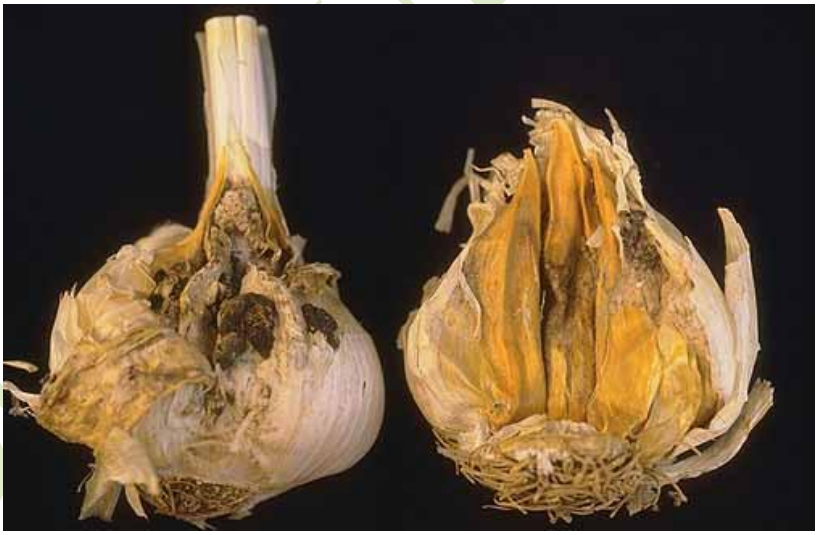


Plate – 7. Symptoms of blast

Pathogen:

- The disease is caused by several species of *Botrytis* like *B. allii* Munn., *B. byssoidea* Walker, *B. squamosa* Walker, *B. cinerea* Pers. and *B. cepa*e Hanzawa.
- *Botrytis* is characterized by its hyaline and septate conidiophores which arise as branches of the mycelium with side branches at the tips, each of which has many ampullae that swell gradually at the tips to form conidia on fine denticles.
- Conidia are hyaline or tinted, single celled and globose to ovoid.

- Sclerotia may form on leaf debris or on the necks of onion bulb and germinate by forming stipes on which conidiophore and conidia are produced.

Disease cycle and epidemiology:

- The fungus perpetuates as sclerotia or mycelia in crop debris.
- Sclerotia upon germination produce conidia at 3 to 27^{o} C (optimum at 9^{o} C) and serve as source of primary inoculum.

Management:

- Collect and destroy the infected plant debris.
- Follow crop rotation avoiding *Allium* spp. in rotation.
- With the initiation of the disease, spray the crop with captan (0.25%) or mancozeb (0.25%) and repeat at 10-14 days interval.

8. ONION SMUDGE

J.C.Walker (1929) gave biochemical resistance i.e. red onion cv. posses catechol and protocatechuic acid that provide resistance against onion smudge pathogen. This disease is more prevalent in white coloured onion var. grown in temperate region of the world. It is one of the important diseases in storage.

Symptoms:

- In seed bed it causes damping off of seedling.
- Disease is characterized by the appearance of dark green to black smudge (minute stromata of the fungus) on bulb, neck or green leaves.
- In humid climate pinkish masses of spore can be seen.

Pathogen

- Disease is caused by *Colletotrichum circinans* (Berk.) Volino.
- Acervuli are formed on stromata just beneath the cuticle by formation of pallisade layer of short conidiophores along with dark satae.
- Conidia are fusiform, falcate and hyaline.

Disease cycle and epidemiology:

- Pathogen survives in stromata and as a saprophytic mycelium in the debris.
- Stromata give rise to acervuli and conidia. Disease development takes place at 10-32ºC but optimum being 26ºC.
- Moist condition favours the production of conidia.

Management:

- Grow coloured variety to minimize disease incidence.
- Dry the bulbs before storage for curing.
- Foliar application of mancozeb (0.25%) in combination with carbendazim (0.05%) will reduce disease development.

Other diseases of importance are:

i. **Rust:** *Puccinia asparagi* DC.
ii. **Pink rot :** *Pyrenochaeta terrestris* (Hansen) Gorenz *et al*.
iii. **Black mould:** *Aspergillus niger* V.Tiegh.
iv. **Bacterial blight:** *Pseudomonas gladioli* pv. *allicola*
v. **Onion yellow dwarf :** Onion Yellow Dwarf Virus
vi. **Garlic mosaic :** Garlic Mosaic Virus
vii. **Aster Yellows :** Phytoplasma

Lecture-14

DISEASES OF CUCURBITS

Cucurbits, member of family Cucurbitaceae, form an important group of vegetable crops in different parts of the world. The family contains 9 genera and 16 species which are cultivated as vegetables. In India, cucurbits like ash gourd, bitter gourd, bottle gourd, cucumber, spong gourd, long melon or snake cucumber, kundru, muskmelon, pointed gourd, pumpkin, ridge gourd, snake gourd, squash, round gourd and watermelon are cultivated round the year in one other region of the country. During cultivation these crops are attacked by a number of fungal, bacterial and virus diseases which cause huge losses. Different diseases affecting cucurbits are described in this lecture.

Aim: To know about different diseases infecting cucurbits and their management

A) FUNGAL DISEASES

1) Downy mildew:

Symptoms:

- Pale green to yellow angular spots restricted by leaf veins appear on the leaves (Plate-1).
- Corresponding underside of these spots is covered by grey to brownish growth of fungus.
- Later on, lesions turn necrotic.

Plate-1. Symptoms of downy mildew of cucumber

Pathogen:

- The disease is caused by *Pseudoperonospora cubensis* (Berk. & Curt.) Rostow.
- The mycelium is hyaline, coenocytic and intercellular.
- Sporangiophores develop in groups of 1-5 and are bulbous at base and branched dichotomously.
- Sporangia form singly on subacutely shaped sporuliferous tips, are pale grey to purple in colour, ovoid to elliptical in shape, possess thin wall with a papilla at distal end.
- Sporangia germinate by producing zoospores.
- Oospores are produced rarely and are spherical, rarely obovoid to ellipsoid ,light yellow to pale yellow with smooth wall.

Disease cycle and epidemiology:

- The pathogen mainly survives as mycelium and sporangiophores in living hosts during off-season.
- Survival through oospore is not common.
- Moderate temperature (20-22^{o} C) coupled with high rainfall and > 80 per cent RH favour disease development and spread.

Management:

- Destruction of plant debris of previous crop.
- Destruction of cucurbitaceous weeds from in and around the field.
- Maintain proper plant density.
- Spray crop with mancozeb (0.25%) followed by one spray of metalaxyl + mancozeb or cymoxanil + mancozeb (0.25%) and two sprays of mancozeb (0.25%) at 10 days interval.

2. Powdery Mildew

Symptoms:

- Disease appear as white, floury coating on the leaves, stems and other succulent parts (Plate-2).
- In *S. fuliginea*, the spots are dirty brown in colour while in *E. cichoracearum* white in colour.

Plate-2. Symptoms of powdery mildew of cucurbits

Pathogen:

- Two fungi namely *Sphaerotheca fuliginea* (Schlecht.ex. Fr.) Poll. and *Erysiphe cichoracearum* DC. are associated with this disease.
- Conidia of S. fuliginea are formed in long chains and often show fibrosin bodies , are ellipsoidal shaped while in E. cichoracearum conidia are single celled, hyaline and barrel to cylindrical and are in long chains.
- Cleistothecia of S. fuliginea contain single ascus with eight ascospores while cleistothecia of E. cichoracearum contain 8-18 asci with 2-3 ascos[pores.

Disease cycle and epidemiology:

- The pathogens overwinter either in the form of cleistothecia or conidia in cucurbitaceous weeds in the neighbouring areas.
- Moderate to warm temperature (25 ^{O} C) coupled with high relative humidity and reduced sunshine hours favour the disease development.

Management:

- Collect and burn the infected plant debris.
- Spray the crop with fungicides like dinocap (0.06%), carbendazim (0.05%) or hexaconazole (0.05%) or difenoconazole (0.03%) and repeat at 10-14 days interva.
- Precaution: Do not spray sulphur fungicides as cucurbits are sensitive to these.

3) Anthracnose

Symptoms:

- Rough, circular, light brown to reddish brown lesions appear on leaves (Plate-3a).
- On fruits, roughly circular, sunken, water-soaked spots with dark borders (Plate-3b) appear.
- Old spots turn black and are covered with pink spore masses under moist weather.

Plate-3a. Symptoms of anthracnose on leaves

Plate-3b. Symptoms of anthracnose on fruits

Pathogen:

- The disease is caused by *Colletotrichum orbiculare* (Berk. & Mont.) Arx. (Perfect stage: *Glomerella lagenarium* F. Stevens)
- The fungus produces black stromata, bearing black setae and hyaline conidiophores.
- Conidia are produced on conidiophores by budding and are one celled, hyaline, oblong to ovate.

Disease cycle and epidemiology:

- The fungus is both soil and seed borne.
- Cucurbitaceous weed hosts serve as source of perennation.
- Conidia are disseminated by moist wind or rain splashes or carried through implements.
- Spore germination is optimum at 22-27^{o} C temperature and 100 per cent RH.

Management:

- Deep ploughing of crop residues immediately after harvest.
- Use healthy seed and treat seed with carbendazim (0.2%).
- Spray the crop with carbendazim (0.1%) or mancozeb (0.25%) or their combination at 10-14 days interval.

4) Fusarium wilt

Symptoms:

- Initial symptoms appear as yellowing and marginal necrosis of leaves from down to upwards.
- Sudden drooping and wilting of plants.
- Vascular discolouration may or not be present.

Pathogen:

- The disease is incited by *Fusarium oxysporum* Schlechtend ex. Fr. f.sp. *cucumerinum* Owen.
- The fungus produces septate mycelium and micro and macro conidia.
- Chlamydospores are also produced.

Disease cycle and epidemiology:

- The fungus is soil borne and survives as chlamydospores.
- A temperature between 18-22^{o} C, high light intensity and relative humidity favour the disease development.
- High nitrogen and low potassium enhance disease incidence.

Management:

- Follow crop rotation with non cucurbitaceous crops.
- Soil solarization for 40-45 days during summer months.

- Apply soil amendments like margosa and mustard cake (250 g/m^2).
- Application of non-pathogenic isolates of *Fusarium* or *Pseudomonas putida* as bioagents also help in the management of this disease.
- Drench the plants with carbendazim (0.1%).

B) BACTERIAL DISEASES

5) Angular leaf spot

Symptoms:

- On leaves water-soaked spots delimited by veins appear (Plate-4).
- A crust of bacterial ooze is formed on them and dead tissue may drop off leaving shot holes.
- The spots on fruits are circular, brown to black and superficial.

Plate-4. Symptoms of angular leaf spot

Pathogen:

- The disease is caused by bacterium *Pseudomonas syringae* pv. *lachrymans* (Smith& Bryan) Young *et al* .
- It is Gram -ve, aerobic, non spore forming, straight rods, motile with 1-5 polar flagella.

Disease cycle and epidemiology

- The bacterium overwinters in infected seed as well as in plant debris.
- Optimum temperature for disease development is 20-24^o C and moisture is most important factor for disease development and spread.

Management:

- Follow crop rotation.
- Collect and destroy infected plant debris.
- Use disease free seed produced in dry areas and soak them in Streptocycline (100 ppm) for 30 minutes and dry them under shade before sowing.
- Spray the crop with Streptocycline (100 ppm) or copper oxychloride (0.3%) and repeat at 10 to 14 days interval.

C) VIRUS DISEASES

6) Cucumber Mosaic

Symptoms:

- Most strains of virus cause systemic infections, which are sometimes symptomless.
- The virus cause severe mosaic, mottling, chlorosis, necrosis and distortion in leaves and fruits.

Pathogen:

- The disease is caused by Cucumber Mosaic Virus which belongs to genus *Cucumovirus.*
- The particle size of virus is 29nm.

Disease cycle and epidemiology:

- The virus has wide host range including members of cucurbitaceae, solanaceae, malvaceae, brassicaceae and leguminosae.
- It is transmitted in non persistent manner by aphids like *Myzus persicae* and *Aphis gossypii.*
- The virus is also known to be transmitted mechanically and through seed.

Management:

- Planting barrier crops, applying sticky traps or covering the ground with an aphid deterrent material like aluminium foil boards can help in reducing the disease incidence.
- Use of virus free seed together with erradication of virus reservoirs are effective.

Other important diseases are: Bacterial wilt: *Erwinia tracheiphila* (Smith) Bergey *et al*.,

Seed rots and damping -off : *Pythium aphanidermatum* (Edson) Fitzp., *Rhizoctonia solani* Kuhn, Squash mosaic : Squash mosaic virus (Comovirus).

Lecture-15

DISEASES OF GINGER AND TURMERIC

Ginger (*Zingiber officinale* Roscoe) and Turmeric (*Curcuma longa* L.) are two of the most important spice crops of India. These are severely affected by rhizome and foliar diseases. The symptoms, pathogen, their disease cycle and epidemiology and management practices are described in detail in this lecture.

Aim: To know about different diseases infecting ginger and turmeric and their management.

DISEASES OF GINGER

1. RHIZOME ROT

Symptoms:

- Two types of symptoms i.e. wet and dry rot (Plate 1a and 1b) are generally noticed on rhizomes.
- The initial symptoms of the disease are noticed on leaves which turn slightly pale.
- The yellowing of the leaves starts from the tip of the blade and spread downwards. Ultimately the infected leaves are killed which droop and hang down along the pseudostem (Plate 1c).
- The basal portion of the plant exhibits pale translucent colouration which becomes water soaked and soft.
- The rotting extends from collar regions to rhizomes.
- Lesions gradually decompose forming a watery mass of putrifying tissues enclosed by the tough rind of the rhizome (Plate 1d) and the fibrovascular strands are not affected.
- Roots arising from the affected regions of rhizome also show typical softening and rotting.

Plate1a. Dry rot

Plate1b. Wet rot

Plate1c. Field showing infection of rhizome rot **Plate1d. Rotten rhizome**

Plate-1. Symptoms of Rhizome rot of ginger

Pathogen (s):

- The disease is caused by different species of *Pythium* and *Fusarium* amongst these important are *P. aphanidermatum* (Edson) Fitz., *P. butleri* Subram., *P. complectens* Braun, *P. deliense* Meurs., *P. gracile* (de Bary) Schenk, *P. graminicolum* Subram., *P. myriotylum* Drechsler, *P. pleroticum* T. Ito; *Fusarium solani* (Mart.) Sacc. and *F. equiseti* (Corda) Sacc.
- The hyphae of *P. aphanidermatum* are hyaline, branched, non septate which form long tapering sporangia by swelling of the hyphae.
- The sporangium blows out into a vesicle to which the protoplasm migrates.
- Zoospores are formed in vesicle and are kidney shaped, biflagellate and slightly depressed at the hilum end.
- The oospores are smooth walled, plerotic and spherical in shape.
- *Fusarium equiseti* produces micro and macro conidia.
- Micro conidia are aseptate whereas macro conidia are 1-3 septate, sickle to spindle shaped.

Disease cycle and epidemiology:

- The pathogen (s) perpetuate through diseased rhizomes as well as through oospores.
- The infected plant debris remaining in the field serves as primary source of infection.
- Oospores have also been detected in the scales of stored rhizomes.
- A warm and humid climate predisposes the plants to infection at sprouting stage.
- The spread of the disease is typical of soil borne diseases.
- For germination of *Pythium* spp. like *P. aphanidermatum* and *P. myriotylum*, the optimum temperature is 34^{o} C and for *P. pleroticum* is 25-30^{o} C whereas for growth and multiplication of *F. equiseti*, the optimum temperature is 30^{o} C.

Management:

- Use disease free seed rhizome for planting and treat by steeping them in the mixture of mancozeb (0.25%) and carbendazim (0.1%) for 60 minutes before storage as well as sowing followed by drying in shade for 48 hours.
- Follow at least 2-3 years crop rotation.
- Apply neem cake (250 g/m^2) and lime as soil amendment.
- The antagonistic species of *Trichoderma* such as *T. viride, T. harzianum* and *T. hamatum* are known to inhibit the growth of the pathogenic fungi (40 g/m^2).
- Drench the affected plants with copper oxychloride (0.3%) or Bordeaux mixture (4:4:50) during rainy season.

2. PHYLLOSTICTA LEAF SPOT

Symptoms:

- On leaves, small oval to elongated spots appear which later on develop white papery centre and dark brown margins with a yellow halo (Plate 2).
- The affected leaves suffer from extensive desiccation and are shredded and disfigured.

Plate 2. Symptoms of Phyllosticta leaf spot of ginger

Pathogen:

- The disease is caused by *Phyllosticta zingiberi* T.S. Ramakr.
- The fungus forms amphigenous, subglobose, dark brown, ostiolate pycnidia on the host. Pycnidiospores are hyaline, unicellular, oblong and biguttulate.

Disease cycle and epidemiology:

- The fungus overwinters in infected plant debris and seed rhizomes.
- The disease begins to appear towards the end of June when temperature varies from 23.4 to 29.6 and RH is in between 83.3 to 90.2 per cent.
- Later, in July when rainy days and total rainfall increases, the disease aggravates and spread fast.

- Higher intensity of rain, accompanied by wind, exert greater impact on target leaves and fungus is splashed to a greater distance resulting in increased disease incidence.

Management:

- Collect and destroy the infected plant debris by burning.
- Use healthy seed rhizomes and by steeping them in the mixture of mancozeb (0.25%) and carbendazim (0.1%) for 60 minutes before sowing and dry in shade for 48 h. Spray the crop with mancozeb (0.25%) or carbendazim (0.1%) or combination of mancozeb (0.25%) and carbendazim (0.05%) and repeat at 10 to 14 days interval.
- Few cultivars like Narasapatom, Tura, Nadia, Tetraploid and Thingpani are moderately resistant to the pathogen.

B) BACTERIAL DISEASES:

3. BACTERIAL WILT

Symptoms:

- Water-soaked patches or streaks appear on collar region which slowly enlarge.
- Bronze colouration on leaf margins is also observed and the leaves slowly become flaccid with intense yellowish bronze colour and ultimately droop down exhibiting typical wilt symptoms (Plate 3a).
- At advanced stages, pseudo stem appears slimy and if infected rhizomes are pressed, a milky bacterial exudates oozes out in clear water (Plate 3b).

Plate 3. a) Symptoms of bacterial wilt b) Milky bacterial ooze of bacteria in water

Pathogen:

- The disease is caused by bacterium *Ralstonia solanacearum* (Smith) Yabuuchi *et al*.
- The bacterium is described in detail under tomato lecture.

Disease cycle and epidemiology:

- The bacterium perpetuates through infected rhizomes and soil. Biotype III of the bacterium is known to cause this disease in India.
- The bacterium has wider host range including both cultivated and weeds.
- The presence of nematodes has a positive and significant effect on the development of bacterial wilt.

Management:

- Use healthy seed rhizomes for planting.
- Eradicate weed hosts and adopt effective crop rotation for at least three years.
- Treat seed rhizomes with Streptocycline (100 ppm) for 30 minutes before planting and spray the crop with Streptocycline (100 ppm) starting from 1 month after transplanting and repeat at fortnightly interval.

C) NEMATODE DISEASES

4. ROOT-KNOT

Nematodes can attack ginger and turmeric such as root-knot nematode (*Meloidogyne incognita*).This nematode has a very wide-host range and heavy infections may render drastic reduction of ginger yield.

Symptoms:

- The symptoms are similar to root gall as on tomatoes, cucurbits, lettuce, and other vegetable crops.
- Root knot infected ginger may have stunted growth with partial yellowing of plants.
- On roots irregular round galls and spindle-shaped enlargements appear on the tap and side roots (Plate-4).
- The nematode larvae feed on roots causing the swellings or knots that are characteristic of root-knot infection.
- Roots are often stunted and deformed

Plate 4. Symptoms of root knot on ginger plant

Pathogen:

- Root-knot nematode is caused by the plant parasitic nematode, *Meloidogyne incognita*.
- Root knot nematodes are sedentary, endoparasitic and gall producing nematodes.
- The infective stage is the second stage juveniles, which have lightly sclerotized cephalic framework.
- The stylet in juveniles and adult female is weak.
- Third and fourth stage juveniles lack stylet and body is saccate.
- Adult females are swollen, pyriform, saccate with short neck, vulva is terminal, perineal cuticular pattern of striae is present, tail is absent, didelphic, prodelphic ovaries are coiled, excretory pore is present near stylet base, large rectal glands produce gelatinous matrix expelled through the anus.
- Males are vermiform with single or paired testis, tail end is twisted and rounded, bursa is absent, reproduction is parthogenic.

Disease cycle and epidemiology:

- The second stage juveniles in the soil after searching a suitable site, normally behind the root cap, start feeding on epidermal cells and penetrate in cortical layers of root.
- They reach upto the stellar region, where they form giant cells for feeding.
- Second stage juveniles initiate gall formation and feed for 7-15 days, undergo three moults to become adult.
- The total time taken for completing one life cycle under optimum conditions is 3-4 weeks.
- Moderate temperature (29-30^{o}C) and higher relative humidity (79-80%) favour *M. incognita* reproduction and spread.

Management:

- Crop rotation with non-hosts, graminaceous poor hosts and a few antagonistic crops for one or two years have been reported to be effective in reducing the population of root knot nematode.
- Groundnut-mustard rotation was found most effective in reducing the population of *M. incognita*.
- Soil amendments (dry or green crop residues, oil cakes, meals, sawdust, FYM etc.) are allowed to decompose in the nematode infested field, which in turn helps in alteration of physical, chemical and biotic conditions of the soil.
- At the time of field preparation, nematicides like Thimet or Phorate (12-15 kg/ha) should be applied and immediately apply light irrigation. After 15-20 days again cultivate the field and sow the crop.
- Efficacy of various biocontrol agents like *Paecilomyces lilacinus*, *Pasteuria penetrans* and *Pseudomonas fluorescens* in managing *M. incognita* has also been reported.

Other diseases of importance:

i. **Yellows :** *Fusarium oxysporum* Schlechtend f.sp. *zingiberi* Treujillo
ii. **Chlorotic fleck :** Ginger Chlorotic Fleck virus
iii. **Big bud :** Tomato Big Bud Phytoplasma

DISEASES OF TURMERIC

1. RHIZOME ROT

The yield is considerably reduced due to the attack of this disease.

Symptoms:

- The affected plants show progressive drying of the leaves, which proceeds first along the margins and later the entire leaf dries up.
- The base of the aerial shoots shows water soaked soft lesions.
- The root system is adversely affected and as the disease progresses, infection gradually passes to the rhizomes, which begin to rot and become soft.
- The colour of the affected rhizomes is changed into different shades of brown.

Pathogen:

- Different fungi like *Pythium aphanidermatum* (Edson) Fitzp., *P. graminicolum* Subram. and *Fusarium solani* (Mart.) Sacc. have been reported to be associated with this disease.
- The pathogen is described in detail in ginger diseases.

Disease cycle and epidemiology:

- These fungi are both seed and soil borne.

- High temperature and soil moisture favour the disease development.

Management:

i. Select healthy seed rhizomes for sowing.
ii. Collect and destroy the infected clumps and burn them.
iii. Follow atleast three years crop rotation.
iv. Use tolerant cvs. like PCT-13, PCT-14, and Shillong.
v. Dip seed rhizomes in the solution of metalaxyl + mancozeb (0.25%) before sowing and with the initiation of the disease, drench the plants with the same chemical.

2. TAPHRINA LEAF BLOTCH

This disease was first described by Butler (1911) and now it has been observed in all the turmeric growing areas of the country. Severe outbreaks of this disease have been reported from Rayalaseema area of Andhra Pradesh.

Symptoms:

- Several spots of the disease appear on both surfaces of leaves, being generally more numerous on the upper surface.
- Spots of 1-2 mm in diameter, first appear as pale yellow discolouration not sharply defined from the rest of the tissue, become dirty yellow and then deepen to the colour of old gold and some times to bay shade.
- The infected leaves are distorted, have a reddish brown appearance and become yellow much sooner in comparison to the normal ones.

Pathogen:

- The disease is caused by *Taphrina maculans* Butler.
- The fungus produces cuboid ascogenous cells several layers in depth in the subcuticular interspaces of the epidermis.

Disease cycle and epidemiology:

- The fungus persists by means of ascogenous cells on the leaf debris and as desiccated ascospores and blastospores on the soil and amongst fallen leaves.
- Ascospores cause primary infections.
- Optimum temperature for infection is 21-23^{o} C along with 80 per cent RH.

Management:

i) Collect and destroy the infected plant debris.
ii) Use resistant cvs. like CLL 324, Amalapuram, Mydukur, Karhadi local, CLL326, Ochira 24 and Alleppey.

iii) With the initiation of the disease, spray the crop with funcides like carbendazim (0.1%) or thiophanate methyl (0.1%) or mancozeb (0.25%) or propineb (0.25%) and repeat at 10-14 days interval.

3. LEAF SPOT

This disease was first reported from Coimbatore districts in Tamil Nadu and is now prevalent in all the turmeric growing tracts in India. The disease mostly appears during August and September when the humidity is high.

Symptoms:

- The disease manifests in the form of elliptic or oblong spots of variable size.
- In the initial stages, the spots remain small but very soon many of them increase in size.
- Many spots coalesce and develop irregular patches often involving a major portion of the leaf which eventually dries up.
- The centre of the spots is greyish white and thin with numerous black dot like acervuli on both surfaces.
- There is an indefinite yellowish region outside the spot forming a halo around.
- The infected field presents a scorched look during the years of epiphytotics.

Pathogen:

- The disease is caused by *Colletotrichum capsici* (Syd.) Butler & Bisby.
- The pathogen is described in detail in bell pepper and chili diseases lecture.

Disease cycle and epidemiology:

- The pathogen may be carried over in the rhizomes as dormant stromata between the scales.
- The disease appears when there is high and continuous humidity in the atmosphere.

Management:

i) Use healthy seed rhizomes.

ii) Use resistant cvs. like Nallkatla, Sugandham Duvvur and Ganikota.

iii) With the initiation of the disease, spray the crop with carbendazim (0.1%) and repeat at 15 days interval.

Lecture-16

DISEASES OF POTATO - I

Potato (*Solanum tuberosum* L.) is an important vegetable crop and is cultivated in an area of about one million hectares in India. One to three crops per year are taken under varied agroclimatic conditions. The cultivation of this crop is often affected due to attack of various diseases caused by fungi, bacteria, viruses and nematodes. This lecture deals with the important fungal disease problems in potato and their management.

Aim: To know about different fungal diseases infecting potato and their management - I.

A. FUNGAL DISEASES - I

1. LATE BLIGHT

Late blight is the most destructive disease of potato throughout the world. It was first recorded in the beginning of nineteenth century in the Andes Mountains of South America. The fungus moved from this place to Europe around the year 1842 and established in Ireland where it caused severe epidemic in 1845-46, resulting in famous Irish famine. Four million people were directly affected by this epidemic and many (approx. 1.5 Million) among them faced death with millions migrating to other parts of the world. Since then, the disease has been reported from almost all parts of the world wherever potato is grown. In India, the disease was first reported from Nilgiri Hills in between 1870 and 1880. Since then, it is appearing in plains as well as in hills regularly. In Himachal Pradesh, disease was first reported from Kumarsain of Shimla District by Butler (1903).

Symptoms:

- The symptoms of the disease appear on leaves, stem and tubers.
- Initially small water-soaked lesions develop near the tips and margins of the leaves which rapidly grow into large, brown to purplish black, necrotic lesions under favourable weather conditions (Plate 1a).
- During morning hours, whitish downy growth of the pathogen consisting of sporangiophores and sporangia can be seen on the edges of the lesions mostly on the underside of the leaves.
- Light brown to dark brown lesion appear on stem and petioles which may elongate later and girdle the affected parts (Plate 1b.)
- Since the disease is polycyclic in nature, the entire crop in the field may be killed in one or two weeks and field give blighted appearance (Plate 1c)..
- The tubers may get infected by rain washed sporangia from the diseased foliage.

- The infected tuber show irregular, small to large, slightly depressed areas of brown to purplish skin which extend deep into the internal tissue of the tubers (Plate 1d).
- The infected tuber tissue which is firm and dry in the beginning is often invaded by secondary pathogens, mainly bacteria both in the field or poorly ventilated storage places resulting in to soft rot of tubers.

Plate1ba. Blightening symptoms on stem Plate1ab. Blightening symptoms on leaves

Plate1c. View of late blight infected field Plate1d. Late blight infected potato tubers

Plate-1. Symptoms of late blight of potato (Courtesy T.S. Thind)

Pathogen:

- The disease is caused by *Phytophthora infestans* (Mont.) de Bary.
- The mycelium is coenocytic, endophytic, hyaline, branched and intercellular.
- Sporangiophores arise from the internal mycelium through stomata and are slender, hyaline, sympodially branched, indeterminate, relatively thick walled, septate having side branches with swollen base (Plate 2).
- Sporangia develop on the tip of the sporangiophores and are hyaline, thin walled, lemon shaped, distinctly papillate.

- On maturity, the sporangium turns to the side, growth of the sporangiophore continues which looks to have zigzag growth with characteristics swellings at the nodes.
- *P. infestans* is heterothallic for sexual reproduction and involves two mating types namely A 1 and A 2.
- In India, although A 1 mating type is of common occurrence, prevalence of A 2 type has been recorded in Shimla Hills after 1984.
- Antheridia and oogonia are produced after A 1 and A 2 mating types come in close contact. Antheridia are amphygynous while oogonia are spherical.
- Oospores are thick walled and develop after fertilization.

Plate-2 Sporangiophore and sporangia of *Phytophthora infestans*

Disease cycle and epidemiology:

- The fungus perpetuates as dormant mycelium in the infected tubers in the cold storage or in infected tubers left in the field at temperatures <30^{o} C (Fig. 1).
- Infected tubers transported from hills may also carry latent infections.
- Oospores even if formed rarely, also serve as source of primary inoculum.
- Tomato growing in the vicinity of potato may produce initial inoculum if infected early and act as collateral host.
- The infected tubers generally give rise to diseased haulms on which the pathogen sporulates during suitable weather conditions, thus providing primary sporangial inoculum for further spread.
- Cool moist conditions favour the disease spread while dry spells kill the fungus quickly.
- Relative humidity >90 per cent coupled with suitable temperature (12-24^{o} C) is most important for disease development.

- The sporangia germinate at temperature ranging from 2-30^{o} C.
- Temperature around 16-18^{o} C are optimum for mycelial growth while 9-16^{o} C are optimum for sporulation.
- At a temp. 12-13^{o} C, the sporangia germinates by producing zoospores while direct germination of sporangia takes place around 24^{o} C.
- Initial low temperatures thus, can help in assuming the high disease severity in the crop.

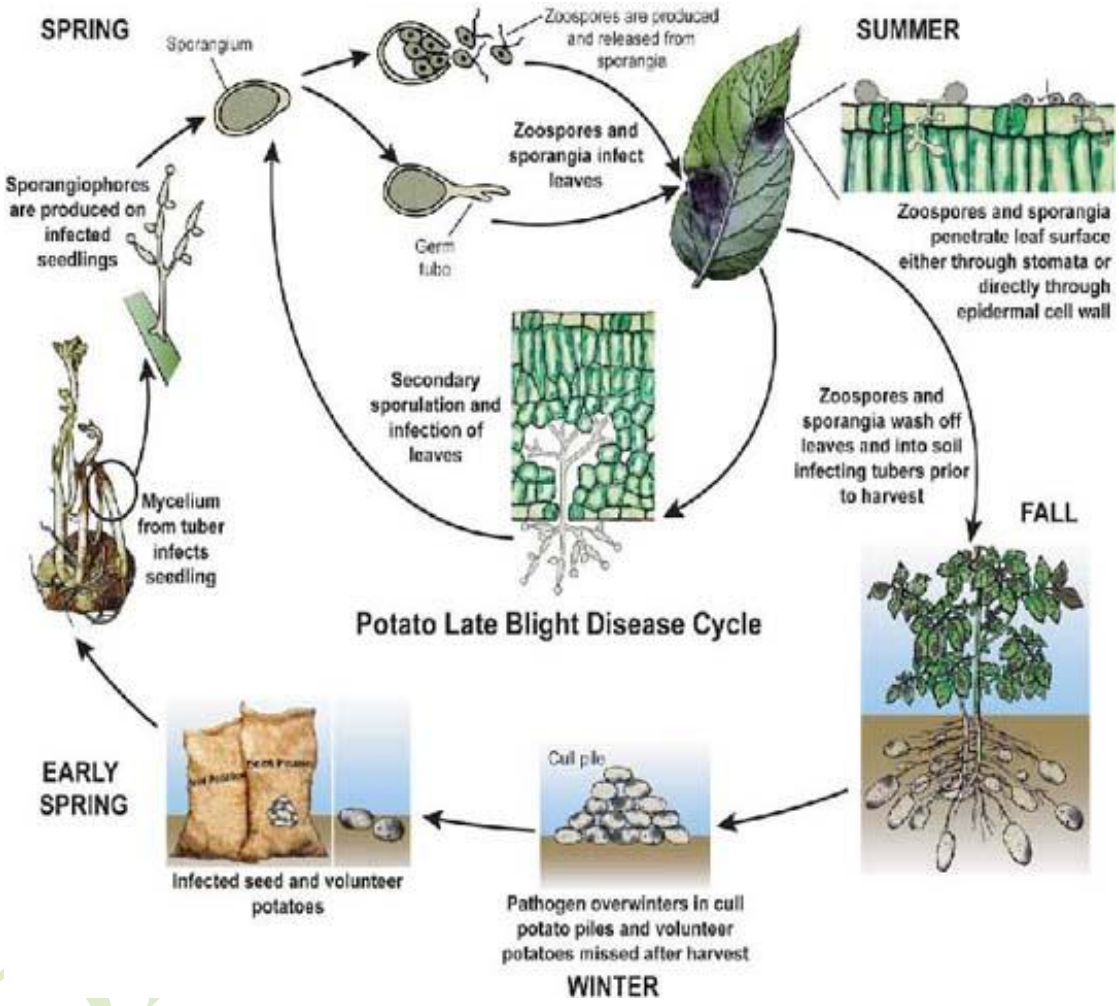


Fig.1. Disease cycle of late blight of potato

Forecasting:

- Several methods of late blight forecasting have been developed for better disease management through proper planning of spray programme.
- These are primarily based on record of temperature and rainfall or relative humidity and predict the probability of the late blight development assuming the presence of inoculum. “BLITECAST”, a forecasting model is being used in North-eastern U.S.A. for timing fungicide application.

- Such disease prediction rules have also been developed in Netherlands (Dutch Rules) and England (Beaumont Rules).
- In India, CPRI people achieved complete success in predicting outbreaks of late blight in the hills. According to them:-

i) the 7 day moving rainfall (30 mm for Shimla, 28.9 mm for Ootacamund and 38.5 mm for Shillong) and mean temperature of 23.9^{o} C or less continues for 7 days, the disease would appear within 3 weeks and if

ii) hourly temperature ranging between 10-20^{o} C associated with 80 per cent or more relative humidity continues for 18 h for 2 consecutive days, the blight would appear within a week.

Management:

- Use healthy seed and cull piles near cold stores should be destroyed.
- High ridges and proper earthing up prevents tuber infection.
- Restrict irrigation during cloudy days.
- Use resistant varieties like Kufri Giriraj.
- Give timely application of fungicides like mancozeb/ chlorothalonil/ propineb (0.25%) as prophylactic sprays (1st week of November for main crop in plains and IIIrd week of June in hills) and repeat at weekly intervals.
- Repeat sprays promptly after rain. Use metalaxyl + mancozeb or cymoxanil + mancozeb (0.25%) only when disease risk is likely to be severe and favourable weather conditions persist.
- Avoid the use of formulations containing metalaxyl alone.
- Digging of tubers should be done after 2-3 weeks of dehaulming.

2. EARLY BLIGHT

Symptoms:

- The symptoms first appear on older leaves as dark brown oval or angular spots or lesions surrounded by chlorotic zone, which may extend much beyond the lesion due to the presence of toxin “alternaric acid” produced by the pathogen (Plate 3).
- Under favourable climatic conditions, they enlarge rapidly, become irregular and may involve entire part of the leaf lamina.
- Concentric ridges appear on the necrotic tissue giving them target board appearance, lesions on leaf coalesce together and give blight appearance.
- Dark brown lesions are produced on stem and petioles, which break at the point of infection.

- On the tubers, the lesions are slightly dark, sunken and round to irregular in shape, with time infected tubers show dry rot symptoms.

Plate 3. Symptoms of early blight

Pathogen:

- The fungus responsible for this disease is *Alternaria solani* Sorauer.
- The mycelium is septate and pale to olivaceous brown.
- Conidiophores arise singly or in small fascicles, through stomata from the mycelium present in the dead centres of the spots.
- Conidia are usually solitary, obclavate, oblong to ellipsoid, tapering to a beak almost of the same length or even longer as that of the conidial body.
- Conidia are muriform, pale to golden or olivaceous brown having 9-11 transverse and 0 to few longitudinal septa.
- No perfect stage of the fungus has been recorded.

Disease cycle and epidemiology:

- The fungus overwinters as mycelium or spores in the infected plant debris.
- The pathogen also infects other hosts like tomato as well as solanaceous weeds which may play an important role in its survival.
- Primary infection occurs on older leaves early in the season.

- In general, hot and humid climate with frequent rains is favourable for disease development.
- A temperature range of 26-28^{o} C with 70-80 per cent RH is favourable for the development of the disease.
- The disease is often more severe when the plants are predisposed by injury, poor nutrition or other types of stress.

Management:

- Various cultural practices like removal and destruction of diseased haulms from the infected field after harvest, use of healthy seed tubers for planting and proper fertilization of the plants should be followed.
- With the initiation of the disease, spray the crop with mancozeb (0.25%) or chlorothalonil (0.2%) and repeat at 10-14 days interval.
- Resistance source is available with *Solanum phureja* and *S. chacoense* which can be exploited for breeding resistant cultivars.
- Cv. Kufri Sindhuri provides good resistance against this disease in plains.

Lecture-17

DISEASES OF POTATO - II

Potato (*Solanum tuberosum* L.) is an important vegetable crop and is cultivated in an area of about one million hectares in India. One to three crops per year are taken under varied agroclimatic conditions. The cultivation of this crop is often affected due to attack of various diseases caused by fungi, bacteria, viruses and nematodes. This lecture deals with the other important fungal disease problems in potato and their management.

Aim: To know about different fungal diseases infecting potato and their management - II.

A. FUNGAL DISEASES - II

3. BLACK SCURF

Black scurf causes qualitative damage as it decreases the quality and market value of the tubers both for table as well as seed purpose.

Symptoms:

- Stem canker and blight phase and the scurf phase are the two distinct phases of the disease.
- In stem canker phase, the growing tips of sprouts show browning.
- Sunken, circular or elongated brown necrotic spots may also be observed on the sprouts.
- Severely affected sprouts are killed.
- Later, when shoots emerge, similar necrotic lesions are observed on the stem which may extend downwards and may completely girdle the stem.
- The most prominent symptom of black scurf is the presence of black crust on tubers due to the formation of sclerotia of the fungus (Plate 4).
- The pathogen produces a large number of sclerotia superficially on the surface of growing tubers.
- These sclerotia may be hard or spongy forming a black crust of scurf on the tuber surface.
- These are normally seated on the skin and do not cause any damage to the tuber inside.
- Black scurf phase is more common than stem canker in India.

Plate-4. Symptoms of black scurf on potato tubers

Pathogen:

- The disease is caused by *Rhizoctonia solani* Kuhn.
- The perfect stage of the fungus is *Thanatephorus cucumeris* (Frank) Donk.
- The fungus is described in detail in bean diseases.

Disease cycle and epidemiology:

- The fungus perpetuates in the form of sclerotia on the seed tubers or in the soil.
- On germination in the presence of moisture, the sclerotia produce mycelium which may cause infection on the sprouts, stem or young tubers.
- The fungus can survive in soil for many years.
- The fungus although has a wide host range but shows some level of specificity depending on the host crops.
- The strains infecting members of graminicolous crops do not generally cause infections on solanaceous crops including potato and *vice verse*.
- Soil moisture and temperature are the two important factors affecting scurf development.
- Low temperature and high soil moisture are conducive for infection on sprouts and stems.
- This phase is not common on early autumn crop in North Indian plains as the temperature remains high.
- The more common phase is black scurf where the sclerotia formation is favoured at temperature 28^{o}C.
- The number of sclerotia on tubers varies with soil temperature and moisture.
- Black scurf on tubers is more common in crops grown in sandy to sandy loam and moderately wet soils.

Management:

- Planting of healthy and disease free tubers is helpful in reducing the incidence of the disease.
- Two to four years rotation with cereals, brassicas and legumes is helpful for the management of this disease.
- The increase in organic matter content of the soil helps in reducing the population of the fungus through enhanced activity of the antagonist microorganisms.
- The normal unsprouted tubers should be dipped in carbendazim (0.1%) or Monceren 25 SC (0.25%) for 10 minutes.
- The seed after treatment should be dried under shade by spreading on the floor of the cold store.

4. POTATO WART

The disease is quite serious in temperate regions of the world. In India, the disease was first reported by Ganguli and Paul (1953) from Darjeeling and is presently restricted to that area only due to restrict domestic quarantine on this disease.

Symptoms:

- All the underground parts except roots are attacked.
- The plant cells multiply rapidly at the infection site and produce hypertrophied tissue masses resulting into wart type symptoms on the tubers and beads like projections on the stems or stolons varying in size from small protuberances to large intricately branched structures (Plate-5 a &b).
- Warts are normally soft, pulpy, spherical and similar in colour to tubers.
- The colour may change to green on exposure to sunlight.
- Sometimes whole tuber may be covered with warts.
- Sometimes secondary microorganisms invade the wart tissues causing their decomposition.

Plate 5a. Warts on tuber

Plate 5b. Wart infected plant

Plate 5. Symptoms of potato wart

Pathogen:

- The disease is caused by fungus *Synchytrium endobioticum* (Schill.) Perc. which is holocarpic and endobiotic in nature.
- The fungus produces sporangia which release a large number of uniflagellate planospores and originate from different sori, these behave as planogametes and copulate to form zygospores which act as resting sporangia of the pathogen.

Disease cycle and epidemiology:

- The resting sporangia are released into the soil on disintegration of warty structures where they can remain dormant for several years.
- When soil becomes wet after irrigation or rainfall in the next spring, these resting sporangia germinate and cause fresh infections.
- The longevity and viability of resting spores are affected by soil type and crop rotation.
- Other factors like soil moisture and pH also affect the process of infection.
- For germination of resting spores and sporangia, a free film of water is necessary.
- When moisture conditions are available, infection can occur at temperatures ranging from 10-24^{o} C and at soil pH ranging from 4 - 8.6.
- The optimum temperature for infection is 21^{o} C and slightly acidic or neutral pH is preferred.

Management:

- Since the pathogen is soil borne, its eradication is very difficult.
- Strict domestic quarantine measures enforced in India have helped to restrict the disease to Darjeeling district only.
- Follow crop rotation with non-host crops for 8-10 years.
- Some potato lines viz., Rondo, Edina, Mira from European countries have shown considerable resistance to wart.
- Varieties developed by CPRI, Shimla like Kufri Jyoti, Kufri Sherpa, Kufri Jeevan and Kufri Muthun have demonstrated resistance to late blight as well as wart.

Lecture-18

DISEASES OF POTATO - III

Potato (*Solanum tuberosum* L.) is an important vegetable crop and is cultivated in an area of about one million hectares in India. One to three crops per year are taken under varied agroclimatic conditions. The cultivation of this crop is often affected due to attack of various diseases caused by fungi, bacteria, viruses and nematodes. This lecture deals with the important bacterial and viral disease problems in potato and their management.

Aim: To know about different bacterial and viral diseases infecting potato and their management.

A) BACTERIAL DISEASES

1. BROWN ROT

Symptoms:

- Both above and underground plant parts are affected.
- There is a sudden wilting and death of the infected plants or collapse of one or more branches is observed.
- Wilting appears during July in the hills about two weeks after the onset of monsoon rains (Plate 1a).
- Many wilted plants also show stem rot at soil level.
- Greyish brown discolouration appears through the stem of affected tubers (Plate 1b).
- Cross-section of such tubers reveals a distinct brown discolouration in the vascular ring.
- A slight pressure on the cut tuber causes oozing of typical grayish white bacterial slime out of the vascular ring.
- Vascular browning is a characteristic symptom of the disease.
- In North-eastern hills, lenticels infection of potato tubers in the form of water-soaked lesions is quite common.

Plate 1a. Symptoms on plant **Plate 1a. Symptoms on tuber**

Pathogen:

- The bacterium associated with this disease is *Ralstonia solanacearum* (Smith) Yabuuchi *et al.*
- It is a rod shaped, Gram negative bacterium measuring 0.5-0.6 x 0.8-1.2 um and having 1-4 polar flagella.
- Three races of the pathogen have been identified and potato is affected by race 1 and 3.

Disease cycle and epidemiology:

- *R. solanacearum* is both soil and seed borne and can survive in infected crop debris, potato tubers, wild host plants and weeds.
- Infested soil and infected seed tubers serve as sources of primary inoculum.
- Bacterial masses released in the soil by brown rot affected plant parts help in the survival of this bacterium.
- This bacterium survives in the soil for extended periods and enters the roots through wounds made by transplanting, cultivation or insects and through natural wounds where secondary roots emerge.
- A high temperature regime (28-30^{o} C) and relatively high soil moisture are favourable for wilt development.
- The bacteria multiply rapidly inside the water-conducting tissue of the plant, filling it with slime.
- This results in a rapid wilt of the plant, while the leaves stay green.
- If an infected stem is cut crosswise, it will look brown and tiny drops of yellowish ooze may be visible.

Management:

- It is difficult disease to manage as the commercial cultivars are highly susceptible and chemical control is not feasible.
- The reduction of the initial inoculums is of prime importance to manage bacterial wilt.

- Use of disease free seed is important for avoiding introduction of this disease to new fields.
- In disease prone areas, plant whole tuber.
- Follow wheat-maize rotation at least for 2-3 years.
- Application of stable bleaching powder @ 12 kg/ ha has been found to reduce bacterial wilt by 80 per cent when applied in furrows at the time of planting.

2. COMMON SCAB

Symptoms:

- Symptoms of scab are generally seen on tubers and are categorized as shallow and deep pitted.
- In shallow scab, the affected tubers show superficial roughened areas, sometimes raised about but often slightly below the skin of the tubers.
- The lesions of scab consist of corky tissue which is the result of abnormal proliferation of the cells of tuber epiderm due to invasion by the pathogen.
- The lesions may vary in shape and size and the color is brown.
- In deep pitted scab, the lesions are dark brown or almost black and measure 3-4 mm or more in depth surrounded by hard corky tissue.
- They may join together involving entire surface of the tuber.
- Quite often, multiple types of symptoms such as slightly brownish roughening of tuber skin, proliferated lenticels with hard corky deposition, concentric series of wrinkled layers of cork around central black core, raised rough and corky pustules and deep pits surrounded by hard corky tissue are produced.
- Russet scab in the form of superficial encrustation is also becoming quite common in the state.

Pathogen:

- The disease is caused by a bacterium *Streptomyces scabies* (Thaxter) Waksman and Henrici.
- The mycelium of the pathogen is slender, branched with few or no cross walls.
- The spores are cylindrical or ellipsoid produced on special hyphae that develop cross walls from tip and spores are pinched off.
- The *S. scabies* is gram positive and aerobic bacteria.

Disease cycle and epidemiology

- *Streptomyces scabies* can survive in soil for several years under favorable conditions.
- The infected tubers play a major role for its survival from one season to the other.
- The diseased plant debris and infested soil help in survival of the pathogen and also in initiating disease in succeeding crop of potato.

- The pathogen can be distributed through movement of soil by wind, water and cultural operations.
- Various soil factors influence the severity of the disease.
- *Streptomyces scabies* is active in the pH range of 5.2-8.0 and the severity of disease increases with the increase in pH.
- A temperature range of 20-30^0 C is most suitable for the growth of the organism.
- Optimum temperature for infection is slightly below 20^0 C and for lesion development slightly above 20^0 C .
- The pathogen is active in dry soils and is suppressed by watering.

Management:

- As the disease is both tuber and soil borne, it is difficult to control it.
- However, some management practices have been adopted to check this disease.
- Healthy and blemish free seed tubers should be selected for sowing in order to reduce the primary source of inoculum.
- Green manuring and cultivation of certain legumes before planting potato has been emphasized as important cultural practices in controlling common scab.
- Green manuring probably increases the activity of certain actinomycetes and other bacteria which are antagonistic to *S. scabies*.
- Soil moisture is important factors in the development of disease, it has been exploited for minimizing the losses.
- Successful control of potato scab can be achieved by frequent irrigations of the field at weekly intervals from tuberisation until maturity.
- The tubers can be disinfected by dipping them for 10 minutes in Emisan (0.25%).

B) VIRAL DISEASES

1. LEAF ROLL VIRUS (POTATO LEAF ROLL VIRUS)

Symptoms

- There is rolling of upper leaves.
- Infected leaves remain upright and turn pale yellow in colour.
- Plants produced from infected tubers show rolling of lower leaves and stunting of plants with upright habit (Plate 1).
- Rolled leaves are leathery, stiff and brittle. Marginal interveinal chlorosis of leaflets is visible on upper leaves.

Plate 1. Symptoms of potato leaf roll virus on plants

Virus characters

Disease cycle and epidemiology:

- Potato leaf roll virus is transmitted through infected tubers and spreads through several species of aphids.
- It is more severe in warm climate.

2. MOSAIC (*POTATO VIRUS X, POTATO VIRUS Y* AND *POTATO VIRUS S*)

Symptoms:

- PVX induces interveinal mild mosaic symptoms with light and dark green patches, mottling with stunting and often crinkling with more virulent strains.
- There is considerable dwarfing of plants with reduction of leaflet size.
- PVY induces severe or rugose mosaic.
- Veinal necrosis may also occur and plant remain stunted.
- Severe mosaic causes rugosity, bunching or twisting of leaves.
- Symptoms caused by PVS are barely perceptible as mottle or faint vein bending.

Plate 1. Symptoms of potato mosaic virus on plants

Virus characters

Disease cycle and epidemiology:

- Mosaic viruses are transmitted by infected tubers, cutting knife and aphid vectors names .
- Many plants of solanaceae, chenopodiaceae and leguminoseae act as alternate and collateral host hosts of this disease.

Management:

- Always use virus free certified seed from reliable sources.
- Rouge out infected plants as soon as these are first noticed in early crop period.
- Keep the aphid population under check by spraying insecticides like Rogor or Metasystox @ 250 ml/acre or apply 5 kg of Thimet 10G/acre at first earthing up.
- Cut the haulms in 4th week of December when aphid infestation starts (20 aphid /100 leaves) and leave the tubers in soil to mature till the end of February.

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