01 - Diseases of Citrus

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

**Symptoms**

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

**Pathogen**

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

**Favourable conditions**

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

**Mode of spread and survival**

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

**Management**

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

Scab/Verucosis: *Elsinoe fawcetti*

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

**Pathogen**

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

**Mode of Spread and Survival**

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

**Management**

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

**Canker : Xanthomonas campestris pv citri**

**Symptoms**

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

**Pathogen**

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

**Favourable conditions**

Free moisture for 20 minutes, 20-30°C.
Mode of survival and spread

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

Management

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

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

Symptoms

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

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

**Pathogen**

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

**Mode of spread**

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

**Management**

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

**Exocortis of scaly butt: Viroid**

**Symptoms**

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

**Pathogen**

Viroid is free RTVA without protein coat.

**Mode of Spread and Survival**

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

**Management**

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

**Greening: Liberobactor asiaticum** (Phloem limited bacteria)

**Symptoms**

This disease affects almost all citrus varieties irrespective of root stock. Stunting of leaf, sparse foliation, twig die back, poor crop of predominantly greened, worthless fruits. Sometimes only a portion of tree is affected. A diversity of foliar chlorosis. A type of mottling resembling
zinc deficiency often predominates. Young leaves appear normal but soon assume an upright position, become leathery and develop prominent veins and dull olive green colour.

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

**Pathogen**

Rickettsia like organism

**Mode of spread**

Infected budwood; psyllid vector- *Diaphorina citri*

**Management**

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

**Anthracnose: Colletotrichum gloeosporioides**

**Symptoms:**

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

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

**Pathogen**

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

**Mode of survival and spread**

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

**Favourable conditions**

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

**Disease cycle**

The survival of pathogen in detached diseased twigs and leaves lying on surface of soil and in diseased twigs attached to the tree. They successfully reproduced the disease by inoculating leaves, petioles, stems and fruits. The optimum temperature for infection was found
to be 25°C. The disease spreads rapidly in the rainy season. Cloudy and misty weather during flowering favors damage to the infected floral parts.

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

**Management**

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

**Powdery mildew: Oidium mangiferae (Acrosporum mangiferae)**

**Symptoms**

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

**Pathogen**

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

**Mode of survival and spread**

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

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

Management

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

Mango malformation: *Fusarium moliliforme var. subglutinans*

Symptoms

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

Pathogen

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

Mode of spread

Diseased propagatives materials.
Disease Cycle

Management

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

**Symptoms**

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

**Pathogen**

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

**Mode of spread and survival**

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

**Management**

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

**Red-rust:** *Cephaleuros virescens*

**Symptoms**

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

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

Management

Bordeaux mixture (0.6%) or Copper oxychloride 0.25%

**Grey Blight : Pestalotia mangiferae**

**Symptoms**

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

Pathogen

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

Mode of survival and spread

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

Favourable conditions

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

Management

Remove and destroy infected plant parts. Spraying copper oxychloride 0.25 Mancozeb 0.25% or Bordeaux mixture 1.0%.
**Sooty mould :** *Capnodium mangiferae*

**Symptoms**

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

**Favourable conditions**

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

**Management**

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

**Downy mildew: Plasmopara viticola**

**Symptoms**

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

**Pathogen**

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

**Mode of Spread and Survival**

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

Management

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

**Powdery mildew**: *Uncinula necator*

**Symptoms**

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

**Pathogen**

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

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

Disease Cycle

Management

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

Bird’s Eye Spot/Anthracnose: *Gloeosporium ampelophagum* (*Elsinoe ampelina*)

Symptoms

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

Pathogen

Mycelium is septate and dark colored. Conidia single celled oval and hyaline.
Mode of Spread and Survival

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

Disease Cycle

Management

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

Scab – *Venturia inaequalis*

Symptoms

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

Pathogen

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

Mode of Spread and Survival

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

Disease cycle

This disease, caused by the fungus *Venturia inaequalis* (anamorph *Spilocaea pomi*), may be quite severe when rainy, cool weather occurs in the spring. Fungal spores are produced in early spring on dead, fallen apple leaves about the time buds begin to develop. These spores are splashed by rain and blown by wind to land on developing plant tissue and initiate infections. After spots appear on the newly formed leaves, more spores are produced that spread infection to other parts of the tree. Again, rainy weather greatly encourages spore spread and infection during the secondary phase of spore production. The fungus over winters on fallen leaves.
Clean cultivation, collection and destruction of fallen leaves and pruned materials in winter to prevent the sexual cycle. Spray Tridemorph 0.1% before flowering. Spray Mancozeb 0.25 % at bearing stage. Spray 5 % urea prior to leaf fall in autumn and 2 % before bud break to hasten the decomposition of leaves.

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

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

**Powdery mildew – Podosphaera leucotricha.**
Symptom

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

Pathogen

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

Mode of Spread and Survival

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

The mildew fungus over winters mainly as mycelium in dormant blossom and shoot buds produced and infected the previous growing season. Conidia are produced and released from the unfolding leaves as they emerge from infected buds at about tight cluster stage. Conidia germinate in the high relative humidity usually available on the leaf surface at 10-25°C with an optimum of 19-22°C. Germination does not occur in free moisture. Early-season mildew development is affected more by temperature than by relative humidity. Abundant sporulation from over wintering shoots and secondary lesions on young foliage leads to a rapid buildup of inoculum. Secondary infection cycles may continue until susceptible tissue is no longer available. Since leaves are most susceptible soon after emergence, infection of new leaves may occur as long as shoot growth continues. Fruit infection occurs from pink to bloom. Over wintering buds are infected soon after bud initiation. Heavily infected shoots and buds are low in vigor and lack winter hardiness, resulting in a reduction of primary inoculum at temperatures below -24°C.
Management:
Spray Dinocap 0.05% or Chinomethionate 0.1%

**Fire blight - *Erwinia amylovora***

**Symptom**

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

**Pathogen**

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

**Mode of Spread and Survival**

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


**Soft rot – Penicillium expansum**

**Symptom**

Young spots starts from stem end of the fruit as light brown watery rot. As the fruit ripens area of the rotting increases, skin becomes wrinkled. A peculiar musty odour is emitted.
under humid condition a bluish green sporulating growth appears. Infection take place by wounds in the skin caused by insects and during handing in storage and transport.

**Pathogen**

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

**Mode of Spread and Survival**

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

**Disease cycle**

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

**Management**

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

**Bitter rot – *Glomerella cingulata***

**Symptom**

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

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

**Management**

Spray Mancozeb 0.25 % in field. Treatment with Mancozeb 0.25 % to check the disease in storage.
Lecture - Diseases of Chilli

**Damping off: Pythium aphanidermatum**

**Symptoms:**

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

**Pathogen**

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

**Mode of spread and survival**

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

**Management**

Soil drenching with Copper oxychloride 0.25%

**Fruit Rot and Die Back: Colletotrichum capsici**

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

**Pathogen**

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

**Mode of spread and survival**

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

**Management**

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

**Powdery mildew: Leveillula taurica**

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

**Disease cycle**

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

**Management**

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

**Bacterial leaf spot:** *Xanthomonas campestris pv. vesicatoria*

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

**Mode of spread and survival**

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

**Management**

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

**Cercospora leaf spot : Cercospora capsici**

**Symptoms**

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

**Pathogen**

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

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

Management

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

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

**Symptoms**

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

**Pathogen**

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

**Management**

Use of wilt resistant varieties. Drenching with 1% Bordeaux mixture or Blue copper or Fytolan 0.25% may give protection. Seed treatment with 4g Trichoderma viride formulation or 2g Carbendazim per kg seed is effective. Mix 2kg T.viride formulation mixed with 50kg FYM,
sprinkle water and cover with a thin polythene sheet. When mycelia growth is visible on the heap after 15 days, apply the mixture in rows of chilli in an area of one acre.

**Leaf curl**

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

**Mosaic Viruses**

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

**Management of viral diseases**

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

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

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

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

**Conditions for Disease Development**

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

**Management**

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

**Alternaria Rot - *Alternaria sp.***

**Symptoms**

The fungus is reported to enter wounds (sunscald or punctures). Dusty black spores on fruit spots are characteristic. In most instances this disease follows blossom-end rot, but it also follows injuries, chilling, and other decays. On the fruit, large greenish-brown to brown lesions covered, with grayish-brown mold are produced. Similar lesions on the lower-part of the fruit are characteristic of *Alternaria* rot following blossom-end rot. The larger lesions may show
alternating light and dark-brown concentric zones. Shipping peppers under standard refrigeration will check the development of this rot, but when the fruit is removed from refrigeration the decay will advance rapidly at moderate to warm temperatures.

**Pathogen**

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

**Mode of spread and survival**

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

**Management**

**Pre storage dry heat**

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

Late blight of potato: *Phytophthora infestans*

**Symptom:**

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

**Pathogen**

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

**Mode of spread and survival**

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

**Favourable conditions**

RH>90% , Temp.-10-25°C and Night temperature:10°C. Cloudiness on the next day Rainfall at least 0.1mm, the following day.
Management

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

Early blight: *Alternaria solani*

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

**Pathogen**

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

**Favourable condition**

Dry warm weather with intermittent rain. Poor vigor. Temperature: 25-30°C. Poorly manured crop.
Mode of spread and survival

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

Management

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

Post-harvest tuber rots - *Sclerotium rolfsii*

Symptoms

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

Pathogen

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

Favorable condition

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

Mode of spread and survival

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

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

**Black scurf- *R. solani***

**Symptoms**

![Image of black scurf symptoms]

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

**Pathogen**

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

**Mode of spread and survival**

The fungus is capable of leading a saprophytic life on the organic material and can remain viable in the soil for several years. The sclerotia on the seed tubers is the principal source
of infection of the subsequent crop raised with these tubers. On return of favourable conditions the mycelium present in the soil may develop producing new hypae.

Management

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

**Symptoms**

![Image of infected potato tuber](image)

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

**Pathogen**

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

**Mode of spread and survival**

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

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

**Symptoms**

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

**Pathogen**

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

**Favourable condition**

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

**Mode of spread and survival**

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

**Disease cycle**

*R. solanacearum* is a soilborne and waterborne pathogen; the bacterium can survive and disperse for various periods of time in infested soil or water, which can form a reservoir source of inoculum. In potato, the brown rot pathogen is also commonly tuber borne. The bacterium usually infects potato plants through the roots (through wounds or at the points of emergence of lateral roots).
Under favorable conditions, potato plants infected with *R. solanacearum* may not show any disease symptoms. In this case, latently infected tubers used for potato seed production may play a major role in spread of the bacterium from infected potato seed production sites to healthy potato-growing sites. *R. solanacearum* can survive for days to years in infected plant material in soils, infested surface irrigation water, infected weeds, and infected potato washings and sewage. From these sources of inoculum, bacteria can spread from infested to healthy fields by soil transfer on machinery, and surface runoff water after irrigation or rainfall. Infected semi-aquatic weeds may also play a major role in disseminating the pathogen by releasing bacteria from roots into irrigation water supplies.

**Soft rot- *Erwinia carotovora* subsp *caratovora***

**Symptoms**

![Erwinia carotovora](image)

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

**Pathogen**

It is a gram negative rod shaped bacterium with 1 to 6 peritrichous flagella.
**Mode of spread and survival**

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

**Management**

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

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

Cucumber and squash

Vascular Wilt: *Erwinia tracheiphila*

Symptoms

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

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

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

Two cut stem ends can also be put together, squeezed, then separated to look for shiny strands of bacteria. The sap of a healthy plant is watery and will not string. Sometimes it helps to wait several minutes after cutting to perform the test. This technique is useful in field diagnosis to separate this disease from other vascular wilts. Beware, however, that the technique may not always work (i.e., no bacterial strings occur yet the plant is still infected). The test works better
for cucumbers than for muskmelons. Fruit may also show symptoms. Small water-soaked patches form on the surface. These patches eventually turn into shiny decayed spots on the fruit.

**Pathogen**

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

**Mode of spread and survival**

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

**Management**

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

**Scab: Cladosporium cucumerinum**

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

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

**Pathogen**

Conidia are oblong, dark, mostly aseptate.

**Mode of spread and survival**

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

**Disease Cycle**

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

**Management**

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

**Musk melon and water melon**

**Gummy Stem Blight** - *Mycosphaerella melonis*

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

**Mode of spread and survival**

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

**Disease Cycle**

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

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

This can be significant to determine when early-season disease scouting should be initiated for future control. Penetration by conidia is probably direct and does not need to occur through stomata or wounds. Wounding, striped cucumber beetles, and aphid feeding, along with
powdery mildew infection, predispose plants to infection. The additional nutrients provided by such injuries enhance gummy stem blight infection.

Management

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

**Bacterial Wilt -*Erwinia tracheiphila*

**Symptoms**

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

Eventually the entire plant wilts and dies. If you cut through the stem of an affected plant and squeeze both cut ends, a white, sticky exudate will often ooze from the water-conducting tissue of the stem. This exudate is composed of bacterial material that plugs the vascular system of the plant. Affected stems do not appear significantly discolored. Bacterial wilt is closely associated with either the striped or the spotted cucumber beetle. The bacteria over winter in the bodies of adult cucumber beetles. The beetles carry the bacteria when they emerge in the spring.
The bacteria are spread either through the feces of the beetle or from contaminated mouthparts. When the beetles feed on young leaves or cotyledons, they open entry points for the pathogen. Once inside the plant, the bacteria travel quickly through the vascular system, causing blockages that in turn result in wilting of the leaves. The disease progresses from plant to plant when a carrier beetle moves through the field or when clean beetles pick up the bacteria from a diseased plant and fly to healthy plants. Larvae are not known to carry the wilt organism.

**Pathogen**

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

**Mode of spread and survival**

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

**Management**

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

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

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

Mode of spread and survival

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

Management

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

Anthracnose Colletotrichum orbiculare (= C. lagenarium)

Symptoms

The diagnostic features of anthracnose vary with the host. Sunken, elongated stem cankers are most prominent on muskmelon, though leaf and fruit lesions also occur. Large lesions girdle the stems and cause the vines to wilt. Stem cankers are less obvious on cucumbers,
but leaf lesions are very distinct. Watermelon foliage affected by anthracnose appears scorched; sunken fruit lesions are easy to recognize. The anthracnose fungus over winters on diseased crop residue. There also reported that the pathogen is carried in or on cucurbit seed. In wet conditions each spring, the fungus releases airborne spores that begin new infections on vines and foliage. Anthracnose usually becomes established in mid-season, after the crop canopy has fully developed.

**Mode of spread and survival**

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

**Management**

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

**Sudden Wilt**

**Symptoms**

Unlike bacterial wilt, which can occur any time during the season, sudden wilt generally occurs late in the season and is closely associated with a heavy fruit load on the plant. Cucumbers and melons appear to be most sensitive to sudden wilt. Initial symptoms are a slight flagging of the plants in midday even when abundant moisture is present. This flagging will continue to worsen so that, by the third or fourth day, many of the plants are completely wilted. Disease progression is rapid, hence the name sudden wilt. After five to six days, all of the vines have melted down and only the immature fruits are left in the fields. Affected plants appear to lack feeder roots; other roots become slightly misshapen and thick. Currently it is thought that
sudden wilt is caused by a root rot complex involving *Pythium* sp., *Rhizoctonia solani* and *Fusarium* sp. that invade the roots and further colonize the root tissue. It is thought that stresses such as excess moisture and drought, prolonged periods of low temperatures (below 50 degrees F) and attack by the several viruses that commonly affect melons and/or cucumbers individually or in combination weaken plants so that soil-borne pathogens can rapidly colonize the root systems.

**Management**

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

**Powdery mildew - Erysiphe cichoracearum**

**Symptoms**

![Powdery Mildew](Image)

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

**Pathogen**

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

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

**Management**

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

**Alternaria Blight - *Alternaria cucumerina***

**Symptoms**

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

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

**Pathogen**

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

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

Management

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

**Downy mildew - Pseudoperonospora cubensis**

Symptoms

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

Pathogen

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

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

Disease cycle

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

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

Management

Spraying with Metalaxyl 500 g or Metalaxyl + Mancozeb 1 kg/ha or Mancozeb 1 kg/ha.
Angular Leaf Spot - *Pseudomonas lacrymans*

**Symptoms**

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

**Pathogen**

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

**Mode of spread and survival**

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

**Management**

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

**Gourds**

Downy mildew: *Pseudoperonospora cubensis*

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

**Pathogen**

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

**Mode of spread and survival**

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

**Management**

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

**Powdery mildew: Erysiphe cichoracearum**

**Symptoms**

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

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

Mode of spread and survival

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

Management

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

Mosaic: PRSV/CMV

Symptoms:

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

Mode of spread and survival

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

Management

The virus is readily transferred by aphids and survives on a wide variety of plants. Varietal resistance is the primary management tool, and eliminating weeds and infected
perennial ornamentals that may harbor the virus is critical. Spray with any one of the systemic insecticide.
Diseases of Crucifers
Radish

**Alternaria Blight**: *Alternaria raphani*

**Symptoms**

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

**Pathogen**

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

**Mode of spread and survival**

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

Spraying with Mancozeb 0.25 %

**White Rust:** *Albugo candida*

**Symptoms**

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

**Pathogen**

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

**Mode of Spread and Survival**

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

**Management**

Regular spraying with Mancozeb 0.25 % effectively controls the disease.

**Cauliflower**

**Downy Mildew:** *Peronospora parasitica*

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

**Pathogen**

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

**Mode of Spread and Survival**

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

**Management**

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

Wire stem: *Rhizoctonia solani*

**Symptoms:**

![Wire stem](image)

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

**Pathogen**

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

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

Cabbage

Black leg: Phoma lingam

Symptoms

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

Pathogen

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

Mode of Spread and Survival

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

Management

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

**Symptoms**

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

**Pathogen**

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

**Mode of Spread and Survival**

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

**Management**

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

**Root rot: Rhizoctonia solani**

**Symptoms**

Young plants show soft, water soaked lesion on the stem near soil level, the cotyledons wither and the plant eventually falls over and perishes. When infection occurs at a later stage of
growth, the lower part shows discoloration over a length of several centimeters, becomes hard and woody, and thinner than usual as the cortical tissue dies and this phenomenon is known as wire stem.

**Pathogen**

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

**Management**

Nursery beds: Soil drenching with Methyl bromide @ 1 kg/10 m² and covered with polythene sheet. Seed treatment with Captan/Thiram 4g/kg, followed by seed treatment with Trichoderma viride 4g/kg.

**Black spot:** *Alternaria* sp.

**Symptoms**

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

**Pathogen**

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

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

Management

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

Club root: *Plasmodiophora brassicae*

Symptoms

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

Pathogen

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

Mode of Spread and Survival

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

Disease Cycle

*P. brassicae* is capable of surviving in the soil for 7-10 years or longer as resting spores. The resting spores of the fungus can be spread from field to field by infested soil, contaminated water supplies, infected transplants, infested soil on farm machinery, and even by roving animals such as cattle. When soil conditions dictate, the resting spores of the pathogen germinate to
produce zoospores, which are able to "swim" by means of flagella to infect susceptible plant root hairs. The germination of resting spores requires moist, acid soil and can occur over a wide temperature range of 12-27°C. Disease development is favored by high soil moisture and soil temperatures between 18-25°C. Although clubroot has been found in soils exhibiting a wide pH range from 4.5-8.1, the disease is primarily associated with acid soils. Within the infected plant roots, the organism develops rapidly, causing an increase in the number and size of cells, which results in "clubbing." During the development of the organism in the plant, new zoospores are produced; these are capable of infecting the same plant or adjacent plants and, thus, repeating the cycle. Eventually, resting spores are formed within the diseased plant tissue, and these are released into the soil when the plant roots disintegrate.

**Management**

Soil fumigation with Methly bromide 1kg/10m² followed by covering with plastic film. Seed treatment with Captan/Thiram 4g/kg, followed by *T.viride* 4g/kg. Application of lime 2.5 t/ha. Soil drenching with Copper oxychloride 0.25%.
**Powdery mildew: Erysiphe polygoni**

**Symptoms**

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

**Pathogen**

Conidiophores are septate. The cleistothecia are sharp and globose.

**Mode of Spread and Survival**

The disease spread through water and wind borne conidia.

**Management**

Spray inorganic sulphur 0.25% or Dinocap 0.05%.

**Bacterial diseases**

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

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

**Pathogen**

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

**Mode of Spread and Survival**

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

**Management**

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

**Turnip**

**Alternaria Leaf Spot: Alternaria** spp.

**Symptoms**

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

**Pathogen**

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

**Mode of Spread and Survival**

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

**Management**

Spraying with Mancozeb 0.25%

**Carrot**

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

**Symptoms**

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

**Pathogen**

The bacterium is rod shaped and polar flagellum.

**Mode of Spread and Survival**

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

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

Management

Spraying early with Copper oxychloride 0.25 %.

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

Symptoms

![Image of soft rot symptoms](image)

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

Pathogen

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

Mode of Spread and Survival

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

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

**Cercospora leaf spot:** *Cercospora carotae*

**Symptom**

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

**Pathogen**

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

**Mode of Spread and Survival**

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

**Management**

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

**Sclerotinia Rot or White mold:** *Sclerotinia sclerotiorum*

**Symptom:**
Mycelia growth and sclerotia (red arrow)

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

Management

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

Asparagus

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

Symptoms

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

Management

The disease is seed- and soil-borne. New plantings should be established on soil (well-drained, sand-loam soils are preferred) where asparagus has not been previously grown for at least five years. Use strong healthy plants (1 year crowns) to start a plantation and to ensure good
plant health by following good planting and growing procedures such as fertilization, insect and weed control and avoid over harvesting.

**Purple Spot**: *Stemphyllium vesicarium*

**Symptoms**

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

**Management**

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

**Rust**: *Puccinia asparagi*

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

**Management**

Plant in areas with good air circulation and irrigate during the day so plants can dry out before evening.
**Diseases of Onion & Garlic**

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

**Symptoms**

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

**Pathogen**

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

**Mode of spread and survival**

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

**Management**

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

**Downy mildew**: *Peronospora destructor*

**Symptoms**

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

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

Mode of spread and survival

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

Disease Cycle

Dormant Period

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

Primary Spread

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

Secondary Spread

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

**Management**

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

**Leaf Blight (Blast): Botrytis spp.**

**Symptoms**

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

**Pathogen**

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

**Disease cycle**

![Disease cycle diagram]
Dormant Period

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

Primary Spread

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

Secondary Spread

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

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

Management

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

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

**Symptoms**

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

**Pathogen**

*Pythium aphanidermatum*

**Epidemiology**

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

**Management**

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

**Smut:** *Urocystis cepulae*

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

Pathogen

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

Mode of spread and survival

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

Management

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

White Rot: Sclerotium cepivorum

Symptoms

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

Mode of spread and survival

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

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

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

Management

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

**Purple blotch:** *Alternaria porri*

Symptoms

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

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

**Management**

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

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

**Symptoms**

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

**Mode of spread and survival**

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

**Blue mould rot**: *Penicillium sp*

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

**Pathogen**

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

**Mode of spread and survival**

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

**Black mould- Aspergillus niger**

**Symptoms**

Infection usually is through neck tissues as foliage dies down at maturity. Infected bulbs are discolored black around the neck, and affected scales shrivel. Masses of powdery black
spores generally are arranged as streaks along veins on and between outer dry scales. Infection may advance from the neck into the central fleshy scales. In advanced disease stages, the entire bulb surface turns black, and secondary bacterial soft rot may make the bulb soft and mushy. No external symptoms may be found with some bulbs.

**Management**

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

Black spot - *Diplocarpon rosae*

**Economic Importance**

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

**Symptoms**

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

**Pathogen**

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

**Mode of spread and survival**

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

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

**Powdery mildew – Sphaerotheca pannosa**

**Economic Importance**

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

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

**Pathogen**

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

**Disease Cycle**
Mode of spread and survival

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

Management

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

Die back – Diplodia rosarum

Economic Importance

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

Symptoms

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

Pathogen

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

Mode of spread and survival

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

Management

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

Rust – Phragmidium mucronatum
Economic Importance

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

Symptoms

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

Pathogen

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

Management

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