PRACTICAL MANUAL

Course Title : **Diseases of Field and Horticultural Crops and their Management - II**

Credits : **3 (2+1)**

Course No. : **PATH - 365 (New)**

Course : **B.Sc. (Hons.) Agriculture**

Semester : **VIth Semester (New)**

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2019

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PARBHANI : 431 402

College of Agriculture, ...

CERTIFICATE

This is to certify that Shri / Miss ..... Reg.No..... a student of VI (New) Semester, **B.Sc. (Hons.) Agriculture** has completed all the exercises successfully for the Course : **Diseases of Field and Horticultural Crops and their Management - II** Course No. : **PATH - 365 (New),** during Academic year 20 - 20 .

Place :

Date : **COURSE TEACHER**

Remarks :

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INDEX

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Identification and histo-pathological studies of selected diseases of Field and Horticultural Crops covered in the theory. Collection and Preservation of diseases specimen.

Note : Students should submit 50 pressed and well-mounted specimens.

I) FIELD CROPS :

Exercise No. 1 :

Study of Wheat Diseases

(1) Rusts (2) Loose smut (3) Karnal bunt,
(4) Powdery mildew (5) Alternaria blight (6) Ear cockle

(1) Rusts :

Causal organisms :
1. **Black stem rust** of wheat .....*Puccinia graminis tritici* Eriks and Henn.
2. **Brown rust** or leaf rust of Wheat.....*Puccinia recondita* Reb.ex. Desm.
3. **Yellow rust** or stripe rust of wheat...*Puccinia striiformis* West.

Black or stem rust	Yellow or stripe rust	Brown or leaf rust
Stalk affected most severely than leaf sheath, leaves and ears.	**Plant parts affected** Attacks leaves most severely than leaf sheath, stalks and ears.	Attacks the leaves almost exclusively, rarely the leaf sheath and very rarely the stalks.
Uredopustules are larger, elongated running together and bursting early throwing up large fragment of the epidermis. Colour dark brown to brick red becoming dark gradually as teliospores are formed in the same pustules. Found on all green parts of the plant.	**Uredosori** Uredopustules are small, oval, do not run together, burst late, and with little displacement of the epidermis. Almost always arranged in long rows. Colour lemon yellow, found on all green parts of the plant.	Uredopustules are small but often larger than in yellow rust, oval or round, do not usually run together, burst early with a fringe of broken epidermis around them. Never arranged in long rows, colour bright orange when fresh, found chiefly on upper surface of leaves.
Uredospores are red to brown in colour. Oval in shape with 4 germ pores	**Uredospores** Uredospores are lemon yellow, round in shape, 6-10 germ pores are present.	Uredospores are bright orange in colour, after maturity becomes brown in colour, round and

which are arranged at equatorial layers. 25-30 x 17-20 um.	23-25 x 20-25 um.	uredospores with 7-10 germ pores 16 x28 um.
Teleutopustules are like uredo but black, burst rather early. Found on all green parts of the plant but least on the leaf blades.	**Teleutosori** Teleutopustules like uredo but more flattened and dull black, do not burst through epidermis, found chiefly on under surface of leaves but also on green parts of the plant.	Teleutopustules often absent, when present, resemble uredo but more flattened and dull black. Do not burst through epidermis. Found chiefly on leaves, very rare else where.
Teliospores are two celled and pointed at the apex. Chestnut brown, 40-46 x 15-20 um.	**Teliospores** Teliospores are round in shape, with flattened apex. 35-63 x 12-20 um.	Teliospores are round at apex.
24 races and biotypes in India, Race 122 is virulent while race 21 and its biotypes are more prevalent.	**Races in India** 18 races and bio types 14, 14A, 20, 20A and 38, 38A races are more prevalent.	18 races, Race 162, 162A, 162B and 77A, 77B more virulent and prevalent.
Black or stem rust	**Yellow or stripe rust**	**Brown or leaf rust**
Berberis vulgaris *Mahonia acauifolia*	**Alternate hosts** *Bromus juponicus*	*Thalictrum polygamum*
Through uredospores on self sown wheat crop in hills, secondary infection through uredospores in the infected fields.	**Perpetuation** **Same as black rust.**	Same as black rust.

Control measures :

1. Use of resistant varieties e.g. HD-2189, HD-4502, HD-2380, HD-2501, HD-2278, MACS-2496, AKW-2501, N-8223, UP215, PBN-42, DWR-162, DWR-195, NI-9947, NIAW-34, NIAW-301.. The resistance may break down and the varieties may become susceptible in the course of time.
2. Spraying of 1) Zineb. 0.25 % + 2% Urea or Mancozeb –0.25 % + 2% Urea. Second spray of fungicides, if infection persists.
3. Eradication and cleaning up of alternate and collateral hosts.
4. Indiscriminate use of nitrogenous fertilizers increase the susceptibility of the crop to rust disease. Application of potash and phosphatic fertilizers give tolerance.

Class Work :

1. Cut transverse sections through stage of black stem rust of wheat, mount the section in lactophenol on glass slide and examine, sketch and label the drawings.
2. Study the comparative symptoms of the three rusts of wheat supplied to you

Exercise :

1. Explain annual recurrence of wheat rusts in India.
2. What is heteroecious rust? What do you understand by races? Why do we have less races of black stem rust in our country ?
3. Draw life cycle of heteroecious black stem rust of wheat.

(2) Loose Smut :

Causal organism – *Ustilago tritici* (Pers.) Rostr.

Symptoms:

Affected ears on diseased plants emerge out of the flag leaf earlier than those of healthy plants. Usually entire ears rarely partial, are transformed into sori, which is covered with thin silvery membrane that bursts as soon as affected ears emerge form host leaf. Almost all the parts of ears except awns are transformed. The olive brown black spores are easily blown away leaving the naked rachis behind.

Etiology:

The spores are olive brown, lighter on one side, spherical or oval, 5-9 um in diam. The epispore has fine spines especially on the lighter side. Germination of spores by germ tube, which soon dies unless the spores have fallen on feathery stigma. No sporidia are produced and diplodization takes place between the cells of germ tubes. In about three weeks, the hyphae reach the lower end of raphe and pass round the bottom of endospore to reach the scutellum and penetrate the embryo, where it remains in dormant state. The dormant mycelium is thick walled, oily and irregularly swollen.

Disease cycle:

The disease is internally seed borne and systemic. The infected grains can not be distinguished apparently from the healthy grains. The pathogen remains dormant within the mature grain. When such seeds are sown, the dormant mycelium becomes active and grows keeping pace with the growing tip of the host. Secondary spread is by chlamydospores which are wind borne.

Control measures :

1. **Solar heat treatment** – Soak the seeds in cold water for four to six hours and then spread in thin layer on threshing floor or on galvanized iron sheets during summer in between 12.00 to 4.00 pm.(This is called as Luthra's(1953)treatment who modified hot water treatment to suit under Indian condition)
2. **Hot water treatment** – Soak the seeds for four to six hours to activate the dormant mycelium. Then dip the seeds in water 53°C for 10 min. Dry the seeds and treat with seed protectants. (This treatment was develoed by Jensen1887)
3. Seed treatment with systemic fungicides like carboxin (Vitavax) 2.0 g/kg seed.
   OR-Carboxin+ Carbendazim at the rate of 2.5g/Kg seed

Use resistant varieties, viz. Kalyan Sona 227, PV-18, WG-307, C-302.

Answer the following:

1) Why soaking of seeds in cold water is necessary in hot water as well as solar hat Treatment?
2) Write a brief note on hot water treatment and solar heat treatment.
3) Which systemic fungicides are used for controlling the internally seed borne loose smut of wheat?

(3) Karnal Bunt:

Causal organism : *Neovossia indica* (Mitra) Mundkur

Symptoms :

Karnal bunt becomes evident when the grains have developed. It is then found that some grains have been partially, rarely wholly, converted into black powdery mass enclosed by the pericarp. Not all the ears in a stool carry the disease and even on the same ear only few grains are smutted. Embryo is not always damaged. Such partially affected seeds are germinated. Due to irregular distribution of infected grains in the ears it has been presumed that they are result of air borne local infection. The spore mass remains covered by the pericarp but later reputures exposing the black powder.

Etiology :

The spores are smooth walled measuring 2-49 micron in diameter requires a long resting period, sporidia are produced in large number on short stout basidium. The primary sporidia are needle shoped, secondary sporidia are sickle shaped.

Perpetuation :

Soil borne and air borne usually affecting only a few spiklets in an ear.

Control measures :

1. Cultivation of susceptible varieties and continuous cropping of wheat in the same field should be avoided.
2. Deep ploughing during summer
3. Crop rotation
4. Common bunt can be controlled by seed treatment with carboxin (Vitavax). However karnal bunt spreading through air borne sporidia is very difficult to control with seed treatment.

(4) Powdery mildew :

The disease is important in areas where humid conditions prevail during the growing season of the crop and is present throughout the world wherever wheat is grown. In India, the disease is serious in the northern as well as southern hill regions and appears sporadically in the North-Western plains.

Symptoms :

The fungus infects all the aerial parts of the plant. On the upper surface of the leaves, the symptoms appear as small flecks on which white to grayish powdery growth appears. Later this turns to tanned mildew growth which consists of fluffy, superficial powdery mass. Such areas enlarge with time, coalesce and cover the complete leaf surface. On these lesions, condiophores are produced which bear conidia. As the disease progresses, small round black perithecial fruiting bodies appear in the powdery mass, which are visible as black dots scattered over the mycelial mat. Under high humidity, the disease spread is very fast and the fungus covers all the plant parts including the leaves, leaf sheaths, stem, glumes etc. The number and size of leaves is reduced which are also twisted, crinkled and deformed. The respiration of the plant is increased. The plants become chlorotic and there is increase in water loss.

Causal organism : *Erysiphe graminis* f. sp. *tritici* (D.C.) E. Marchal.

Etiology : The fungus perpetuates in the form of perithecia in the soil during the off season which release ascospores. The ascospores germinate to produce a germ tube which enters the wheat leaves through stomata and results in primary infection. The mycelium continues to grow ectophytically and small lesions are immediately produced which increase in size and produce a large number of conidia in short time. The conidia are released from the conidiophores and become air borne. The conidia on germination produce a germ tube, which forms an appressorium from where the infection peg arises which penetrates the cuticle and sub cuticular walls of the host.

Epidemiology : The optimum temperature for conidial germination is 15-20 ^{0}C. The infection takes place successfully at temperatures around 20 ^{0}C at a relative humidity 25-75 per cent. After production, the conidia loose their viability within 24 hours of liberation at temperature above 20 ^{0}C. The disease spread is increased under high relative humidity, prolonged rains, high plant population and high fertilizers doses.

Control measures :

i) Grow resistant varieties.
ii) Burn the crop refuse in the field after harvesting
iii) Spray fungicide mixture of Dithane M-45 and Karathane 25 WP @ 2 kg/ha in 800-1000 litres of water. For this make solution of 8 g Dithane M-45 and 2 g Karathane 25 WP per 5 litres of water. About two to three sprays should be given at an interval of 10-15 days if the losses are expected to be very high.

(5) Alternaria blight :

Causal organism: *Alternaria triticina* Prasad and Prabhu

Symptoms: Small, chlorotic, oval- or elliptical lesions appear. As they enlarge, these lesions become irregular in shape. The chlorotic borders of the lesions may become diffused and turn light to dark brown in colour. Lesions are difficult to distinguish from those caused by

Helminthosporium spp. Infection usually starts on the lower leaves, but symptoms can be found on all plant parts.

Etiology: The fungus survives as conidia on seed or as mycelia within seed. Sporulation on lower leaves provides inoculum that can be dispersed by wind, leading to secondary spread of the disease. Seed-borne inoculum often results in spike infections late in the crop cycle.

Predisposition:

High humidity or irrigation, as well as warmer temperatures (20 to 25 ^{0}C) favor infection and disease development.

Bread wheat and durum wheat, as well as several related grasses, are the primary hosts. The disease is common in the eastern and central areas of the Asian Subcontinent. Alternaria leaf blight can be very severe if environmental conditions are favorable for disease development; major losses can result when susceptible cultivars are grown.

Control measures :

i) Grow resistant varieties.

ii) Disease is externally as well as internally seed borne. Seed treatment by pre-soaking for four hours followed by a 10 minute dip in hot water at 52^{0}C

iii) Foliar application of zineb OR mancozeb 0.2% can reduce disease severity.

iv) Give adequate NPK fertilizers and irrigation.

6) Ear Cockle :

Cause : *Anguina tritici* (Stein) Filipjev.

Symptoms :

The infested plants may remain dwarfed and show generally twisted, rolled and crinkled leaf blades. The glumes become more divergent and grains replaced by hard, dark coloured galls which are full of nematodes. The diseased ears are shorter and thicker than the healthy ones and keep green longer.

Control measures :

Use gall free seeds. This can be done by floating the seeds in salt water. The seeds are treated with 20 % Salt solution and stirred vigorously. The affected seeds along with galls floating on the surface can be separated with seives. The healthy seeds settle down and can be used for sowing after washing and drying.

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Exercise No. 2

Sugarcane

(1) Red Rot :

Causal organism : Imperfect stage: *Colletotrichum falcatum* Went
Perfect stage :*Glomerella tucumanensis* Von Arx and Muller

Symptoms :

The first symptom of red rot is discolouration of young leaves. The margins and tips of the leaves wither and leaves droop. The withering will proceed and finally whole crown withers and cane dies within weeks time. In single stool most of the canes become evident. The tissues are reddened through out the basal portion, mostly the vascular bundles. There may be crosswise white patches interrupting the reddened tissue. As the disease advances the entire stem rot and central tissue become pithy. The internodes shrink and when the cane split open; large cavities may be found in the center. Pithy tissues turn brown with whitish mycelial growth of the fungus. Also embedded with are black minute bodies representing the acervuli. Leaves show symptoms in the form of dark red lesions in the midrib, which elongate, turning blood red with dark margins and later straw coloured center with minute black dots of acervuli.

Etiology :

The mycelium is thin, septate, hyaline and intracellular formation of acervuli is the character of genus. They are minute black dots with setae. Conidia are borne singly on the conidiophores are single celled, falcate thin walled. Sometimes terminal or intercalary chlamydospores are formed. The primary source of infection is planting material, plant debris and chlamydospores. Secondary infection through conidia spread through irrigation water.

Perfect stage *G. tucumanensis* produces globose perithecia in which clavate asci are produced. Each ascus contains 8 ascospores.

Control measures :

1) Selection of planting material from healthy cane and disease free area.
2) Setts are treated by dipping in 0.1% Carbendazim for 18 minutes at 52^0C (Rangaswami 2002)
3) In standing crop if one of the stool infected, the canes are to be collected and destroyed by burning.

Answer the following questions:

1)Describe in short the red rot symptoms.
2)Describe the etiology of asexual and sexual stage.
3)Comment on feasible sett treatment for the control of red rot.

(2) Smut :

Causal organism – *Ustilago scitaminea* Syd.

Symptoms:

Affected plants produce a whip like structure, several feet in length and much curved on it self. It comes out from the central spindle at the apex. In its earlier stages, the smut like

powder on this whip-like outgrowth is covered by a white, silvery, thin membrane, which soon ruptures and flakes off exposing dense black powdery dust.

Etiology:

The black powdery mass formed in the whip like structure is the mass of chlamydospores(teleutospores). The chlamydospores are enchinulate, light brown and spherical, 5-10 microns in diameter. The sugarcane has no dead season and hence the pathogen gets ready host. Blown off spores fall on the base of the leaf sheath, buds or injured canes and get the entry and travel upwards. The disease perpetuates by either:

i)Planting smutted canes.
ii)By spores borne on buds.
iii)By infection of buds on standing canes.
iv)Ratooning of smutted canes.

Secondary spread of the pathogen is by wind and irrigation water.

Control measures :

1. Removal of smutted canes in thick cloth of gunny bags and their destruction.
2. Discourage the practice of ratooning
3. Avoid planting of sets from smutted canes.
4. Use setts from healthy fields.
5. Disinfect the setts before planting by (a) H.W.T. at 52°C for 1 hour or Hot moist air treatment at 54°C for 8 hours.(Vala *et al* ., 1992,Singh 2005)
6. Dipping the setts in 0. 5% vitavax or Bavistin solution for 10 min.(Waraitch 1986)
7. Use of resistant varieties
   i) Co 449,527,658,974,1148,6806,7108,7319 (Agnihotri1983)
   ii) Bo11,22,24 (Singh 2005)
   iii) CO. 7219, COM-8014 and COM- 7125, CO-62175, CO-7704.

The varieties which are resistant may break down resistance and become susceptible in due course of time.

Exercise:

1.Why this smut is called as whip smut?
2. How primary and secondary infection of this smut occurs?
3.Descibe i)hot water treatment ii) Hot moist air treatment for sugarcane setts.

(3) Sugarcane Wilt :

Cause : *Cephalosporium sacchari* Butl.

Symptoms :

The earliest symptoms appears late in the season as yellowing and withering of crown leaves, followed by rapid drying of the cane. The internal tissue of the cane becomes brown or reddish brown in colour but there is no transverse white bands as seen in the red rot diseases. The stem becomes light and hollow making them worthless for milling. A foul smell is noticed after splitting the infected canes. Cottony mycelial growth can be seen in the pith cavities.

The pathogen persists as mycelium in plant residue. Infected setts serve as the means of primary inoculum.

Etiology :

Mycelium grows profusely inside the affected canes. They are hyaline, thin walled and septate and produce many coidiophores as lateral branches. The conidiophores and are simple or branched and aseptate bear the numerous micro conidia on the tips. Micro conidia are hyaline, ovoid or oblong ellipsoidal, aseptate, single celled , and measure 4-12 x 1-3 µm. No macro conidia are produced.

Predisposing factor / Favourable conditions : -

The disease symptoms appear during the monsoon and post monsoon periods, affected plants are present either singly or in small groups. High day temperature is 30-35 ^{0}C. Low humidity - 50-60 %. Low soil moisture and alkaline soils.

Control measures :

i) Select canes from completely healthy fields for planting.
ii) All the infested canes together with their leaves and roots should be completely burnt.
iii) No ratoon should be kept in the infested fields.
iv) Dip cane setts in 0.25 per cent solution of Agallol or Aretan for five minutes before planting.
v) Grow resistant varieties recommended for the particular area.
vi) Soil treatment or setts treatment with boron and zinc at the rate of 40 ppm has been reported to reduce the wilt infestation.

(4) Grassy shoot :

Causal organism: *Mycoplasma like Organism (MLO)*

Symptoms :

The disease is characterised by the production of number of thin tillers from the base of the infected cane. This growth gives rise to bunch of tillers having pale yellow or completely chlorotic and white leaves. Cane formation does not take place in such tillers. Disease seriously affect the yield and quality of juice.

Etiolgy:

MLO is found to present in the sieve cells of infected plants. Two types of bodies are noticed, spherical bodies of 300-400 nm diameter and filamentous bodies of 30-53 µm diameter.

Transmission :

Disease is readily transmitted by sap inoculation and by cutting knife but mostly it spreads by the use of infected cane setts. In nature, it spreads by aphids, *Aphis maydis, Aphis indosacchari, Aphis sacchari.* The disease is caused by a phytoplasma. In addition to sugarcane, the disease also infect number of hosts.

Control measures :

1) Eradication of diseased cane.

2) Use of healthy cane setts for replanting.
3) Giving up practice of ratooning for 4 years at least.
4) Moist hot air 54°C for 8 hours or hot water treatment at 52°C for 1 hour (Rangaswami 2002).
5) Spraying with metasystox or Follidal in standing crop twice a month for control of aphids.

(5) Ratoon stunting :

Cause : *Clavibacte xyli* sub sp. *xyli*
Leifsonia xyli (Davis *et al.*)

Symptoms :

The affected crops remain stunted with short and thin canes. The leaves are pale and roots are poorly developed. The characteristic symptom can be seen in the split canes as pink discoloration of the growing point. A yellow-orange, pink-red or reddish brown discolouration occurs with individual vascular bundles in the nodes of mature canes. The ratoons display symptoms more than the older canes.

The disease spreads through seed setts and ratoon crops.

Etiology :

The disease, which was originally thought to be caused by a virus or by a mycoplasma like organism, has now been definitely identified as *Clavibacter xyli* sub sp. *xyli* or *Leifsonia xyli* (Davis *et al.*).

The fastidious, xylem-inhabiting, gram positive, coryne form bacteria, measure 1.0 - 4.0 x 0.3 - 0.5 μ in size. There are non-motile and non-spore forming. Sometimes, they occur in chains and appear like filaments.

Mode of survival and spread :

The pathogen over seasons in infected sugarcane plants and propagative materials, such as seed-canes. Plants growing from infected setts develop the disease invariably. The bacterium is sap transmissible and is spread by cutting knives, by implements used in cultural operations and also by harvesting equipments. Rodents, such as field rats, rabbits, wild pigs etc. also aid in the transmission of the bacteria through injuries they cause to the standing canes. The disease continues to spread to different countries through infected sugarcane germ plasm.

Control measures :

i) Grow resistant varieties.
ii) Plant setts only from healthy canes.
iii) Hot air treatment of cane seeds at 54 °C for eight hours is effective in destroying the causative agent.
iv) Do not ratoon the diseased crop.
v) Treat seed canes with Agallol or Aretan after hot air treatment before planting.

(6) Pokka Boeng :

Causal organism : *Fusarium moniliforme*

Symptoms :

The characteristics symptoms of Pokkah Boeng disease are the appearance of chlorotic patches towards the base of the young leaves, in acute cases disease shows distortion of stalk with external and internal cut like lesions and rotting of apical part of stalk. Under field conditions, the disease may develop many variations from the general symptoms, but the final result is usually a malformed or damaged top and stalk. The base of affected leaves is often narrower as compared to normal leaves. Knife cut symptoms of the disease were reported.

Characteristics of pathogen - Etiology :

Fusarium moniliforme showed different colour for its mycelium pale white, pink and purple mycilium and for pigmentation of metabolites also during the growth on artificial medium. The growth of conidia range from 9.3 - 29.7 µM in length and 2.7 - 6.0 µM in width, mycelium width of ranged in between 1.75 - 7.00 µM. Mycelium of F. moniliforme was generally dense in delicately floccose to felted with powdery appearance due to formation of macro conidia.

Effect of environmental factors :

Temperature is an important natural factor governing the distribution of a pathogen and it grows and sporulates luxuriantly in a temperature range of 20-30 ^{0}C in both *in-vitro* and *in-vivo* condition. Minimum, optimum and maximum temperature for growth pathogen are 10-15^{0}C, 30 0C and 35-40 ^{0}C, respectively. The severe incidence of the disease occurred in the range of temperature between 20 to 32 ^{0}C with high humidity up to 70-80 % and cloudy weather in rainy season from July to September. We also found the incidence of the disease in the month of July to September when humidity range from 79.0 - 85.5, temperature ranging 29.0 - 30.0 ^{0}C and rain fall is high. Temperature ranging from 20-30 ^{0}C and humidity ranging from 75-85 % is the best suitable for the growth of *Fusaium pathogen.*

Control measures :

i) Spraying of Bavistin @ 1 gm/lit. of water or Blitox 0.2% or Copper oxychloride or 0.3 % Dithane M-45 3 gm/lit. of water.

ii) Two to three sprayings with an interval of 15 days interval.

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OILSEEDS

Exercise No. 3

Sunflower

(1) Sclerotinia stem rot :

Cause : *Sclerotinia sclerotiorum* (Lib) & de Bary.

Symptoms :

Rabi crop is generally affected by this disease. There is similar symptom of wilt as in case of sclerotium wilt except that the sclerotinia are black, large and irregular on the affected portions. Rotting of the head and stem can also be seen sometimes instead of wilting. This is evident with the shredding of the affected head and stem. Initial symptoms are sudden wilting of plant, particularly during grain fill and light brown, water-soaked area at the stem base will become obvious. The stem will often break at this point of infection and when ripped upon, black sclerotinia can be seen. Heads do not fill properly due to nutrients and water not being taken up by the plant.

Etiology and Epidemiology :

Sclerotinia are hard small black bodies produced in a host of broad leaf cops. Wet soil conditions over a period of 10-14 days in stimulate the Sclerotinia to germinate. Wet and cloudy conditions are favourable for disease advancement. Temperature 11-15 ^{0}C is required to germinate sclerotinia and trigger spore release.

Control measures :

i) Use certified seeds.
ii) Collect and burn the affected plants.
iii) Follow up crop rotation with cereals.
iv) Keep the field free from weed.

(2) Alternaria blight :

Causal organism: *Alternaria helianthi* Hansf.

Symptoms:

The fungus produces brown spots on the leaves, but the spots can also be seen on the stem, sepals and petals. The lesions on the leaves are dark brown with pale margin surrounded by a yellow halo. The spots later enlarge in size with concentric rings and become irregular in shape. Several spots coalesce to show bigger irregular lesions leading to drying and defoliation.

Etiology:

The fungus produces cylindrical conidiophores, which are pale grey-yellow coloured, straight or curved, geniculate, simple or branched, septate and bear single conidium. Conidia are pale grey-yellow to pale brown, muriform, having many transverse septa and 1 to 2 longitudinal septa.

Predisposition:

Rainy weather, cool winter climate and late sown crops are highly susceptible.

Perpetuation:

The fungus survives in the infected host tissues and on weed hosts. The fungus is also seed-borne. The secondary spread is mainly through wind blown conidia.

Control measures :

i) Remove and destroy infected plant debris.
ii) Rogue out weeds at periodical intervals.
iii) Sow the crop early in the season (June sowing).
iv) Treat the seeds with Thiram 6 g/kg for preventing primary seed borne infection
v) Prevent secondary infection by spraying 0.2(%) Mancozeb immediately after appearance of the disease.

(3) Rusts

Causal organism : *Puccinia helianthi* Schw.

Symptoms:

Uredial pustules usually appear first on the lower leaves. They are small, circular, powdery, orange to black and when the infection is severe the uredo pustules are produced on the younger leaves and become scattered over the entire vegetative surface covering stem, petiole, floral bracts and flower parts. Chlorotic areas usually surround the pustules even on the susceptible varieties. The uredia may coalesce to occupy large areas on the affected plant parts. The leaves are particularly dry under such conditions, as the plant approaches maturity or is subjected to physiological stress. Teliospores appear in the uredia and develop into telia and the black stage appear.

Etiology:

This pathogen is macrocyclic, heterothallic, autoecious, producing all stages of spores on sunflower have been reported from India.

Stage-II: Uredospores are brown and vary from subglobose to ovate. The wall of spores is smooth, chestnut brown. The spores are pedicellate.
Stage-III: Teleutospores bicelled and black in colour
Stage-IV: Basidiospores: infect sunflower to produce pynia of + and – types.
Stage-0: Pycniospores are small, oval, hyaline and appear as shining viscous mass.
Stage-I: Aeciospores are orange, typically ellipsoidal and finally echinulate.

Perpetuation:

It survives through teliospores on leaves left in field or on the soil surface. Uredia, sporidia, pycnia and aecia survive on volunteer seedlings among plant debris of the previous years crop.

Primary infection results from primary inoculum source such as sporidia from germination of surviving teliospores, or from aeciospores on volunteer seedlings or uredospores.

Secondary infection occurs usually through repeatedly produced uredospores in a crop season.

Control measures :

1. (a) Grow varieties with R_1 and R_2 genes.
   (b) Morden, EC. 684142, Surya.
2. Spraying with Mancozeb or Zineb @ 0.25 %
3. Removal and destruction of volunteer seedlings.

Answer the following questions:

1) Define following:
   i) Macrocyclic ii) Heterothallic iii) Autoecious
2) Comment on:1) Pycnia 2)Aecia iii) Uredosorus iv) Teleutosorus v) Basidiospores or sporidia
3) How will you manage recurrence of sunflower rust?

(4) Downy Mildew :

Causal organism : *Plasmopara halstedii* (Fort.) Berl. & de Tom.

Symptoms :

Symptoms of the disease become evident as seedling damping off, systemic symptoms, local foliar lesions and basal root or stem galls.

(a) **Damping off :** Seedlings are killed before or soon after the emergence due to subterranean infection by the downy mildew fungus. Affected plants dry and become wind blown.

(b) **Systemic symptoms :** Sunflower plants carrying systemic infection are severely stunted and at flowering the upper leaves become entirely chlorotic. The stem becomes brittle. Flower heads of infected plants remain erect, become small in size, remain sterile and produce no seeds or only few seeds are produced on such heads.

(c) **Local foliar lesions :** Small angular greenish-yellow spots appear on leaves as a result of secondary infection. The spots may enlarge and coalesce to infect a large part of the leaf. The fungal growth becomes visible at lower surface of the diseased area.

(d) **Basal root or stem galls :** The root infection may result in formation of galls at the base of the plants on primary roots. Such plants are less vigorous and subject to lodging.

Etiology:

Mycelium nonseptate, sporangiophores are typically branched at right angles, Produce brownish, thick walled, resting, sexual oospores.

Perpetuation :

The oospores in the residues of the preceding sunflower crop or oospores in or on seeds from the systemically infected plants serve as primary source of infection. Some oospores have been reported to remain dormant upto 14 years. Secondary infection may be caused by the zoosporangia produced in great numbers on systemically infected plants.

Control measures :

i) Early planting at the onset of the rainy season decreased disease incidence.
ii) Treat the seeds with metalaxyl compound (Apron 35 SD) @ 5.7 g/kg of seed.
iii) Apply foliar spray of metalaxyl (0.025%) + Mancozeb (0.2%) (Ridomil MZ 72 WP) at the concentration of (0.25 % of formulation RidomilMZ-75WP) on 20 days after sowing.

Questions

1) Describe the following phases of symptom expression:
   i) systemic leaf chlorosis
   ii) Downey phase
   iii) Erect head and stunting
   iv) Basal and root galls
2) How will you control the primary and secondary infection of downey mildew of Sunflower?
3) Draw the life cycle/diseases cycle of downey mildew fungus.

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Exercise No. : 4

Mustard

(1) Alternaria blight :

This is also known as **Dark Leaf** and **Pod spot.**

Causal organism :

Alternaria brassicae (Berk.) Sacc., and *Alternaria brassicicola* Wiltshire.

Symptoms :

Initial symptom is the production of small brown circular necrotic spots with yellow halo around them on the leaves. The spots increase in size and form light brown to brown or black concentric rings on leaves, stems and pods. In the years of severe outbreak, pods turn black in colour and may also rot. Seeds are shrivelled and undersized in such pods.

Etiology :

The mycelium of the pathogens is inter and intracellular and effused. Hyphae are olive in colour, branched, septate and smooth. Conidiophores arise from hyphae, which gather beneath the host epidermis. In *Alternaria brassicae*, the conidiophores arise in fascicles through the stomata. They are simple, erect, septate, olivaceous and up to 170 µ in length. Conidia are produced in acropetal succession, either singly or in short chains of up to 4. They are dark coloured, straight or slightly curved, smooth, obclavate to muriform, with 16-19 transverse septa and 0-8 longitudinal septa. They measure 125-225 x 16 - 28 µ in size. The beak is rather long. The fungus perpetuates through seed and affected plant debris.

Mode of survival, spread and Epdemiology :

The conidia and mycelium present in the diseased plant debris serve as the main means of perennation of the pathogen. Conidia are formed abundantly in moist atmosphere and are disseminated by wind. Seed-borne infection is of no significance. The weed hosts viz., *Anagallis arvensis* and *Convolvulus arvensis* and various other crop hosts provide enough inoculum all through the year. Conidia are formed abundantly in moist atmosphere and are disseminated by wind.

Control measures :

i) Grow resistant varieties like YSPB-24.
ii) Sow healthy and bold seeds from the disease free plots.
iii) Spray Dithane M-45 or Difolatan or Duter at the rate of 2 g per litre of water at 10 days interval with the appearance of the disease.
iv) Collect and destroy the affected plant parts after harvesting of the crop.

(2) White rust :

This is also called as **White Blister.**

Cause : *Albugo candida* (Pers.) Kuntz.

Symptoms :

Prominent white or cream yellow scattered pustules appear on the undersurface of the leaves. These pustules are raised blisters found on the leaves, stems and floral parts. These

blisters burst and liberate a white powder. Floral parts are much deformed. The flowers get malformed (hypertrophied) and become sterile. The petals become green and stamens are transformed into leaves like structures which become thick and club shaped. All parts of plants except roots are attacked. The peduncle, pedicel, sepals; petals and carpels become hypertrophied, thickened and leaf-like. The ovules and pollens are usually atrophied, resulting in sterility. The formation of chlorophyll in these parts, increases the photosynthetic activity and starch gets accumulated in these parts, which imparts a characteristic violet discoloration in these parts. In the hypertrophied parts oospores are formed in large numbers in the intercellular spaces. When systematic infection occurs early; the entire plant becomes dwarfed and only small leaves are formed. Normally lateral buds may be stimulated and they grow in to abnormal lateral shoots. The disease becomes more serious if it occurs along with the downy mildew.

Survival and spread :

The pathogen survives through oospores in affected host tissue and soil. Secondary infection is carried out by sporangia and zoospores which produce new infection.

Favourable conditions :

Moist more than 70 % relative humidity coupled with warm weather 12-25 ^{0}C and intermittent rains favours disease development.

Control measures

i) All crops refuse should be destroyed carefully after harvest.
ii) Follow crop rotation with non-cruciferous crops.
iii) Give two to three sprayings with 0.3 % Blitox-50 or Dithane Z-78 or Difolatan at the rate of 2 gm per litre of water as soon as symptoms are noticed.

(3) Downy mildew :

Cause : *Peronospora brassiocae* Gaumann.

Symptoms :

All aerial plant parts are affected but the foliage and inflorescence are more severely infected. The yellow, irregular spots appear on the upper surface of the leaves and white mycelial growth is visible on the under-surface of leaf opposite to the spots. In severe infection the inflorescence is malformed, twisted and covered with a white powder. No pod formation occurs on the infected inflorescence. Sometimes the stems are swelled and may be smaller or several inches long.

Survival and spread :

The pathogen survives as oospores on the affected plant tissue in soil and on weed hosts.

Favourable condition :

Atmospheric temperature in the range of 10-20 ^{0}C and relative humidity 90 % favours disease development.

Control measures :

i) Use healthy seeds for sowing.

ii) Spray the crop with Dithane Z-78 (0.2%) or Karathane (0.1%) with the appearance of the disease and repeat the spray two to three times at 10 days interval.

(4) Sclerotina stem rot :

Cause :

Sclerotinia selerotiorum (Lib.) de Bary. It is a soil borne fungus and over winters in the form of sclerotia.

Symptoms :

All parts of plants are attacked. Symptoms develop in the form of buff to light brown water soaked lesion which rot and later on covered with white frosty growth of fungus. A large number of black sclerotia appear in the fungal growth around the rotted stem. The seeds may become smaller and shrivelled. The disease is more serious in cool and dry weather.

Plant look likes whitish from distance at internodes or base. Premature ripening and shredding of stem, wilting and drying. Brown to black sclerotial bodies may also be seen in the later stage on the infected plant parts.

Survival and spread :

The pathogen survives as mycelium in dead or live plants and as sclerotia in infected plant parts or on the soil surface or with seed as contaminant.

Favourable condition :

High humidity 90-95 % and average temperature 18-25 ^{0}C along with wind current favours the disease development.

Control measures

i) Apply Benonyl at the rate of 0.2 % concentration.

ii) Spraying of Benlate (0.025 %) three times of 10-12 days interval has been recommended.

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PULSES

Exercise No. 5

(5) (A) Gram (5) (B) Pea

(5) (A) Gram :

(5) (A) (1) Wilt :

Cause : *Fusarium oxysporum* var. *ciceri* or *Ozonium* sp.
It is a soil borne fungus.

Symptoms :

The disease can affect the crop at any stage. The symptoms can be seen on the seedlings as well as on the mature plants. The leaves in the initial stages become pale yellow and ultimately dry up. The plants also become yellowish and finally dry out. Roots turn black and decompose ultimately. Dark brown or dark discoloration of the internal stem tissue is visible. At adult stage, dropping of petioles, rachis and leaflets and finally entire plant wiltd.

Epidemiology :

F. oxysporum f. sp. *ciceri* is both soil and seed-borne in nature. It can survive in the soil on infected plant parts and also carried over by other host plants. Several environmental factors such as soil temperature, soil moisture, soil nutrients, soil pH and inoculum play important role in the disease development. Adequate information is lacking on the role of biotic factors such as inoculum density, plant age, plant density and races on the epidemology of the disease. The fungus grows well at 25 ^{0}C and pH range of 5-7. Sandy and alkaline soils favour wilt development. The pathogen within the host tissues could not survive in the flooded soil for more than 65 days. However, it could survive for over 200 days in constantly wet soil.

Control measures :

i) Deep summer ploughing..
i) Grow resistant varieties like G-24 or C-214.
iii) Seed treatment with *T. viride* @ 4 g/kg or *P. fluorescens* @ 10 g/kg of seed or Carbendazim or Thiram 2 gm/kg of seed.
iv) Crop rotation of three to four years may be followed in the fields of heavy incidence of gram wilt.
v) Deep planting of gram seeds about 8-10 centimetre deep in the light soils reduces the gram wilt incidence.
vi) Sowing of gram should be done after 2nd week of October.
vii) Spot drenching with Carbendazim 1 g/litre or *P. fluorescens* / *T. viride* @ 2.5 kg/ha with 50 kg FYM.

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(5) (A) (2) Gray Mould :

Cause : *Botrytis cinerea.*

Symptoms :
Lack of pod setting is the first indication. Under favourable conditions, foliage shows symptoms and plants often die in patches. Shedding of flowers and leaves, covered with spore mass can be seen. Lesions on stem are 10-30 mm long and girdle the stem fully. Tender branches break off at the point where the gray mold has caused rotting. Affected flowers turn in to a rotting mass. Lesions on the pod are water soaked and irregular. On infected plants, the pods contain either small, shriveled seeds or no seeds at all.

Favourable factors :
Leftover plant residue and infected soil provides inoculum for infection. The optimal conditions are cool temperature 15-23 ^{0}C dense canopies, wet weather or leaves, and high humidity, especially as the stand approaches maturity.

Control measures :
i) Avoid excessive vegetative growth.
ii) Intercrop with linseed.
iii) Avoid excessive irrigation. Use compact varieties.
iii) Seed treatment with Carbendazim + Thiram (1:1) @ 3g/kg of seed is recommended or Spray the crop with Captan 5-6 kg/ha at 15 days interval / Spray of Carbendazim @ 1.5 g/litre of water is recommended / Spray Manocozeb @ 3 g/litre of water.

(5) (A) (3) Ascochyta blight :

Cause : *Ascochyta rabiei.*

Symptoms :
All plant parts are affected. Symptoms appear on leaves as water soaked lesions. Symptoms include smaller circular brown spots on leaves. Under favourable conditions, these spots enlarge rapidly and coalesce, blighting the leaves and buds. In case of severe infection, the entire plant dries up suddenly. The lesions are also developed on stems and petioles. Late infections result in shriveled and infected seed. The disease is seed borne in nature. Left over debris in the fields serve as a source. Wet and warm weather, and dense crop canopy are conducive to he spread of the disease.

Favouring factors :
Infected seeds, infected debris and infected fields nearby. Moist weather conditions, rain splash, wind and sprinkler irrigation. Moderate temperatures between 15-25 ^{0}C. Leaf wetness for 18-24 hours. Dense stands of chickpeas.

Control measures :
i) Sow disease-free seed.
ii) Follow rotation crop.

iii) Intercrop with wheat, barley, mustard.
iv) Seed treatment with Carbendazim @ 1 g/kg of seed or Hot water seed treatment (52 ^{0}C for 10 minutes) to lower the infestation.
v) Spray the crop with Mancozeb @ 2.5 g/litre if noticed during the growth period **or** Spray wettable sulphur at the rate of 2.3 g/litre of water.

(5) (B) Pea :

(5) (B) (1) Downy mildew :

Cause : *Peronospora pisi* Sydow.

Symptoms :

The symptoms appear on the upper surface of the leaves as scattered yellow to brown patches of indeterminate shape. The infected tissues soon die and turn brown. The white to greyish violet downy growth of the fungus is covering the under surface of the infected leaf from the early stages. The pods are also infected and the seeds are smaller in size and aborted.

Etiology and Epidemiology :

Cool humid condition is very favourable for the disease development, while the disease incidence is slowed down with warm condition. This is due to the fact that the sporangia which behave as conidia spread the disease, but can not stand desiccation. The sporangia are blown about freely by the wind and spread the disease rapidly from plant to plant.

Control measures :

i) Follow crop rotation and field sanitation by burning the crop debris after harvest.
ii) Crops may be sprayed with Dithane Z-78 or Dithane M45 in case of heavy infestation.
iii) Deep tillage to bury crop residue.
iv) Use tolerant cultivar.
v) Use metalaxyl for seed treatment.

(5) (B) (2) Powdery mildew :

Cause : *Erysiphe polygoni* DC. Syn. *E. pisi*.

Symptoms :

The disease first appears on the leaves and then on the other green parts of the plant. The characteristic symptoms are the formation of white floury patches on both sides of the leaves and also on the tendrils, stems and pods. In advanced stages, the entire plant surface may be covered with white powder like mass. The superficial mass consists of mycelium and spores of the fungus. Later on black dots may appear late in the season when the crop reaches maturity. The pod and grain formation is adversely affected.

Etiology and Epidemiology :

Primary source of infection is infected debris. Secondary source of infection is wind blown conidia. Fungus survive in summer leaf over debris. Seed is not considered as main source of inoculum.

Control measures :

i) Spray Sulfex (0.25 %), or Elosal (0.5 %) or Thiovit (0.5 %), Mildex (0.2 %), Milstem (0.2 %), Cosan (0.2 %), Morocide (0.1 %), Karathane (0.2 %), Hexasul or any of the wettable sulfur at the rate of 3 g/litre of water. Give first spray after appearance of the disease in the crop. Second spray should be given after 14 days of first spray. Repeat spraying only if needed.

iii) Avoid late planting.

iv) After harvest collect and destroy the plant debris.

v) Resistant varieties like T-10, T-56, P-185, P-388, P-6583 and P-6587 may be grown.

(5) (B) (3) Rust :

Cause : *Uromyces fabae* (pers.) de Bary and *U. pisi* (Pers.) de Bary.

Symptoms :

All the green parts of the plant are affected. The earliest symptoms of the disease are the production of yellow spots (aecia) in round or elongated clusters. The uredo pustules develop on both surfaces of the leaf as well as on the other parts in form of powdery, light brown appearance. The teleuto sorus or pustules develop in brown or almost black in colour. The stem of the plant becomes malformed and the affected plant dies out.

Etiology :

Uromyces fabae is macrocyclic rust fungus, it exhibits all five spore forms known for the uredinales. It is autoecious, as all spores are produced by single host. After over wintering on residual plant material, diploid teliospores germinates in the spring with a metabasidium. After meiosis, the latter produces four haploid basidiospores with two different mating types. These spores after landing on a leaf of a host germinate and produce infection structure. Pycnia are produced which contain pycniospores. Pycniospore are exchanged between pycnia of different mating types and after spermatization, dikaryotization occurs in aecial primordial. An aecium differentiates and dikaryotic aeciospores are produced. These aeciospores germinate and form infection structure from which uredia develop which produce urediospores.

Epidemiology :

Low temperature 17-22 ^{0}C results in formation of secondary aecia while at 25 ^{0}C development of uredia takes place. Relative humidity 60-70 % favour uromyces fabae development and spread whereas temperature above 25 ^{0}C and below 7-8 ^{0}C along with rains disfavour rust spread.

Control measures :

i) Spray the crop with Dithane M-45 at the rae of 2 g per litre of water. Two to three sprays will be sufficient and should be given at 15-20 days interval.

ii) Grow resistant varieties.
iii) Affected plant debris should be collected and burnt after harvest.
iv) Spraying with Bayleton (0.05%) and Calixin (0.2 %) has shown effective in controlling the disease.

(5) (B) (4) Fusarium Wilt :

Causual organism : *Fusarium oxysporum* f. pisi (Linford) Snyder and Hansen.

Symptoms :

The earliest symptoms are seed near blossoming stage plants. Plant growth is checked, leaves become yellow and there is down ward curling of stipules and leaflets. Later on the leaves show loss of turgidity and the entire plant wilts and the stem shrivels.

These diseases caused by *Fusarium* sp. are more severe when the crops are sown early. They are not very serious diseases.

Etiology :

Fusarium oxysporum is a common soil pathogen and saprophyte that feeds on dead and decaying organic matter. It survive in the soil debris as a mycelium and all spore types but is most commonly recovered from the soil as chlomydospores. This pathogen spreads in two basic ways : By water splash and by planting equipments and long distance by infected transplants and seeds. *Fusarium oxysporum* infects a healthy plants by means of mycelia or by germinating spores penetrating the plants root tips, root wounds or lateral roots.

Control measures :

i) Seed treatment with Brassicol, Thiram or Agrosan GN or Bavistin at the rate of 2 gm/kg of seed before sowing.
ii) Avoid early sowing in badly infected areas.
iii) Grow resistant varieties.
iv) Follow suitable crop rotation for 3 to 4 years using crops other than peas.
v) Use certified seeds only.

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ii) Grow resistant varieties.
iii) Affected plant debris should be collected and burnt after harvest.
iv) Spraying with Bayleton (0.05%) and Calixin (0.2 %) has shown effective in controlling the disease.

(5) (B) (4) Fusarium Wilt :

Causual organism : *Fusarium oxysporum* f. pisi (Linford) Snyder and Hansen.

Symptoms :

The earliest symptoms are seed near blossoming stage plants. Plant growth is checked, leaves become yellow and there is down ward curling of stipules and leaflets. Later on the leaves show loss of turgidity and the entire plant wilts and the stem shrivels.

These diseases caused by *Fusarium* sp. are more severe when the crops are sown early. They are not very serious diseases.

Etiology :

Fusarium oxysporum is a common soil pathogen and saprophyte that feeds on dead and decaying organic matter. It survive in the soil debris as a mycelium and all spore types but is most commonly recovered from the soil as chlomydospores. This pathogen spreads in two basic ways : By water splash and by planting equipments and long distance by infected transplants and seeds. *Fusarium oxysporum* infects a healthy plants by means of mycelia or by germinating spores penetrating the plants root tips, root wounds or lateral roots.

Control measures :

i) Seed treatment with Brassicol, Thiram or Agrosan GN or Bavistin at the rate of 2 gm/kg of seed before sowing.
ii) Avoid early sowing in badly infected areas.
iii) Grow resistant varieties.
iv) Follow suitable crop rotation for 3 to 4 years using crops other than peas.
v) Use certified seeds only.

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Exercise No. 6

(A) Lentil and (B) Linseed

(6) (A) Lentil :

(6) (A) (1) Rust :

Cause : *Uromyces fabae*

Symptoms :

Rust disease is a potential threat to lentil cultivation and causes substantial yield losses ranging from 60-69 per cent. Rust pustules can be seen on leaf blade, petiole and stem. Rust starts with the formation of yellowish-white pycnidia and accial cups on the lower surface of leaflets and on pods, singly or in small groups in a circular form. Later, brown uredial pustules emerge on either surface of leaflets, stem and pods. Pustules are oval to circular and up to 1 mm in diameter. They may coalesce to form larger pustules. In severe infections leaves are shed and plants dry prematurely, the affected plant dries without forming any seeds in pods or with small shriveled seeds.

Epidemiology :

The disease generally starts from low-lying patches in the paddock and radiates towards the border. Rust is an autoecious fungus, completing its life cycle on lentil. High humidity, cloudy or drizzly weather with temperatures 20 to 22 ^{0}C favour disease development. The disease generally occurs during the flowering / early podding stage.

Control measures :

i) Use of foliar fungicides as Hexaferb and Dithane M-45 give best control.

ii) Fungicides as Mancozeb (0.2 % a.i.), Bayleon (0.05 % a.i.) and Calixin (0.2 % a.i.) are found effective against the pathogen.

ii) Foliar spray of Benomyl, Carboxin, Metalaxyl, Oxycarboxin.

(6) (A) (2) Wilt :

Cause : *Fusarium oxysporum* f. sp. *lentis*.

Symptoms :

Early sown crops are damaged much. In this disease the growth of the plants is checked, leaves start yellowing. Infected plants start drying and finally die. The roots of infected plants remain under developed and look light brown in colour. Seedlings characterized by sudden dropping, followed by drying of leaves and seedling death. The roots appear healthy, with reduced proliferation and nodulation and usually no internal discoloration of vascular system. Seed from plants affected in mid-pod-fill to late pod-fill are often shriveled.

Epidemiology :

The fungus is born which can survive in the soil and plant debris in the absence of its host for a period of 3-4 years. The disease is favoured by low soil temperature, 30 % soil water holding capacity and increasing plant maturity. Yield losses depend on the stage at which the plant wilts. It can be 100 % when wilt occurs at pre-pod stage, about 67 % when it occurs at pre-harvest stage.

Control measures :

i) Follow three years crop rotation and clear cultivation.
ii) Use healthy seeds.
iii) Grow moderately resistant variety like Pant-4-406.
iv) Early sowing may be avoided.
v) Seed treatment with benomyl (0.3 %) reduces wilt incidence.
vi) Using antagonistic microflora like *Bacillus sabtilis, Tricoderma harziarum, T. viride* @ 4 g/kg seed.

(6) (B) Linseed :

(6) (B) (1) Alternaria bud blight :

Cause : *Alternaria lini* Dey.

Symptoms :

All the aerial parts of the plants are infected but the chief symptom of the disease appears on the floral parts. In the diseased plants the buds fail to open which is later on followed by the appearance of small, dark, black spot near the calyx. The spots increase in size and reach up to the pedicel. The floral parts shrink and rot away and the closed buds die out ultimately.

Control measures :

i) Seed treatment with Thiram or Captan at the rate of 2.5 g per kg of seed before sowing.
ii) Spray the crop with Zineb (Dithane Z-78) @ 2.5 g per litre of water. The first spray should be given with the appearance of initial symptom. Sprays may be repeated according to the economic loss.
iii) Grow late maturing varieties.

(6) (B) (2) Rust :

Cause : *Melamposora lini* (Ethrenb.) Lev.

Symptoms :

The rust appears on all the aerial parts of the plant including the capsules. The characteristic symptom is the appearance of the yellowish orange pustules on both the surfaces of leaves. The leaves become chlorotic and fall off. Later on reddish-brown to black elongated lesions appear on the stems and the young twigs.

Etiology :

In temperate countries, primary infection takes place through basidiospores which are produced as a result of germination of teleutospores perennating in the soil. Uredospores are killed due to excessive temperatures. It is presumed that the uredospores produced on linseed at hills come down to plains to cause infection. Thus, the primary inoculum, windblown, fall on the host, germinate the cause infection.

Control measures :

i) Avoid excessive us of nitrogenous fertilizers.
ii) Spray the crop with Dithane Z-78 @ 2g per litre of water or dust the crop with sulphur dust @ 15-20 kg per hectare.
iii) Grow resistant varieties like Neelum, Hira, Mukta, K2, and LC-185.
iv) Affected plant portions after harvest should be collected and burnt.

(6) (B) (3) Powdery mildew :

Cause : Oidium lini Skorik.

Symptoms :

The first symptom of the disease is the appearance of greyish-white powdery growth on the youngest growing tips of the plant. The branches, leaves and flowers are also infected in case of severe attack. The infection results in the defoliation of infected plants and shrivelling of grains.

Epidemiology :

Primary inoculum may be present in the form of mycelium on infected tissue, mycelium within buds, or ascospores contained within ascocarps.

Control measures :

i) Affected plant debris should be collected and burnt after harvest.
ii) Spray the crop with Sulfex or Elosal or Thiovit at the rate of 3 g per litre of water. Repeat spraying according to the disease intensity and economic loss.

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CASH CROPS

Exercise No. 7

Cotton

(1) Root rot

Causal organism – Sterile stage : *Rhizoctonia bataticola* (Taub.) Butl.
Imperfect stage: *Macrophomina phaseolina* (Tassi) Goid.

Symptoms:

The symptoms may appear in seedling stage, where brownish spots on the cotyledons are seen. At collar region there is browning, which may extend downward. The fibrous roots undergo decaying. The bark of the roots show rotting and shredding. Affected plants can be pulled out with ease. The disease appears in patches in the field.

Etiology:

The hyphae are thick, septate and produce black and irregular sclerotial bodies. Crop residue with sclerotia act at primary source of infection.

Control measures :

1. Seed treatment with Thiram or Captan @ 3 g +1 g Carbendazim/ kg seed.
2. Soil mulching after rains.
3. Mixed cropping with legumes and Sorghum.
5. Soil disinfection with 0.1% Carbendazim helps in controlling the disease.

(2) Wilt :

Cause : *Fusarium oxysporum f. vasinfectum* (Atk).
Snyder and Hansen.

Symptoms :

The chief symptoms are the yellowing, wilting and drooping of the leaves. Affected plants are stunted . They gradually wilt and die. The vascular system becomes brown. Often the diseased plants are small with smaller leaves and bolls. Leaf discolouration appears around the edges and progresses towards the midrib and the leaves gradually drop.

The fungus survives in the soil as chlamydospores and sclerotia and in the infected plant parts as mycelium.

Etiology :

Pathogen survives for long period in soil and on cotton debris or other organic materials in the soil as saprophyte for many years. Secondary spread may be through wind, water and other physical and biological agents. The fungus grows at a wide range of temperature from 11 to 35 ^{0}C, pH required is 5.2 to 7.2, optimum temperature range is 24-28 ^{0}C.

Control measures :

i) Grow resistant varieties. American cottons are more resistant to the disease. C-C-1-1-35, NA-3 (77) (*G. arboreum*) and EZ-13-02 (*G. herbaceum*),
ii) Sow healthy seeds from healthy plants.
iii) Seed treatment with fungicide like Vitavax and Bavistin.
iv) Apply organic manure and potash in sufficient amount in the field before sowing.
v) Follow crop rotation with millets, groundnut, sorghum and small grains.
vi) Soil amendments with Zinc also reduce the wilt.

(3) Anthracnose

Causal organism : Imperfect stage: *Colletotrichum gossypii* Southw.
Colletotrichum capsici (Syd.) Butl & Bisby
Perfect stage : *Glomerella gossypii* (Southw) Edgerton

Symptoms :

In seedling stage, small, reddish circular spots appear on the cotyledons and primary leaves. When the lesions are on the collar region, the stem may be girdled, causing seedlings to wilt and die. In mature plants, the fungus attacks the stem, causing it to split and shred the bark. The symptoms are prominent on bolls as water-soaked, circular, slightly sunken reddish-brown spots which turn black in colour. As a result of boll infection, they open prematurely. The lint becomes stained, hard and compact.

Etiology:

The mycelium is septate. The fungus produces acervuli in which the setae are present. Conidia are hyaline, single-celled, sickle shaped.

Perpetuation:

Primary infection takes place through infected seeds and plant-debris, secondary infection by means of conidia disseminated by wind.

Control measures :

1. Seed treatment with Thiram 3 g OR Carbendazimm 1g + Thiram 3 g/kg of seed.
2. Spray the crop once or twice with copper oxychloride (0.25%) or Zineb (0.25%) after boll formation.

(4) Black arm :

Causal organism : *Xanthomonas axonopodis* pv. *malvacearum* (Smith)Vauterin et al.,

Symptoms :

Small water-soaked spots appear on the under surface of cotyledons, which may dry and wither. Such spots also appear on the leaves. They become angular bound by veinlets and turn brown to black in colour. Several small spots may coalesce. The infected petiole may collapse. Elongated, sunken and dark brown to black lesions appear on stem, petioles and branches. The young stems may be girdled and killed in the black arm phase. Sunken black lesions may be seen on the bolls. Young boll may fall-off. The attacked stem becomes weak. Bacterial slime is exuded on the brown lesions. Discolouration of lint may take place.

Perpetuation :

The pathogen can remain as slimy mass inside the seed or on the fuzz. The disease may be carried over through infected leaves, bolls and twigs on the soil surface. The secondary infection is through water, wind.

Control measures

1. Field sanitation.
2. a) Externally seed borne infection can be eradicated by delinting the seed with Conc H_2SO_4 for 5 minutes, wash with lime solution to neutralise the effect and finally washing with running water to remove the residue and drying seeds.
   b) Internally seed borne infection canbe eradicated by soaking seeds overnight in 100 ppm streptomycin sulphate or agrimycin.
3. Secondary spread of the diseasecan be controlled by spraying the crop with streptomycin sulphate 100 ppm+ Copper oxychloride (0.25%) at an interval of 15 days.

(5) Dahiya diseases

Causal organism: Imperfect stage : *Ramularia areola* Atk.
Perfect stage : *Mycosphaerella areola.*

Symptoms :

The fungus usually attacks the older leaves causing irregular to angular, pale, translucent spots. They are usually restricted by the veinlets and appear mostly on the lower surface of the leaf, though occasionally on the upper surface. A few to over a hundred spots may be found on a single leaf. In severe infections the leaves turn yellowish brown and fall off prematurely.

Etiology: A frosty or whitish grey mildew growth consists of conidia and conidiophores and mycelium of the fungus. The mycelium is endophytic, septate. The conidiophores are short, septate, branched at the base. The conidia are borne singly or in short chains at the tip of the conidiophores and are colourless, irregularly oblong, with pointed, rounded, or flattened end, unicellular to three septate.

Control measures :

1. Destruction of infected plant debris.
2) Dust the crop with 300 mesh sulphur @ 20 kg/ha or spray crop with0.05% propiconazole.

(6) Leaf curl of cotton

Causal agent : The causal agent is Gemini virus and is called as **Cotton Leaf Curl Virus** .

Leaf curl disease was first observed on *G. barbadense* in 1989 in Delhi and on *G. hirsutum* cultivars during 1993 from Sriganganagar area of Rajasthan. Now it is prevalent in Punjab and Haryana also.

Symptoms :

The affected leaves show curling both upward and downward, the veins on lower side are thick and raised and are dark green compared to normal transluscent veins healthy plants.

On the main veins of lower side, the presence of small cup shaped secondary leaves is a typical feature prominently on middle main vein and invariably near the nectary point. This cup shaped enation can also be seen on other veins on lower side of the leaves. The affected plants are stunted in growth with less number of bolls. The disease is spread by the whitely Bremisia tobaci Genn.

Epidemiology : Cotton is grown from May to December in North zone. For the remaining period some weeds and crops serve as alternate hosts of the virus. Most common alternate host is okra (*Abelmoschus esculentus*). The buildup of whitefly early in the season on alternate hosts leads to severe infection of CLCV on cotton. The climate favouring whitefly population build up is epidemiologically important.

Control measures :

i) The spread of the disease can be checked by managing the whitefly with the application of botanical pesticides and insecticides. The repeated use of same insecticide should be discouraged to avoid resistance development in whitefly.

ii) Care should be taken to spray the under surface of the leaves at whiteflies harbour mostly on lower surface.

iii) Weeds harbouring whitefly near cotton fields should be immediately destroyed..

iv) Okra should not be grown between March to June to avoid possible build up of the virus and its vector.

v) Excessive application of nitrogenous fertilizers should be avoided.

v) The CLCV infected plants should be removed and burnt.

(7) 2-4-D Injury

2-4-D is a broad spectrum selective post emergence herbicide used for control of herbaceous annual dicot weeds. But crops like cotton, tomato, tobacco are extremely sensitive to 2-4-D which can cause harmful damage to crops (cotton).

Symptoms :

1) Leaves are greatly modified. They become narrow and deeply lobed. Scorching, rolling and puckering of leaves which hamper normal photosynthesis in plants. As a result of 2-4-D injury there can be elongation of leaves giving string like slender appearance.

2) Disruption of phloem tissues and dislocation of normal translocation of food material- so plants looks like wilted.

3) Alternation of nucleic acid (protein metabolism)- Growth ceases

4) Blocking of gaseous exchange between leaf and atmosphere so ultimately plant die.

5) Malformation is also observed in crop.

6) If 2,4-D concentration is high then the symptoms are seen within 2-3 days.

7) If 2,4-D concentration is low then the symptoms are seen within 8-10 days.

Control measures :

1) Urea (2%) spray may show little recovery if the damage is in early stage

HORTICULTURAL CROPS

Exercise No. 8

Mango

(1) Die back :

Causal organism: *Botryodiplodia theobromae*

Symptoms:

The disease is characterized by drying of branches and twigs from tip downwards followed by complete defoliation. The onset of dieback becomes evident by discolouration and darkening of the bark some distance from the tip leading to death of the branches or twigs. Internal browning in the wood tissue is observed on splitting open the twigs along the long axis.

Perpetuation:

Diseased twigs bearing fruiting bodies are the main source of perpetuation and survival of the pathogen and thus serve as initial inoculum for next season.

Control measures :

1. Prunning of diseased twigs (3 inch below the infection site) followed by spraying of 1 per cent Bordeaux mixture.
2. Selection of scion from healthy trees helps in keeping the disease under check.

(2) Anthracnose :

Causal organism: *Colletotrichum gloeosporioides*
(Perfect stage : *Glomerella cingulata*)

Symptoms:

The fungus attacks the young leaves, stem, inflorescence and the fruits, the damage being maximum when the fruits are attacked. Small brown spots appear on the leaves and inflorescences. Twigs show dieback symptoms. Flowers shed when inflorescence is infected. When the fruits are attacked, black, round or irregular sunken spots are formed and the skin becomes discoloured. Older infected fruits rot and decay.

Etiology:

Mycelium is septate. Acervuli develop profusely in diseased parts of the plant. The conidia in mass are pinkish but hyaline individually. Conidia are straight, cylindrical or oval, hyaline, single celled with round ends and sometime contain one or two oil globules.

Perpetuation:

Infected plant parts (diseased twigs, leaves and fruits) are primary source of infection. Secondary spread is through air-borne conidia. The optimum temperature for infection was found to be 25^0 C and relative humidity from 95-97 %.

Control measures :

1. Spraying Bordeaux mixture (1%) or copper oxy-chloride 50 WP (0.25%) or Carbendazim 50 WP (0.1%) or Captan (0.2%) at 15 days interval until harvest effectively controls anthracnose.
2. Before storage, dip fruits in Carbendazim 50 WP solution 0.05 % i.e, 5g/10 litres of water) for 5 minutes.

(3) Mango malformation :

Causal organism: *Fusarium moniliforme* var. *subglutinans* wollenw and Reink.

Mango malformation is also known as bunchy top is a very serious threat to mango industry.

Symptoms: Three distinct types of symptoms are produced. They are bunchy top seedling, repetitive malformation and floral malformation.

Bunchy top phase:

Appears on young plants in the nursery beds when they are 4-5 months old. The growth of the plant is stopped and it gives an appearance of bunchy top.

Vegetative malformation: Induces excessive receptive branches of limited gowth in young seedlings.

Malformation of Inflorescence : Shows variation in panicle formation.

Etiology:

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

Perpetuation:

The fungus does not sporulate in situ but it sporulates on drying malformed panicles. A tree once affected can not escape from the disease in subsequent years. Diseased propogating material help in the spread of the disease.

Control measures :

1. The disease incidence is reduced by spraying with NAA at 100 to 200 ppm during October.
   Prunning of diseased parts followed by spraying of Carbendazim 0.1 per cent or Captafol 0.2 per cent effectively control the disease.

(4) Bacterial blight :
Bacterial Leaf spot of Mango :

The Causal organism : *Pseudomonas mangiferae pv. indicae*

The disease occurs in almost all the states of India, where mango is grown and causes considerable loss in yield during some seasons.

Symptoms :

The symptoms appear as minute, dark-grown or black, water-soaked spots in large numbers in groups, mostly near the leaf tips. The spots enlarge under favourable conditions and may reach 1.0-4.0 mm in size. The spots become black and are surrounded by a yellow halo. As the spots enlarge, they are limited by the veins and become angular in shape. Many such spots may coalesce to form large, black patches. On the surface of the spots, bacterial ooze comes out, which on drying forms a rough coating. When large areas of the leaves are affected, they turn yellow and fall off prematurely. The bacteria attack the petioles, twigs of branches and fruits. On the surface of fruits in the early stages of development, small dark-brown or black, water-soaked spots develop. Small cracks may be formed on the skin of affected fruits. Severely affected fruits drop off prematurely. The bacteria causing the disease are rod-shaped, occurring singly or in short chains, motile by a single polar flagellum and measure 0.45-1.4 x 0.36-0.54 μ in size. They are non-spore forming and are aerobic in nature.

Mode of survival, spread and epidemiology :

The host being a perennial tree, the bacteria survive in the affected leaves and twigs of branches throughout the year. The pathogen is disseminated by strong winds, lashing rains and by insects mechanically. The bacteria enter the leaves through the stomata, through injuries caused by insect pests, especially the sucking insects and through lenticels in the twigs and fruits.

Humid and moist conditions, continuous rains and moderate temperature favour occurrence and development of the disease.

Disease management :

Agronomic practices :

i) Severely affected branches may be pruned during the off season.

ii) Healthy and disease-free seedlings should be used for planting.

Chemical control :

i) Spraying the crop with Streptocycline at 100-200 ppm (1.0-2.0 gm/10 litres of water), twice, at 20 days interval, when the fruits are young reduces the fruit infection.

ii) Suitable insecticides may be applied to control insect pests causing injuries to the leaves and young fruits, which helps to minimize infection by the bacteria.

(5) Powdery mildew :

Causal organism: *Oidium mangiferae* Berth.

It is a serious disease of mango affecting almost every variety.

Symptoms:

The disease is easily recognized by the whitish, sometimes grayish powdery growth on the inflorescence and tender leaves. Generally the infection starts from the tip of the inflorescence and spreads downwards covering the floral axis, tender leaves and thin stem. Infected floral parts are severely damaged and drop off. If the fruit is already set it may drop off prematurely. The fruits are also sometimes malformed and discoloured due to severe mildew attack. The axis may begin to dry showing characteristic die back symptoms.

Etiology:

The mycelium is ectophytic, branched with haustoria and hyaline. Conidia are born in chains on short conidiophores. They are hyaline, single celled. Pathogen is an obligate parasite. Perfect stage of fungus has not been observed.

Perpetuation:

Primary infection is through dormant mycelium on the tree. Secondary infection takes place through air borne conidia.

Control measures :

Spray the crop with wettable sulphur 80 WP (0.2 %) or Carbendazim 50 WP (0.1%) or Karthane 50 WP (0.1%) or Tridemorph (Calixin) 75 EC (0.05%) or Hexaconazole (Contaf) 5 EC (0.05%) as soon as incidence is noticed.

(6) Spongy tissue :

Symptoms :

Apparently normal and attractive fruits on cutting reveal spongy developed in the flesh. The fruits have a bad colour and become unfit for consumption. This type of disorder is mostly encountered in variety **Alphonso,** where a pulp patch fails to ripen and the area affected will appear white and soft like sponge.

Spongy tissue formation in mangoes is caused by the shift of the seed to germination mode inside the fruit. Germination is the process by which a seed development in to a plant. During this stage, the seed requires nutrients to develop. So when the seed inside the mango shifts into germination mode, it sucks the nutrients from the surrounding fleshy part of the fruit, making it appear white and spongy. Hence as the fruit ripens, the patch affected by this malady can be seen clearly.

Since the spot is devoid of nutrients, it begins to rot (identified by the appearance of black colour). If identified earlier, the particular patch can be removed and the rest of the mango can be consumed. But it is difficult to indentify from the outside and hence the rot spreads, making the mangoes inedible. When the rotten mangoes are stored along with other

mangoes, the rot spreads to other mangoes too. This has been the cause for decrease in the quality and also in production of mango in India.

Control measures :

It is major problem in Alphonso, due to inactivity of ripening enzymes due to high temperature, convective heat and post harvest exposure to sunlight.

Harvesting the fruit when it is three-fourth matured instead of waiting for it to mature fully, and then storing it in a cool place will prevent any rotting'.

Mulching has shown promising results in the prevention of spongy tissue effect.

(7) Red rust :

Causal organism: *Cephaleuros virescens*

Symptoms:

Disease can be easily recognized by the rusty- red spots mainly on leaves and sometimes on petioles and young twigs. Initially the spots are greenish-gray in colour and velvety in texture. Later they turn to reddish brown. Spots are slightly raised and coalesce to form larger ones. The upper surface of spots consists of unbranched filaments which project through cuticle. Some filaments represent sterile cells while some are fertile. The fertile cells bear cluster of spores at the apex which are brown in colour.

Reduction in photosynthetic activity and defoliation as a result of algal attack, lower vitality of host plant.

Spread and perpetuation:

Besides mango, algae attack many types of other plant species. The disease is more common on closely planted mother plants. Fruiting bodies of algae are formed in moist atmosphere. Zoospores formed by sporangium initiate fresh infections. Pathogen invades cortical cells of host plant and mainly spreads with rain splashes and winds.

Control measures :

Avoidance of close planting. Sprays of Bordeaux minture (1 %) or copper oxychloride (0.25%) helps in checking the disease.

(8) Pink diseases :

Causal organism: *Botryobasidium salmonicolor*

Symptoms:

The disease is noticed as pinkish powdery coating on the twigs and branches. Later, the fungus invades bark to get established in the internal tissues and interferes with the transport of nutrients. Often the fungal growth spreads to girdle the stem. Severely infected bark gets shredded and the wood exposed. Leaves turn yellow and dry. Roots are not infected. The pink colour on the tissues represents profuse conidial production by the fungus and hence the name 'pink disease' is given.

Perpetuation:

The fungus persists from one season to another through dormant mycelium inside the bark and the cankerous tissues serve as a potential source of infection in wet season. The disease is seen after rainy season.

Control measures:

The disease can be kept under control by cutting and removing the affected branches. Cut ends should be protected with Bordeaux paste. The disease can also be controlled by lime sulphur and oil based copper.

(9) Loranthus :

It is also known as Mistletoes. It is a partial stem parasite of many perennial dicot trees having woody nature. The parasite has true functional leaves however, it lacks true root system and therefore, it is unable to sustain in absence of host plants. The parasite has to depend on the host for nutrition and water. Nutrients and water absorbed by roots of the host plant is diverted for the growth of the parasite. As a result, the growth of the host above the point of penetration show marked reduction. Simultaneously, parasite develops in a faster rate. Development of many loranthus branches completely weaken the host. Vigour of the host plant is markedly reduced and automatically, there is reduction in yield and quality of fruits of the host plant.

Life cycle of the parasite:

Berry type fruits are produced on loranthus branches in summer. Birds upon consuming these berries disseminate the seeds, which remain adhered on tree trunks at the branching junctions of the host. Seeds on the host surface (tree trunk) germinate on the onset of monsoon and directly penetrate the host. Initial growth of the parasite is slow. Upon penetration into the host, sucking organ 'haustorium' is produced by the parasite within the host tissue, which penetrates and absorbs nutrients from the xylem of the host. Establishment of parasitic relationship results into development of big knot or gall like over growth and the point of contact of the parasite and host.

Host range:

There are many cultivated and wild hosts of the parasite.

Cultivated hosts: Mango, Citrus, Jackfruit, Sapota.

Wild Hosts: Teak, Shiwan, Harda, Beheda.

Control measures :

1. Removal by scrapping of the parasite form infected branch before flowering with the help of 'Amar' loranthus cutter.
2. Well established loranthus bushes be cut below the point of penetration and destroyed.
3. Application of 0.5% Glyphosate at the point from where the loranthus growth has been scrapped to avoid further re-emergence of the parasite from the established haustorium.

(10) Stone graft Mortality :

Stone grafting is the efficient and popular technique of rapid multiplication of quality planting material in mango. Severe mortality of sprouted grafts takes place if proper care is not taken.

Symptoms:

Newly sprouted grafts having light green coloured leaves show sudden drooping and loss of turgidity followed by wilting of the graft in next two to three days. There is longitudinal shrinkage of the stem followed by drying of leaves. Normally such type of wilting is observed 15-20 days after sprouting. Rootstock of such wilted plants many times sprouts from the base indicating the incompatibility of root stock and scion.

Causal organism: During the investigation it was initially thought that the disease is caused by the fungus *Fusarium oxysporium*. However, it was observed that, the wilting continued after fungicidal covering. The detail investigations revealed that, different non-biological (abiotic) causes are associated with this disease. These are

1. Use of improper potting mixture and poor drainage in polybags.
2. Incompatibility between rootstock and scion.
3. Exposure of graft to intense rain showers.

Control measures :

1. Selection of good quality seed (stone).
2. Deep the stones in 0.1 per cent Carbendazim to control *Sclerotium* growing on the stone.
3. Grafting should be done at a proper stage when the stem is red-brown in colour.
4. Select healthy matured scion stick of proper girth (pencil size).
5. It scion is too broad two root stocks should be used to supply adequate nutrients to the scion.
6. Use well drained soil and FYM in 3:1 proportion as a potting mixture.
7. Provide six punched holes at the base of plastic bag for proper drainage.
8. Drench 0.1% Emisan or 1% Bordeaux mixture solution 50-100 ml in each bag to avoid development of *Sclerotium* in soil.
9. Follow regular plant protection measures in the nursery to avoid pest and disease problem in the initial stage of graft growth.

(11) Lime induced chlorosis :

The lime induced iron chlorosis in fruit trees is a widespread and up to now unresolved problem in plant nutrition on calcareous soils in arid and semiarid areas. On average some 20% to 50% of different kinds of fruit trees suffer from this type of physiological disorder and show symptoms of iron deficiency. This is accompanied by poor yields, low fruit quality and the complete loss of trees.

Symptoms :

The leaves loose green colour and turn white and is called **Bleaching**. The size of the leaf is reduced. In severe cases of iron deficiency, the leaves dry from tip downwards. The

deficiency is cimmon in soils with high calcium content. Hence, the effect is known as **Calcium induced iron chlorosis.** Symptoms are first seen in the youngest leaves. Initially the veins remain green, which produces reticulate pattern of green veins on yellow leaves. The leaves eventually turn completely chlorotic but there is no associated necrosis.

Control measures:

Rhododendron-Lime-induced chlorosis.

1. Adjust soil pH to 4.5 to 6.0. Adding sulfur or organic matter, especially conifer needle mulch, which is highly acidic, may help.
2. Check plant's proximity to newly poured concrete.
3. Use iron chelate either as a soil treatment or by foliar feeding to quickly help chlorotic plants.
4. **CULTAN Method to solve problems of iron chlorosis :** In order to overcome these difficulties we propose to solve the problem of iron chlorosis in fruit trees by a newly developed technique based on the **CULTAN (Controlled uptake Long Term Ammoium Nutrition).** With this technique a proportion of the roots of a fruit tree is directed to a certain spot in the root zone by ammonium attraction. There for every tree about one liter of calcareous soil is replaced by compost material, which is enriched with iron and ammonium as sulfates and acidified by sulfuric acid to yield a pH-value of 3.0 . The ammonium in this mixture is stabilized by a nitrification inhibitor. From these spots trees take up ammonium, iron and sulfate - the latter being taken up as active sulfate in high surplus according to needs of sulfur of the plants. Therefore, a large amount of sulfate is available in the trees for the bonding calcium, which has been taken up from soil solutions rich in calcium carbonate or calcium bicarbonate. Thus metabolic carboxylation processes of the plants are supplemented with the surplus of sulfate to stabilize the pH-value of the plant sap. This is the main idea of our approach to solving the problem of iron deficiency. In a number of countries this technique has proved to be an effective and economical way of overcoming lime induced iron chlorosis.

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Exercise No. 9

Citrus

(1) Citrus canker :

The canker disease was first recorded in 1933 on herbarium of *Citrus medica* in England and United States., collected at Dehra Dun during 1827-1831. In India citrus canker was first reported from Punjab in 1940. It occurs in Assam, Andhra Pradesh, Karnataka, Madhya Pradesh, Tamil Nadu and Uttar Pradesh. Now the disease is known to occur in almost all the citrus growing areas. Acid lime is highly susceptible to citrus canker.

Symptoms: The disease attacks seedlings and grown up trees.

a. Seedlings: in young plants, especially in the nursery, the disease causes serious damage. Badly cankered leaves are shed and the assimilating surface of leaves is reduced while the canker spots often girdle the stems to cause partial or complete death of the plants.

b. Trees- On leaves: The disease affects all the plant parts of grown up trees *viz.*, leaves, twigs, thorns, older branches and fruits. On the leaves the disease first appears as small watery, translucent spots of yellow colour, than the surrounding tissue and with raised convex surface. As the spot mature, the surface becomes white or grayish and finally ruptures in the centre giving a rough, hard, corky and crater-like appearance. The lesions which are circular when young, become irregular when old. The old lesions are light brown in grape fruit, dark brown in sweet orange, mandarin orange and trifoliate, almost black on lime and lemon leaves. The spots increase in size from 1.0 to 10.0 mm in diameter and may coalesce to form elongated lesions. The size and abundance of lesions vary with the kinds of citrus trees and conditions of growth. Lesions are largest on grape fruit leaves nearly 13 mm in diameter. On leaves of lime and lemons, they are much smaller, often not more than 3 mm in diameter.

On twigs and branches: Lesions on twigs and branches are quite similar. Branches of 50 to 75 mm in diameter are commonly infected. The canker growth often encircles the twigs causing the death of the portions above the infected area leading to die-back of shoots.

On fruits: The infection spreads to the fruits on which typical cankerous spots are formed. Yellow halo around the canker is absent in fruits. The cankers may be scattered all over the surface or several cankers may occur together forming an irregular scurfy mass. Gumming is sometimes associated with spots on fruits. More effect on pulp is noticed and juice content is much reduced. The market value is very much reduced because of the canker spots on the fruits. Cankers provide point of entry to secondary rotting organisms. The affected plants are stunted and fruit yields are reduced considerably.

On roots: Canker has never been observed occurring naturally on roots of even badly diseased trees. However, the disease has been found on grape fruit roots exposed above ground surface.

Causal organism:

Xanthomonas axonopodis pv. citri (Hasse) Vauterin *et al.* [Syn. *Xanthomonas campestris pv. citri* (Hasse) Dye.]. The bacterium is a rod shaped, 1.5 to 2.0 x 0.5 to 0.75 µm in size. It forms chains and capsules and is motile by single polar flagellum. No spores are formed. The bacterium is Gram-negative and aerobic. Bacterial colonies on beef agar are circular, straw yellow, slightly raised and glistening. Nitrate is not reduced by it. Three strains of the bacterium are identified in India.

Perpetuation:

The bacterium spreads mainly through wind splashed rains. Long distance dissemination takes place through diseased planting material. It survives in cankerous leaves, twigs and branches. It survives in the infected leaves for five months and infected twigs upto 76 months. Injury caused by leaf miner (*Phyllocnistis citrella*) paves the way for the entry of pathogenic bacterium.

Epidemiology:

The disease is serious in acid lime, lemon and grape fruit. Rarely it if found on sweet oranges and mandarins. Temperature between 20 and 35^{0}C with evenly distributed rains favour the disease. Presence of free moisture for 20 min on the host surface is essential for successful infection.

Control measures:

1. Dropped off canker affected leaves and twigs should be collected and burnt.
2. Disease free nursery stocks should be used for planting in new orchards.
3. The plants before planting in new orchards should be sprayed with Bordeaux mixture 1.0 per cent. In old orchards pruning of affected plant parts before the onset of monsoon and spraying with Bordeaux mixture 1.0 per cent at periodical intervals depending upon weather conditions controls the disease. Spraying should be done immediately after the appearance of every new flush of leaves.
4. The vigour of the plant should always be maintained by proper fertilization and irrigation. Manuring should be done in such a way that its maximum effect is felt during wet weather.
5. Proper care should be taken to minimize the attack of leaf miner which disseminate the disease.
6. The disease can be effectively controlled by spraying with streptomycin sulphate 500 to 1000 ppm at 15 days interval.
7. Spraying with neem cake solution has been found highly effective in checking citrus canker as well as leaf miner. Number of sprays for one year may range from 10 to 20.

Three sprayings with streptomycin 100 ppm + 0.3 per cent copper oxychloride (1.5 kg/ha) check the canker disease.

(2) Gummosis & Fruit rot :

The disease seems to occur especially in the high rainfall areas.

Symptoms:

Profuse gumming on the surface of the attacked bark is the main symptom. When gumming occurs on the stem, droplets of gum trickle down the stem. The bark shows conspicuous brown staining along with hardened masses of gum on the surface. It gradually turns dark brown and develops longitudinal cracks. When scrapped, the affected portion of the bark appears brown. A thin layer of wood tissues is also affected. When gumming starts close to the soil, the disease spreads on the main roots and then around the base of the trunk. As a result of severe gumming, the bark becomes completely rotten and the tree dries owing to girdling effect. Prior to death, the tree usually blossoms heavily and dies before the fruits mature. In such cases the disease is called foot rot or collar rot. The fungus produces blight symptoms on leaves. Blighted leaves drop off. The fruits lying on the ground are invaded by the fungus. Affected fruits develop brown rot.

Leaf fall and fruit rot:

This is a severe disease of mandarin oranges in heavy rainfall areas of south India. The pathogen becomes active after the outbreak of monsoon. Quick shedding of leaves is the earliest symptom. The infection starts as water-soaked lesion at the leaf base. By the time, the lesions extend to the whole leaf, the leaf drops off. The infection may spread to young twigs which are killed. Fruits of all stages are infected. The affected leaves show water-soaked patches on rind and subsequently such fruits drop off and rot. The surface of dropped fruit gets covered by cottony growth of the fungus. Foul odour is emitted by the rotting leaves and fruits. The pathogen may cause bark-rot, crown rot and girdling of the base of stem. Repeated attacks by the pathogen reduce vigour of the tree which may die.

Causal organism:

There are at least six different species of *Phytophthora* known to be associated with the disease, gummosis. They are *P. citrophthora* (Sm. & Sm.) Leon, *P. parasitica* Dastur, *P. palmivora* Butler, *P. hibernalis* Carne, *P. syringae* Kleb. and *P. cactorum* Sm. And Sm. The first three are more commonly present and are believed to be the chief causal agents of citrus gummosis.

Leaf fall and fruit rot is caused by *Phytophthora palmivora* Butl. The hypha is 3 µm in diameter. They are smooth without hyphal swellings. The hyphae are usually intercellular and intracellular. Sporangia are ellipsoid or ovoid with the widest part near the base, papillate and with a short pedicel. The sporangia measure 35 to 60 x 20 to 70 µm. The base of the sporangium is usually rounded and attached with the sporangiophore almost at right angles. Sporangiophores are upto 6.0 µm. It shows sympodial growth with a sporangium at the tip and a branch from immediately below. The sporangia germinate within 3 hrs and release zoospores, which are biflagellate and motile. Chlamydospores are 30 to 40 µm in diameter. Oospores are 30 µm in size.

Perpetuation:

The fungi survives on fallen fruits, twigs, leaves and cracks of the tree and spread by irrigation water, rain splashes, wind and insects to stems, leaves and fruits.

Epidemiology:

Serious occurrence of this disease is noticed in sweet oranges, acid lime and lemon. Heavy soil, high water table, high soil moisture, soil pH of 5.4 to 7.5 and temperature of 25 to 28°C are conducive for disease development. Low grafting, deep planting and nearness of bud union to ground level increases the chances for soil borne infection.

Control measures :

1. Resistant rootstocks like sour orange (*Citrus aurantium*) and Cleopatra mandarin may be used.
2. Proper drainage facilities are to be provided. Excess irrigations should be avoided. Continuous contact of trunk with water has to be avoided by adopting ring system of irrigation or by heaping earth around the base of
   Plants. Double ring basin system around the trunk may be provided. The inner ring being 45 cm away from the trunk. This prevents direct contact of water with the trunk.
3. Injuries to the plant should be avoided.
4. Planting pits may be dusted with a mixture of zinc sulphate, copper sulphate and quick lime (5:1:4) just before planting.
5. The diseased bark along with a little portion of healthy tissue may be removed with a knife and the exposed tissue is painted with zinc sulphate- copper sulphate - quick lime paste (0.6 :0.2 :0.5 kg in 100 litres of water).
6. Healthy trees should be protected by painting with Bordeaux mixture upto a height of about 50 to 75 cm above ground level in the trunk once in a year.
7. Rootstocks like *Poncirus trifoliate* or its hybrids like citranges can be used.
8. Drenching the soil around plant base with Bordeaux mixture 1.0 per cent controls the disease.
9. Diseased leaves and fruits should be collected and burnt.

Spraying with Bordeaux mixture 1 per cent alone or with tin sulphate or difolatan 0.3 per cent or metalaxyl-mancozeb 0.2 per cent and Fosetyl-Al (Aliette) controls the disease.

(3) Citrus greening :

The name 'Greening' is not descriptive of the tree symptoms but only of the fruits of affected which remain mostly green even on maturity but with a conspicuous yellow patch on the rind surface directly exposed to sun. This disease is reported to be widespread in India.

Symptoms:

This disease affects almost all citrus varieties irrespective of rootstock. Stunting of leaves, sparse foliation, twig die-back, poor crop of predominantly greened and worthless fruits are important symptoms. Sometimes only a portion of tree is affected. A diversity of foliar chlorosis occurs. A type of mottling resembling zinc deficiency often predominates. Young leaves appear normal but soon assumes on outright position, become leathery and develop prominent veins and dull olive green colour. Green circular dots are found on leaves. Many twigs become upright and produce smaller leaves. The side of fruit exposed to direct sunlight develops full orange colour but the other side remain dull olive green. Fruits are low in juice and soluble solids and high in acid. Fruits are worthless either as fresh fruit or for processing. Seeds are poorly developed, dark coloured and aborted.

Causal organism: Rickettsia- like organism.

Perpetuation:

All exotic and indigenous varieties of citrus and its relatives are susceptible to greening disease. It is transmitted by infected budwoods and through citrus psylla (*Diaphorina citri*). Even a single psylla is capable of spreading the pathogen. The initial symptoms of greening appear in 20 to 45 days after the feeding by psyllid.

Control measures :

1. The disease can be controlled by removal of affected and unproductive trees and by replanting disease-free budded plants raised on improved root stock.
2. The insect vector can be controlled by spraying monocrotophos 0.05 per cent at periodical intervals helps to check the spread of the disease.
3. Tetracycline 500 ppm sprays at fortnightly interval reduces the incidence by inhibiting the multiplication of the pathogen.
4. Infection of nursery stock should be avoided by restricting citrus nurseries to those localities where psyllid vector is virtually absent. Certified pathogen free budwood should be used for propogation.

(4) Anthracnose :

Symptoms:

The disease affects branches, leaves and fruits. The plant is affected at all stages. The weakened or injured twigs and branches are generally affected. The branches begin to wither from tip downwards. The drying back generally progresses downwards with the leaves turning yellow, withering and drooping and gum formation on the affected stem. Black dot-like acervuli appear in large numbers on the dead twigs. The spots on fruits may vary from small specks to one cm in diameter. They are reddish brown initially and become dark brown to black later. Circular and sunken acervuli develop on these spots. The leaves also may show spots with dark brown marginal ring and grayish white central patch with numerous black acervuli arranged in concentric rings.

Causal organism: *Colletotrichum gloeosporioides* Penz., *Gloeosporium bimetticolum* and *G. follicolim.*

Perpetuation:

The fungus *Colletotrichum gloeosporioides* survives in a dormant condition in the dead twigs and branches.

Epidemiology:

It causes decline of acid lime trees in North India. Malta oranges and grapefruits suffer more. Deficiency of nitrogen and unfavourable soil conditions make the plant weak and susceptible to the disease. The disease is severe during July-January.

Control measures :

1. Dried twigs should be pruned. The cut ends should be protected by painting with Bordeaux paste or any other copper fungicide.

2. Such trees may be sprayed thrice with Carbendazim 0.1 per cent or Captafol 0.2 per cent after pruning.
3. The trees should be adequately manured with urea. Urea at 100 g per every 10 litres of spray solution should be mixed for increasing the vigour of the plant.
4. Drainage facilities should be improved. Trees should be properly irrigated.
5. Periodical spraying with Bordeaux mixture 1.0 per cent or ferbam or zineb or captan 0.15 to 0.2 per cent gives good control.
6. Zinc sulphate, copper sulphate and lime mixture at 0.6: 0.2: 0.5 kg in 100 litres of water is also effective.

(5) Tristeza :

This disease was reported in *Citrus aurantifolia* and *C. sinensis* from Italy and California in USA. The name 'tristeza' was suggested to describe the sad appearance of the diseased citrus trees. The disease is widespread in Argentina, Australia, Brazil, India, Indinesia, Java, Malaysia, Pakistan, Sri Lanka, Panama, Thailand, USA and Venezuela.

Symptoms:

Lime is susceptible both as seedling or budding on any rootstock. But mandarin and sweet orange seedlings or rough lemon, trifoliate orange and citringes and Rangpur lime rootstocks are tolerant. Susceptible root stocks are grapefruit and sour orange. In sweet orange or mandarin, on susceptible rootstocks, leaves develop deficiency symptoms and abscise. Roots decay, twigs die-back. Fruit set diminishes and only skeleton remains. Fine pitting is noticed on inner surface of the bark of sour orange stock. Grape fruit and acid lime are susceptible irrespective of rootstock. Acid lime leaves have more number of vein flecks (elongated transculent area). Affected trees remain stunted and die. Fruits are small in size and insipid. Yield is highly reduced.

Causal organism: *Citrus tristeza virus (CTV).* The virus is long, flexous rod and measures 2000 x 12 nm in size. Three strains viz., mild, severe and seedling yellow are reported.

Perpetuation:

The virus is transmitted by mechanical inoculation. Transmission of the virus usually occurs through infected bud wood. Under field conditions, it is transmitted by aphid vector. The most efficient vector is the black citrus aphid *Toxoptera citricidus* Kirk.. Other species are *Toxoptera aurantii* Fonse., *Aphis gossypii* Glover, *Aphis craccivora* Koch., *A. spiraecola* and *Dactynotus jaceae* and *Myzus persicae* Sulz. It is transmitted in a non-persistant manner. The virus is also transmitted by the parasitic dodder, *Cuscuta reflexa* Roxb. The virus is not transmitted through sweet orange, acid lime and grape fruit seeds.

Control measures :

1. All diseased trees should be identified and removed as and when the disease is noticed.
2. Fresh planting is done with virus free materials on tolerant rootstocks. For sweet orange and mandarin this method of management is highly useful.
3. For acid lime seedlings immunized with mild strain of tristeza virus (cross protection) is highly useful. Tristeza virus is not seed-borne. Hence nuclear seedlings which are

virus free and true to type can be used in the control of the disease. Nucellar seedlings of sweet orange remained free from infection for more than six years after planting.

4. Periodic spraying of insecticides like monocrotophos 0.05 % reduces the secondary spread of tristeza in the orchards.

(6) Citrus exocortis :

Symptoms:

The plants show cracking and scaling of the bark. Scaling is characterized by narrow vertical strips of the outer portion of the bark. The symptoms appears first on the rootstock near the soil line (collar region) and gradually extends upward to the bud union and down to the roots. Bark becomes dead and dries. Diseased trees remain are stunted.

Causal organism:

Viroid (Free RNA without protein coat with a molecular weight of 119,000 and 371 nucletides)

Perpetuation:

Rangpur lime (Citrus limonia Osbeck) and it's hybrids are susceptible. Transmission normally occurs through infected budwood, contaminated tools (budding knife) and sap inoculation and is not through vector and seed.

Control measures :

1. Removal of unproductive trees and replanting viroid free certified bud woods on tolerant rootstocks.
2. Budding knives should be cleaned and periodically washed with Trisodium phosphate-soap solution.

(7) Scab of citrus :

Cause : *Elsinoe fawcetti* Bitan court and Jenkins.

Symptoms :

The typical scab symptoms occur on the leaves, twigs, and fruits. The disease is confused with bacterial canker in being warty but differs in that the wartiness occurs only on one side of the leaves and that its attack causes distortion and wrinkling of leaves. The infected fruits become hard and often drop prematurely. In the north-eastern hill regions and parts of West Bengal the disease is commonly observed on the mandrins, citrons, lemons etc. The opposite surface of leaf corresponding to warty growth shows a circular depression with a pink red centre.

The type of scab prevalent in India is similar to the sour orange scab. The pathogen perpetuates or survives in off season as ascospores.

Control measures :

i) Remove the infected leaves, twigs and fruits.

ii) Spray with Blitox-50 or Fytolan @ 3g/litre water.

iii) Spraying with Difolatan or Benomyl or Perenox or Feram or Ferbam + Triphenyltin will also control the disease.

iv) Spraying of Macuprax / Burcop @ 0.3 % is also quite effective.

(8) Mottle leaf of citrus :

Cause : Zinc deficiency causes Mottle leaf in citrus. The first symptom to appear on orchard trees is the yellowing of leaves of one or more branches. This led to the use of the name citrus yellowing. In later stages, mottle leaf and yellowing becomes general and is accompanied by some defoliation, twig dieback, and multiple shoot growth with small, dull, chlorotic, or mottled leaves which tend to grow in an erect position. Affected trees display some of the symptoms described for greening disease, stubborn disease; such symptoms include multiple bud development, small upright leaves, and undersized, lopsided fruits. Fruits on affected trees are often more acid to the taste than normal, and color unevenly. In affected fruits, many seeds are small and dark-colored or undeveloped. the average fresh weights of the mottled leaves are less than those of the healthy leaves, the average fresh weights of the root systems of the mottled leaves are greater than those of the corresponding healthy leaves

Control measures :

1. Use an annual foliar spray (Zinc sulphate) on the spring flushs leaves when they are about two -thirds their full size @ Sprays Zinc sulphte (23 % Zinc) + manganese sulphate 150 g/100.
2. The use of zinc in the culture solution in not too high a concentration was followed by a dark green color in the leaves. By means of coating with a mixture of zinc the surfaces of leaves of rooted Valencia leafy-twig cuttings grown in culture solutions, it was possible to note the toxic effect of zinc or its temporary inhibiting or retarding effect on growth. Such effects may partially account for the variations that frequently are observed in the rate of response of trees to zinc applications.

3) Spray 2 1/2 kg $ZnSO_4$ + 1.5 kg of Slaked lime in 400 liters or water.

4) Soil application of ZSO_4 @ 120-300 g/plant.

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Exercise No. 10

Grape vine

(1) Downy mildew :

Downy mildew is the most serious disease of grapevine. It is prevalent in America, Europe, North and South Africa. It also occurs in Australia and New Zealand. It is serious throughout South India, especially in AP and Karnataka and is a chief limiting factor in grape production.

Symptoms:

The disease is usually first observed as small translucent, pale yellow spots with indefinite borders on the upper surface of leaves. On the under surface of leaves and directly under the spots, a downy growth of the fungus appears. The tissue in the spot is traversed by reddish lines. Later, the infected areas are killed and turn brown. The growth on the lower surface becomes dirty grey. The necrotic lesions are irregular in outline and they enlarge and coalesce to form larger necrotic areas on the leaves, frequently resulting in defoliation. Diseased shoots remain stunted. Infected leaves, shoots and tendrils are covered with whitish growth of the fungus. Flowers and berries are also affected. Flowers may blight or rot.

During blossom or early fruiting stages, centre clusters or part of them may be attacked and become quickly covered with the downy growth and die. If infection takes place after the berries are half-grown, the fungus grows mostly internal. The berries become leathery and wrinkle and develop a reddish marbling to brown colouration. The fruits shed if the attack is very severe. The juicy quality of fruit is found to be reduced. Infection of green young shoots, tendrils, stems and fruit stalks results in stunting, distortion and thickening of the tissues. Infected tissues turn brown and die.

Causal organism:

Plasmopara viticola (Berk. and Curt.) Berl. and deT. Mycelium is intercellular with spherical haustoria, coenocytic, thin walled and hyaline. Sporangiophores arise from hyphae in the sub-stomal spaces. The number may be from 1 to 20 from each stoma. Sometimes they emerge directly through the cuticle. On young berries of grapevine they emerge through the lenticels. The sporangiophores are 300 to 500 µm long and 7 to 9 µm wide. Branching is almost at right angles to the main axis and at regular intervals. Secondary branches arise from lower branches. From the apex of each branch 2 to 3 sterigmata arise and bear sporangia singly. The sporangia are thin walled, oval or lemon-shaped. The zoospores are pear shaped, biflagellate. The oospores are thick walled.

Perpetuation: The pathogen survives on the infected leaves and vines as oospores and dormant mycelium. The secondary spread is through wind-borne sporangia and zoospores which are found on the new flush.

Epidemiology: The most favourable temperature for germination of sporangia is between 10 and 23^{0}C. Disease development is favoured during rainy season when there is heavy dew, relative humidity is above 80 per cent and temperature is between 23 and 27^{0}C.

Control measures :

1. Sanitation is very important in the management of this disease. Removing and burning all diseased leaves, shoots, flowers and berries that may contain hibernating oospores help in preventing the disease.

2. After pruning, the vines should be sprayed with Bordeaux mixture 1% or Difolatan 0.2 per cent or Chlorothalonil 0.2 per cent.
3. When the flushes are formed, spraying the Difolatan 0.2 per cent or Chlorothalonil 0.2 per cent or Metalaxyl 0.2 per cent is effective. It may be repeated at weekly intervals. When the non-systemic fungicide is used during humid and rainy period spraying should be repeated for every two or three days.

(2) Powdery mildew :

The disease was first reported from England and later in France. Powdery mildew is prevalent almost throughout India, Italy and is more problematic in relatively dry areas. It is prevalent in Australia, Greece, Hungary, India, Syria and the USA.

Symptoms:

The fungus attacks all the stages of the crop growth. The characteristic symptom of this disease is the appearance of white powdery patches on affected parts. On the leaves the disease starts as small, white patches which later become larger in size and powdery in appearance. Sometimes the entire leaf is covered with dusty white fungal growth. Diseased leaves become discoloured and malformed. Similar powdery growth appears on the stem, tendrils, flowers and young fruit bunches. The stem turns grey and finally dark. When the blossom is affected, the flowers wither and dry up. Affected plants show a wilted appearance and remain dwarf. The affected bunches show whitish powdery growth on the berries. If the fruits nearing maturity are attacked they become mis-shapped, crack and only few of them ripen. In case of early infection the young fruits may not develop.

Causal organism:

Uncinula necator (Schw.) Burr.(syn. *Oidium tuckeri* Berk.). The mycelium is superficial and adheres to the host surface by means of appressoria. The hyphae are slender, branched, hyaline and turn darker when formation of conidia is completed. Conidiophores are simple, errect and bear a chain of 3 to 4 conidia which are oval, ellipsoid to cylindrical. Cleistothecia are rarely formed in India conditions. They are embedded in the mycelium and are dark when fully mature, globose with flattened top.

Perpetuation:

It survives as dormant mycelium and as cleistothecia on the shoots and buds from season to season. The disease spreads by the air-borne conidia.

Epidemiology:

The disease occurs in severe form during Oct-Nov in North India and Feb.-June in South India.. Disease is favoured by warm, cloudy weather and retarded by sunshine. Warm winter temperature from 20^0 to 33.5^0 C has been found to be the cause for epidemic in Hyderabad. Disease development is adversely affected by rain.

Control measures:

1. Over-crowding and dense growth of the vines should be avoided by proper pruning.
2. Effective control can be achieved by spraying Wettable Sulphur 0.2 per cent or Dinocap 0.7 per cent or Carbendazim 0.1 per cent or Sulphur dusting at berry development stage with a prophylactic treatment.

3. Red sultana, Saint George and No. 1613 are the highly resistant varieties.

(3) Anthracnose / Bird's Eye Spot :

Symptoms:

The fungus attacks shoots, tendrils, petioles, leaves, veins and stems. Numerous spots occur on the young shoots. These spots may unite to girdle the stem and cause death of the tips. Spots on petioles and leaves appear as small, irregular, dark brown. The central tissue turns grey and falls off. The disease appears as dark red spots on the berry. Later these spots are circular, sunken and ashy grey and in late stages these spots are surrounded by a dark margin which gives it the bird's eye appearance. The spots are 7 mm in diameter but they may involve about half of the fruit.

Causal organism:

Elsinoe ampelina Shear (Syn. *Manginia ampelina* v. & p.). Conidial stage is *Sphaceloma ampelinum* de Bary [Syn. *Gloeosporium ampelophagum* (Pass.) Sacc.]. Conidia are formed in pink Perithecia (pseudothecia) and are small and unconspicuous. Asci are globular and ascospores are hyaline, 3-celled and are 15 to 16 x 4 to 4.5 µm.

Perpetuation:

The pathogen survives as mycelium in the cankers on the stem and on the infected twigs. Secondary spread is through conidia which are carried by wind and rain water.

Epidemiology:

The disease is severe during July-August and Nov-Dec months. Infection in new sprouts takes place during rainy season. Heavy rains after pruning leads to more incidence.

Control measures :

1. The diseased leaves and twigs should be pruned and burnt.
2. Spraying pruned canes and leaves with a mixture of ferrous sulphate 2.5 kg + 0.5 pint sulphuric acid in 4.5 litres of water controls the disease.
3. Spraying at 10 to 15 days with Bordeaux mixture 1.0 per cent or copper oxychloride 0.25 per cent or Carbendazim 0.1 per cent or Mancozeb 0.2 per cent or Difolatan 0.2 per cent effectively controls this disease. The interval may be reduced during days.
4 Angur kalan, Bangalore blue, Beauty seedless, Bharat early, Delight, Golden Muscat, Golden Queen, Hussaini, Karachi Niagra, Khalili, Large white, Muscat, Schuyler white, White Muscat are resistant varieties.

(4) Bacterial canker :

Symptoms:

The disease appears as small water soaked spots surrounded by yellowish halo at lower surface of the leaf. These spots enlarge in size and become dark brown and angular. Sometimes these spots coalesce to form larger patches. Leaves also show vein infection. Infected leaves after drying remain attached to the stem. Lesions are brown to black, elongated and cankerous on petioles and canes. In advanced stages of infection, stunting, cracking and abnormal growth of canes are seen. Berries show brown to black and cankerous lesions. Severely affected ones become small and shrivelled.

Bacterium: *Xanthomonas campestris pv. Viticola* (Nayudu.) Dye. Bacterium is Gram negative, rod shaped with rounded ends, motile by single polar flagellum (monotrichous) and measures 0.4 to 1.2 x 2 to 3 µm.

Perpetuation:

The alternate hosts are neem, mango and *Phyllanthus maderaspatensis*. The bacterium survives in the infected dry leaves up to 65 days. Secondary infection takes place through wind splashed rain. Disease spreads to distant places by diseased cuttings.

Epidemiology:

Temperature range of 25 to 30^{0}C is favourable for disease development. Free water from dew, irrigation or rain on leaf are more important for pathogenesis. The disease frequency is positively correlated with number of rainy days.

Control measures :

1. Regular inspection of vineyard, destruction of infected plant materials, use of disease-free cuttings and late October pruning are recommended for its management.
2. Spraying with Streptocycline 300 ppm starting from two leaf stage upto 70 days at an interval of 15 days is also effective.

(5) Grape fan-leaf virus :

Symptoms:

Affected young leaves show variegated mottling. The malformed leaves have open petiolar sinuses. Widening of the petiolar sinuses and reduction of areas between the veins gives the impression of a half-closed fan. Affected leaves stand upright along the axis of young shoots and become cup like. The dark green areas in the mottled leaf bulge upward and leaf surface become rough. The characteristic symptom of the disease is reduction in size of the leaves. The affected leaves turn light yellow and show cupping. The distance between internodes is reduced and nodes and internodes become thin and weak. Growth become more zig-zag at the internodes. Few lateral branches are produced. Infected plants do not produce flowers and fruits even at the age of three years. Plants become stunted and produce very few and weak rootlets.

Causal organism: Grapevine fan leaf virus (GFLV)

Virus particles are isometric and 25 to 30 nm in diameter. Thermal inactivation point is 60 to 65^{0}C. Longevity *in vitro* is 15 to 30 days at 20^{0}C and the dilution end point is between 10^{-3} to 10^{-4}. The vectors are nematodes, *Xiphinema index* and *X.italiae*. The virus is mechanically transmissible to *Cucumis sativus*, *Chenopodium amarantocolor*, *Gomphrena globosa*, *Nicotiana tabacum cv.* 'White Burley', *Phaseolus vulgaris cv.* Prince.

Perpetuation:

The disease has been transmitted by grafting or budding of the diseased scion onto the healthy rootstock of variety Emperor. It is not transmitted through pollen.

Control measures : Soil application with nematicide controls the nematode vectors and reduce the spread of the disease.

Exercise No. 11

(A) Peach (B) Apple and (C) Strawberry

(11) (A) PEACH :

(11) (A) (1) Leaf curl

Taphrina (Exoascus) deformans

Symptoms :

The first symptoms appear in the early spring, soon after the leaves come out of the buds. Some of the leaves become distorted and fold over, so that the tips are directed backwards, while others curl and the whole lamina except the tip becomes puckered and blistered. In some leaves, only paft of the lamina may show such distortions. The blistered portions are thicker and softer than the normal portions of the leaf blade. The affected areas may remain green for a while, but gradually become yellow and finally turn reddish-purple, which makes the affected leaves very conspicuous against the green colour of healthy foliage. The reddish-purple surface of the lamina soon becomes covered with a whitish-grey bloom consisting of the fructifications of the fungus on the upper surface, but sometimes on the lower surface also. Sometimes, the young shoots may be attacked and they become swollen and distorted. No lesions are usually formed on the fruits.

Affected leaves fall off prematurely and in case of severe infections, the trees may suffer from acute defoliation in the late spring. Recurrent attacks of leaf curl from season to season, results in weakening of the trees and consequent loss in yield and quality of the fruits.

The causal organism :

The fungus does not form true conidia. The whitish-grey bloom formed on the leaf surface consists of a large number of asci, which are not developed and protected within a fruiting body, but merely break through the leaf cuticle under which they arise from the mycelium within the leaf. The hyphae enter the leaf mostly by cuticular penetration, but stomatal entry also occurs. Since early infections take place as soon as the buds break into leaves, infections occur largely through the lower leaf surface, but later on infection may take place through the upper surface also. After entry into the leaf, the mycelium travels across the mesophyll intercellularly and spreads out chiefly between the palisade layer and the upper epidermis, where it develops widely. Infection of young leaves stimulates cell multiplication in the mesophyll tissue, resulting in distortion of the leaf surface. All the starch content in the leaf tissue are used up by the fungus and with the gradual degeneration of chloroplasts, the reddish-purple pigmentation appears in the infected cells.

Mode of survival, spread and Epidemiology :

Primary infection may occur through resting conidia, which have been sheltered on some part of the host during the winter. The conidia, which are produced in masses and have yeast like consistency, can adhere to the buds and cause primary infection during the ensuing spring. It is also probable that the fungus can survive during winter in the form of agglutinated masses of conidia on bud scales and twig surfaces, as they are capable of withstanding dessication and low temperatures.

The optimum temperature for growth of the fungus is 20 ^{0}C. Cold and wet weather conditions at the time of bud burst to form new leaves favour severe infection.

Disease management :

i) Agronomic practices :

Orchard and tree sanitation, removal and destruction of fallen leaves and other plant refuse, clean cultivation etc. help to minimize the occurrence of the disease.

ii) Chemical control :

Thorough spraying with Bordeaux mixture - 1 % or Copper oxychloride 2.5 gm or Manocozeb 2.0 gm or Captan 1.25 gm, or Tridemorph 1.0 ml, or Chlorothalonil 1.25 gm per litre of water before bud-break in early spring, followed by a second spring after a fortnight controls the disease.

(11) (B) APPLE :

(11) (B) (1) Scab :

Cause : *Venturia inaequalis* (Cke) Wint,

Conidial stage : *Spilocaea pomi* Ft.

Symptoms :

The characteristic symptoms of the disease are the development of scattered, roughly circular brown or olive green spots with a concentric margin on the lower surface of the leaves. Most of these spots do not have a definite margin in contrast to those on the upper side of the leaf. On the leaves the spots have a radiating appearance with a feathery edge. In case of heavy infection the leaf blade may become curled, dwarfed and distorted. The spots on the fruits are smaller and darker in early stage but turn to almost black. In older spots the centre turns brown and corky. The loosened cuticle of the fruit looks like a whitish hand around the spots. Distorted growth and cracking of the surface are seen in fruit infected in the early stage. Barks of twigs may show rupturing and blistering symptoms.

The fungus overwinters in saprophytic phase (winter spore stage) on the fallen leaves. The mycelium penetrates from the subcuticular stroma into the interior of the leaf. Perithecia formed in autumn and winter mature in spring to produce ascospores which is the chief source of primary inoculum. The fungus has parasitic phase (summer spore stage) on the leaves and fruits.

Control :

The routine scab control by the following spray schedule is very essential for apple growing.

i) The spray schedule is given below :

Spray No.	Tree stage / month	Chemicals / 100 litre water
1st	Bud swell to green tip (March-April)	Difolatan / Foltaf (300 g) or Dithane M-45 (400 g) or Captan (400 g) or Delan (100 g)
2nd	Pink bud to partial bloom (April)	Dithane M-45 / Captan (300 g) or Delan (100 g)
3rd	After Petal fall or fruitlet stage (April-May)	Bavistin / Topsin M (50 g)
4th	Fruit development stage (May-June)	Bavistin 25 g + Dithane M-45 (250 g) or Dithane M-45 / Captan 300 g or Detan (50 g)
5th	14 days later	Dithane M-45 / Captan (300 g) or Difolatan (150 g) or Delan (50 g)
6th	Pre harvest or nearing fruit maturity about 15-18 days before harvest	Repeat 4th spray
7th	Post harvest but few days prior to general leaf fall (November - December)	Urea 5 kg.

Add stickers like Sandovit (50-75 ml) or Sel-wet-E (50-75 ml) / Teepol (25 ml) or Triton, Uphaar (50-75 ml) in 100 litre water in each fungicidal spray for improving the solubility, retention and spreading of the fungicide.

Besides the above operation care should be taken to collect and destroy the fallen leaves, pruned materials and also to plough the orchard to remove debris harbouring the mycelium.

Spraying urea (5%) or Benlate (0.2%) once in autumn prior to leaf fall and again just before bud break (2% urea) followed by two sprays of Captan (0.2%) or Dithane M-45 (0.25%) at 10-15 days interval after petal fall will effectively control the disease.

Benzimidazole fungicides provide excellent eradicative activity and other better fungicides are dodine, guaziatine and EBIs (Ergosterol biosynthesis inhibitors like bitertanol, fenarimol etc.)

(11) (B) (2) Powdery mildew :

Cause : *Podosphaeria leucotricha* (E & E) Salm.

Symptoms :

The disease affects the growth of buds, new shoots and leaves. The symptom appears as small white or greyish felt like patches on the underside of the leaves. The infected leaves becomes crinkled and curled. The mycelium and the powdery coating of spores soon become evident on the entire leaf. Infected foliage becomes hard and brittle. The powdery coating of twigs later on disappears and a brown, felt like covering with black fruiting bodies is seen.

Diseased twigs remain stunted or get killed. Floral parts become shrivelled and blighted. Young infected apple fruits show stunting, russetting and cracking.

The fungus survives in the form of resting mycelium or encapsulated haustoria in the buds.

Control :

i) Remove the dormant shoot tip and silver terminals and destroy them.

ii) Spray the plants with wettable sulphur (2-3 g/litre) or Carbendazim fungicide (Bavistin / B-stien / JK-stein / Agrozim / Benuguard) or Topsim M @ 1 g/ 2 itres water or Karathane 1 ml / 2 litre water :

a) during late dormancy

b) at bud swell.

c) at petal fall and

d) two weeks later.

In nurseries the spraying should be done at 7 days interval. Stickers like Sandovit / Triton / Teepol / Set - wel E or Set - wel 99 may be added in the fungicide solution.

iii) Dikar, Dinocaop, Benomyl, Thiophanate and 10 ppm Aurcofungin sprays also effectively control the powdery mildew of apple.

iv) Spraying of like sulphur in

a) 1 : 15 dilution at green tip stage;

b) 1 : 35 dilution at open cluster stage ;

c) 1 : 60 dilution at full pink stage;

d) 1 : 100 dilution at half petal fall stage also give significant control of the disease.

(11) (B) (3) Fire blight :

Casual organism : ***Erwinia amylovora***

The bacteria causing the disease are rod-shaped, occurring singly or in pairs and measure 1.0-2.0x0.8-1.2 μ in size. The anaerobic, non-spore forming, encapsulated and motile by 2-8 peritrichous flagella.

Symptoms :

The pathogen attacks flowers and twigs and kills them. Large branches and trunks may be girdled and eventually killed. Young trees may be killed in a single season.

Infected flowers become water-soaked, shrivel, turn brownish-black and fall down or remain clinging to the tree. The lighted flowers appear scorched, as if burnt by fire and hence the disease is called "fire blight". Soon, leaves on the same branch or nearby twigs develop brown to black blotches along the mid rib and main veins or along the margins. As the blackening progresses, the leaves curl, shrivel, hang down and cling to the curled, blighted twigs. Terminal twigs wilt from the tip downward, their bark becoming brownish-black and soft at first, but shrinks and hardens later. From the fruit spurs and twigs the symptoms progress down to the branches, where cankers are formed. The bark of cankers is water-soaked in the beginning and becomes darker, sunken and dry later on. The canker may

enlarge and encircle the branch. In such cases, the branches above the point of infection die. Infected, small, immature fruits become water-soaked, turn brown to black, shrink and may cling to the tree for several months.

Under humid conditions, droplets of milky white, sweet, sticky bacterial ooze may appear on the surface of recently infected parts and may run down the surface of the infected twigs. The ooze usually turns brown soon after exposure to air.

Mode of survival, spread and Epidemiology :

The bacteria overwinter at the margins cankers, in buds and in the wood tissue. In the spring, they become active again, multiply and spread to the adjoining healthy bark. During humid or wet weather, the bacteria exude through lenticels and cracks usually at the time, when the flowers are opening. Various insects, such as bees, flies, ants etc. are attracted to the sweet, sticky, bacterial exudate and carry the bacteria sticking on to their bodies to flowers they visit afterwards. The bacteria may also be disseminated on to other flowers by lashing rains. The dry bacterial ooze may also be carried by wind to flowers. The bacteria multiply rapidly in the nectar and through nectarthodes enter the floral tissues. The bacteria multiplying inside the floral tissues kill the flowers. Then, the bacteria move through the intercellular spaces of through the macerated middle lamella and flower cells down the pedicel into the fruit spur. Infection of the fruit spur, results in the death of all flowers, leaves and fruits on it. The bacteria may also enter the leaves through stomata, hydathodes or through wounds. From the leaf, the bacteria pass in to the petiole and the stem. After entering the tissues, they move through the vessels, attack and kill other cells, causing blight and canker symptoms in the process.

Warm, humid conditions favour infection and development of the disease.

Disease management :

Agronomic practices :

i) During the winter, all blighted twigs, branches and cankers should be cut about 10 cm below the point of infection and destroyed burning. The cut ends should be smeared with Bordeaux paste or copper oxychloride paste.

ii) The cutting tools should be disinfected after each cut with mercuric chloride (1:1000) solution or commercial sodium hypochlorite-10% solution.

iii) The crop should be maintained properly by judicious application of fertilizers.

iv) Suitable insecticides should be applied to control insects, so as to prevent the spread of bacteria to succulent twigs and flowers by such insects.

v) Resistant varieties should be grown in new areas.

Chemical control :

Spraying with Streptomycin or Oxytetracycline at 100 ppm is effective in controlling the disease to some extent. Spraying with Bordeaux mixture - 0.75 % or Copper oxychloride at 2.5 gm/liter of water affords fairly good control of the disease.

(11) (B) (4) Crown gall :

Caususal organism : *Agrobacterium tumefacience* (E.F. Smith and Towns) Conn.

Crown gall is a common plant disease caused by the soil-borne bacterium *Agrobacterium tumefacience*. It is found throughout the world and occurs on woody shrubs and heraceous plants including grapes, raspberries, blackberries and roses.

Symptoms :

Round, wart-like growths two inches or larger in diameter that appear at or just above the soil line, or on lower branches and stems. Plants with several galls may be unable to move water and nutrients up the trunk and become weakened, stunted and unproductive. Young plants can be killed by developing gall tissue. The bacteria responsible for crown gall can persist in the soil for many years and are released when galls become saturated with moisture or as older galls decompose. Susceptible plants are infected through fresh wounds or abrasions, many of which are a result of pruning, freeze injury, soil insects, cultivation and other factors that may damage plants. Nursery stock is often infected through grafting and budding scars.

Control measures :

1) Select resistant cultivars when possible and purchase plants from a reputable nursery.
2) Do not buy plants that shows signs of swelling or galling.
3) When carying for susceptible plants, avoid injury or pruning wounds that may come in contact with the soil.
4) Use Tree Wrap to protect against string trimmer damage and keep your garden tools clean.
5) Provide winter protection with natural burlap so bark does not crack.
6) In many cases, existing galls can be removed with a sharp pruning knife. Destroy the infected plant tissue and treat thwe wound with pruning sealer. If the plant does not recover, remove and destroy it.
7) Avoid injury to the roots and collars.
8) Dip healthy grafted plants in 1 per cent Copper sulphate solution for 1 1/2 hours.
9) Rotate the nursery sites to new place.
10) Rogue out infested plants.

(11) (B) (5) Mosaic :

Causual organism : Apple mosaic disease is caused by *Apple mosaic virus*. It is often found in mixed infections with several other viruses. There is no indication of field spread other than potentially through root grafting. Apple Mosaic virus, besides many *Malus* spp. and pear, occurs naturally in more than 30 mostly woody hosts including hazelnut, hop, *Prunus* spp., *Rubus* spp.; *Rosa* spp., *Betula* spp., *Chenomeles* spp. and *Aesculus* spp.

Symptoms :

Creamy white or yellow patches appear on the lamina. The spots may be so numerous that a part or whole leaf may turn yellow. The leaves become necrotic and finally wither

away. Yellow band appears along the primary and secondary veins. The affected trees may remain stunted. Vein banding sometimes occur on the apical leaves. Affected leaves exhibit dramatic white banding patterns on the leaves of many cultivars. Pale yellow to bright cream irregular spots or bands along major veins can be found on leaves in the spring. Symptomatic leaves may drop prematurely. The distribution of symptomatic leaves may be erractic throughout the tree or limited to a single limb. The number and severity of affected leaves also depends on seasonal temperatures, with the symptoms being more seere in years with moderate springtime temperatures. The disease can result in the loss of up to 40 % of the crop depending on the cultivar. Usually symptomless in pear.

Control measures :

1) Use of certified virus-tested (and found to be free of all known viruses) planting material is the preferred strategy for protection from this disease.
2) Thermotherapy (24 to 32 days at 38 ^{0}C) and/or apical meristem culture have been used to eliminate various viruses.
3) Avoid the use of graft wood or bud wood from the infected trees,

(11) (C) Strawberry :

(11) (C) (1) Leaf spot

a) Mycosphaerella Leaf Spot :

Casual organism : *Mycosphaerella fragariae Tul.*
(= Ramularia tulasnii Sace, imperfect stage)

White centered purple margined leaf spot is a fungal disease caused by *Mycosphaerella fragariae Tul: (= Ramularia tulasnii* Sace, imperfect stage). In India this disease was first reported from Niglar, Bhiwadi hill of Uttar Pradesh during October 1952. In initial stage, circular purple scattered spots appear on upper surface of young leaves with an average diameter of 2-6 mm. The spots are most frequent on blades of leaflets but also appear on petioles, fruits and fruit stems. The leaf spots enlarge, then turn to white and are surrounded by dark purple margins, rendering birds eye effect. On lower surface of leaves, prominent veins touching the spots become reddish purple and entire leaf may die in advanced stages. The lesion on stem fruit stalks are elongated as well as circular. On berries infection is not common however, the pulp of berry becomes discolored and render the fruits unmarketable under high pressure of disease .

Etiology :

The fungus belongs to class - ascomycetes. The ascomata are usually very small, mostly emerging the host tissues in leaves. The single septate hyaline ascospores measure 11-45x2-3 µm dimensions, and are usually formed on the upper surface of the leaf, sometimes formed on petioles and calyx also. The sclerotia on dead leaves may also produce conidia.

Epidemiology :

The fungus overwinters through perithecia and sclerotia. Perithecia are produced at the edge of leaf in autumn season, whereas, sclerotia are produce on infected stolons and petioles. The prolonged wet period with low temperature in winter and spring favours the disease development. Continuous rainfall in month of April is mainly responsible for the occurrence of epiphytotics. The disease spreads in a very short period of time when strawberry plants are grown during warm days and cold nights . The optimum temperature for the conidial germination ranges from 13 ^{0}C to 21 ^{0}C. Six virulence groups on nine strawberry cultivars and four races of fungus were noted. Lower temperature delayed the maturation of young leaflets, thus, extending their susceptible period.

Management

1) Cultural practices to reduce disease incidence include planting the **strawberry** plants in well drained soil, keeping out the weeds and maintaining proper spacing.
2) Avoid excessive use of nitrogenous fertilizers. During sprouting in spring and budding, the spray of micronutrients like manganese, copper and boron can reduce the infection and enhance the yield upto 13.20 Percent.
3) Chemically this disease is managed by spraying of Bordeaux mixture (0.8 per cent), Zineb Ferbam, Captan, Cuprex (0.2 per cent) and by benomyl.
4) Spray of ditianon and Euparen M is effective against the disease and Vincolozolin and Iprodione have also been found effective.
5) The application of soil sterilants, methyl bromide, Chloropicrin or Basamide can completely control the disease caused by sclerotia.
6) The use of benomy and thiophanate-M as dips to nursery plants or as pray after planting renders best control.
7) The varieties like Combridge, Takane Kurumal 103, Horida's wonder, Hukuda and Benzuru, Arking, Premier, Coaliar, Dilpasand and Albitron, Elista and Tioga, Tarada vicoda and Maramalda, Joliette were reported as resistant against disease.

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VEGETABLES

Exercise No. 12

Potato

(1) Early and Late blight

Causal organism: *Phytophthora infestans.*

Symptoms:

The disease first appears as circular or irregular, water soaked, light brown lesions, which develop usually at the tips or edges of the lower leaves. In moist weather, the spots enlarge rapidly and form brown blighted areas. If the infected leaves are examined closely, whitish growth of the fungus can be seen on the lower surface. The growing shoots are blightened in the beginning and then the disease spreads up to tubers, which show brown or purple discolouration of the skin of the tubers turn brown and decay before harvest. In dry soils, spread of the disease is slow and may result in the dry rot of tubers.

Etiology:

Mycelium is coenocytic, hyaline, branched and both inter and intracellular. The sporangiophores are aerial and arise from the internal mycelium through stomata and lenticles on the tubers. They are slender, hyaline, branched and indeterminate. The sporangia are thin walled, hyaline, oval or pear shaped with a definite papilla at the apex germinating by zoospores or by germ tube. Oospores are thick walled and yellowish.

Perpetuation:

The primary infection is through dormant mycelium in infected tubers or through oospores present in the soil or in the debris. Secondary infection through sporangia spread by wind or irrigation water. The disease is favoured by moderate temperature and high relative humidity.

Management:

1. Use healthy tubers for planting. Selected seed material from the disease free seeds.
2. Follow clean cultivation.
3. Dip the seed tubers for 5 minutes in solution of Ridomil @ 0.2 per cent before planting.
4. Give two to three sprays at an interval of 15 to 21 days starting from 25 days after planting with Bordeaux mixture 1 per cent or copper oxychloride 0.3 per cent or Zineb 0.2 per cent or Mancozeb 0.3 per cent or Ridomil 0.2 per cent.

(2) Black scurf

Cause : *Rhizoctonia solani* Kuhn.

Symptoms :

The disease appears in **two phases.** (i) In stem canker phase the sprouts are killed before they emerge and (ii) this delays the germination resulting in loss of yield. The cankers may cause wilting of the plants also. In black scurf phase the surface of the diseased tubers

has rough black encrustations. This reduces the market value of potato. This disease not only spoils tubers but induces heavy wilting of the plants from such tubers.

Etiology :

Rhizoctonia solani frequently exists as thread like growth on plants in culture, and is considered soil borne pathogen. The pathogen is not currently known to produce any asexual spores though it is considered to have on asexual life cycle. Occasionally sexual spores (Basidiospores) are produced on infected plants.

Epidemiology :

It is more severe in soils that are cool and moist. Acid soil is most favourable. Development of disease is favoured by soil temperature between 16-23 ^{0}C, while soil temperature is above 25 ^{0}C reduce the severity of disease.

Control :

i) Use disease free tubers for sowing.

ii) Tubers should be disinfected with 0.5 % Agallol or 0.25 % Aretan for 4-5 minutes before sowing.

iii) Soil can be treated with Brassicol (PCNB) at the rate of 20-30 kg/ha. Combination of seed and soil treatment gives the best control of the disease.

iv) Soil application of saw-dust at the rate of 25 quintals per ha with 20 kg of additional nitrogen at least 15 days before planting will reduce the disease incidence.

(3) Leaf roll :

Cause : Leaf roll virus (Luteo viruses group).

Symptoms :

Plants infected with leaf-roll virus show rolling of leaflet. The leaflets roll upwards from the margins and progress towards the mid ribs until the entire lamina is involved. The leaves become leathery and brittle and produce a rattling sound when brushed with hand. The number of tubers per plant and their size is greatly reduced. The disease may lead to necrosis of conducting tissues with the result that the food material assimilated in the leaves can not be translocated to the tubers.

Control :

i) Use healthy certified seeds for sowing. Do not plant very small sized tubers since they are more likely to be from diseased plants.

ii) Inspect the fields regularly and rogue out the plants showing the initial symptoms and should be burnt or buried deep.

iii) Spray the crop with systemic insecticides like Metasystox or Rogor at the rate of 600-750 ml in 500 to 600 litres of water. Dimecron in 750 litres of water per hectare at 10-15 days interval to check the insect vectors which transmit the disease.

iv) The granular insecticides viz. Thimet 10 G @ 15-20 kg/ha can be used along with the fertilizer at the time of planting.

v) Dig out infected plants and destroy them.

(4) Mosaic :

1. Mild mosaic: It is caused by Potato Virus- X. also known as potato mottle virus, potato virus, and Solanum Virus- 1.

Symptoms:

The disease results into interveinal mottling or mosaic under favourable growth conditions of the host, the symptoms are masked thus making the recognition of the disease difficult. There is little dwarfing of the plant or deformation of the foliage. Button flower and wilt chilli are the local lesion host of potato virus. The virus infects tomato, datura, tobacco and *Solanum nigrum*.

2. Vein banding severe mosaic: Varietal reactions vary from mild mosaic to severe mosaic and veinal necrosis followed by leaf-drop streak. The affected plants are stunted and the size and number of tubers are reduced. *Solanum demissum* is a local lesion host.

3. Rugose mosaic of potato: It causes very severe damage to individual plants. The foliage is not only mottled but is also severely wrinkled, puckered and markedly reduced in the size. The leaflet margin is rolled downwards and the entire plant is severely dwarfed. The lower leaves generally have black necrotic veins.

4. Crinkle of potato: It is caused by combination of potato virus – x but the yellowish patches on the foliage are bigger and more prominent. As death approaches, this colour becomes more pronounced and is accompanied by rusty brown spot, beginning near the tip of the leaves. The foliage is brittle and easily injured.

Transmission- Mild mosaic:

1. Sap inoculation.
2. Contact between healthy and diseased plants.
3. Diseased seed-stock.
4. Cutting knives.

Vein banding severe mosaic:

1. Sap.
2. *Myzus persicae*
3. *Aphis gossypii*.

Rugose mosaic:

1. Small tubers from diseased plants.
2. *Myzus persicae*

Crinkle mosaic:

1. Tuber borne.
2. Sap-inoculation transmits potato virus-x.
3. Myzus persicae transmits potato virus- A.

i.e. certified seed and early planting.

Management:

1. Use disease free seed tubers.
2. Control of vectors by granular application of Phorate 10 G @ 10 kg/ha or Carbofuran 3G @ 16.5 kg/ha.
3. Rouge out sick looking plants as soon as located.
4. Detop the plants in the third or fourth week of December.
5. Use large sized potatoes for planting.

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Exercise No. 13

Cucurbits

(1) Downy mildew :

Causal organism: *Pseudoperenospora cubensis* (Berk. & Curt.) Rostow.

Symptoms:

Downy mildew symptoms are variable. Sometimes, the first symptoms on leaves remind one of mosaic; pale green areas are separated by islands of darker green. Soon the pale green areas change to yellow angular spots bounded by leaf veins. During moist weather, the corresponding lower surface is covered with a faint purplish fungus-fruiting layer. Occasionally, the purplish hue is lacking and the colour ranges from white to almost black. The entire leaf dries quickly. Usually leaves near the centre of the hill are affected first. Then the symptoms appear progressively on younger leaves until most leaves are killed.

Etiology:

It is an obligate parasite. The mycelium is coenocytic and intercellular with small, ovate or finger-like haustoria. One to five sporangiophores arise through the stomata. The upper third of the sporangiophore is branched either dichotomously or intermediately between dichotomous and monochotomous branching. Spore bearing tips are subacute. Sporangia are grayish to olivaceous purple, ovoid to ellipsoidal, thin walled with a distal papilla and 21 to 39 x 14 to 23 µm. Zoospores are 10 to 13 µm in diameter. Oospores are not common.

Perpetuation:

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 summer alternate with cooler and wet winters, year round survival is possible on summer irrigated crops. They may overwinter as thick walled oospores. Sporangia are dessiminated by wind.

Epidemiology:

The fungus can infect plants at temperatures from 10 to 27^{0}C with an optimum of 16.1 to 22.2^{0}C. They survive several days when temperatures are over 37^{0}C. Maximum lesion development occurs at cycles of about 25^{0}C day time, 15^{0}C night temperature and a photoperiod of 6 h darkness.

Management:

1. Use of bed system of cultivation, wider spacing and planting sites with good drainage, air movement and exposure to sun light helps to check disease development by promoting good aeration and rapid drying of plant surfaces.
2. Avoiding successive plantings in adjacent fields during a single season reduce spread from early planting to late planting.
3. The removal and destruction of infected vines helps in reducing the spreaed of the disease.
4. Difolatan 0.2 per cent or mancozeb 0.2 per cent or chlorothalonil 0.2 per cent spray has been found to be effective.

5. Seed treatment with Apron at 2 g/kg followed by spraying with 0.3 per cent mancozeb or daconil 2 kg/ha is effective in controlling the disease.

(2) POWDERY MILDEW

Causal organism: *Erysiphe cichoracearum* DC. Abd *Sphaerotheca fuliginea* (Schlecht) Poll.

Symptoms:

The leaves on the upper surface show small white or grayish superficial spots. These spots enlarge forming large patches of powdery growth on the fungus. Large area, sometimes the entire leaves, are covered by the fungal growth. Black pin point bodies, representing the ascigerous stage of the fungus, appear during winter months. When the attack is severe defoliation occurs. The fruits from affected plants are very small. Considerable reduction in yield has been noted.

Etiology:

In *Erysiphe cichoracearum*, the conidia measure 63.8 x 31.9 µm, the Cleistochecia are globose 80 to 100 µm in diameter containing 10 to 15 asci, which are 58 to 90 x 30 to 50 µm in size. Each ascus, contains two ascospores and are oval or subcylindrical. In *S. fuliginea*, mycelium is hyaline, occasionally brownish when old. Conidia are in chains with distinct fibrosin bodies, ellipsoid to barrel-shaped and 25 to 37 x 14 to 25 µm. Conidial germ tubes are mostly forked. Perithecia are round, 66 to 98 µm in diameter, with various number of tortous appendages. Asci are broadly elliptic and are 50 to 80 x 30 to 60 µm. In each ascus eight ellipsoid ascospores are found and they are 17 to 22 x 12 to 20 µm.

Perpetuation:

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 dispersed by wind, thrips and other insects.

Epidemiology:

The disease is favoured by cloudy weather, moderate temperature, reduced light intensity and succulent plant growth. Good conidial germination occurs within a temperature range of 22 to 31^{0}C (with an optimum of 28^{0}C). germination takes place in low relative humidity of 20 per cent in less than 2 hr. Incubation period is 3 days.

Management:

1. Spraying with carbendazim 0.1 per cent or dinocap 0.2 per cent or thiophanate methyl 0.1 per cent or benomyl 0.1 per cent controls powdery mildew effectively.
2. All the affected plant parts should be destroyed.
3. Use tolerant/resistant varieties of cucurbits.

(3) Wilt :

Causal organism: *Fusarium oxysporum* f. sp. *niveum* (E.F. Smith) Snyder and Hansen.

Symptoms:

Watermelon plants are attacked at all stages of growth. When young seedlings are invaded, they may damp-off and die or the cotyledons may wilt and seedlings grow slowly and become stunted. Older plants wilt quickly, severely and permanently and they die within 10 days after symptoms appear. Inside wilted stems, the vascular tissue is discoloured. In a wet weather, a white or pinkish fungus growth develops on the surface of dead stems. In advanced stages of the disease, roots decompose.

Etiology:

The fungus produces three types of spores, small, colourless, oval to narrowly elliptical, non-septate microconidia, large, sickle shaped, septate macroconidia and thich walled chlamydospores.

Perpetuation:

The pathogen survives in soil and on seeds. In soil, it survives upto 16 years in the absence of watermelon crop. The fungus spreads in soil, compost and manure and can be carried by water and on tools, machinery and feet of workers and animals. The thick walled chlamydospores are the fungus structures that persist in field soils.

Epidemiology:

Plants are infected through root tips and breaks in roots where lateral roots emerge. The presence of root-knot nematodes results in increased incidence of wilt. After penetration, the fungus grows to the xylem, where it accumulates and produces gum-like material, tyloses and mycelium which can help plug the xylem to results in wilting. The optimum temperature for infection and disease development is 27^0C with little occurring above 30^0C. In field, plant wilting is promoted by high air temperature, high light intensity, low relative humidity and high evaporation rate. Disease is promoted by a high level of organic matter in the soil.

Management:

1. Spread of infested soil carried by equipment, tools, feet of animals and human beings and running water should be prevented.
2. Affected plants should not be put in compost or manure pits and do not introduce high levels of inoculum with plant debris, compost or manure.
3. Good water drainage should be provided to minimize secondary spore formation .
4. Disease free seeds should be used.
5. Resistant varieties like Crimson sweet, Early resistant queen, Fairfax, Sunshade, Summit, White hope etc. should be grown.

(4) Angular leaf spot :

Causal organism: *Pseudomonas syringae* pv. *lachrymans* (Smith and Byran) Young *et al.* [Syn. *P. lachrymans* (Smith and Byran) Carsner].

Symptoms:

Symptoms appear as water soaked lesions on the leaves and are confined by the veins. They turn grey to tan and form an exudates on the lower surface. Many of the spots loosen and fall out. Infected fruits show a brown, circular, superficial, firm rot, which causes rapid deterioration. The rot may extend into the flesh.

Etiology:

The bacterium is rod shaped with 1 to 5 polar flagella and 0.8 to 1.2 μm in length. It forms capsule and a green fluorescent pigment in culture. The colonies on beef-peptone agar are circular, smooth, glistening, transparent and white.

Perpetuation:

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

Epidemiology:

Optimum temperature for development is found as 24 to 26.5^{0}C. Moisture is the environmental factor that is most limiting to disease development. Angular leaf spot is promoted by wet conditions frequently associated with rainfall and sprinkler irrigation. Excessive nitrogen levels in the plant result in increased levels of disease.

Management:

1. Use of diseased free seed and crop rotations are major control measures.
2. The chances of seedling infection can be reduced by soaking the seeds for 5 to 10 minutes in mercuric chloride 0.1 per cent solution, rinsing in water and drying quickly.
3. Spraying the plants with 400 ppm solution of streptomycin sulphate or copper fungicide effectively controls the disease.
4. Crop rotation is useful when one or two years without cucurbits is recommended.
5. Resistance or tolerance to angular leaf spot is reported in cucumber varieties/lines such as Cherokee, Chipe, Dasher, Early Truimph, Turkey, Premier, Raider, Sweet slice etc.

(5) Mosaic :

Causal organisms: *Cucumber mosaic virus* (CMV).

Symptoms:

Mostly the infection begins when the plants are about 6 weeks old and growing vigorously. At this time they have 6 to 8 leaves and are commencing to run. The first symptoms appear in the young leaves which develop small greenish yellow areas, 1 to 2 mm in diameter, limited in outline by the smaller veins of the leaf. These spots are slightly more translucent than the remainder of the leaf and are often scarcely visible except by transmitted light. The characteristic symptom is yellow mottle on all leaves after infection. Leaf distortion and stumping of the plant are also noticed. Affected leaves are wrinkled and savoyed in appearance and become distorted and curled. The stem internodes are shortened, the leaves attain only about one half normal size and the petioles are reduced in length. Plants infected at an early stage blossom sparingly and set few fruits.

The stem and young fruits first become mottled with yellowish green and this gradually spread over the entire fruit. The body of the fruit becomes a light yellowish green, intermingled with spots of a much darker green colour. These dark portions are usually raised above the surrounding surface to form wart-like projection and produce distortions on the plant. In the later stages of the disease, fruits become smooth greenish white and somewhat mishappen with irregular green areas. The white fruits are responsible for the name 'white pickle'. The flowers are dwarfed and reduced in number.

Etiology:

The virus particle is isometric and 28 to 30 nm in diameter. Molecular weight of the virus is 4 to 9 x 5 to 8 million. RNA content is 18.5 per cent and the protein is 81.5 per cent. The thermal inactivation point is between 55^{0}C to 70^{0}C, the dilution end point is $\log_{10}$ 3 to 6 and the longevity *in vitro* is one to 10 days. The virus is destroyed by drying.

Perpetuation:

The virus is transmitted through sap and rarely through seeds. Aphids (*Myzus persicae, Aphis craccivora*) and spotted and striped cucumber beetles transmit the virus. The reservoir hosts are banana, clover, corn, passion fruit, safflower, spinach, sugarbeet, wild cucumber, *Commelina cucumis, C. diffusa*, blind weed, ragweed, white cockle, common motherwort, periwinkle, *Gladiolus* sp.

Management:

1. Initial inoculum should be reduced or avoided by eliminating as many reservoir hosts as possible in areas where cucurbits are grown, by avoiding double cropping.
2. Aluminium mulch is the reflective material used to control aphid-borne viruses. The mulch repels aphid vectors and thus delay virus infection.
3. Plant sanitation is very essential to keep down the disease incidence.
4. Eradication of weed hosts like *Commelina nudiflora, Physalis* sp., *Phytolacca americana* etc. should be carried out.
5. Infected plants should be pulled out and destroyed.
6. Vectors should be controlled by spraying of suitable insecticides.

(6) TOSPO virus

Symptoms :

Early symptoms of infection are difficult to diagnose. In young infected plants the characteristic symptoms consist of inward cupping of leaves and leaves that develop a bronze cast followed by dark spots. As the infection progresses additional symptoms develop which include dark streaks on the main stem and wilting of the top portion of the plant. Fruit may be deformed, show uneven ripening and often have raised bumps on the surface. Once a plant becomes infected the disease cannot be controlled.

Serological and molecular tests are commercially available to diagnose TSWV as well as a second common tospo virus found in ornamentals, *Impatiens necrotic spot virus* (INSV). Cytological studies of TSWV and INSV have shown that these viruses produce granular inclusions in the cytoplasm of infected plants. These inclusions can be seen in the light microscope with proper staining techniques. These inclusions can be diagnostic.

Epidemiology :

Tospo viruses are prevalent in warm climates in regions with a high population of thrips. For instance TSWV is an agricultural pest in Asia, America, Europe and Africa. Over the past 15 years outbreaks of Tomato spotted wilt disease have become more prevalent in these regions. Therefore, TSWV is described as an emerging viral disease of plants. The increased prevalence is largely because of the successful survival of the thrips vector *Frankliniella occidentalis*. Another thrips, *Scirtothrips dorsalis*, has also been implicated in the transmission of at least three tospo viruses, but there remains some controversy over its efficiency as a vector.

Control measures :

To prevent spread of the virus, infected plants should be immediately removed away from neighbouring plants. Control of insects, especially thrips, is important to reduce spread of the virus by vectors.

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Exercise No. 14

Onion

(1) Purple blotch / Scald :

Causal organism: *Alternaria porri* (Ell. Neergaard and *A. palandui* Ayyangar).

Symptoms:

This 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, then circular to oblong concentric, black velvety rings appear in the chlorotic area. These are fructifications of the fungus. The lesions develop towards the base of the leaf. The spots join together and spread quickly to the entire leaf area. Sometimes a yellow halo develops around each lesion. The leaves gradually die from the tip downwards. The affected leaves gradually die from the tip downwards. The diseased leaves break at the point of infection and hang down. The infection is also seen on the outer scales of the bulb. The disease causes premature drying of the foliage which results in poor development of bulbs. Bulbs become dry and papery.

Etiology:

Mycelium is branched, coloured and septate. Conidiophores arise singly or in groups. They are straight or flexuous, sometimes geniculate. They are spetate, pale to mid brown. 120 x 5 to 10 µm and are with one to several conidial scars. Conidia are solitary, straight or curved and obclavate. The body of the conidium may be ellipsoid, tapering to the beak. The beak is about the same length of the body, pale to mid- golden brown, smooth or minutely cerruculose and 100 to 300 µm in length and 15 to 20 µm thick at the broaden part. They are with 8 to 12 tranverse septa and zero to several longitudinal or oblique septa. The beak is flexuous, pale, 2 to 4 µm in diameter and is tapering.

Perpetuation:

It is also carried through seed bulbs collected from infected areas. The fungus spreads mainly through air-borne spores. Warm weather with humid conditions caused by rain and heavy dew helps in the spread of the disease and is favourable for the development of the fungus. The pathogen enters the plant through stomata or wounds. The fungus survives in the plant debris for 8 months

Epidemiology:

The fungus requires rain or persistent dew for reproduction and penetration. The optimum temperature for development of disease is 21 to 30^0C and relative humidity 90 per cent favours the disease.

Management:

1. Disease free bulbs should be selected for planting.
2. Seeds should be treated with thiram 2.5 g/kg of seed.
3. The field should be well drained.
4. The disease can be controlled by three foliar sprayings with copper oxychloride 0.25 per cent or chlorothalonil 0.2 per cent or zineb 0.2 per cent or mancozeb 0.2 per cent or difolatan 0.3 per cent.
5. Use resistant varieties for planting (Red Creole, New selection, VL 67, 33-2, 53-5).

(2) Stemphylium blight :

Cause : *Stemphylium vesicarium* (Wallr.) Simson.

Symptoms :

Initial infections on the leaves and leaf sheaths are small, light yellow to brown, and water-soaked. As the lesions expand they coalesce causing extensive blighting of the leaves. Typicllly, lesions are found in higher numbers on the side of leaves facing the prevailing wind. The centres of lesions turn brown to tn, then drk olive brown and finally black as the fungus sproulates. Sometimes fruiting bodies called perithecia may appear in infected tissue as small, black, pinhead-like raised bodies. Symptoms of stemphylium leaf blight are very similar to those of purple blotch, which ofen results in misidentification.

Etiology : Conditions for disease development :

The fungus survives in plant debris or soil.

Epidemiology :

Warm (18-25 ^{0}C) humid conditions and long periods of leaf wetness (16 hours or more) favour disease development. Extended periods of leaf wetness from dew formation, rainfall or overhead irrigation during bulb formation and development can result in severe leaf blighting. Bulb size can be greatly reduced due to loss of foliage. Infection is usually limited to leaves and does not extend down to the scales of the bulb.

Control measures :

Chemical control with fungicides is effective in reducing disease developmet. Long term rotation with unrelqated crops may reduce losses. Also, good field drainage and reduced plant density may lessen disease severity.

Spray Dithane M-45 @ 2 g/litre water mixed with Sandovit or Triton @ 1 ml/2 litre of solution.

(3) Downy mildew :

Causal organism: *Peronospora destructor* (Berk. Casp.)

Symptoms:

The symptoms vary with the type of infection. In systemic infection which occurs when the plants are grown from a diseased bulb, the plants remain stunted, become distorted and pale green. In humid atmosphere the downy growth of the fungus develops over the entire leaf surface. In dry weather this growth is absent and only white spots are seen. In local, infections caused by wind-borne conidia, oval to cylindrical pale spots are formed on the leaves. Usually these spots contain alternating green and chlorotic zones. In humid weather, the fungus develops as white to purplish downy growth of these spores. Usually the older leaves are attacked first and the infection spreads to the sheath. Inner leaves are then affected but new and young central leaves remain healthy. The bulbs produced on diseased plants can remain small with succulent necks. Succulent necks are subjected to attack by fungi and bacteria in storage.

Etiology:

The sporangiophores are aseptate, 122 to 820 µm long, swollen at the base to a diameter of 7 to 8 µm. Branching is dichotomous. Sterigmata are subacute or acute. Sporangia are pyriform to fusiform, attached to the sterigmata by their pointed end and measure 40 to 72 x 18 to 20 µm. Sporangia germinate by one or two germ tubes. The coenocytic mycelium is intercellular with filamentous haustoria. Oogonia are formed in the intercellular spaces. Oospores germinate by means of germ tube.

Perpetuation:

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 inoculum are the diseased bulbs used for propogating 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.

Epidemiology:

The fungus requires cool, moist nights and only moderately warm days for best development. Cloudy days are also favourable because eight hours of light kills the sporangia. The sporangia are produced in humid atmosphere at 4 to 25°C with optimum at 13°C. they usually develop during the night and mature early in the morning. Dissemination occurs throughout the day.

Control measures :

1. Bulbs used for propogation should be obtained from healthy fields. Wild onions should be destroyed.
2. The fields should be well drained.
3. Three sprayings with mancozeb 0.2 per cent or ziram 0.1 per cent or difolatan 0.1 per cent is found effective. Spraying should be started 20 days after transplanting and repeated at 10 to 12 days interval.

(4) Smut :

Causal organism: *Urocystis cepulae* Frost [Syn. *Urocystis colchici* var. *cepulae*; *Tuburcina cepulae* (Frost Liro].

Symptoms:

The fungus attacks seedlings. Dark lesions occur on the cotyledons as they emerge. These lesions develop into thickened areas of several millimeters in size. Eventually the lesions burst open releasing masses of black smut spores. Most infected plants are killed outright within 3 to 4 weeks of emergence. Surviving plants remain permanently stunted with short, brittle, distorted leaves bearing lesions throughout their length. In the mature plants numerous blisters are found both on the leaves and bulb scales. Bulbs are usually small. Infected plants which survive have low resistance to secondary infections from other pathogens.

Etiology:

The sori of *Urocystis cepulae* contain dark coloured and powdery spore masses. The spores are found in permanent balls. Each ball consists of an enveloping cortex of tined, sterile, bladder like cells with one or two central dark coloured thick walled chlamydospores (smut spores). The sterile cells are smaller than the spores. The spores germinate by means of short promycelium while still in the ball. These chlamydospores are 12 to 15 µm in diameter. The promycelium does not produce the apical whorl of sporidia, instead hyphae grow out from the promycelium.

Perpetuation:

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. Wind-born soil and surface drainage water are important means of local dissemination.

Epidemiology:

Optimum temperature for the development are 10 to 20^{0}C but the pathogen can occur at temperatures upto 20^{0}C. Infection of the plant from the soil can only takes place through the first leaf. The plant, therefore, is only susceptible for about 2 to 3 weeks from the time of sowing. Soil moisture has direct effect on the amount of diseases.

Management:

1. Seed treatment with thiram @ 30 g or 30 to 50 per cent carboxin + thiram at 25 g per kg of seed is proved effective in the control of the disease.
2. Crop rotation and use of diseased free seeds in healthy fields are recommended. Since the smut spores remain viable in the soil for number of years, long crop rotations are recommended.
3. Crop debris should be burnt.
4. The cultivar Hardy White Bunching is completely resistant.

(5) Smudge :

Causal organism: *Colletotrichum circinans* (Berk. Cogl. *C. chadoniana* Nolla and *C. allii* Averna)

Symptoms:

Smudge is mainly a disease of scales of the bulb. In the seed bed the fungus may cause damping off if the soil is wet and warm. The most common symptom of the disease is the appearance of subcuticular, dark green to almost black smudge. The black colour may be uniform on the lesion but normally the circular lesions show concentric rings of dark stroma and mycelium. The outermost ring is the broadest. Inner scales are also attacked. The lesions are small, sunken and yellow, later they enlarge and coalesce. In humid weather pinkish masses of spore develop on the black stroma.

Etiology:

The mycelium is septate and branched. In culture the fungus produces dark brown colonies with aerial mycelium through which abundant sclerotia (stroma protrude). Intercalary chlamydospores are also formed. The thick walled hyphae interwine and form dark green to

black stromata. They sometimes coalesce. Acervuli are formed on stromata beneath the cuticle by formation of a palisade layer of short, hyaline conidiophores which later rupture the cuticle. Scattered all over the acervulus are numerous thick walled, dark coloured, zero to three septate setae which may be 80 to 315 µm long. Conidia are borne on the tip one at a time. The conidia are fusiform, falcate, with acute apices, guttulate, hyaline, one celled and 19 to 21 x 3.5 µm. They germinate by one or two germ tubes which form appressoria as soon as they come in contact with a hard surface.

Perpetuation:

The fungus overwinters on onions in soils as stromata and also as saprophytic mycelium for several years. Individually the conidia are sensitive to freezing and desiccation but in mass they can withstand drying. In favorable conditions the stromata develop acervuli and conidia, causing infection of the bulbs. Conidia formed on these infected parts further spread the disease.

Epidemiology:

The fungus need a fairly wet soil for good growth. Optimum temperature for germination of conidia is 20^0C. Best growth of the fungus on agar medium occurs at about 26^0C. The disease can develop at any temperature from 10 to 32^0C but the optimum is about 26^0C. moist temperature is essential for production of conidia. Rain drop splashes help in their dispersion.

Management:

1. Red onion (Nasik Red, Pusa Ratna, Pusa Red) is resistant to the disease.
2. Protection from rains after harvest is necessary.
3. Before storage the bulbs should be dried properly. Sun drying is not desirable. The drying of the bulbs before storage should be by hot air at 37 to 48^0C.
4. The crop may be sprayed with zineb 0.2 per cent or maneb at seven to ten days interval to reduce inoculum load.
5. Chemical treatment of the bulbs before storage can be done with 0.2 per cent suspension of thiram, captan or difolatan. However, before use such bulbs need utmost care.

(6) Erwinla rot
Bacterial soft rot of Onion :

Causal organism : *Erwinia carotovora* pv. *carotovora*

The bacteria causing this disease are rod-shaped and measure 1.5 - 5.0 x 0.9 µ in size. They occur, either singly or in pairs of in long chains and have peritrichous flagella numbering 2-8. They are non-spore forming.

The disease occurs in all countries, where onion is grown. The pathogen attacks the bulbs in the fields, as well as in transit and storage.

Symptoms :

The initial symptoms appear as small, irregular, water- soaked lesions near the points of infection. The infection, which starts at the collar region or through senescent leaves, spreads rapidly and progresses downward into the bulb and very soon after all the tissues of the bulb. The affected areas become soft and mushy. The outer scales become completely rotten and slimy ooze is seen on the surface. When the infected bulb is pressed slightly, a turbid, slimy liquid comes out. Bulbs infected by the soft rot bacteria gives off a repulsive odor.

Mode of survival, spread and Epidemiology:

The bacteria causing this disease are rod-shaped and measure 1.5 - 5.0 x 0.9 μ in size. They occur, either singly or in pairs of in long chains and have peritrichous flagella numbering 2-8. They are non-spore forming.

The soil inhabiting bacteria are faculative parasites and can survive in the soil up to 20 years in the diseased plant parts and in dead organic matter present in the soil as a saprophyte. When conditions are favourable for the bacteria and when suitable hosts are present they become parasitic and attack the hosts. They are primarily wound parasites and can enter the host only through injuries or bruises caused during cultural operations or while handling during transit or storage. Once the bacteria enter the host tissue, they multiply enormously within a short period. They produce certain enzymes, which dissolve the cell walls and the tissues become a mushy mass of disintegrated, rotten cells. They are spread by direct contact, through agricultural implements, soil, water and insects, and by hands.

The bacteria can grow and are active over a wide range of temperatures from 5-35 ^{0}C and moisture levels. However, temperatures between 28-35 ^{0}C and high soil moisture favour the occurrence and spread of the disease. Besides onion, the pathogen can attack several other crops, such as carrot, turnip, radish, potatoes etc.

Disease management :

Agronomic practices :

i) The pathogen is a typical wound parasite. So precautionary measures should be taken to avoid causing possible damages, such as injuries or bruishes during cultural operations or while handling during transit or storage.

ii) Diseased bulbs should be sorted out at the time of harvest and before storing.

iii) Affected bulbs should no be allowed to be mixed with sound bulbs.

iv) Diseased plant debris should be removed from the field and destroyed.

v) Irrigation water should not be allowed to flow from diseased fields to other fields.

vi) The store houses should be kept clean, moisture free and well ventilated.

vii) The bulbs should be sufficiently dry before being stored.

viii) It is better to store the bulbs in air conditioned ware houses.

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Exercise No. 15

Garlic

(1) Neck and bulb rot :

Causal organism: *Botrytis allii* and *Fusarium oxysporum.*

Symptoms:

Neck rot is found commonly upon the bulbs at the time of harvest. Affected scale tissue become soft. Dense layer of grey mould appear at the neck. The infection progresses most rapidly down the scales which have been originally infected. Dark sclerotia appears on the older decayed tissue. In case of bulb rot caused by *Fusarium* failure of germination and drying of leaves from the tip can be noticed. Rotting of bulbs and production of side shoots produces clump of shoots.

Perpetuation:

Both the pathogens survive in the field as well in storage. *Botrytis allii* survives as sclerotia while *Fusarium* survives as chlamydospores in soil and also in bulbs.

Epidemiology:

Botrytis allii requires cool and moist weather. Infection and proliferation decay of bulbs are favoured by the temperature between 15 to 20^{0}C. Fusarial bulb rot is favoured by excess rainfall followed by a dry spell for 10 to 15 days. The feeding injury by garlic mite, *Rhizoglyphus echinopus* favours bulb rot.

Control measures:

1. Selection and collection of seed materials should be from disease free areas.

Bulb treatement with thiram or captan or carbendazim 2g/kg and spraying with carbendazim 0.1 per cent controls the disease.

(2) Stemphylium blight :

Disease symptoms :

Infections occurs on radial leaves of transplanted seedings at 3-4 leaf stage during late March and early April. The symptoms appear as small yelloowish to orange flecks or streaks in the middle of the leaves, which soon develop into elongated spindle shaped spots surrounded by pinkish margin. The disease on the inflorescence stalk causes severe damage to the seed crop.

Survival and spread : The fungus survives in plant debris or soil.

Favourable conditions : Warm (18-25 ^{0}C) humid conditions and long periods of leaf wetness (16 hours or more) favour disease development.

Control measures :

1) Use healthy planting material.
2) Two to three years crop rotation with non-host crop.
3) Collectionn and destruction of infected plant debris to reduce inoculum load.
4) Spraying crop with Carbendazim 0.2 % propineb. Chlorothalonil, Difenconozole etc. for effective management of this disease.
5) Foliar application of Propiconazole (0.05 %) followed by Manozeb (0.2 %) is effective for control of the disease.

(3) Blemish :

Causal organism : *Sclenotium cepivorum*

Sytmptoms :

* It usually affects patches of plants rather than individuals.
* Stunted plant growth observed initially which is followed by early yellowing and death first of the outer leaves then the reset of leaves and the central stem.
* Obvious rotting of the stem above the bulb.
* The disease much more appearent on the bulb itself where rotting appears as white, fluffy mycelial growth around the basal plate that moves upwards and quickly develops small, black, poppy seed sized *Sclerotia* in and on decaying tissues,

(4) Black mould :

Causal organism: *Aspergillus alliaceous* Thorn and Church, *A. niger*, *A. repens* (Corda de Bary) and *A. sclerotiorum* Huber.

Symptoms:

Black mould blemish is common throughout the year. It manifests itself with the copious growth of a dust-like fungal mass which remains concealed mostly between the scales. The whole tissue gradually transform into a black powdery mass. The individual bulbs shrivel and become light in weight. Under high humid conditions the inner tissues become moderately soft. Infected bulbs loose their pungency and smell. Rotten garlic show black, brown, pink or white coloured rotting. Rotting may be partial or complete.

Control measures :

1. Garlic bulbs should be stored in a dry airy place.
2. Infected bulbs should be discarded before storage.
3. Fumigation of bulbs with formation 0.03 per cent for table purpose is effective.

Exercise No. 16

(A) Chilli (B) Coriander and (C) Turmeric

(16) (A) Chilli

(16) (A) (1) Anthracnose and Ripe Fruit Rot :

Causal organism: *Colletotrichum capsici* (syd.) Butler and Bisby

Symptoms: The disease appears in the die back and ripe fruit rot phases.

i) Die back phase : 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. In diseased plant, flowers dry up. This drying up spreads from the flower stalk to the stem and subsequently causes die-back of the branches and stem and the branches wither. The entire branch or the entire top of the plant may wither away.

ii) Ripe rot phase : The disease symptoms appear mostly on ripened (turning red) fruits. A small, black, circular spot appears on the skin of the fruits and spreads in direction of the long axis thus becoming more or less elliptical. The spots are usually sunken with black margins. Badly diseased fruits turn straw coloured or pale white from normal red. On this discoloured area numerous black acervuli are found scattered. Ultimately the diseased fruits shrivel and dry up.

Etiology:

The mycelium is septate and inter and intracellular. The acervuli are produced on the affected portion. Conidia in mass appear pinkish. Conidia are borne singly at the tip of conidiophores. Conidia are falcate, hyaline, unicellular, curved with narrow ends.

Perpetuation:

The fungus is seed borne. Primary infection takes place through infected seed and plant debris and secondary spread is by air-borne conidia.

Control measures:

1. All diseased fruit should be collected and destroyed as soon as noticed.
2. Use disease free seeds.
3. Seed treatment with Thiram 2 g/kg of seed.
4. Spray the crop with Zineb 0.25 per cent or Mancozeb 0.25 per cent or Carbendazim 0.1 per cent. the first spraying should be given jest before flowering and the second at the time of fruit formation. A third spraying may be given a fortnight after second spray.

(16) (A) (2) Wilt

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

Weightage (%)
6
6
6
3
2
3
5
5
6
3
5
2
3
3
3
100

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 atached. By the time above - ground symptoms are evident, the vascular system of the plant is discoloured, particularly in the lower stem and roots.

Etiology : Primary source of inoculum : Chlymydospores, Soil, Seed.
Secondary source of inoculum : Micro conidia, Macro conidia, water.

Epidemiology :

Fusarium is a soil borne fungus. Once a field is infected, the pathogen may survive in the soil for many years. The fungus can be transported by farm equipent, drainage water, wind, or animals, including humans. The fungus is seed and soil borne. Warmer and drier climates (> 25 ^{0}C) favour disease and also when crop rotations are not practiced.

Control measures :

i) Use of wilt resistant varieties.

ii) Drenching with 1 % Bordeaux mixture or Blue copper or Fytolan 0.25 % may give protection. Seed treatment with 4 g Trichoderma viride formulation or 2 g Carbendazim per kg seed is effective. Mix 2 kg *T. viorode* formulation with 50 kg 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.

(16) (A) (3) Leaf curl

Causal organism: *Gemini virus.*

Symptoms:

Leaves curl towreds 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. Son control measures of whitefly in this regard would be helpful.

Mosaic virus :

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

Control measures of viral diseases :

1) Control measures are not known for majority of viral diseases.
2) Hence mechanical, cultural methods are mostly recommended.
3) The infected plants should be uprooted and burnt or buried to avoid further infection.
4) Avoid monoculture of chilli crop.
5) Selection of healthy and disease-free seed.
6) Suitable insecticidal sprays reduce the incidence of viral disease, since majority of viral diseases are transmitted by insect vectors.

7) Soaking seeds in a solution containing 150 g Trisodium orthriphosphate per litre of water for 30 minutes inhibits seed-borne inoculum.
8) Treated seed should be washed with fresh water and dried before sowing.
9) Nursery beds should be covered with nylon net or straw to protect the seedlings from viral infection.
10) Raise 2-3 rows of maize or sorghum as border crop to restrict the spread of aphid v ectors.
11) Apply Carbofuran 3G @ 4-5 kg/acre in the mainfield to control sucking complex and insect vectors selectively.
12) If it is not possible spray the crop with systemic insecticides. Dimethoate 2 ml of Acephate 1 g per litre of water.
13) Collect and destroy infected virus plants as soon as they are noticed.

(16) (B) Coriander

(Coriandrum sativum L.)

(16) (B) (1) Stem gall

Causal organism : *Protomyces macrosporus* Unger

Symptoms :

Galls appear o the leaves and stems of the plants affected by the disease. Shape of coriander seeds change due to effect of the disease. The disease appears in the form of tumour like swellings on the last veins, leaf stalks, stems and on the fruits. These tumours are at first glossy but later on rupture and become rough. The plants may be killed in case of severe attack.

Control :

i) To control the disease, sowing may be done only after treating the seeds with 4 g Thiram and 2 g Bavistin / kg seeds.
ii) Spray 0.1 % solution of Carbendazim when the symptoms start appearing and repeat the spraying at an interval of 20 days till the disease is completely controlled.
iii) Use clean and healthy seeds collected from healthy fields.
iv) Follow suitable crop rotation and field sanitation.

(16) (B) (2) Powdery mildew of Coriander

Causal organism: *Erysiphe polygoni*

Symptoms:

It appears as small, white circular patches on young parts of stem and leaves. These increase in size, often coalesce to cover extensive area of leaf surface. Affected leaves reduce in size. Appearance of whitish powdery mass on leaves and stem is the characteristic of disease. If the disease is unchecked at early stage, the whole plant becomes almost white with powdery coating. Early infection does not permit seed formation. However, if the infection

occurs at late stage, seed formation may take place but the seeds will be small and shriveled thus affecting the yield and quality.

Etiology:

Fungus is ectoparasite, spreading over the surface of the host and sending haustoria into the epidermal cell. Conidia are produced in chains on conidiophores. The conidia are ovate to rectangular, hyaline and thin walled. In advance stage of fungal growth on the host tissue, the perithecial stage is found. The cleistothecium contain two the eight asci and each ascus contain four to eight ascospores.

Perpetuation:

Fungus survives on collateral host. The cleistothecium can also survive on the host tissue during off season which serve as primary source of infection. Secondary spread is through air borne conidia.

Disease appears late in the season and observed during February to March. Dry season and high temperature favours the disease development. Early sown crop show less incidence as compared to late sown crop.

Control measures:

1. Removal and destruction of left over plant debris after the crop is harvested.
2. Adjustment in sowing dates is advocated and early sowing in October is recommended.
3. Sulphur dusting (25 kg/ha) once when the crop is about 2 months and once again after fruit setting was found effective.
4. Spray the crop with 0.25 per cent wettable sulphur or Karathane (0.2 per cent).
5. Use of resistant varieties.

(16) (B) (3) Wilt

Casual organism : Wilt caused by *Fusarium oxysporum Schech. f. corianderii* Kulkarni *et al.* was reported in Central India for first time in 1936 and from Madhya Pradesh in 1953. The pathogen is soil as sell as seed borne.

The plants of coriander are attacked by wilt at all stages of plant growth from seedlings to maturity. It can cause pre-emergence mortality in germinating seeds and wilting of germinating seedlings. Old plants show drooping of terminal portion to base followed by withering and drying up of leaves eventually leading to death of plants.

The disease is systematic and also soil borne in nature. Discolouration of vascular bundles of root system and vascular plugging with fungus can be seen. Sterility is a major and common feature in wilted plants. Seeds if formed are light, immature, shriveled with out any symptoms. Localization of pathogen is restricted to carpophores, pericarp, seed coat and endosperm in asymptomatic seeds infected with *Fusarium oxysporum f.* sp. *corianderii*. In moderately discoloured seeds, it was observed in carpophores, percarp seed coat and endospserm. In heavy infected discoloured seeds pathogen colonizes embryo also besides all these components.

Etiology : The fungus produces macro, micro conidia and chlamydospores. The micro conidia are scattered freely in mycelial mats and macro conidia in sporodochia.

Epidemiology : The fungus is capable of growing at 12-35 ^{0}C and best growth is observed at 19.5 ^{0}C. Factors like irrigation, soil pH between 5.8-6.9 temperature between 29.8 to 33.0 ^{0}C and soil moisture content between 60-70 per cent favours the disease development.

Control measures :

i) Fertilizer levels also play an important role in incidence of wilt disease.

ii) Summer ploughing, soil solarization and proper three years crop rotation (e.g. cluster bean; cumin-cluster bean, wheat-cluster bean-mustard) may be an effective measure to reduce the wilt incidence.

iii) Sowing of seeds in first to second week of November and use of resistant variety CO-3, Sadhana and Surabhi have been recommended to minimize the disease incidence.

iv) According to incidence of wilt disease can be reduced by use of healthy and disease free seeds.

iv) Seed treatment with bavistin 1.0 g/kg of seed or Trichoderma @ 10 g/kg of seeds or seed treatment with 1:1 mixture of Bavistin + Captan @ 4.0 g/kg of seeds.

(16) (C) Turmeric

(*Curcuma longa* L.)

(16) (C) (1) Leaf spot (Taphrina leaf spot of Turmeric)

Causal organism: *Taphrina maculans* Butler.

Symptoms:

The spots appear in great numbers, thickly covering both the sides of the leaves. The attacked leaves bear a reddish brown appearance instead of the normal green. Individual spots are minute and are arranged in rows along the leaf veins, often they run together to form linear irregular lesions. Infected leaves appear reddish-brown, in contrast to the golden yellow or yellowish-green of healthy leaves. The leaves later become chlorotic and pale yellow. As the disease advances, the affected tissues wither and dry and although the diseased plants are not killed the leaves are severely blotched and blight.

Etiology:

The mycelium is septate, endophytic and intercellular between cutical layer and epidermis producing haustoria. Asexual reproduction is by means of budding i.e. bud-like structures are formed (conidia) which are called blastospores. Sexual reproduction by ascospores in asci. Asci are naked and contain unicellular, oval ascospores.

Perpetuation:

Primary infection takes place through dormant mycelium in rhizomes. It is also possible that the dried leaves having spots and laying about in the field with the asci might functioning as the chief source of primary inoculum for the next crop. Secondary infection by asci and ascospores carried by wind and rain drops from spots. The fungus is homothallic and favoured cool and moist weather.

Control measures: 1. Clean cultivation.

2. Spray with 0.6% Bordeaux mixture or copper oxychloride (0.25%) or Zineb (0.2%) or Ziram (0.2%).

Exercise No. 17 ORNAMENTAL CROPS

(17) (A) Marigold and (B) Rose

(17) (A) Marigold

(17) (A) (1) Botrytis blight

Causal organism : Botrytis cinerea

Symptoms :

* Symptoms appeared as dead blotches on leaves, flowers and stems.
* Rotting of stems may cause plants to collapse.
* Flower buds may fail to open and diseased flowers that open become decayed and drop prematurely.
* A covering of gray fuzzy fungal growth and spores appears on infected plant tissues.

Etiology :

Botrytis cinerea persists in the green house year around as mycelium conidia on sclerotica on living or dead plants and sclerotia or conidia in infested soil. Outdoors the fungus over seasons on decayed plant material or in infested soil. In rare cases, seed lot may be contaminated with sclerotia.

Epidemiology :

- Sclerotia are the main structures for field survival, although conidia may over season in the field and can survive a temperature rage of 39 to 131 ^{0}F (4 to 54 ^{0}C).
- The overwintering stage can be spread by anything that moves soil on plant debris and transplants scierotia, mycelium on conidia.
- Botrytis is often considered a cool weather pathogen with best growth spopulation, spore release germination and establishmnet of infection occurring at an optimum of 18 to 23 ^{0}C.

Control measures :

1) Avoid splashing water on the foliage when watering.
2) Strict sanitation is of the utmost importance.
3) Provide maximum air circulation in the green house and the plant beds.
4) Properly space plants to allow for maximum air circulation.
5) Avoid over fertilization (especially with nitrogen) and wet mulches.
6) Avoid unnecessarily wounding plants.
7) Use of different fungicides at intervals of 5 to 7 days in rainy. Oven cast weather and every 7 to 10 days in warm, dry weather.

(17) (A) (2) Alternaria blight

Causal organism : *Alternaria zinnae*.

Symptoms :

The infection can lead to premature deloliation and finally death of the plant. *Alternaria zinnae* cause inflorescence blight of marigold in which elongated lessions are formed in inflorescence Light tan to dark brown, large irregular blotches appears on leaves with zonation.

Etiology : The fungus grows optimally at 25 ^{0}C and growth was almost inhibited at temperature 35 ^{0}C.

Epidemiology : The favourable temperature for germination of conidia of Alternaria spp is 25-28 ^{0}C and presence of free water on leaf surface.

Control measures :

1) Diathane M-45 (0.2%) and Carbendazim (0.05 %) spray can be used to control the disease.
2) Volatile oils from Eulalyptus sp. found effective against the disease.
3) Some plants and plant products found to be useful in controlling the disease.

(17) (B) Rose

(17) (B) (1) Dieback

Causal organism: *Diplodia rosarum* Fr.

Symptoms:

The pruned surface of the twig dries from tip downwards. Twigs become brown to dark brown or black. The disease passes from the branch twig to the main stem and from where it spreads to the roots. Finally it kills the whole plant. Stem and roots show browning of the internal tissues.

Etiology:

The fungus produces round, black pycnidia which bear spores. The pycnidiospores are dark coloured and 2-celled. Perithecia are immersed in the host tissue and are surrounded by a pseudostroma. Ascospores are ellipsoidal or fusoid, hyaline, 2-celled with the septum in or near the middle.

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

Epidemiology:

Older plants and neglected and weak bushes are most frequently attacked. Disease spread is faster at 30 to 32^{0}C.

Management:

1. Pruning should be done so that the lesions on the shoots are climinated. Partially diseased twigs should be pruned atleast 3 to 5 cm below the visible

symptoms of the disease. In all the cases, the pruned ends should be immediately coated with Chaubattia paint to avoid attack of digger wasp which lays eggs on the softer inner tissues of the canes.

2. Spraying with copper oxychloride 0.2 per cent or difolatan 0.2 per cent or chlorothalonil 0.2 per cent or mancozeb 0.2 per cent once in early September and again in later October is recommended for the control of the disease.

(17) (B) (2) Powdery mildew

Causal organism: *Sphaerotheca pannosa* var. *rosae* (Wallr.) Lev.

Symptoms:

The disease appears as slightly raised blister-like areas on the young leaves. Soon leaves are covered with a grayish white, powdery fungal growth. When the leaves expand, they become curled and distorted. On older leaves, large white patches of fungal growth appear. But usually there is little distortion. Lesions on leaves may appear more or less discoloured and many become necrotic. White patches of the fungal growth appear on young, green shoot and they may coalesce and cover the entire terminal portions of the growing shoots which may become arched or covered at their tip. Buds may also be attacked and covered with white mildew before they open. Diseased buds fail to open. The infection spreads to the flower parts and they become discoloured, dwarfed and dried.

Etiology:

Mycelium is white, septate, ectophytic and send 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 to 29.0 x 12.9 to 14.5 µm. 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 8 ascospores.

Perpetuation:

The fungus overwinters 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.

Epidemiology:

Infection occurs when the air is saturated with moisture and the temperature is about 20^0C. Optimum conidial germination occurs at 97 to 99 per cent relative humidity and at temperature ranging between 17 to 24^0C. The disease is favoured by dry weather with maximum day temperature of 20 to 25^0C with cool nights.

Management:

1. The diseased and fallen leaves should be collected and burnt.
2. Four sprayings at 10 days interval of wettable sulphur 0.3 per cent or dinocap 0.07 per cent or carbendazim 0.1 per cent controls the disease effectively.
3. Spraying with phaltan 0.3 per cent + carbendazim 0.025 per cent also controls the disease.
4. Spraying with benomyl 0.1 per cent or triademefon 0.1 per cent at 30 days interval controls the disease.

5. Sulphur dust can be used at 25 kg/ha. Wettable sulphur or sulphur dust should not be used when the temperature is above 30^{0}C as it may cause scorching.

(17) (B) (3) Black leaf spot

Causal organism: *Diplocarpon rosae* Wolf. (Conidial stage: *Marssonina rosae*).

Symptoms:

The disease is characterized by the presence of black spots on the leaves. These spots are more or less circular in outline. They have a very irregular fibrillose border due to the radiating strands of mycelium which occur beneath the leaves and leaf buds which open late in the season. The plant blossoms poorly. They may not flower in the following season. Besides appearing on the leaves, the fungus may manifests itself on the stems and flowers of rose bushes. On the stem the infected areas present a blackened, blistered appearance, dotted with pustules.

Causal organism: *Diplocarpon rosae* Wolf. (Conidial stage: *Marssonina rosae*).

Etiology:

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 µm in diameter. Stroma is thin. Conidiophores are hyaline, short and cylindrical. Conidia are hyaline, 2-celled, fusiform or allantoid to obclavate, upper end round, base narrow, guttulate, 18 to 25 x 5 to 6 µm. Perithecia are spherical to disciform. Asci are oblong or subclavate. Paraphyses ae slender and enlarged abruptly at the tip. Ascospores are oblong, hyaline, unequally 2-celled, constricted at the septum and 20 to 25 x 5 to 6 µm.

Perpetuation:

The fungus hibernates in the old leaves fallen on the ground and in the lesions on the stem in the temperate regions. In tropical and warm weather, the fungus survives in the infected leaves on the plants. Either conidia or ascospores act as primary inoculum.

Epidemiology:

The disease is favoured by high humidity and low temperature (21^{0}C). winter frosts favour the disease. In North India it is prevalent during January-April but in South Indian conditions it is noticed from August.

Control measures :

1. As the fungus perpetuates on old diseased leaves and stems it is necessary to collect and destroy them at the end of the season. Diseased plants should be pruned carefully, collected and burned.
2. Spraying with tridemorph 0.025 per cent or captan 0.2 per cent or ferbam 0.2 per cent or benomyl 0.1 per cent at weekly intervals starting with the sprouting of the plants till the appearance of the new foliage and continuing during humid weather effectively controls the disease.
3. Rose cultivars Belaya, John Cabot and Carefree Beauty are resistant.

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Syllabus

Course :	PATH 365	Credit:	3(2+1)	Semester-VI
Course title:	Diseases of Field and Horticultural Crops and their Management – II			

Theory : Symptoms, etiology, disease cycle and management of following diseases:

Field Crops:Wheat: rusts, loose smut, karnal bunt, powdery mildew, alternaria blight, and ear cockle;Sugarcane: red rot, smut, wilt, grassy shoot, ratoon stunting and PokkahBoeng;Sunflower: Sclerotinia stem rot and Alternaria blight; Rust, Downy mildewMustard: Alternaria blight, white rust, downy mildew and Sclerotinia stem rot;Gram: wilt, grey mould and Ascochytablight;Lentil: rust and wilt;Cotton: anthracnose, vascular wilt, and black arm;Pea: downy mildew, powdery mildew and rust

Horticultural Crops: Mango: anthracnose, malformation, bacterial blight and powdery mildew;Citrus: canker and gummosis,Grape vine: downy mildew, Powdery mildew and anthracnose;Apple: scab, powdery mildew, fire blight and crown gall;Peach: leaf curl,Strawberry: leaf spot Potato: early and late blight, black scurf, leaf roll, and mosaic;

Cucurbits: Downy mildew, powdery mildew, wilt;Onion and garlic: purple blotch, and Stemphylium blight;Chillies: anthracnose and fruit rot, wilt and leaf curl;Turmeric: leaf spot, Coriander: stem gall,Marigold: Botrytis blight; Rose: dieback, powdery mildew and black leaf spot.

Practical : Identification and histopathological studies of selected diseases of field and horticultural crops covered in theory. Field visit for the diagnosis of field problems.Collection and preservation of plant diseased specimens for herbarium.Note: Students should submit 50 pressed and well-mounted specimens.

Teaching Schedule : Theory

Lecture	Topic	Weightage (%)
	Symptoms, etiology, disease cycle and management of major diseases of following crops	
	Field crops	
1,2,3	Wheat: Rusts, loose smut, Karnal bunt, powdery mildew, Alternaria blight, and ear cockle	5
4,5	Sugarcane: Red rot, Smut, Wilt, Grassy shoot, Ratoon stunting and Pokka Boeng	5
	Oilseed	
6,7	Sunflower: Sclerotinia stem rot and Alternaria blight, Rust, Downy mildew	3
8,9	Mustard: Alternaria blight, White rust, Downy mildew and Sclerotinia stem rot	3
	Pulses	
10	Gram: wilt, grey mould and Ascochyta blight	5
	Lentil: rust and wilt	4
11	Linseed :Alternaria bud blight, Rust ,Powdery mildew	2
12	Pea: Downy mildew, Powdery mildew and Rust, wilt	5
	Cash Crop	
13,14	Cotton: Root rot, Wilt, Anthracnose, and black arm, Dahiya diseases, leaf curl of cotton, 2-4-D injury	7
	Horticultural Crops	

Lecture	Topic	Weightage (%)
15,16,17,	Mango: Die back, Anthracnose, Mango-malformation, bacterial blight and powdery mildew, Spongy tissue, Red rust, Pink diseases, Loranthus, Stone graft Mortality, Lime induced chlorosis	6
18,19	Citrus : Citrus canker, Gummosis, Fruit rot, Citrus greening, Anthracnose, Tristeza, Citrus Exocortis, Scab of citrus, Mottle leaf of citrus	6
20,21	Grape vine: Downy mildew, Powdery mildew, Anthracnose, Bacterial Canker, Grape fan-leaf virus	6
22	Apple: Scab, Powdery mildew, Fire blight and Crown gall, Mosaic	3
23	Peach: leaf curl	2
23	Strawberry: Leaf spot	3
	Vegetables	
24	Potato: Early and late blight, black scurf, leaf roll, and Mosaic	5
25,26	Cucurbits: Downy mildew, powdery mildew, wilt, Angular leaf spot, Mosaic, TOSPO virus	5
27	Onion: Purple blotch, and Stemphylium blight, Downy mildew, Smut, Smudge, Erwinia rot	6
28	Garlic : Neck and bulb rot, and Stemphylium blight, Blemish, Black mould	3
29	Chilli : Anthracnose and fruit rot, Wilt and leaf curl	5
30	Coriander : Stem gall, Powdery mildew, Wilt	2
30	Turmeric: leaf spot	3
	Ornamental Crops	
31	Marigold :Botrytis blight, Alternaria blight	3
32	Rose: Dieback, Powdery mildew and Black leaf spot	3
	Total	**100**

Practical

Exp.	Topic
	Identification and histopathological studies of selected diseases of field and horticultural crops covered in theory. Collection and preservation of disease specimen. **(Note: Students should submit 50 pressed and well-mounted specimens)**
1	Wheat: Rusts, loose smut, Karnal bunt, powdery mildew, Alternaria blight, and ear cockle
2	Sugarcane: Red rot, Smut, Wilt, Grassy shoot, Ratoon stunting and Pokka Boeng
3	Sunflower: Sclerotinia stem rot and Alternaria blight, Rust, Downy mildew
4	Mustard: Alternaria blight, White rust, Downy mildew and Sclerotinia stem rot
5	Gram: wilt, grey mould and Ascochyta blight, Pea: Downy mildew, Powdery mildew and Rust, wilt
6	Lentil: rust and wilt, Linseed :Alternaria bud blight, Rust ,Powdery mildew
7	Cotton: Root rot, Wilt, Anthracnose, and black arm, Dahiya diseases, leaf curl of cotton, 2-4-D injury
8	Mango: Die back, Anthracnose, Mango-malformation, bacterial blight and powdery mildew, Spongy tissue, Red rust, Pink diseases, Loranthus, Stone graft Mortality, Lime induced chlorosis
9	Citrus : Citrus canker, Gummosis, Fruit rot, Citrus greening, Anthracnose,

Exp.	Topic
	Tristeza, Citrus Exocortis, Scab of citrus, Mottle leaf of citrus
10	Grape vine: Downy mildew, Powdery mildew, Anthracnose, Bacterial Canker, Grape fan-leaf virus
11	Peach: leaf curl, Apple: Scab, Powdery mildew, Fire blight and Crown gall, Mosaic. Strawberry: Leaf spot
12	Potato: Early and late blight, black scurf, leaf roll, and Mosaic
13	Cucurbits: Downy mildew, powdery mildew, wilt, Angular leaf spot, Mosaic, TOSPO virus
14	Onion: Purple blotch, and Stemphylium blight, Downy mildew, Smut, .Smudge, Erwinia rot
15	Garlic : Neck and bulb rot, and Stemphylium blight, Blemish, Black mould
16	Chilli :Anthracnose and fruit rot, Wilt and leaf curl. Coriander : Stem gall Powdery mildew, Wilt. Turmeric: leaf spot
17	Marigold :Botrytis blight, Alternaria blight, Rose: Dieback, Powdery mildew and Black leaf spot
18	Field visit for the diagnosis of field problems

Suggested Readings

1) Agrios, GN. 2010. *Plant Pathology*. Acad. Press
2) Diseases of Horticultural Crops fruits (1999) By Verma L.R and Sharma R.c,Indus Publishing company, New Delhi
3) Diseases of fruit crops (1986) By V.N.Pathak ,Oxford & IBH publication, New Delh
4) Diseases of fruit crops (1986) By R.S.Singh ,Oxford & IBH publication, New Delhi
5) Diseases of Fruits and vegetables (2007) S.A.M.H. Naqvi,Springer Science & Business Media
6) Diseases of Plantation Crops (2014) By P.Chowdappa, Pratibha Sharma IPS 263pp
7) Diseases of Horticulture Crops and their management ,ICAR e-book for B.Sc.(Agri) &B.Tech (Agri) By TNAU pp172
8) Advances in the diseases of Plantation crops & spices (2004) P.SanthaKumari,International Book Distributing Company , 247 pp
9) Mehrotra RS &Aggarwal A. 2007. *Plant Pathology*. 7th Ed. Tata McGraw Hill Publ Co. Ltd
10) Vegetable Diseases : A Colour full Hand book (2006) by Steven T.Koike ,Peter Gladers and Albert Paulus ,Academic Press, pp448
11) Diseases of Vegetables crops by R.S.Singh (1987) Oxford & IBH publication, New Delhi
12) Plant Diseases.(2008) Singh RS. 20088th Ed. Oxford & IBH. Pub. Co.
13) Diseases of Crops Plants in India (2009) By PHI learning Pvt. Ltd, pp 548
14) Diseases of Vegetable crops (2005) by AlferdSteferud ,Biotech Books ,New Delhi
15) Mehrotra RS &Aggarwal A. 2007. *Plant Pathology*. 7th Ed. Tata McGraw Hill Publ Co. Ltd
16) Diseases of Vegetable Crops ,Diagonosis and Management (2014) Dinesh Singh an P.Chodappa, Today and Tomorrow Printers ,pp734
17) Singh H. 1984. *House-hold and Kitchen Garden Pests - Principles and Practices*.Kalyani Publishers.