

INTRODUCTORY PLANT PATHOLOGY PP 402)

Plant Pathology:

It is derived from a two Greek words i.e. Pathos = disease or Suffering and Logos = to study. It is the branch of agriculture, which deals with the study of plant diseases. The detailed study includes the importance and occurrence, symptoms, cause, etiology, disease cycle, epidemiology and management of diseases.

Pathogen:

Any organism or micro-organism which lives feeds and multiplies on host and also cause diseases.

Parasite:

Any organism or insect which lives feeds and multiplies on host and may cause disease.

Disease:

Any deviation from normal functioning is called disease. OR

“It is a continuous process due to some biotic or non biotic factors which cause abnormality in the physiology, morphology and genetics of plant or its part that reduces quality or quantity or both above economic levels”.

HISTORY OF PLANT PATHOLOGY

The history of Plant Pathology is as old as human civilization. Even when the humans lived as nomads and used to eat only leaves, fruits and seeds, plant diseases took their toll, causing leaves to mildew and blight and fruit and seeds to rot. When man began to grow one or few kinds of food plants, part of the crop was lost to diseases thus delimiting food supplies and hunger was common.

Homer (1000 B.C.) mentioned the therapeutic properties of sulphur on plant diseases. Democritus (470 B.C.) recommended the control of blights by sprinkling plants with the olive grounds left after extraction of the olive oil.

The Greek philosopher Theophrastus (300 B.C.) was the first to study and write about diseases of trees, cereals and legumes.

The role of fungi in plant diseases

Spores of wheat rust fungus were observed with the compound microscope for the first time in England (Leeuwenhoek, 1667).

In Italy, Micheli (1729) described many new genera of fungi. He proposed that fungi arose from their own spores rather than spontaneously, but nobody believed him.

In 1755, Tillet in France showed that he could increase the number of wheat plants developing smut by adding smut dust to the wheat seed before planting and that he could reduce the number of wheat plants developing smut by treating the seed with copper sulphate before planting.

In 1807, Prevost (also in France) repeated Tillet's inoculation and copper sulphate treatment experiments and he also observed with the microscope, the production and germination of smut spores, as well as the inhibition of spore germination with the addition of a drop of copper sulphate.

Late blight of potato caused severe losses in much of Northern Europe in 1840s, but it absolutely destroyed the potato crop in Ireland in 1845 and 1846. Several scientists described various aspects of this disease and of a fungus that seemed to be always associated with the disease. However, DeBary (1861) finally established experimentally that a fungus (*Phytophthora infestans*) was the cause of this plant disease.

Louis Pasteur (1860-1863) finally provided evidence that microorganisms arise only from the pre-existing microorganisms and that fermentation is a biological phenomenon.

Discovery of other pathogens as causes of plant diseases

Nematodes

Needham (1743) observed nematodes within small, abnormally rounded wheat kernels (wheat galls) for the first time. In 1855, a second nematode, the root-knot nematode, was observed in cucumber root

galls. In the next four years two other plant parasitic nematodes, the bulb and stem nematode and the sugar beet cyst nematode, were reported. Cobb described several more nematodes during the 20th century.

Bacteria

Pasteur and Koch (1876) for the first time showed that an animal disease, known as anthrax, was caused by a bacterium. Based on his experiments of this disease, Koch proposed "Koch's Postulates" in 1882, which are used to confirm the pathogenicity. Erwin F. Smith recorded several other bacterial plant diseases. In 1972, Windsor and Black observed a new kind of phloem inhabiting bacterium causing the club leaf disease of clover. The next year, a still different kind of xylem inhabiting bacterium was observed in grape plants infected with a disease (Pierce's disease), which until then was thought to be caused by viruses.

Viruses

In 1886, Mayer reproduced the 'Tobacco Mosaic' disease by injecting juice from infected tobacco plants into healthy tobacco plants. In 1892, Ivanowski showed that whatever caused the Tobacco mosaic disease, could pass through a filter that retains bacteria, so he concluded that the disease was caused by a toxin secreted by bacteria or by unusually small bacteria that passed through the pores of the filter. Beijerinck (1898) finally concluded that tobacco mosaic disease was caused not by a microorganism but by a 'contagious living fluid', which he called a virus.

In 1935, Stanley added ammonium sulphate to tobacco juice extracted from infected tobacco leaves and obtained as a sediment in a flask (a crystalline protein), which when rubbed on healthy tobacco leaves, caused the tobacco mosaic disease. He was awarded a Noble Prize in chemistry in 1946.

Finally, in 1956, Gierrer and Schramm showed that it was the ribonucleic acid, which enabled the viruses to cause infection and not the proteins.

In 1982, an even smaller type of infectious agent was recorded by Prusiner, which he named as 'prion'. This apparently consists of only a small (~55,00 Da) proteinaceous infectious particle, the protein of which is encoded by a chromosomal gene of the host.

Mollicutes

In 1967, Doi and his colleagues in Japan observed mollicutes i.e. wall less mycoplasma like bodies in the phloem of plants exhibiting yellows and witches' broom symptoms. The same year, the same group showed that the mycoplasma like bodies and the symptoms disappeared temporarily when the plants were treated with tetracycline antibiotics.

Control of plant diseases

Chemical control

Ancient Greeks, Homer (1000 B.C.), Democritus (470 B.C.) and Theophrastus (300 B.C.) were among the first ones who gave realistic recommendations for disease control. In 1600s farmers used brine (sodium chloride solution) to control the bunt of wheat. In mid 1700s, sodium chloride was replaced with copper sulphate. In early 1800s, lime-sulphur and aqueous suspensions of sulphur were recommended for the control of mildew of fruit trees. In 1885, Millardet discovered the magical Bordeaux Mixture for the control of downy mildew of grapes in France. This fungicide is still widely used particularly in the developing countries.

In 1913, organic mercurical compounds were introduced for seed treatment. Such chemicals have been in extensive use until 1960s, when these were banned due to their toxicity. In 1934, the first dithiocarbamate fungicide (thiram) was discovered, which led to the development of a series of effective and widely used fungicides including ferbam, zineb and maneb.

In 1965, the first systemic fungicide 'carboxin' was discovered followed by the introduction of several other systemic fungicides, such as benomyl.

Antibiotics, primarily streptomycin, were first used to control the plant diseases in 1950. Soon after, the antibiotic Actidione was shown to be effective against several plant pathogenic fungi. In 1967 tetracycline antibiotics were shown to control plant diseases caused by fastidious bacteria that live in the xylem of their host plants.

In 1954, it was noticed that some strains of phytopathogenic bacteria were resistant to certain antibiotics, and, in 1963, strains of fungal plant pathogens were found, which were resistant to certain protective fungicides. This prompted to development of new strategies for plant disease control.

Biological control

Flemming (1928) reported that *Penicillium notatum* inhibits the growth of other fungi and bacteria. Biological control with antagonistic microorganisms was started in 1963. It involved inoculation of the surface of stumps of freshly cut pines with the spores of *Pneophora gigantea* (a fungus) that protected them from infection of another fungus (*Heterobasidium annosum*). In 1972, control of the crown gall bacterium was obtained by preinoculating the seeds or roots of transplants of stone fruit trees with a related but nonpathogenic bacterium. Similarly, the control of tobacco mosaic virus in tomato was obtained by preinoculating tomato seedlings with a nonpathogenic strain of the virus. Produced by artificially mutating the virus. Similarly, control of citrus tristeza virus and some other viral diseases has been demonstrated by cross protection.

In the late 1980s, genetic engineering was introduced to control plant diseases. In the early 1990s, non-toxic chemicals called plant defense activators were synthesized which activate the plant defense system. The first such compound (CGA-245704) was tested and marketed in 1996.

Importance of Plant Pathology

Plant diseases are of immense importance to human beings by making the difference between a happy life and a life haunted by hunger or even death from starvation. On global basis, of about 35 percent annual crop loss, 12 percent is due to diseases other than nematodes, 11 percent due to nematodes, 7 percent due to insect pests and 3 percent due to weeds (Cramer, 1967)

- The death and migration of millions of people due to late blight of potato which starvation in Ireland in 1845 is just one example.
- Wheat rusts have been appearing in epidemic form from time-to-time in many countries forcing the farmers to change their cropping pattern and food habits.
- Brown leaf spot of rice (*Helminthosporium oryzae*) was one of the main reasons contributing to the Bengal famine in 1942.
- Regular occurrence of coffee rust in Sri Lanka was responsible to reduce the coffee export by 93 percent from 1871 to 1893, which ultimately forced the growers to substitute it with tea. This situation promoted coffee cultivation in Brazil and it became the major coffee producing and exporting country.
- In Pakistan Cotton leaf curl virus become epidemic problem since 1992 and cause a lose of 7 millions bails of cotton annually
- Gram blight and Bacterial leaf blight of rice are also emerging threats for Pakistan economy.
- More recently tree decline (Shisham, Mango, Guava and Citrus) problem also faced by our farmers.

The major objectives of plant pathology are:

- i. To study the causes (biotic & abiotic) of plant diseases.
- ii. To study the mechanism of plant disease development.
- iii. To study the interaction between plant and pathogen in relation to environment and time.
- iv. To develop effective system of management of plant diseases to minimize the losses caused by them.

PLANT VIRUSES

A virus is a super molecular complex that can replicate within appropriate host cells. It is a sub-microscopic, obligate, infective and filterable entity which is nucleo-proteinic in nature. It placed in mesobiotic group of plant pathogens. They consist of DNA or RNA surrounded by a protein capsid or membranous envelope.

Viroid:

It is a smaller, low molecular weight ribo-nucleic acids that can infect plant cells, replicate themselves and cause diseases OR Naked RNA having no protein coat.

Virion:

A complete infectious virus particle is called virion.

Prion:

It is the infectious protein which produced in plants by itself in nature.

Viral Morphology:

Virus consists of nucleic-acid and protein. Protein forms a protective coat around the nucleic acid called capsid. If lipid layer is present in protein coat of virus it is called envelop. Its Morphological shapes are

1. Rod-shaped e.g. TMV
2. Flexible thread e.g. Maize Dwarf Viruses
3. Geminate or Circular e.g. CLCuV
4. Rhabdo-shaped e.g. Cucumber Mosaic Viruses

1-COTTON LEAF CURL

Causal Agent: Cotton Leaf Curl Virus (CLCuV). It is a ss-DNA (circular) virus which belongs to family Geminiviridae and Begomo virus group.

Occurrence and Importance: The disease appeared firstly in Multan in 1967 in severe form and caused heavy losses. Then in 1988 these again appeared in epidemic form. Then the disease caused so much heavy losses in 1993-94 that the farmers avoided the cultivation of cotton and our economy was badly affected. It caused losses of about 28 billion rupees. All of our resistant varieties became susceptible in 2002 to a new strain of virus known as Booray Wala Strain. So far, no variety has been developed which is resistant to this strain.

Symptoms:

1. There is upward and downward curling of leaves of infected plant that starts from the apical portion of plant.
2. Veins of the infected leaves become thickened.
3. Vein thickening is more prominent on the lower side of leaf.
4. Vein thickening may be;
 - a. Small Vein thickening
 - b. Main vein thickening
5. In our conditions small vein thickening is more common and results in bead like modification of veins.
6. In severe conditions, one or more "cup-shaped" or leaf laminar outgrowths called "Enations" appear on the veins on the underside of leaves.
7. Infected leaves show reduction in size.
8. Internodal length of plant is also reduced, so the plant remains stunted with adverse effect on fruit/bolls.

Disease Cycle:

Vector: Bemisia tabaci. (Whitefly)

In nature virus is transmitted by whitefly and it has no transovarial transmission i.e. the virus is not transmissible by any other insect, seed or sap inoculation. Incubation period for the virus varies from 15-30 days.

Alternate Hosts: There are many alternate hosts but most common are; Okra, Eggplant, French bean, Lehi, Watermelon, Cowpea etc.

Epidemiology:

Maximum Temperature: 33-45°C

Minimum Temperature: 25-30°C

Optimum Temperature: 32°C

In our conditions early infection occurs in Late July while the late infection occurs in Late September when temperature becomes favourable for infection.

Management of Disease:

1. Cultivation of resistant varieties e.g. FH-900, FH-1000, VH-148, CIM-446
2. Destruction of the alternate hosts.
3. Timely cultivation and harvest of the spring crops. The spring crops must be harvested one month before the sowing of cotton to avoid insect's attack that act as vectors.
4. Timely sowing of cotton. It should never be sown before 15th of April.
5. Management of vector. Apply such pesticides, which do not produce resistance in the vector.
6. Destruction of stubbles to check the overwintering of the vector.
7. Seed dressing with proper pesticide and then delay the first spray up to the Economic Threshold Level is reached.
8. Balanced fertilization and irrigation.

BANANA BUNCHY TOP

Causal Agent: *Banana Bunchy Top Virus* (BBTV). It is an ss-DNA (isometric) virus which belongs to family *Cercoviridae* and *Nanovirus* group.

Occurrence and Importance: It is the most important disease of banana crop. Except few it occurs in all banana-producing countries of the world. The importance of the disease is more because the infected plants produce no fruit. The virus causes complete destruction of plantations.

Symptoms:

1. Dark green streaks of broken line can be seen on the petioles and veins of new leaves by looking against the light.
2. Margins of leaves become chlorotic.
3. Leaves at the top of plant become narrow, upright, and closer so that the top appears bunchy due to the densely crowded rosette at the apex.
4. Infected plants also show stunted growth.
5. Inflorescence fails to develop or it fails to emerge from the pseudo-stem and resultantly there is no fruit formation.

Disease Cycle

Vector: Black Banana Aphid (*Pentalonia nigronervosa*)

- The virus is also transmitted through propagative materials such as rhizomes, suckers or tissue-cultured meristems.
- In our conditions the main source of primary infection are diseased suckers while the secondary spread takes place by the Aphid in persistent manner.

- Aphid has to feed for minimum 17 hours on the diseased plant to get infective.
- Maximum Vector concentration is at the base of the stem.

Disease Management:

1. Management of this disease depends upon, adopting cultural measures which reduce virus infection, such measures include;
 - a. Quarantine measures to keep the virus out of virus-free area.
 - b. The Use of Virus free propagative material.
 - c. Locating the new plantation away from older infected one.
 - d. Destroying all volunteer banana plants.
 - e. Uprooting the infected plant and burning it to avoid further spread.
2. Control of vector with insecticides has little effect on the spread of this virus.
3. Cultivate resistant varieties e.g. Gross Michel etc.

POTATO LEAF ROLL

Causal Agent: *Potato Leaf Roll Virus* (PLRV). It is an ss-RNA (isometric) virus which belongs to family Luteoviridae and Luteovirus group.

Occurrence and Importance: The disease occurs as world wide. It causes high yield losses and can be the most devastating virus of potato.

Symptoms:

1. The symptoms particularly appear in the lower leaves of affected plants.
2. The leaflets roll upwards;
 - a. Rolling starts from the margins
 - b. Rolling progresses towards the mid-rib until the entire lamina is involved.
3. If the plants are infected late in the season, there are two stages of disease;
 - a. **Primary Stage:** It occurs in the same season when the virus is transmitted to the plants by the vector.
 - b. **Secondary Stage:** If the primary stage infected plants survive to produce tubers, these tubers, when sown in the next season produce diseased seedling at early stage and damage is very much severe.
4. On certain varieties, a reddish bronze discolouration may develop along the tips and edges of rolled leaves.
5. The leaves become leathery and brittle and produce a rattling sound when brushed with hand.
6. Plants are stunted and have stiff upright growth.
7. In some varieties, phloem becomes necrotic and carbohydrates accumulate in the leaves.
8. The number of tubers per plant and their size is greatly reduced.
9. There is also phloem necrosis in tubers.

Disease Cycle:

Vector: *Myzus persicae* (Aphids)

- More than 10 species of Aphids act as the vectors. They transmit the virus in a persistent manner.
- Infected potato seed tubers act as primary source of infection while the secondary spread is by the Aphids.
- Aphid has to feed for several hours (usually 12-14 hours) to acquire the virus and several more

hours (usually 48-72 hours) to infect the plants with the virus.

- Alternate hosts such as;
 - *Datura stramonium* (Datura)
 - *Physalis floridana* (Peelak) etc.

Also play an important role in infection initiation.

- Thermal inactivation point of the virus is 70-80°C.

Management of Disease:

1. Use healthy, certified seed for sowing. Don't plant very small sized tubers since they are more likely to be obtained from diseased plants.
2. Inspect the fields regularly and rogue out the plants showing the initial symptoms and should be burnt or buried deep.
3. Spray the crop with systemic insecticides like Metasystox or Rogor @ 600-750 ml in 500-600 liter of water per hectare at 10-15 days interval to check the insect vectors which transmit the disease.
4. The granular insecticides viz. Thimet 10G @ 15-20kg per hectare can be used along with the fertilizer at the time planting.
5. Use the resistant varieties e.g. i). Cordinal ii). Faisalabad red etc.

TOBACCO MOSAIC DISEASE:

Causal Agent: *Tobacco Mosaic Virus* (TMV). It is a ss-RNA (rod shape) virus which belongs to Tobamovirus group.

Occurrence and Importance: The disease is wide spread in occurrence and is well known in countries where tobacco is grown. As high as 55% reduction in produce has been observed in different countries of the world. The quality of the produce also reduced considerably making the produce sub-standard in the market.

Symptoms:

1. Leaves of infected plants show mild clearing of veins.
2. Clearly visible mottle and mosaic occur in younger leaves later in the season.
3. Light discolouration along the veins of young leaves.
4. Golden yellow mosaic or light green spots are seen on *Nicotiana sylvestris* and tissue necrosis occurs within these spots in some other cultivars.
5. Wrinkling, crinkling, twisting margins and narrowing of leaflets are also observed.
6. There is dwarfing of entire plant.
7. There is marked reduction in growth and substantial reduction in yield.

Disease Cycle:

- The virus is preserved in nature in herbaceous and woody plants.
- Vegetables including tomato, potato and pepper grown indoors, usually transmit the virus from crop to crop.
- Virus overwinters in infected plant stacks, plant debris, in soil, on the surface of the seed, on the seedbed, cloths, natural leaf and manufactured tobacco like cigarette and cigar.
- Contact of virus with wounded tissues of tobacco seedlings or transplants in the field results in initial infection of few plants.
- In the host tissues, after multiplication, the virus systemically spreads to other plant parts through Plasmodesmata and Phloem.

- No insect vector is known to exist, however, grasshoppers, butterflies and caterpillars are known to transmit the virus through mechanical means. Mechanical transmission of TMV is unique among viruses.
- The virus has a wide host range and can infect more than 550 species of flowering plants.

Disease Management:

1. Use clean and healthy seed for nursery.
2. Use uncontaminated soil for seed production.
3. All workers should disinfect their hands at regular intervals. Restrict smoking by the workers.
4. All infected and suspected plants should be removed from the vicinity of nurseries.
5. Grow tobacco in three year crop rotation with maize and wheat, avoiding tomato and pepper crops.
6. Infection by mechanical inoculation can be reduced in limited area by spraying plants with skim milk or butter milk.
7. Leaves from healthy plants should be harvested first then those from infected plants.
8. Grow resistant varieties.
9. Field sanitation practices should be followed.

MOSAIC OF SUGARCANE

Occurrence and Importance: This disease was first recorded in Java in 1892 and from Punjab in 1926-27. The disease caused by sugarcane mosaic virus (SCMV) is commonly referred to "mosaic." Estimated yield losses due to the disease vary greatly depending on the time period and sugarcane growing area involved.

Causal agent: *Sugarcane mosaic potyvirus* I or *Saccharum Virus* I. It is a ss-RNA (filamentous) virus which belongs to family Potyviridae. The host range includes maize, sugarcane, sorghum and many other wild grasses.

Symptoms: The first symptoms appear about 6 weeks after sowing in the form of pale patches in the green tissue of the leaf. Disease leaves show characteristic mottling of chlorotic or light coloured elongated more or less irregular stripes or streaks. The mosaic symptoms are more clearly apparent on the younger basal portion than on older leaves. These symptoms can be seen on the leaf sheath and stalk as well. Highly susceptible varieties show yellow stripes and may be seen even on canes, which may lead to stem splitting. The entire plant becomes stunted and chlorotic.

Disease Cycle: The disease perpetuates, either by the diseased setts or by ratooning the diseased crop. There are three principal modes of spread of SCMV:

- by aphid vectors,
- by infected seed cane and
- by mechanical inoculation.
- Only aphid vectors and infected seed cane are important in the field.
- Mechanical transmission, for the most part, is important only in greenhouse and lab. research.

Disease Management:

1. Use of healthy setts of resistant varieties
2. Roguing of diseased canes
3. Elimination of weed hosts
4. Abstain from ratooning
5. Insect control

TRISTEZA VIRUS DISEASE OF CITRUS (QUICK DECLINE OF CITRUS)

Occurrence and Importance: The first tristeza disaster was reported in the 1930's in Argentina, where 90% of the citrus was planted on sour orange rootstock. CTV is today widespread in Israel, Morocco,

India, China, Japan, Southern California, Florida, Argentina, Brazil, South Africa, Australia and southern Spain, and is moving into previously free, northern Spain. The severity of the strains can change, and the tolerance of certain scion/rootstock combinations to this virus can no longer be taken for granted. Careless introduction of severe strains, such as the orange stem pitting strain of tristeza recently found in Asia and Europe is a constant risk.

Causal agent: *Citrus tristeza closterovirus*. It is an ss-RNA (flexuous rod shape) virus which belongs to family closteroviridae.

Symptoms: Symptoms of virus vary depending upon the virus species involved, root stock and strain of virus present. The most economically important symptom is the quick decline or death of orange, grapefruit and mandarin trees on sour orange rootstock.

- Virus infection in the scion causes dieback of the phloem in the sour orange rootstock below the bud union.
- This results in the rootstock being girdled and therefore starved as the starch reserves are depleted.
- A second symptom of the disease is a 'slow decline' where the trees decline in health over a period of years. This decline is accompanied by a loss in productivity but the tree does not necessarily die.
- A third common symptom is stunting where the virus does not kill the tree, but the tree does not grow.
- Either deep pits are present under depressed areas of the bark, or more severe strains may cause a more general distribution of the stem pitting and vein clearing.

Disease Cycle:

CTV is transmitted by several aphid species with the most effective being the citrus aphid (*Toxoptera citricida*). Due to the aphid introduction, CTV rate of spread has increased dramatically. Additionally, growers have aided in the spread of the virus with the propagation of trees using infected budwood.

Disease Management:

It is hard not to sound alarmist when discussing the potential threat of vector-transmitted viruses such as CTV.

- Two important are:
 - (1) the need to stringently exclude any introduction of severe strains,
 - (2) the need for mild strain protection in the future.
- Strict control by quarantine must be continued in order to prevent the introduction of more severe strains.
- Rootstocks that offer resistance

BACTERIAL DISEASES OF PLANTS

BACTERIA

Bacteria are belonging to prokaryotes. These are extremely minute, rigid, unicellular micro-organisms without definite nucleus i.e. the nuclear material is not bounded by nuclear wall but have a single circular chromosome. And have no chloroplasts and mitochondria.

Structure:

- Surface appendages e.g. flagella which are used for locomotion.
- Surface adherents e.g. capsule.
- Cell-wall which is thin, hard and made up of hemi cellulose and pectin.
- Cytoplasm and cell organelles.

Morphological shapes:

Eubacteria have three basic shapes i.e.

1. Coccus: these are round or Spherical e.g. micro-coccus.

- a - Monococcus: Single coccus is called monococcus.
- b - Diplococcus: if cocci occur in group of two then it is called diplococcus.
- c - Staphylococcus: if large no. of cocci occurs in groups.

2. Bacillus: these are elongated Rod-shaped e.g. bacillus

3. Spirillum: these are Spiral coiled filaments thread-like e.g. *Streptomyces*

Some other important plant pathogens closely resembling with bacteria known as mollicutes or micoplasma like organisms (MLOs) which are pleomorphic organisms that lack a cell wall.

CITRUS CANCKER

Causal organism: *Xanthomonas axonopodis* pv *citri*.

Order: *Pseudomonadales*

Family: *Pseudomonadaceae*

Symptoms:

1. The disease appears on leaves, branches and fruits.
2. The damage is particularly severe in young trees that may destroy many angular shoots, resulting in significantly retarded growth or sometimes death of the affected plants.
3. On young leaves small yellowish water soaked spots develop first on the lower surface and then on the upper surface.
4. In the beginning, these spots are small and scattered.
5. These later turn brown, corky, hard and raised with a yellow halo around them.
6. These spots increase in number and size.
7. The diseased areas become dead and sometimes drop out leaving holes in the leaves.
8. Similar diseased spots (without yellow halo) develop on twigs and fruits.
9. Fruit fall is also common.

Disease Cycle

Primary Infection: Diseased nursery plants act as source of primary inoculum. Diseased plant material is also the potential source of primary inoculum.

Secondary Infection: Secondary infection takes place by splashing rain as well as by contact. Leaf miner and injury by thorns are also potent agents, aiding in the dissemination of the pathogen.

Entrance: Bacteria enter through stomata, and wounds caused by the spines. They multiply in the cortex and remain confined there.

Epidemiology

The disease is favoured by mild (20-30 °C) and wet weather. Presence of free moisture for at least 20 minutes is essential for successful infection.

Control:

1. Use of healthy nursery plants.
2. Removal and burning of diseased plant portions.
3. Windbreaks should be set around the citrus groves.
4. Periodic spraying of insecticide to control citrus leaf miner.
5. Spray of Bordeaux mixture or any other copper compound or streptomycin compounds during the active growth of new shoots (from January to May) and rosin Bordeaux mixture in July and September.

ANGULAR LEAF SPOT OF COTTON

Causal organism: *Xanthomonas axonopodis* pv *malvacearum*

Order: *Pseudomonadales*

Family: *Pseudomonadaceae*

Symptoms

On seedling: Small, Water soaked and circular lesions appear on cotyledons. Afterwards these spots/lesions become enlarge, irregular and brown causing wilting and withering of seedling.

On Leaves of mature plants: Reddish, water soaked lesions appear as angular spots, which are bounded by the vein-lets. Later on they turn dark brown to black and coalesce to make patches.

On stem and branching wood: Dark brown to black patches are formed which cause girdling of branches and stem. It ultimately results in pre-mature falling of leaves. Blackening of stem occurs and hence the disease is also termed as *Black Arm Disease*.

On Fruit/Boll: Dark brown to black, sunken lesions are formed on fruit/boll. Bolls infected when young, may drop prematurely. Older bolls when infected may become distorted and the lint may be discolored. If bacterium gets deep-seated into the fruit/boll, the lint, the fiber and seed all become infected.

Disease Cycle: The bacteria enter the mature seed through the basal end of the chalaza. They over winter in this manner and as contaminants on the surface of the seeds or in the lint attached to it. Volunteer seedlings are the chief source of primary inoculum when cotton is planted after cotton. Wind blown soil, rain and irregular water are the means of dissemination. Insects have little importance.

Epidemiology: High humidity and moderate temperature (28 °C) favours the development of the disease. Primary infection is favoured by 30 °C and secondary infection is better at 35 °C. presence of moisture is very important for the first 48 hours. Dry and hot weather retards disease development.

Control:

1. Use of healthy seed from healthy plants.
2. Delinting seeds with concentrated sulphuric acid then floating the delinted seeds in water and removal of the floating seeds.
3. Disinfections of seeds with 1000-ppm streptomycin sulphate solution overnight.
4. Destruction of diseased plant debris, and
5. Killing of volunteer seedlings.

BACTERIAL BLIGHT OF RICE

Causal organism: *Xanthomonas campestris* pv. *oryzae*

Order: *Pseudomonadales*

Family: *Pseudomonadaceae*

Symptoms: The disease may induce either wilting of leaf blight. Wilting commonly appears sporadically in the field causing serious damage, and is known as “Kresek”. This is due to early systemic infection of from infected seeds and the bacterium brought in contact with the germinating seedlings. It commonly occurs within 3-4 weeks after planting resulting in either the death of whole the plants or wilting of only a few leaves.

Leaf blight is the most predominant phase of the disease. This usually occurs between tillering and heading stages of the crop accompanied by yellowing of leaves, which later on show blighted symptoms.

The earliest symptoms are in the form of appearance of dull greenish water soaked or yellow spots 5-10 mm in length towards the tip or margins, which later start drying. This involves the leaf sheath at later stages. Spots formed on leaf sheath coalesce to form straw brown large lesions. Occasionally, the lesions may extend from tip downwards along the midrib, leaf margins remaining green. Bacterial ooze, pale amber in colour, is found on the affected portions. On drying, these drops make crust and make the leaf surface rough.

Disease Cycle: The bacterium may survive, in addition to volunteer rice plants, on some related grasses and other plants like *Leersia hexandra*, *Cyperus rotundus*, *C. deformis* etc. Seed may also serve as primary source of infection. The secondary infection takes place through wounds and stomata by bacteria disseminated by wind borne rain splashes, irrigation water or rain water coming from infected fields and by contact between diseased and healthy leaves. In the initial stages, there is patchy appearance of the disease in the field due to more rapid vertical spread than horizontal spread. Later due to a large number of such patches, the field appears to be infected uniformly.

Epidemiology: The development of disease is favoured by a temperature above 25°C. Symptoms never appear at temperatures below 20°C. combination of rainy weather, strong winds and temperatures of 22-26°C favour rapid development of disease. Kresek development occurs when there is a combination of of maximum temperature of 30-35°C, minimum temperature of 24-26°C, uniform high humidity of 64-84 percent RH, short sunny days and heavy well distributed rainfall.

Control:

1. Use of disease free seed.
2. Soaking of seeds in 0.025% Agrimycin + 0.05% wettable Ceresan solution for 12 hours and then transferring to hot water at 52-54°C for 30 minutes.
3. Avoiding excessive application of nitrogenous and less application of potash and phosphoric fertilizers.
4. Spraying the crop with Agrimycin (3g/100 litres of water).

SOFT ROT OF POTATO

Symptoms: It causes characteristic soft decay of the fleshy tissues of several crops, e.g., potato tubers, carrot roots, onion bulbs and cabbage that generally have high carbohydrate and nitrogen contents. When soft rot affects, the tissue softens. It becomes watery or slimy and as the rot progresses, the water extrudes, or in a dry atmosphere the water is lost rapidly by evaporation. This is a most common and destructive vegetable disease in storage and transit. Some diseased tissues give an offensive sulfurous odour, which may be due in part to the invasion of secondary bacteria. When the seed tuber of potato are affected in the soil the same types of decay follows. The shoots arising from the seed tuber may be similarly affected if the soil is moist for a long period. However, if the soil moisture is reduced as the disease progresses, the shoot develops a blackened, shriveled carto and its growth is stunted, but is not promptly killed. The leaflets become reddish in colour at the tips and branches more upright than normal and the entire shoot gradually becomes pale and yellowish. This phase of disease is commonly known as balckleg.

Causal organism: *Erwinia carotovora*

- a. Order: *Eubacteriales*
- b. Family: *Enterobacteriaceae*

Disease cycle: Wound caused by harvest bruises, freezing injury and insect activity is the most common avenue of infection; the organism lives over in soil and plant refuse. Abundant moisture at the surface of the tissue, where wounds are present, is essential for invasion; Penetration through lenticels of freshly harvested potatoes has been observed but is less common. Fairly high humidity is essential for the progress of the disease. Progress of disease is checked in a dry atmosphere.

Several maggot fly carry the organism. Adult flies lay their eggs on decayed vegetable material and as the eggs hatch, the larvae feed upon decayed debris, ingesting the bacteria including the soft rot organism. The adults developed from then are internally contaminated. The eggs laid by them are smeared with soft rot organism. The mouthparts and the intestines of the larvae hatched from these eggs become contaminated. As they feed on potato seeds pieces, or fleshy organs of vegetable, they carry the bacteria and infect the plant tissue. The larvae can penetrate any cork par the plant may form. Thus the insect becomes a very important factor of the causal organism.

The bacteria produce an enzyme, which is capable of dissolving the middle lamella. The continuation of this process accounts for the watery condition and for the loss of consistency of the decayed tissue.

Epidemiology: The presence of wounds and high humidity are most essential for the progress of this disease. The disease development takes place between a temperature range of 7-35°C.

Control:

1. Bruising of plant parts should be avoided.
2. Provision should be made for healing of wounds and for drying of surfaces.
3. Storage should be at the lowest temperature at which the particular product can be expected to retain its culinary quality.
4. Potatoes should not be washed but may be passed through a current of warm air before transportation.
5. Plant refuse should be destroyed especially for the control of black leg.
6. Careful selection of seed pieces, and
7. Treating seed pieces with streptomycin sulphate or streptomycin sulphate + tetramycin.

RING ROT OF POTATO

Symptoms: The disease has been found in nature only on potato but has been shown experimentally to be infectious on tomato, brinjal, and other related species of solanaceae. The disease appears first in the field as a diffuse progressive pale yellow chlorosis of leaflets. Marginal necrosis appears next and progresses along with chlorosis. The entire plant shows stunting and may wilt in some cases. The vascular system of stem shows browning in advanced stage. Milky bacterial exudates may come out from cut stem on squeezing. The disease may not show any symptom in tubers before harvest but may appear later in storage. The first sign in tubers is a vascular discolouration, which is light yellow, becoming brown with age. Later bacterial ooze may be evident. The tissues of the vascular ring break down to form macroscopic lysigenous cavities. Dehydration then results in a dry powdery appearance or the invasion of soft rot bacteria may result into a soft, meshy appearance. The vacuolar cavities may extend to pith and cortex and in the latter case may result into cankers.

Causal organism: *Corynebacterium sepedonicum*

Disease Cycle: The organism lives from season to season chiefly in tubers. It may also subsist in the dried slime on farm machinery and tools and stacks. Volunteer plants may also be a source of primary inoculum. The initial invasion is through wounds. The disease becomes established in the large vessels of the vascular system of stem and tubers and progresses systematically. Eventually the bacteria get into xylem parenchyma and contiguous tissue when lysigenous cavities are formed. The bacteria may remain for a long time in tubers without producing visible symptoms.

Epidemiology: No top symptoms develop in infected plants at an air temperature of 16°C. There is a maximum infection in plants growing at a soil temperature of 18°C stunting is severe at 24°C air temperature.

Control:

1. Use of disease free seed.
2. Disinfections of tools etc. with formaldehyde.
3. Use of resistant varieties, e.g. teton.
4. Destroy plant debris.
5. Use certified seed
6. Fumigate stores with etylene oxide 5 lb./1000 ft for 24 hours.
7. Boil bags in CuSO₄ (25:100) for 15 minutes.
8. Disinfect cutting knife tools, etc. with sodium hypochlorite (1:1000), HgCl (1:5000).

PLANT PATHOGENIC NEMATODES

Nematodes are generally multicellular, microscopic, worm-like non segmented animals that live saprophytically in water or soil and as parasites of plants and animals.

These are multi-cellular and transparent in color and belong to animal kingdom. They possess all the physiological systems like animals excepting circulatory and respiratory systems. Feed organ called hypodermal needle or stylet. These have hollow stylet or spear which is used to puncture plant cells, after puncturing the cell they inject saliva into the cells then suck-part of the cell contents and move on within a few seconds. Larvae of nematode are called juvenile. All the nematodes have four juvenile stages in its life. Only the second stage (J2) is infective stage. The estimated losses due to nematodes in life sustaining crops are 11%.

Morphological Characteristics:

- These are small-sized having diameter 300-1000µm. With some up to 4 mm long, 15-35µm wide.
- These are invisible to naked eye because of their small diameter and only can be observed under microscope.
- These are eel-shaped and round in cross section.
- These have smooth and unsegmented body without legs or appendages.
- The females of some species become swollen at maturity, which form pear-shaped bodies. e.g. *Meloidogyne* sp. and *Heterodera* sp.

ROOT-KNOT NEMATODE

- i. *Meloidogyne javanica*
- ii. *M. arenaria*
- iii. *M. incognita*
- iv. *M. hapla* etc.

Family: Heteroderidae

Order: Tylenchida

These are among the most spread plant parasitic nematodes and cause heavy losses, particularly to vegetable, even more than all other diseases combined together.

Symptoms: The plant growth is reduced, unthrifty growth and tendency of plant to wilt during warm days. In case of high populations, young seedlings may be killed over large areas without any trace of gall formation. If the suspected plant is carefully lifted up with a shovel. The plant roots will show galls on them. The galls can be split open and white pearly, typically elongate pyriform females can easily be seen under a microscope.

Crop damage in warm and long season parts of the world is generally much more conspicuous than the cooler and short season areas. Nematode injury is usually associated with plant parasitic fungi, which find their way into the weakened roots. In 1955, Sasser showed that tobacco plants inoculated with Black Shank of Tobacco (*Phytophthora parasitica* var. *nicotianae*) fungus caused mortality 0-30% while other inoculated with nematode and fungus showed 75-100% mortality after 3 weeks of planting.

Life cycle: Elongate ovate eggs are laid in a gelatinous egg sac. On an average female lays 200-500 eggs. This nematode passes through 5 stages. 1st and 2nd stage develop within the egg. 2nd stage larvae come out. They are slender, worm like and move in soil in search of the host. The larvae may find their way in the mother gall or somewhere else. In a single gall, all the stages of the nematode may be found. Larvae prefer root tips for penetration. They are unable to penetrate into large roots. Females remain near the cortex while larvae penetrate to near about the central cylinder.

After the establishment of larvae giant cells develop Female larvae become obese spindle shaped. 3rd and 4th stage cuticles are also produced without any moult. The larvae at this time are flask shaped. When gonads develop, final moult takes place and 2nd, 3rd, and 4th stage cuticles are shed simultaneously. After 3rd stage, male remains active, slender, with small spear, and bluntly tail without bursa. Reproduction can take place without males and a single larva may produce as many as seven generations.

Control:

1. Crop rotation: Not successful to a great extent. In case of cotton and peanut nematodes, a three years rotation of alfalfa has proved useful. Anyhow rotation of alfalfa, tobacco, corn, and cereals will produce satisfactory yield.
2. Fallowing of land: Dry fallowing with 2-3 deep ploughing during hotter summer months in long season areas, is an excellent measure. winter vegetable should be short season.
3. Soil fumigation: DD mixture @ 7 lbs/10,000 sq.ft. EDB+DD mixture @ 8 lbs/10,000 sq.ft. VAPAN @ 3 lbs/100 sq. ft. Nemagon (DBCP) 10 gl/acre; etc.
4. Root residues should be collected and removed or allow them to decay before the application is made, other wise, a large number of population will escape during fumigation.

CITRUS NEMATODE

Causal Organism: *Tylenclulus semipenetrans*,

Family: Tylenchulidae

Order: Tylenchida

This nematode is a serious pathogen of citrus orchards and is invariably present in all the plantations with those trees, which are in various stages of decline and dieback. The nematode causes a disease known as slow decline of citrus which is worldwide in distribution in all the citrus growing areas of the world including Pakistan, India Brazil, S. Africa, Egypt, Lebanon, Iraq, Sri-Lanka, Thailand, China, Australia, Philippines and USA. Losses are sometime very high reaching 40-50%.

Symptoms: Plantings at early stage produced excellent yield for 10-15 years before injury becomes

evident. If the populations are low at initial stage no prominent symptoms appear until population reaching to damaging level. Not until there is competition for fertility and moisture between roots & tress then the nematode overtake root production and begin to check top growth. The first indication of injury is a reduction in terminal growth accompanied by reduced vigor, yellowing and dying of leaves and twigs. The trees are not killed but maintain life on a limited scale and produce a reduced crop of inferior fruit. Young nursery stock if planted on infested land begin to show yellow leaves defoliated twigs a small fruit within 2-3 years and may fail to survive. Roots of infested plants show dirty appearance due to females' gelatinous secretions at the time of reproduction and egg laying.

Life cycle: Females are found on thick, started rootlets to which a layer of soil particle is clinging. These particles are held in place by gelatinous mucus secreted by the female, which protects the eggs. Females can be seen clinging to the roots with head & neck buried in the cortical layer. Second stage larvae required 14 days to locate & feed on epidermal root cells. The third and 4th stages end in shorter immature female. Later the last cuticle is moulted and the young female emerges. Within a week young females had penetrated to the pericycle of the root and developed to fully enlarged female. Second stage male usually are formed before emerging from egg mass, after 3rd moult the fourth stage male increase in size, male sex organs develop. From this stage the adult male emerges. The complete life cycle from egg to egg required 6-8 weeks.

Control:

1. Exclusion: i. Avoid run off water from infested to non-infested fields. ii. Contaminated equipment should not be used. iii. Use nematode tree nursery.
2. Crop management: Correcting such factors like poor drainage, drought stress, excessive salinity, cold damage. Proper management of orchard, weeding and hoeing must be carried out.
3. Direct management: i. Use of resistant root stock Troyer & Carrizo citrange. ii. Preplant nematicides: Fumigants methyle bromide, metan sodium, 1,3 dichlosopropane and Dibromochloropropane (DBSP) 5-10 gallons/Acre with irrigation water. Iii. Post plant nematicides: Aldicarb, femaniphos, oxanyl are translocated systemically.

CYST FORMING NEMATODE

Causal Organism: Sugarbeet cyst nematode: *Heterodera schachtii*,

Potato cyst nematode: *Globodera rostochiensis*

Family: Heteroderidae

Order: Tylenchida

Potato cyst nematodes were first found in 1881 in Germany and in 1941 in USA. Because of its yellow or golden phase during development it was named as golden nematode of potato.

Symptoms: Potato cyst nematodes do not cause distinctive above ground symptoms that are of diagnostic value. Because the nematodes attack root, infected plant exhibit typical symptoms of water or mineral stress. Plants show sickly yellow appearance the plants may die prematurely. High nematode densities cause stunting and in some cases stop growth completely. Close examination of the roots of infected plants at the time of flowering reveals minute pearly white bodies, which are immature females that have erupted the root epidermis. When the females mature they turn dark brown and most of them are dislodged from the roots when plants are lifted for examination. First sign of nematode attack appears in a field with patchy area of poor growth, which year after year enlarges and spread by cultivation to entire field. Yield of infested plants is drastically reduced in terms of number and size of potato. Damage is caused by disruption of the conducting tissues of the roots. This disruption is brought about by formation of feeding sites (syncytia) of phloem parenchyma through cell wall dissolution.

Life cycle: During spring, 2nd stage juveniles emerge from the eggs after stimulation by substances emanating from host plant roots. They invade host root near the tip. Eventually they come to rest with then heads towards the stele and begin feeding on pericycle or cortical and endodermis cells. By injecting saliva, there is breakdown of the cell wall, which forms large "Syncytia transfer cell" with dense granular cytoplasm. The nematode continues feeding until its development is complete (a period of 2-3 months for completion the life cycle depending upon temperature). The juvenile after becoming

sedentary undergoes a series of three months through the 3rd and 4th juvenile stages to the adult. Sex is distinguishable at the 3rd juvenile stage and once sex is determined, it is irreversible.

Fourth stage males remain coiled within the sac-like third stage cuticle and emerge from the root after the final molt. They are vermiform and live freely in the soil. The fourth stage females enlarge as their gonads increase in size eventually rupturing the root cortex exposing spherical body outside the root with only head and neck embedded in the root. The female dies later on and cuticle tends to form a tough leathery cyst protecting eggs and larvae within its body "the cyst". When potatoes are harvested, the cysts are detached from the roots and become free in the soil, where they overwinter. Only one generation is produced each year due to low temperature.

Control Measures:

1. Crop rotation: Nematode has limited host range (potato tomato egg-plant and wild solanum) other crops can be used for rotation of 6-7 years, then potato can be grown profitably.
2. Chemical Treatment: Treating the soil with chemicals fumigants 1,3 dichloropropane (Telone) Methyl bromide, vapam vorlex, dazomet. Oximecarbarnates: Aldicarb and oxangl (3-5 kg/hect). Phenamiphos and ethoprop (organophosphates) are also effective.

ABITOC DISEASES

Tirak or Bad Opening of Bolls of Cotton

The main cause of this disease is nitrogen deficiency, particularly during the flowering and boll formation phases of plant growth. Nitrogen is a constituent of such compounds as proteins, amino acids, enzymes, hormones, chlorophyll and vitamins. Sugar accumulates in the tissues because of deficiency of nitrogen and affects respiration.

Apart from nitrogen some other factors are also responsible to further aggravate the situation. These include poor and sandy soil texture, accumulation of exchangeable sodium salts in the soil in concentration of more than 0.2% in the root zone. A high sodium: calcium ration has also been found to enhance the disease.

Symptoms: There is reddening of leaves in August-September. Premature or incomplete opening of the bolls. Accumulation of tannins in the leaves during flowering stage due to nitrogen deficiency.

Khaira Disease of Rice (Zn Deficiency)

Symptoms: The disease usually appears 10-15 days after transplanting, a time, which coincides with the peak period of decomposition of last year's stubbles in the flooded field. Leaves of diseased plants show chlorosis at the base. Large number of small brown or bronze spots appears on leaf lamina. These spots coalesce to form bigger spots and ultimately the entire leaf turns bronze coloured and dies. The diseased plant remains stunted. Root growth is also restricted and usually the main roots turn brown. Fine roots are destroyed. In severe cases plants fail to grow further and produce ears with few grains.

Control: The disease may be controlled with two foliar sprays of a mixture of 2 Kg of ZnSO₄ and one Kg of slaked lime in 400 liters of water/acre. The first spray should be done as soon as the symptoms of the disease appear followed by a second spray 10 days later.

The disease can be checked by spreading 5 Kg of ZnSO₄ at the time of land preparation.

INTRODUCTION TO PHANEROGAMIC PARASITES

The most important phanerogamic parasites are as under:

1. Stem Parasites
 - i. Dodder of *Cuscuta* spp. (holo parasite)
 - ii. Mistletoes or *Dendrophthoe* spp. (partial parasite)
2. Root Parasites
 - i. *Orobanch* spp. (holo parasite)
 - ii. *Striga* spp. (partial parasite)

DODDER

These are achlorophyllous, leafless, twining parasites, which attach their threads like stem to the stems or other parts of host plants. When their stem comes in contact with the host, haustoria (minute root like organs) penetrate the host cortex reaching up to vascular bundles. Haustoria serve as an anchor as well as food organs. They commonly attack clover, berseem, flax, many oilseed crops, ornamentals and hedge plants.

Dodder perpetuates through seeds, which fall on the ground and remain dormant until a favourable season returns. Portions of stems are also resistant to adverse weather. Its introduction and further spread may be due to:

1. Seeds of Dodder often go un-noticed in seeds of crops like berseem.
2. As seeds and stem pieces moving through irrigation water.
3. As stem pieces present on the dry straw from infested fields.
4. As seeds in the manure.
5. As stem pieces transported by cattle, birds, strong winds and farm implements.

Control

1. Crop seed should be free from dodder
2. Dodder infested cattle's fodder should not be used.
3. Grazing animals should not be allowed to move through infested fields.
4. Badly infested crop should be burnt before the parasite produces seeds.
5. Field should be left fallow after selected eradication measures have been taken.
6. Five years rotation beginning with a non-host crop.
7. If the dodder is present in patches, application of contact herbicides such as diesel oil fortified with DNBP (4,6-dinitro-o-butylphenol), PCP (pentachlorophenol) or 2,4-D.
8. Herbicide Glyphosate is effective even after dodder has established connection with the host.

MISTLETOES

These are common partial parasites of tree trunks and branches. Their leathery and evergreen leaves possess chlorophyll and synthesize carbohydrates but for water and mineral nutrients, these are dependent on host, which is obtained through haustoria. The continuous drain on nutrients by the parasite deprives the host of what is required for its own growth.

The seeds of parasite are dispersed by birds and some other animals. The birds are attracted by brilliant coloured fruits and by eating these fruits; seeds are carried in their droppings. Whenever, the seeds are deposited at the junction of branches, these germinate and give rise to haustoria, thus ensuring the establishment of parasite.

Control

1. Top off the infected branches by cutting them sufficiently low so that the haustorial system is eradicated.
2. Injection CuSO_4 and 2,4-D into affected branches.
3. Spray of diesel oil emulsion in soap water.

OROBANCHE

It is holoparasite affecting tobacco, brinjal, tomato, cabbage, cauliflower, turnips and many other solanaceous and cruciferous crop plants. It usually occurs in patches in the field and affected plants look stunted.

The parasite survives through seeds, which may survive for more than 10 years. These germinate only when roots of certain plants (host or non-host) grow near them. The parasitic roots (haustoria) penetrate into the roots of the host and draw nourishment from there. A large number of parasitic stems may be seen breaking the soil around the host plants. As a result of drain on the food supply, the growth of host plant is checked; it remains stunted and may even die.

Control

1. It would be the best if the parasite is destroyed before seed formation.

2. Long crop rotations, if possible, may be practiced.
3. Spraying the soil with 25% CuSO₄ solution.
4. Using chilli, mothbean, sorghum or cowpeas as trap crops.

STRIGA (WITCHWEED)

It is well known partial parasite of sugarcane, cereals, maize and millets. The leaves of Striga possess chlorophyll and thus they depend on their host only for water and other mineral nutrients. Many of Striga plants are fairly high (9" to 2'), and usually develop in clusters around the host plant. Striga can be found on light as well as heavy soil both in rabi and kharif seasons. The growth and flowering is however, influenced by temperature in different soil types.

The seeds of Striga remain viable for quite a long time. Short distance of seeds takes place through rain and irrigation water while floods and wind are the chief agents of long distance dissemination. Seed germinates when stimulated by root exudates of the specific hosts. After germination, it grows below the soil surface for about 4-8 weeks and produces underground stems and roots. The roots of parasite produce haustoria, which penetrate the host roots from which the parasite draws water and nutrients, eventually draining and destroying the host.

Control

1. Weeding and interculture during early stages of parasitic growth. Within two months 4-6 weedings may be needed.
2. Keeping the field flooded for sometime and then draining out the water also helps in the control of the parasite.
3. Sowing "false host crops" such as cotton, soybean and groundnut, which will induce Striga seed germination but do not support the growth of the parasite. This should be augmented with the use of weedicides to keep away the weeds, which could serve as alternate host of the parasite.
4. Soaking the upper 4-6 layer of soil with 2-3% CuSO₄ solution.
5. Spray of Fernoxone (80% sodium salt of 2,4-D) @ 450 g in 500 litres of water.
6. Application of tetrachloro-dimethyl phenoxyacetic acid (28 Kg/ha).

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