# PAPER – 1

# MICROBIOLOGY, MYCOLOGY AND PLANT PATHOLOGY

## UNIT - V

# Plant pathology

**Plant pathology** (also **phytopathology**) is the scientific study of diseases in plants caused by pathogens (infectious organisms) and environmental conditions (physiological factors). Organisms that cause infectious disease include fungi, oomycetes, bacteria, viruses, viroids, virus-like organisms, phytoplasmas, protozoa, nematodes and parasitic plants.

## Plant pathogens

### Fungi

Most phytopathogenic fungi belong to the Ascomycetes and the Basidiomycetes.

### Ascomycetes

- *Fusarium* spp. (Fusarium wilt disease)
- *Thielaviopsis* spp. (canker rot, black root rot, *Thielaviopsis* root rot)
- *Verticillium* spp.
- *Magnaporthe grisea* (rice blast)
- *Sclerotinia sclerotiorum* (cottony rot)

### Basidiomycetes

- *Ustilago* spp. (smuts) smut of barley
- *Rhizoctonia* spp.
- *Phakospora pachyrhizi* (soybean rust)
- *Puccinia* spp. (severe rusts of cereals and grasses)
- *Armillaria* spp. (honey fungus species, virulent pathogens of trees)

## Fungus-like organisms

### Oomycetes

The oomycetes are fungus-like organisms. They include some of the most destructive plant pathogens including the genus *Phytophthora*, which includes the causal agents of potato late blightand sudden oak death. Particular species of oomycetes are responsible for root rot.

### Significant oomycete plant pathogens include:

- *Pythium* spp.
- *Phytophthora* spp., including the potato blight of the Great Irish Famine (1845–1849)

### Phytomyxea

Some slime molds in Phytomyxea cause important diseases, including club root in cabbage and its relatives and powdery scab in potatoes. These are caused by species of *Plasmodiophora* and *Spongospora*, respectively.

**Bacteria**

Most plant pathogenic bacteria are rod-shaped (bacilli). In order to be able to colonize the plant they have specific pathogenicity factors. Five main types of bacterial pathogenicity factors are known: uses of cell wall–degrading enzymes, toxins, effector proteins, phytohormones and exopolysaccharides.

Pathogens such as *Erwinia* species use cell wall–degrading enzymes to cause soft rot. *Agrobacterium* species change the level of auxins to cause tumours with phytohormones. Exopolysaccharides are produced by bacteria and block xylem vessels, often leading to the death of the plant.

**Significant bacterial plant pathogens:**

- *Burkholderia*[
- *Proteobacteria*
  - *Xanthomonas* spp.
  - *Pseudomonas* spp.
- *Pseudomonas syringae* pv. tomato

**Phytoplasmas and spiroplasmas**

*Phytoplasma* and *Spiroplasma* are genera of bacteria that lack cell walls and are related to the mycoplasmas, which are human pathogens. Together they are referred to as the mollicutes. They are normally transmitted by sap-sucking insects, being transferred into the plant's phloem where it reproduces.

**Viruses, viroids and virus-like organisms**

Plant viruses are generally transmitted from plant to plant by a vector, but mechanical and seed transmission also occur. Vector transmission is often by an insect (for example, aphids), but some fungi, nematodes, and protozoa have been shown to be viral vectors. In many cases, the insect and virus are specific for virus transmission such as the beet leafhopper that transmits the curly top virus causing disease in several crop plants.

**Nematodes**

Root knot nematodes have quite a large host range, they parasitize plant root systems and thus directly affect the uptake of water and nutrients needed for normal plant growth and reproduction, whereas cyst nematodes tend to be able to infect only a few species.

**Protozoa and algae**

There are a few examples of plant diseases caused by protozoa (e.g., *Phytomonas*, a kinetoplastid). They are transmitted as durable zoospores that may be able to survive in a resting state in the soil for many years.

**Parasitic plants**

Parasitic plants such as broomrape, mistletoe and dodder are included in the study of phytopathology. Dodder, for example, can be a conduit for the transmission of viruses or virus-like agents from a host plant to a plant that is not typically a host, or for an agent that is not graft-transmissible.

**Common pathogenic infection methods**

- **Cell wall-degrading enzymes**: These are used to break down the plant cell wall in order to release the nutrients inside.
- **Toxins**: These can be non-host-specific, which damage all plants, or host-specific, which cause damage only on a host plant.
- **Effector proteins**: These can be secreted into the extracellular environment or directly into the host cell, often via the Type three secretion system. Some effectors are known to suppress host defense processes. This can include: reducing the plants internal signaling mechanisms or reduction of phytochemicals production.Bacteria, fungus and oomycetes are known for this function.

**Spores**: Spores of phytopathogenic fungi can be a source of infection on host plants. Spores first adhere to the cuticular layer on leaves and stems of host plant. In order for this to happen the infectious spore must be transported from the pathogen source, this occurs via wind, water, and vectors such as insects and humans. When favourable conditions are present, the spore will produce a modified hyphae called a germ tube. This germ tube later forms a bulge called an appressorium, which forms melanized cell walls to build up tugour pressure. Based on the pathogens life cycle, this haustorium can invade and feed neighbouring cells intracellularly or exist intercellulary within a host.

**Some of the examples of plant disease**

- **Blight**
- **Citrus Canker**
- **Rust**
- **Smut**
- **Tobacco mosaic**
- **Yellow vein mosaic**

# Plant diseases caused by plant pathogen

Infectious plant diseases are caused by bacteria, fungi, or viruses and can range in severity from mild leaf or fruit damage to death..

**Bacterial**

- aster yellows
- bacterial wilt
- blight
    - fire blight
    - rice bacterial blight
- canker
- crown gall
- rot
    - basal rot
- scab

**Fungal**

- anthracnose
- black knot
- blight
    - chestnut blight
    - late blight
- canker
- clubroot
- damping-off
- Dutch elm disease
- ergot
- Fusarium wilt
    - Panama disease
- leaf blister
- mildew
    - downy mildew
    - powdery mildew
- oak wilt
- rot
    - basal rot
    - gray mold rot
    - heart rot

- rust
    - blister rust
    - cedar-apple rust
    - coffee rust
- scab
    - apple scab
- smut
    - bunt
    - corn smut
- snow mold
- sooty mold
- *Verticillium* wilt

**Viral**

- curly top
- mosaic
- psorosis
- spotted wilt

# Defense Mechanism in Plants

***I. Physical Defense***

In plants some structures are already present to defend the attack while in others, the structures to defend the host develops after the infection. In this way, structural defense can be characterised as (A) Preexisting defense structures and (B) Defense structures developed after the attack of the pathogen.

**(A) Preexisting Defense Structures:**

**(i) Cuticular Wax:**

Deposition of wax on the cuticular surface is thought to play a defensive role by forming a hydrophobic surface where water is repelled. As a result, the pathogen does not get sufficient water to germinate or multiply. In addition, a negative charge usually develops on the leaf surface due to the presence of fatty acids – the main component of cuticle. The negative charge prevents/reduces the chance of infection by many pathogens.

**(ii) Cuticle Thickness:**

The thickness of cuticle is most important for those which try to enter the host through the leaf surface. The cuticle thickness obstructs the path of pathogen. In addition, a thick cuticle checks the exit of the pathogen from inside the host, thus reducing the secondary infection.

**(iii) Structure of Epidermal Cell Walls:**

Tough and thick outer walls of epidermal cells may directly prevent the entry of the pathogen completely or make the entry difficult. The presence or absence of lignin and silicic acid in the cell walls may show variation in resistance to penetration of the pathogen. Most outer walls of epidermal cells of rice plants are lignified and are seldom penetrated by blast disease of rice pathogen. In resistant varieties of potato tubers (resistant to *Pythium debaryanum*) the epidermal cells contain higher fibre content than the susceptible ones.

**(iv) Structure of Natural openings:**

Structure of natural openings like stomata lenticels etc. also decide the fate of the entry of the pathogen. In *Szincum* variety of citrus, the stomata are small and possess very narrow openings surrounded by broad lipped raised structures which prevent entry of water drops containing citrus canker bacterium. In the same way, the size and internal structures of lenticels may play a defensive role against the pathogens. Varieties having small lenticels in the apple fruits prevent the entry of the pathogen while those having large openings easily allow the pathogen to enter. Nectaries provide openings in the epidermis and may play a defensive role due to high osmotic concentration of the nectar. In resistant varieties of apple, presence of abundant hairs in the nectaries acts as a defense mechanism while susceptible varieties are devoid of abundant hairs.

**Internal Defense Structures:**

There are many preexisting internal defense structures inside the plant that prevent the entry of pathogen beyond these structures. In some plants, cell walls of certain tissues become thick and tough due to environmental conditions and this makes the advance of the pathogen quite

difficult. In case of stems of cereal crops, vascular bundles or extended areas of sclerenchyma cells checks the progress of rust pathogen. Leaf veins effectively obstruct the spread of pathogen like the angular leaf spot pathogen.

**(B) Defense Structures Developed after the Attack of the Pathogen:**

After the pathogen has successfully managed to overcome the preexisting defense mechanisms of the host, it invades the cells and tissues of the host. In order to check the further invasion by the pathogen, the host plants develop some structures/mechanisms which may be defense reactions in the cytoplasm, cell wall defense structures, defense structures developed by the tissues and ultimately the death of the invaded cell i.e. necrosis.

**(i) Defense Reactions in the Cytoplasm:**

The cytoplasm of the invaded cell surrounds the hyphae of the pathogen and the nucleus of the host cell gets stretched to break into two. In some host cells, the cytoplasm and the nucleus of the infected cells enlarge. The cytoplasm becomes granular and dense and develops granular particles. These result in the disintegration of the pathogen mycelium and thus the invasion stops. Such cytoplasmic defence mechanisms can be seen in weak pathogens like Annillaria and some mycorrhizal fungi.

**(ii) Cell Wall Defense Structures:**

Cell wall defense structures are of limited help to the host. These include morphological changes in the cell wall of the host.

**Three types of cell wall defense structures are generally observed:**

(i) Cell walls thicken in response to the pathogen by producing a cellulose material, thus preventing the entry of the pathogen

(ii) The outer layer of cell walls of the parenchyma cells in contact with invading bacterial cells produce an amorphous fibrillar material that traps the bacteria thus preventing them to multiply and

(iii) Callose papillae get deposited on the inner layers of the cell walls due to invasion by fungal pathogens.

**(iii) Defense Structures Developed by the Tissues:**

**The following four developments take place in the tissues after penetration:**

**(a) Gum Deposition:**

Plants produce a variety of gummy substances around lesions or spots as a result of infection. These gummy substances inhibit the progress of the pathogen. The gummy substances are commonly produced in stone fruits.

**(b) Abcission Layers:**

Abscission layers are usually formed to separate the ripe fruits and old leaves from the plant. But in some stone fruit trees, these layers develop in their young leaves in response to infection by several fungi, bacteria or viruses. An abscission layer is a gap formed between two circular layers of cells surrounding the point of infection.

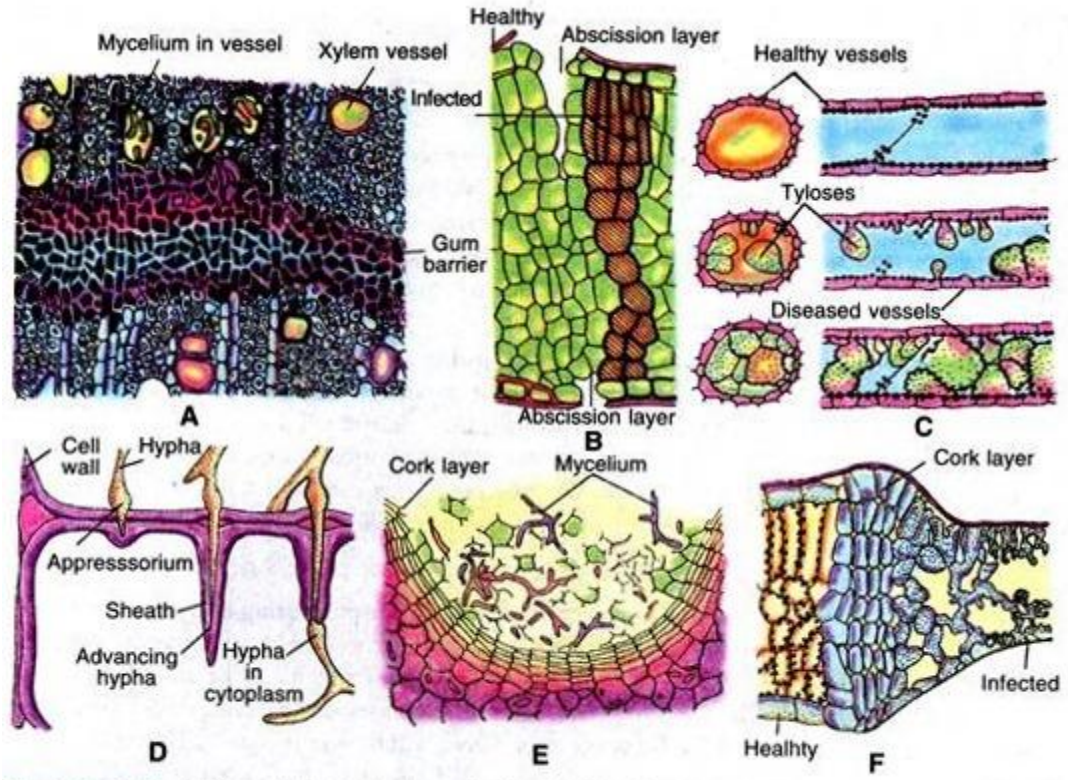



Fig. 21.1. (A-F). Different defence structures developed after infection : (A) Gum deposition in cells ; (B) Abscission layer ; (C) Tyloses ; (D) Formation of sheath around developing hyphae (E, F)./ Development of cork layer in tuber (E) and leaf (F).

**(C) Tyloses:**

Tyloses are out growths of protoplasts of adjacent live parenchyma cells protruding into xylem vessels through pits under stress or in response to attack by the vascular pathogens. Their development blocks the Xylem vessels, obstructing the flow of water and resulting in the development of wilt symptoms.

**(D) Formation of Layers:**

Some pathogens like certain bacteria, some fungi and even some viruses and nematodes stimulate the host to form multilayered cork cells in response to infection, these develop as a result of stimulation of host cells by substances secreted by thus, pathogen. These layers inhibit the further invasion by the pathogen and also block the flow of toxic substances secreted by the pathogen. Cork layers also stop the flow of nutrients of the host thus also depriving the pathogen of the nutrients. Examples of cork layer formation as a result of infection are: soft not of potato

caused by *Rhizopus* sp., potato tuber disease caused by *Rhizoctonia* sp., Scab of potato caused by Streptomyces scabies and necrotic lesions on tobacco caused by tobacco mosaic virus.

**IV. Necrosis or Hypersensitive Type of Defense:**

Necrosis or hypersensitive type of defense is another defense mechanism adopted by some pathogens like *Synchytrium endobioticum* causing wart disease of potato, *Phytophthora infestans* causing late blight disease of potato and *Pyricularia oryzae* causing blast of rice etc. In such diseases, the host nucleus moves toward the pathogen when the latter comes in contact with the protoplasm of the host. The nucleus soon disintegrates into brown granules which first accumulate around the pathogen, later dispersing throughout the host cytoplasm. Soon the cell membrane swells and finally the cell bursts and dies. These cause the pathogen nucleus to disintegrate into a homogenous mass and its cytoplasm dense. As a result, the pathogen fails to grow beyond the necrotic or dead cells and the further growth of the pathogen is stopped.

***II. Biochemical Defense:***

Although structural defense mechanisms do prevent the attack of the pathogen, the defense mechanism also includes the chemical substances produced in the plant cells before or after the infection.

**(A) Preexisting Biochemical Defense:**

**(i) Inhibitors Released in the Prepenetration Stage:**

Plant generally exudes organic substance through above ground parts (phyllosphere) and roots (rhizosphere). Some of the compounds released by some plants are known to have an inhibitory effect on certain pathogens during the prepenetration stage. For example fungistatic chemicals released by tomato and sugar beet prevent the germination of *Botrytis* and *Cercospora.* Presence of phenolics like protocatechuic acid and catechol in scales of red onion variety inhibit the germination of conidia of *Colletotrichum circinans* on the surface of red onion.Inhibitors present in high concentrations in the plant cells also play an important role in defense of plants. Presence of several phenolics, tannins and some fatty acid like compounds such as dienes in cells of young fruits, leaves or seeds afford them resistance to *Botrytis.*

The tubers of resistance vars of potato against potato scab disease contain higher concentrations of chlorogenic acid around the lenticels and tubers than the susceptible vars. Several other compounds like saponin tomatin in tomato and avinacin in oats have antifungal activity. Some enzymes like glucanases and chitinases present in cells of some plants may break down the cell wall components of pathogens.

(ii) Lack of nutrients essential for the pathogen is another preexisting biochemical defense mechanism. Plant varieties or species which do not produce any of the chemicals essential for the growth of pathogen may act as resistant variety.

For example, a substance present in seedling varieties susceptible to Rhizoctonia initiates hyphae cushion formation from which the fungus sends penetration hyphae inside the host plants. When this substance is not present, hyphal cushions are not formed and the infection does not occur.

**(iii) Absence of Common Antigen in Host plant:**

It is now clear that the presence of a common protein (antigen) in both the pathogen and host determines diseases occurrence in the host. But if the antigen is present in the host and absent in the host or vice-versa, it makes the host resistant to the pathogen.

For example, varieties of linseed which have an antigen common to their pathogen are susceptible to the disease rust of linseed caused by Melampsora lini.

In contrast, the absence of antigen in linseed varieties but occurring in the pathogen are resistant to the pathogen. Another example is leaf spot disease of cotton caused by Xanthomonas campestris pv. malvacearum.

**(B) Post-Infection-Biochemical Defense Mechanism:**

In order to sight infections caused by pathogens or injuries caused by any other means, the plant cells and tissues produce by synthesis many substances (chemicals) which inhibit the growth of causal organism. These substances are generally produced around the site of infection or injury with the main aim at overcoming the problem.

**Some such important chemicals are described below:**

**(i) Phenolic Compounds:**

These are the most common compounds produced by plants in response to injury or infection. The synthesis of phenolic compounds takes place either through "acetic acid pathway" or "Shikimic acid pathway".

Some common phenolic compounds toxic to pathogens are chlorgenic acid, caffeic acid and ferulic acid. These phenolic compounds are produced at a much faster rate in resistant varieties than in susceptible varieties.

Probably that the combined effect of all phenolics present is responsible for inhibiting the growth of the infection.

**(ii) Phytoalexins:**

Phytoalexins are toxic antimicrobial substances synthesized 'de novo' in the plants in response to injury, infectious agents or their products and physiological stimuli. The term phytoalexin was first used by the two phytopathologists Muller and Borger (1940) for fungi static compounds produced by plants in response to mechanical or chemical injury or infection.

All phytoalexins are lipophilic compounds and were first detected after a study of late blight of potato caused by Phytophthora infestans. Phytoalexins are believed to be synthesized in living cells but surprisingly necrosis follows very quickly.

Phytoalexins are considered to stop the growth of pathogens by altering the plasma membrane and inhibiting the oxidative phosphorylation.

Phytoalexins have been identified in a wide variety of species of plants such as Soyabean, Potato, sweet potato, barley, carrot, cotton etc. are being investigated. Some common phytoalexins are Ipomeamarone, Orchinol, Pistatin, Phaseolin, Medicarpin, Rishitin, Isocoumarin, 'Gossypol' Cicerin, Glyceolin, Capisidiol etc.

**(iii) Substances Produced in Host to Resist Enzymes Produced by Pathogen:**

Some hosts produce chemicals which neutralise the enzymes produced by pathogen, thus defending the host. Therefore these substances help plants to defend themselves from the attack of the pathogen. In bean plants, infection with *Rhizoctonia solani* causes necrosis. In resistant bean varieties, the entry of pathogen causes the separation of methyl group from methylated pectic substances and forms polyvalent cations of pectic salts which contain calcium. The calcium ions accumulate in infected as well as neighbouring healthy tissues and because of the calcium accumulation, the pathogen fails to disintegrate middle lamella by its polygalacturonase enzymes. These are known to dissolve the middle lamella of healthy tissue in susceptible varieties.

**(iv) Detoxification of Pathogen Toxins and Enzymes:**

In some cases, the plants produce chemicals which deactivate the toxins produced by the pathogens. For example, *Pyricularia oryzae* which causes blast disease of rice produces Picolinic acid and pyricularin as toxins. Although resistant varieties convert these toxins into N-methyl picolininic acid pyrecularin into other compounds, the susceptible varieties do get affected by these toxins. Similarly in case of cotton and tomato wilts, the toxin fusaric acid produced by the pathogen gets converted into non-toxic N-methyl-fusaric acid amide in resistant varieties.

As in case of detoxification of toxins, the toxic enzymes produced by the pathogen is deactivated by phenolic compounds or their oxidation products. Some varieties of cider apple are resistant to brown not disease caused by *Sclereotinia fructigena.* It may be because of the resistant varieties producing pheolic oxidation products which inactivate the pectinolytic enzymes produced by the pathogen.

**(v) Biochemical Alterations:**

It has been observed that infection of the host by the pathogen brings about biochemical changes in the host which may prove toxic to the pathogenic microorganisms and cause resistance to the pathogen. Production of certain new enzymes and other compounds are synthesized and accumulated in higher concentration. This may also add to the resistance of the plant by being toxic to pathogenic microorganisms.

**C. Physiological defense mechanism**

Broadly defined, **disease** is any physiological abnormality or significant disruption in the "normal" health of a plant. Disease can be caused by living (**biotic**) agents, including fungi and bacteria, or by environmental (**abiotic**) factors such as nutrient deficiency, drought, lack of oxygen, excessive temperature, ultraviolet radiation, or pollution. In order to protect themselves from damage, plants have developed a wide variety of constitutive and inducible defenses.

**Constitutive** (continuous) defenses include many preformed barriers such as cell walls, waxy epidermal cuticles, and bark. These substances not only protect the plant from invasion, they also give the plant strength and rigidity. In addition to preformed barriers, virtually all living plant cells have the ability to detect invading pathogens and respond with **inducible** defenses including the production of toxic chemicals, pathogen-degrading enzymes, and deliberate cell suicide. Plants often wait until pathogens are detected before producing toxic chemicals or defense-related proteins because of the high energy costs and nutrient requirements associated with their production and maintenance.

**D. Molecular defense mechanism**

Pathogenesis -Related Proteins (PR-proteins)

Pathogenesis related proteins, Called PR-proteins-A group of plant coded proteins are structurally diverse group toxic to invading pathogens.produced under stress They are widely distributed in plants in trace amounts but are produced in high concentration following pathogen attack or stress .The better known PR proteins are:–PR1proteins,B-1,3-glucanases,chitinases,lysozymes,–PR4proteins, thaumatinelike proteins,osmotin like proteins,cysteine-rich proteins,glycine-rich proteins,proteinase inhibitors,proteinases,chitosanases and peroxidases .There are often numerous isoforms of eachPR-protein in various host plants.

The significance of PR –proteins lies in the fact that they show strong antifungal and other antimicrobial activity.•Although healthy plants may contain trace amounts of several PR proteins, attack by pathogens,treatment with elicitors,wounding,or stress induces transcription of a battery of genes that code for PR –proteins.

This occurs as a part of a massive switch in the over all pattern of gene expression during which normal protein production nearely ceases.Some of them inhibit spore release and germination, whereas others are associated with strengthening of the Host cell wall and its out growths and papillae.

Some of the PR-proteins, for example,B-1,3-glucanase and chitinase,diffuse towards and affect (break down) the chitin supported structure of the cell walls of several plant pathogenic fungi,whereas lysozymes degrade the glucosmine and muramic acid components of bacterial cell wall

Plants genetically engineered to express chitinase genes show good resistance against The soil – borne pathogen *Rhizoctonia solani*

Signal molecules that induce PR proteins synthesis seem to be transported systemically to other parts of the plant and to reduce disease initiation and intensity in those parts for several days or even weeks.

# Plant disease epidemiology

**Plant disease epidemiology** is the study of disease in plant populations. plant diseases occur due to pathogens such as bacteria, viruses, fungi, oomycetes, nematodes, phytoplasmas, protozoa, and parasitic plants. Plant disease epidemiologists strive for an understanding of the cause and effects of disease and develop strategies to intervene in situations where crop losses may occur. Destructive and non-destructive methods are used to detect diseases in plants. Additionally, understanding the responses of the immune system in plants will further benefit and limit the loss of crops. Typically successful intervention will lead to a low enough level of disease to be acceptable, depending upon the value of the crop.

Plant disease epidemiology is often looked at from a multi-disciplinary approach, requiring biological, statistical, agronomic and ecological perspectives. Biology is necessary for understanding the pathogen and its life cycle. It is also necessary for understanding the physiology of the crop and how the pathogen is adversely affecting it. Agronomic practices often influence disease incidence for better or for worse. Ecological influences are numerous. Native species of plants may serve as reservoirs for pathogens that cause disease in crops. Statistical models are often applied in order to summarize and describe the complexity of plant disease epidemiology, so that disease processes can be more readily understood. For example, comparisons between patterns of disease progress for different diseases, cultivars, management strategies, or environmental settings can help in determining how plant diseases may best be managed. Policy can be influential in the occurrence of diseases, through actions such as restrictions on imports from sources where a disease occurs.

# Transmission and spread of Plant Pathogen

Wherever a disease is established in a particular area or country, transmission of the pathogen from host to host or from one place to another is termed as 'dissemination' or 'dispersal' of the pathogen.

1. **Primary infection:** Contact of a pathogen with a suitable host plant and initiation of the disease first time in the season of a crop is called 'primary infection'. Often a few or several plants in the crop are likely to get primarily infected.
2. **Secondary spread**: When a plant or few plants are primarily infected, rapid multiplication of the pathogen sets in under favorable climatic conditions, which helps 'secondary spread' of the disease.

**Modes of primary infections:**

1. Soil borne
2. Seed borne, including diseases carried with planting material.
3. Wind borne
4. Insect borne etc.

**Continuous and discontinuous transmission: -**

Transmission of disease is termed as 'continuous' when it occurs naturally by way of growth, multiplication and spread of the pathogen in an area or country where the disease is established.

At times, however, in an area or country where a particular disease has never occurred, it may get introduced through the agency of man carrying diseased material to a new locality or to a distant country for the purpose of introduction of new plants, crops, varieties etc. such transmission, of course, is unnatural and regarded as 'discontinuous' transmission.

**Direct and indirect transmission:-**

1. **Direct transmission: -** Disease transmission where the pathogen is carried externally or internally on the seed or planting material like cuttings, sets, tubers, bulbs etc.
2. **Indirect transmission:-** The pathogen spreading itself by way of its persistent growth or certain structures of the pathogen carried independently by natural agencies like wind, water, animals, insects, mites, nematodes, birds etc. are the different methods of indirect transmissions.

**Direct transmission: -**

1. **Internal transmission through seed or planting material:-** False smut disease as well as Helminthosporin Blight disease of wheat are the common examples of fungal diseases carried internally through apparently healthy seed. Ring rot and Brown rot of potato caused by bacteria are carried internally through the tubers. The well known whip smut and red rot of sugarcane are fungal diseases carried internally in the planting sets. Mosaic and leaf roll of potato which are viral diseases are also carried inside the infected tubers.

2. **External transmission through seed or planting material:-** In this mode of transmission the pathogen is carried externally over the surface of seed or vegetatively propagated plant parts like sets, tubers, bulbs etc. or may even be carried as a physical mixture of fungal structures with the seed. The common grain smut of jowar is an example of the former type while the fungal structures called 'sclerotia' having the size of a grain or slightly bigger in case of the Ergot disease of bajra are often likely to be transmitted in the form of physical mixture with the seed.

**Indirect transmission: -**

1. **Autonomous transmission:-** It takes place by continuous and persistent growth of the threads or 'hyphae' of the causal fungi in soil, characteristic of several wood rotting fungi attacking forest trees and some fruit plants. Some root rotting fungi infecting certain seasonal crops also are transmitted by this method.
2. **Wind dispersal: -** Fungal spores produced externally on host surfaces are most easily carried by wind currents and this is the most dangerous mode of transmission of plant pathogenic fungi like those causing powdery and downy mildews, leaf spots, blasts, blights and rust diseases.
3. **Water dissemination:** Disease transmission through the agency of water in different ways is comparatively less important as compared to the wind transmission. Splashing rain drops mostly transmit the foliar diseases from leaf to leaf, from shoot to shoot and even from plant to plant in case of closely spaced crops. Such transmission is usually accompanied by wind dispersal as well. Plant pathogens requiring high humidity conditions like the fungi causing downy mildew diseases or bacteria causing canker of citrus are well adapted to this kind of short distance water dispersal.
4. **Animals:** Farm animals serve as disease transmitting agents in some cases. They are likely to carry the pathogen externally on their body surface, particularly on legs and hoofs, etc. or internally through their intestinal tract. Commonly, the soil inhabiting fungi causing rots and wilts are carried externally while certain smut fungi causing diseases to grain crops are transmitted through the intestinal tract.
5. **Birds:** Although birds play a very minor role in disease transmission, in cases of dispersal of seeds of higher flowering parasite. Loranthus sp. Parasitising certain trees like mango, etc. their role is of great significance. They transmit loranthus both externally and internally.
6. **Implements and Tools:** Farm implements used for cultivation of soil are often likely to transmit plant pathogens from one place to another. The pathogens in this case are usually carried in the form of bits of plant disease debris lying in the soil. Similarly tools used for carrying out operations like cutting, pruning, budding, grafting, thinning,etc. also help in the transmission of certain diseases from plant to plant. Several viral diseases are disseminated through the budding and grafting operations.
7. **Insects:** Most of the viral diseases of plants are transmitted through the agency of different insects. Both types of insects viz. sucking and chewing or/biting are capable of transmitting viral diseases. Viruses carried `biologically' by the insect vectors are of two types:

1. Non-persistent-viral pathogen requiring no latent or incubation period in the insect body.

2. Persistent: viral pathogens requiring certain incubation period inside the vector body before they are inoculated or transmitted to healthy host.The insects responsible for transmission of viral diseases belong to the species of aphids, jassids (leaf hoppers), white flies, mealy bugs, etc. Certain bacterial and several fungal pathogens are also known to be carried by insects.

1. **Mites:** Mites in contrast to insects are wingless anthropods resembling ticks and having four pairs of legs and no antennae. It is suspected that some viral diseases of chillies, tomato, brinjal, etc. have vector relationship with mites.
2. **Nematodes:** Nematodes have been observed to transmit viral, bacterial and fungal plant diseases. Nematodes feeding externally on host plant roots cause injuries to roots which become the avenues for entrance of fungal and bacterial pathogens infecting plant roots. The Fan-leaf virus of grapevine is a well known example of transmission through a species of nematodes.
3. **Biological transmission**: Dodder which is higher flowering parasite is known to transmit certain viral diseases which remain `persistent' in the dodder plant. The flowering parasite after acquiring the virus from infected plant does not show any symptom itself but remains capable of transmitting the virus to healthy hosts.
4. **Human dispersal**: Man is often responsible for transmission of plant diseases in two ways viz.

1. Workers handling seedlings, other planting material or fruits are likely to get personally in contact with plant pathogens like fungi or bacteria. While handling the diseased material and unknowingly and indirectly transmit the pathogens to healthy seedlings or plant parts through his contaminated hands. This is a kind of `continuous' mode of transmission.
2. The other or`discontinous' mode of transmission for which only man is responsible is the most efficient and equally dangerous phenomenon of transmission of plant diseases between distant geographical areas often separated by physical barriers like oceans, mountains or deserts, etc. Such long distances transmission of a disease to an area or country hitherto free from the disease is usually accomplished by the transport of infected seed, nursery stock or timber, etc. Thus it is a kind of direct transmission through propagating material.

# Disease cycle

The disease cycle is a series of definite events, which lead to the disease development and pathogen propagation. These events include inoculation, prepenetration, penetration, infection, colonization (invasion), and growth and reproduction of the pathogen.

## Inoculation

Inoculation is the pathogen or any part of the pathogen that contacts with the plant at certain site to initiate the infection process, such as spores, sclerotia, or fragments of mycelium of fungi may be fungal inoculum.

**There are two types of inoculum: primary and secondary inoculum,** which in turn cause primary and secondary infection.

The primary inoculum lives dormant in the winter or summer and causes the original infections in the spring or in the autumn.

The secondary inoculum is that produced from primary infections..

**The inoculum has two sources: inside and outside sources.** The inside source in which the inoculum is produced on the plant, plant debris, or on the soil, such as fungal and bacterial inocula of perennial plants, is produced on the branches, trunks, or roots of the plants.

The outside source of inoculum is in which the inoculum comes into the field with the seed, transplants, tubers, or other propagative organs or it may come from sources outside the field. In some cases, the inoculum is produced on the plant surface as in fungi, bacteria, parasitic higher plants, and nematodes, which either produce their inoculum on the surface of infected plants or their inoculum reaches the plant surface when the infected tissue breaks down

## Prepenetration

### Attachment of pathogen to host

Some pathogens directly penetrate the plant tissues by their vectors and then are surrounded by cytoplasm, cell membrane, or cell wall of plant cell, such as mollicutes, fastidious bacteria, protozoa, and most viruses.

In other cases, the pathogen firstly makes contact with the external surface of the plant, and then penetration process takes place, such as fungi, bacteria, and parasitic higher plants. The adhesion of the pathogen with plant surface is carried out by mucilaginous substances found on the pathogen surface or at its tip. These substances are composed of mixture of water-insoluble polysaccharides, glycoproteins, lipids, and fibrillary materials, which, when moistened, become

sticky and help the pathogen adhere to the plant. In some fungi as powdery mildew, adhesion is carried out by the release of cutinase enzyme from the spore, which makes the plant and spore areas of attachment more hydrophilic and cements the spore to the plant surface.

**Spore germination**

Spore germination process initiates by growth stimulation, which takes place with the availability of proper environmental conditions.. When appropriate physical and chemical signals, such as surface hardness, hydrophobicity, surface topography, and plant signals, are present, germ tube extension and differentiation take place.

**Appressorium formation and maturation**

Appressorium is a specialized cell typical to many fungal plant pathogens that is used to infect the plant host. Once appressoria are formed, they adhere tightly to the leaf surface and then penetrate the plant cell wall via lysozyme secretion.

**Recognition between host and pathogen**

When a pathogen comes in contact with a host cell, the plant triggers a signal that either allows or retards the pathogen growth and development of disease. This signal is a biochemical reaction, which acts as a receptor to a pathogen contact.

**Spores and seed germination**

The availability and ability of host infection are increased by vegetative pathogen. The infection by fungal spores or parasitic higher plant seeds is carried out after germination has achieved. Fungal spores' germination is carried out by releasing either a mycelium or a germ tube that grows into the plant cell and cause infection.

**Growth of nematodes**

The growth of nematodes starts with hatching of eggs, which essentially requires convenient environmental conditions such as temperature and moisture. After hatching of the eggs, the larvae penetrate the plant cell and grow to form the adults.

**Penetration**

Phytopathogens penetrate plant surfaces either through natural openings such as fungi and nematodes or through wounds in cell wall such as bacteria, viruses, viroids, mollicutes, fastidious bacteria, and protozoa.

**Infection**

The intimate contact of phytopathogen with its host is called infection process. The infection process is either successful or unsuccessful depending on the type of host, whether susceptible or resistant, respectively. Successful infection results in the appearance of symptoms, such as

discoloration, necrosis, dwarfism, and so on of the host. While unsuccessful (latent) infection does not lead to any observations for the symptoms

**Invasion**

The phytopathogens can invade the plant tissues by producing mycelia which grow between the cuticle and epidermis, other phytopathogens such as those causing powdery mildews produce mycelia which grow on the plant surface, and then extend to form a structure called haustoria, which in turn extend into the epidermal cells. Therefore, plant pathogenic fungi can invade their host either by intracellular mycelia, which directly grow through the cells, or by intercellular mycelia, which grow between the cells.

**Growth and reproduction of the pathogen**

Most phytopathogens especially fungi and parasitic higher plants invade and infect plant tissues through the point of inoculation. Therefore, these pathogens can easily grow and spread within the plant tissues until a certain limit or death occurs.

# Epidemics

Disease epidemics in plants can cause huge losses in yield of crops as well threatening to wipe out an entire species such as was the case with Dutch Elm Disease and could occur with Sudden Oak Death. An epidemic of potato late blight, caused by *Phytophthora infestans*, led to the Great Irish Famine and the loss of many lives.

Commonly the elements of an epidemic are referred to as the "disease triangle": a susceptible host, pathogen, and conducive environment. For a disease to occur all three of these must be present. Below is an illustration of this point. Where all three items meet, there is a disease. The fourth element missing from this illustration for an epidemic to occur is time. As long as all three of these elements are present disease can initiate, an epidemic will only ensure if all three continue to be present. Anyone of the three might be removed from the equation though. The host might out-grow susceptibility as with high-temperature adult-plant resistance,the environment changes and is not conducive for the pathogen to cause disease, or the pathogen is controlled through a fungicide application for instance.

Sometimes a fourth factor of time is added as the time at which a particular infection occurs, and the length of time conditions remain viable for that infection, can also play an important role in epidemics. The age of the plant species can also play a role, as certain species change in their levels of disease resistance as they mature; in a process known as ontogenic resistance.

If all of the criteria are not met, such as a susceptible host and pathogen are present, but the environment is not conducive to the pathogen infecting and causing disease, a disease cannot occur. Likewise, it stands to reason if the host is susceptible and the environment favours the development of disease but the pathogen is not present there is no disease. When a pathogen requires a vector to be spread then for an epidemic to occur the vector must be plentiful and active.

**Types of epidemics**

- Pathogens cause monocyclic epidemics with a low birth rate and death rate, meaning they only have one infection cycle per season. They are typical of soil-borne diseases such as Fusarium wilt of flax. Polycyclic epidemics are caused by pathogens capable of several infection cycles a season. They are most often caused by airborne diseases such as powdery mildew. Bimodal polycyclic epidemics can also occur. For example, in brown rot of stone fruits the blossoms and the fruits are infected at different times.

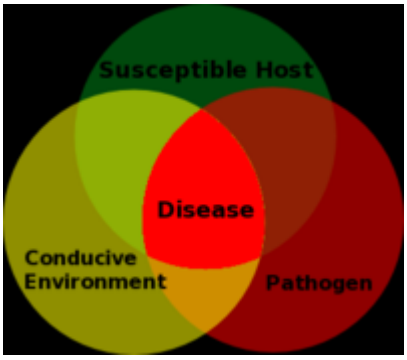
- For some diseases it is important to consider the disease occurrence over several growing seasons, especially if growing the crops in monoculture year after year or growing perennial plants. Such conditions can mean that the inoculum produced in one season can be carried over to the next leading to a build of inoculum over the years. In the tropics there are no clear-cut breaks between growing seasons as there are in temperate regions and this can lead to accumulation of inoculum.

- Epidemics that occur under these conditions are referred to as *polyetic* epidemics and can be caused by both monocyclic and polycyclic pathogens. Apple powdery mildew is an example of a polyetic epidemic caused by a polycyclic pathogen and Dutch Elm disease a polyetic epidemic caused by a monocyclic pathogen.

# Modeling and disease forecasting

Forecasting models are often based on a relationship like simple linear regression where x is used to predict y. Other relationships can be modelled using population growth curves. The growth curve that is used will depend on the nature of the epidemic. Polycyclic epidemics such as potato late blight are usually best modelled by using the logistic model, whereas monocyclic epidemics may be best modelled using the monomolecular model. Correct choice of a model is essential for a disease forecasting system to be useful.

Plant disease forecasting models must be thoroughly tested and validated after being developed. Interest has arisen lately in model validation through the quantification of the economic costs of false positives and false negatives, where disease prevention measures may be used when unnecessary or not applied when needed respectively. The costs of these two types of errors need to be weighed carefully before deciding to use a disease forecasting system.




The plant disease triangle represents the factors necessary for disease to occur

**Plant disease forecasting** is a management system used to predict the occurrence or change in severity of plant diseases. At the field scale, these systems are used by growers to make economic decisions about disease treatments for control. Often the systems ask the grower a series of questions about the susceptibility of the host crop, and incorporate current and forecast weather conditions to make a recommendation. Typically a recommendation is made about whether disease treatment is necessary or not. Usually treatment is a pesticide application.

Forecasting systems are based on assumptions about the pathogen's interactions with the host and environment, the disease triangle. The objective is to accurately predict when the three factors – host, environment, and pathogen – all interact in such a fashion that disease can occur and cause economic losses.

In most cases the host can be suitably defined as resistant or susceptible, and the presence of the pathogen may often be reasonably ascertained based on previous cropping history or perhaps survey data. The environment is usually the factor that controls whether disease develops or not. Environmental conditions may determine the presence of the pathogen in a particular season through their effects on processes such as overwintering. Environmental conditions also affect the ability of the pathogen to cause disease, e.g. a minimum leaf wetness duration is required for grey leaf spot of corn to occur. In these cases a disease forecasting system attempts to define when the environment will be conducive to disease development.

Good disease forecasting systems must be reliable, simple, cost-effective and applicable to many diseases. As such they are normally only designed for diseases that are irregular enough to warrant a prediction system, rather than diseases that occur every year for which regular treatment should be employed. Forecasting systems can only be designed if there is also an understanding on the actual disease triangle parameters.

**Examples of disease forecasting systems**

Forecasting systems may use one of several parameters in order to work out disease risk, or a combination of factors. One of the first forecasting systems designed was for Stewart's wilt and based on winter temperature index as low temperatures would kill the vector of the disease so there would be no outbreak. An example of a multiple disease/pest forecasting system is the EPIdemiology, PREdiction, and PREvention (EPIPRE) system developed in the Netherlands for winter wheat that focused on multiple pathogens. USPEST.org graphs risks of various plants diseases based on weather forecasts with hourly resolution of leaf wetness.

# Blast disease of paddy

## Symptoms

Initial symptoms are white to gray-green lesions or spots with darker borders produced on all parts of the shoot, while older lesions are elliptical or spindle-shaped and whitish to gray with necrotic borders. Lesions may enlarge and coalesce to kill the entire leaf. Symptoms are observed on all above-ground parts of the plant. Lesions can be seen on the leaf collar, culm, culm nodes, and panicle neck node. Internodal infection of the culm occurs in a banded pattern. Nodal infection causes the culm to break at the infected node (rotten neck). It also affects reproduction by causing the host to produce fewer seeds.

## Causative agent

***Magnaporthe grisea***, also known as **rice blast fungus**, **rice rotten neck**, **rice seedling blight**, **blast of rice**, **oval leaf spot of graminea**, **pitting disease**, **ryegrass blast**, **Johnson spot**, and **neck blast** is a plant-pathogenic fungus that causes a serious disease affecting rice. *M. grisea* is an ascomycete fungus. It is an extremely effective plant pathogen as it can reproduce both sexually and asexually to produce specialized infectious structures known as appressoria that infect aerial tissues and hyphae that can infect root tissues.

## Control measures

The fungus has been able to establish resistance to both chemical treatments and genetic resistance in some types of rice developed by plant breeders. It is thought that the fungus can achieve this by genetic change through mutation. Another strategy would be to plant resistant rice varieties that are not as susceptible to infection by *M. grisea*. Knowledge of the pathogenicity of *M. grisea* and its need for free moisture suggest other control strategies such as regulated irrigation and a combination of chemical treatments with different modes of action. Managing the amount of water supplied to the crops limits spore mobility thus dampening the opportunity for infection. Chemical controls such as Carpropamid have been shown to prevent penetration of the appressoria into rice epidermal cells.

# Powdery Mildew of Grape

**Symptoms**

The powdery mildew fungus can infect all green tissues of the vine. Small, white or grayish-white patches of fungal growth appear on the upper or lower leaf surface . These patches usually enlarge until the entire upper leaf surface has a powdery, white to gray coating . The patches may remain limited throughout most of the season. Severely affected leaves may curl upward during hot, dry weather. Expanding leaves that are infected may become distorted and stunted.On young shoots, infections are more likely to be limited, and they appear as dark-brown to black patches that remain as dark patches on the surface of dormant canes.

If blossom clusters are affected, the flowers may wither and drop without setting fruit. Infections on cluster stems often go unnoticed, but can be very damaging. Infected cluster stems may wither and dry up, resulting in berry drop (shelling). Affected berries may have patches of fungal growth on the surface similar to those on the leaves, or the entire berry may be covered with the white, powdery growth. Infected berries often are misshapen or have rusty spots on the surface. Severely affected fruit often split open .When berries of purple or red cultivars are infected as they begin to ripen, they fail to color properly and have a blotchy appearance at harvest. Berries are susceptible to infection from early bloom through three to four weeks after bloom.

Late in the season, many black specks may develop on the surface of infected areas. These are the sexual fruiting bodies (cleistothecia) of the fungus.

**Causal Organism**

Powdery mildew is caused by the fungus *Uncinula necator*

**Control measures**

Select an open planting site with direct sunlight. Plant rows in the direction of the prevailing wind in order to promote good air circulation and faster drying of foliage and fruit. Prune and train vines properly in such as way as to reduce shading and increase air circulation.

On susceptible varieties, control is based on the use of properly timed applications of effective fungicides. Early season (prebloom through bloom) control of primary infections caused by ascospores must be emphasized. For the most current fungicide recommendations and spray schedules, commercial growers are referred to Bulletin 506, *Midwest Fruit Pest Management Guide*, and backyard growers are referred to Bulletin 780, *Controlling Diseases and Insects in Home Fruit Plantings*.

# COTTON BLIGHT

**Symptoms**

The bacterium attacks all stages from seed to harvest. Usually five common phases of symptoms are noticed.

**i) Seedling blight:**

Small, water-soaked, circular or irregular lesions develop on the cotyledons, later, the infection spreads to stem through petiole and cause withering and death of seedlings.

**ii) Angular leaf spot:**

Small, dark green, water soaked areas develop on lower surface of leaves, enlarge gradually and become angular when restricted by veins and veinlets and spots are visible on both the surface of leaves. As the lesions become older, they turn to reddish brown colour and infection spreads to veins and veinlets.

**iii) Vein blight or vein necrosis or black vein:**

The infection of veins cause blackening of the veins and veinlets, gives a typical 'blighting' appearance. On the lower surface of the leaf, bacterial oozes are formed as crusts or scales. The affected leaves become crinkled and twisted inward and show withering. The infection also spreads from veins to petiole and cause blighting leading to defoliation.

**iv) Black arm:**

On the stem and fruiting branches, dark brown to black lesions are formed, which may girdle the stem and branches to cause premature drooping off of the leaves, cracking of stem and gummosis, resulting in breaking of the stem and hang typically as dry black twig to give a characteristic "black arm" symptom.

**v) Square rot / Boll rot:**

On the bolls, water soaked lesions appear and turn into dark black and sunken irregular spots. The infection slowly spreads to entire boll and shedding occurs. The infection on mature bolls lead to premature bursting. The bacterium spreads inside the boll and lint gets stained yellow

because of bacterial ooze and looses its appearance and market value. The pathogen also infects the seed and causes reduction in size and viability of the seeds.

**Causative agent**

**Bacterial blight -** bacterium ***Xanthomonas axonopodis* pv. *malvacearum***

**Control measures**

- Delint the cotton seeds with concentrated sulphuric acid at 100ml/kg of seed. Treat the delinted seeds with carboxin or oxycarboxin at 2 g/kg or soak the seeds in 1000 ppm Streptomycin sulphate overnight.
- Remove and destory the infected plant debris. Rogue out the volunteer cotton plants and weed hosts.
- Follow crop rotation with non-host crops.
- Early thinning and early earthing up with potash.
- Grow resistant varieties like Sujatha, 1412 and CRH 71.
- Spray with Streptomycin sulphate +Ttetracycline mixture 100g along with Copper oxychloride at 1.25 Kg/ha.

# Banana bunchy top virus

## Symptoms

Symptoms are the short, narrow and upright clustering of leaves which form at the top of an infected banana plant .Banana leaves infected with BBTV have dark green streaks along the small veins on the underside of the leaf. As BBTV progresses dark green streaks appear on the infected leaves, midribs and stalks. In older banana plants dark green streaks may be seen on flower bracts.Banana plants which are mature when infected with BBTV may produce fruit but the bunches will be stunted and deformed.

## Causative organism

Banana bunchy top virus infects cultivated and wild bananas in the Musaceae plant family.

## Control measures

BBTV can be controlled by promptly treating the banana aphid infestation and then destroying the infected banana plant.Banana aphid infestations must be treated before destroying the infected banana plant. If the infected plant is destroyed first the banana aphids will simply fly to nearby healthy plants and spread the disease.

## Banana aphid control

It require that banana aphids on banana plants infected with BBTV are treated within 3 days of detection of the disease.

Banana aphids can be treated by either:

- injecting the infested banana plant with imidacloprid (PER14850, expires 30 Sept 2024); or
- spraying the entire infested banana plant with paraffinic oil. All banana plants within 10 metres of the infested banana plant should also be sprayed with paraffinic oil (PER14850, expires 30 Sept 2024).

**Bunchy top virus control**

The Control that after the aphid treatment, the banana plants infected with BBTV are immediately destroyed.Banana plants infected with BBTV can be treated by either:

- injecting the infected banana plant with glyphosate . The whole banana plant, including the corm, attached suckers and pseudo stems, should be treated (PER14850, expires 30 Sept 2024); or
- removing the entire infected banana plant from the ground. The pseudostems should be split and the corms cut into pieces no more than 5 centimetres in diameter.

# Phyllody Sesame

**Phyllody** is the abnormal development of floral parts into leafy structures.

### Symptoms

A plant affected by sesame phyllody bears clusters of leaves and a malformed flower at the tip. Plants looks like a witches broom. In severe infection, the entire flower shoot is replaced by short twisted leaves closely arranged on a stem with abnormal branches bending downwards.

### Causative agent

This disease is caused by Phytoplasma which is transmitted by leaf hoppers. It converts the flowering parts into green leafy structures then causes severe vein clearing. Though the capsules are formed on the lower portion, the plant does not yield quality seeds. This is the major yield-reducing problem affecting sesame.

### Control measures

- Intercrop sesame with pigeon pea (6:1)
- Remove and burn infected plants
- Seed treatment with imidacloprid or carbosulfan protects the crop from all sucking pests including leaf hoppers for about a month
- Spray dimethoate 30 EC @ 500 ml/ ha at 30, 40 and 60 days after sowing to control the leaf hopper