

**COURSE CONTENTS**  
**INSECTS IN RELATION TO PLANT DISEASES (ENT-710)**

**OBJECTIVES:**

To provide the broad overview of insects in relation to plant diseases with special emphasis on vectorial status.

**THEORY:**

Introduction; identification, biology of insect and mite vectors of plant diseases; mode of transmission of plant pathogens by insects and mites; study of causal organisms, etiology, symptoms and control of important fungal, bacterial and viral diseases of crop plants transmitted by insects and mites.

**PRACTICAL:**

Identification of insect and mite vectors and pathogens; rearing and handling of insect vectors for plant pathological studies. Study of mode of transmission of plant pathogens by insect and mite vectors.

**BOOKS RECOMMENDED:**

1. Boucias, D.G. 1998. Principles of Insect Pathology. Chapman Hall, London.
2. Jeppson, L.R. Keifer, H.H. and Baker, E. W. 1975. Mites Injurious to Economic Plants. Univ. Calif. Press.
3. Leach, J.B. 2007. Insect Transmission of Plant Diseases. Biotech Books. Delhi, India.
4. Tanada, Y. 1992. Insect Pathology. Academic Press.

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## TOPIC 1. INSECTS TRANSMIT PATHOGENS IN THREE MAIN WAYS.

- 1) Many insects transmit bacteria and fungal spores passively by feeding in or walking through an infected plant area that has on its surface plant pathogenic bacteria or fungal spores as a result of the infection. The bacteria and spores are often sticky, cling to the insect as it moves about, and are carried by it to other plants or parts of the same plant where they may start a new infection.
- 2) Some insects transmit certain bacteria, fungi, and viruses by feeding on infected plant tissues and carrying the pathogen on their mouthparts as they visit and feed on other plants or plant parts.
- 3) Several insects transmit specific viruses, phytoplasmas, protozoa, nematodes, and xylem- and phloem-inhabiting bacteria by ingesting (sucking) the pathogen with the plant sap they eat. Subsequently, the pathogen circulates through the body of the insect until, with or without further multiplication in the insect, the pathogen reaches the salivary glands and the mouthparts of the insect through which it is injected into the next plant on which the insect feeds

## TOPIC 2. INSECT TRANSMISSION OF PLANT PATHOGENIC VIRUSES

Plant viruses cause many and severe diseases of plants, their number and importance being second only to fungal diseases of plants. Most viruses infect their host plants systemically, that is, the virus multiplies internally throughout the plant. Almost all viruses enter and multiply in phloem and in parenchyma cells. Viruses do not produce spores, nor do they come to the surface of the plant. More than half of the plant viruses, numbering more than 400, are transmitted from diseased to healthy plants by insects.

The most important vector groups, with the number of vector species and viruses transmitted, are listed below

Order	Insect	Number of Species	Number of viruses	Importance
Homoptera	aphids	192 species	275 viruses	Most Important
	leafhoppers	49 species	31 viruses	
	Planthoppers	28 species	24 viruses	
	Whiteflies	3 species	43 viruses	
	Mealybugs	19 species	10 viruses	

	Treehoppers	1 species	1 virus	
Hemiptera	true bugs	4 species	Unknown	Occasional or less important
Thysanoptera	thrips	10 species	11 viruses	
Coleoptera	beetles	60 species	42 viruses	
Orthoptera	Grasshoppers	27 species	Unknown	

## **APHIDS AND APHID TRANSMITTED VIRUSES**

Aphids mouthparts consist of four stylets present inside a groove of labium. In feeding, stylets are drawn-out from labium with saliva then stylets quickly enter the epidermis. Penetration can stop at epidermis or it can carry on into internal layers of cells with saliva forming a sheath around the stylets. The stylets travel among cells till they reach a phloem sieve tube from where aphids get food. Aphids differ in their capability to spread virus to different plants. An infected plant with virus often becomes attractive for the aphids for their growth and reproduction. Different factors like temperature, humidity and light affect acquisition and transmission of virus by aphids.

### **Aphids' life cycle**

Aphids are most successfully exploit plants as food sources. These are much more prevalent in temperate regions. There are many variations in their life cycles.

### **Virus vector relationships**

Insect with sucking mouthparts bring viruses on their stylets while these viruses are recognized stylet-borne, externally borne, or non-circulative as they do not go through vector's internal body. Other viruses are internally taken up by vector and are called inside borne persistent circulative or persistent propagative viruses.

### **Stylet borne non persistent transmission**

The insect obtains virus from plant via feeding on and only seconds or in minutes. This insect can spread virus straightaway following the acquisition and it is lacking any incubation period needed

for transmission. The insect holds virus and is capable of transmitting it for only a few minutes after it attained it. There are 300 known cases of aphid borne plant viruses which are stylet borne non-persistent. Most significant examples of these viruses are in the genera *Potyvirus*, *Cucumovirus*, *Alfamovirus*, and the *Caulimovirus* which are transmitted by *Myzus persicae*. These are stylet borne non-persistent viruses

### **Semi persistent viruses**

Some external non persistent viruses are recognized as semi persistent as these can go up to foregut of vector. In this case, vector must forage on infected plant for several minutes or hours (acquisition period) before it transmits virus. Vector can keep and transmit the virus to healthy plants for several hours (retention time). Semi persistent viruses are helped in their transmission by a helper protein or coat protein structure. The best examples of such viruses include caulimoviruses. These are present in most cell types. Others are beet yellows virus and curly top virus, found chiefly in phloem cells. In some semi-persistent viruses like cauliflower mosaic virus, the assistant element comprise two non-capsid proteins, one is linked with virus particles and other has two binding domains that interact strongly with microtubules.

### **Persistent viruses**

Inside borne viruses can be either persistent circulative and or persistent propagative.

**Persistent circulative viruses:** These need a latent period of some hours to some days after the acquisition phase before these are transmitted by insect vector. They spread the hemolymph of vector. Then these go through different stages of insect, but not from the ovaries to egg. Examples are the luteoviruses e.g., barley yellow dwarf virus; nanoviruses which has example of banana bunchy top virus.

**Persistent propagative viruses:** These are picked up by insect after a feeding time of some hours to some days and remain inside vector for some weeks to some months. These increase in vector. They have a latent period of some to several weeks. These can go through many stages of insect which include transovarial channel to the egg. Persistent viruses are generally transmitted by one

or a few species of aphids and cause symptoms characterized by leaf yellowing and leaf rolling. Examples are rhabdoviruses.

## **LEAFHOPPERS AND PLANTHOPPERS, AND TRANSMISSION OF PLANT VIRUSES**

They all feed by sucking sap from phloem elements of plants. Their feeding behavior is similar to that of aphids in that the mouthparts, surrounded by the salivary sheath, penetrate the phloem of host plants.

### **Virus vector relationships**

Hopper spread viruses are either persistent circulative or persistent propagative. These are spread by a few closely associated hopper species. Only two sub families Agalliinae and the Deltocephalinae spread it. Only one plant hopper family Delphacidae include species as vectors of viruses and many of which give rise to diseases on crops like rice, corn and wheat.

### **Semi-persistent transmission**

*Graminella nigrifrons* and *Nephotettix virescens*, respectively gain viruses as maize chlorotic dwarf virus and rice tungro spherical virus. MCDV particles have been seen in the foregut and a few other tissues but not beyond.

### **Persistent transmission**

#### **Circulative viruses**

Two genera of geminiviruses namely *Mastrevirus* and *Curtovirus* are spread by leafhoppers in the persistent circular manner. The viruses are attained by vector after sucking for a few seconds to one hour. Latent period is of around one day, apparently for the virus to spread the salivary glands. Interior movement of viruses is affected by coat protein and endocytosis which is receptor mediated.

**Propagative viruses.** Two families namely Rhabdoviridae and Reoviridae. These have a latent period of around fourteen days. The virus in this time replicate and invade maximum tissues of insect. When it reaches salivary glands, vector can spread virus to fresh plants and then stay to spread for their life. Slight percentage of hoppers which feed on infested plants convert as vectors and one % of these pass the virus to the next generation through their eggs. For passage of viruses, many capsid proteins appear essential.

## **WHITEFLY TRANSMISSION OF PLANT VIRUSES**

Only a few species of whiteflies transmit viruses, mostly in the tropics and subtropics, but the viruses they transmit cause very severe diseases.

*Bemisia tabaci* whiteflies transmit Begomoviruses. Criniviruses and closteroviruses are transmitted by *Trialeuroides vaporariorum*, *T. abutilonea*, *B. tabaci*, and the type B of *B. tabaci* (*B. argentifolii*).

Whitefly mouthparts and feeding behavior look like aphids. Begomoviruses are spread by whiteflies in persistent circulative manner. Whitefly spread closteroviruses and criniviruses go only insect foregut and are spread in semi persistent manner. These viruses are kept in vector body for around three to nine days. Transmission from the vector is helped by 2 capsid proteins.

## **THRIPS TRANSMISSION OF PLANT VIRUSES**

Four genera of viruses namely *Carmovirus*, *Sobemovirus*, *Ilarvirus* and *Tospovirus* are transmitted by thrips family thripidae.

The larvae are sedentary but adults having wings are active. Their adults feed by sucking the cell sap of cell in sub epidermis. These live for 3 weeks and have twenty generations per year.

Tospoviruses are transmitted in persistent propagative way although viruses from other genera are spread in pollens taken by thrips and through mechanical damage while feeding by vector.

In tospoviruses, it is the larvae and not adults which get virus and their capability decreases with age. Larvae get virus after feeding for only five minutes but typically they must feed for more than hour for both obtaining and in injecting virus. Latent period of three to four days is there beforehand larvae can spread virus. Virus is handed from larvae to adults which can spread it. These viruses appear to multiply in the vector but are not passed through the egg. Structural proteins of the virus appear to be related with the gaining, way through, and injection of virus by its larval as well as adult stages.

## **MEALYBUG AND OTHER BUG TRANSMISSION OF PLANT VIRUSES**

They move typically by way of crawling nymphs, from leaves of neighboring plants in contact by each other. Ants involved with mealybugs and move them from plant to plant and rarely by wind. These feed on phloem and are vectors of badnaviruses.

These include cacao swollen shoot virus, closteroviruses, grapevine leafroll associated viruses and the pineapple mealybug wilt associated virus, and the trichoviruses, such as grape viruses A and B.

Mealybugs obtain viruses after sucking on infected plants only a few or around twenty minutes and hold virus up to twenty four hours. This transmission look like the non-persistent or semi-persistent method of spread in case of aphids.

## **VIRUS TRANSMISSION BY INSECTS THAT HAVE BITING/CHEWING MOUTHPARTS**

Sixty vector species are in Coleoptera order and thirty of these are in Chrysomelidae family. Maximum beetle eat plant cells among leaf veins and vomit through feeding, thus swim their mouthparts in sap and virus. Viruses by beetle spread are in the genera *Tymovirus*, *Bromovirus*, *Comovirus*, and *Sobemovirus*

## **VIRUS TRANSMISSION BY MITES**

Eriophyidae transmit viruses of the genus *Rymovirus*. Two mite species of the family Tetranychidae transmit two plant viruses. All mites in these families feed by piercing plant cells and sucking their contents. Eriophyid mites are minor. These mostly move little but they are speeded by wind. These take two instars of nymphs followed by a inactive pseudopupa. They complete life cycle in fourteen days. Mites can obtain virus from diseased plants in fifteen minutes from beginning of feeding and can spread to vigorous plant in a parallel period. These get virus when they are nymphs but as adults. They transport virus through molts and persist infectious for six to nine days.

Tetranychid mites are larger in size. Pre adult mites get virus and them in addition to the adults, spread virus proficiently

## **VIRUS TRANSMISSION BY POLLINATING INSECTS**

Honey bees and related insects as pollination agents look to dispensing virus infected pollens from diseased plants to vigorous ones however it seems that no particular methods or connection of insects are existing in virus spread.

## **TOPIC 3. INSECT TRANSMISSION OF PLANT PATHOGENIC PROTOZOA**

Three diseases in plants namely phloem necrosis of coffee, sudden wilt of oil palms and heartrot of coconut palms are produced due to protozoa (flagellate) belong to *Phytomonas* genus. In these cases, protozoa enter phloem of diseased plants. These multiply in them and reach population of variable masses. Sieve tubes convert plugged due to protozoa. Normally, the more obvious are the symptoms of diseased plants, the advanced the populations of protozoa in phloem. This pathogen spread from diseased to vigorous plants rarely through normal root grafting and mainly due to stink bugs like in genera *Lincus* and *Oclenus*.



## TOPIC 4. INSECT TRANSMISSION OF PLANT PATHOGENIC NEMATODES

Two severe plant diseases due to nematodes are in genus *Bursaphelenchus* are spread by insects. In both diseases there is a symbiotic relationship between the fungal pathogen and the insect vector.

### Pine wilt

This is a lethal disease of numerous species of pines. It is due to *Bursaphelenchus xylophilus* called as pinewood nematode. It is 800 µm in length and with 22 µm diameter and it multiplies rapidly. It produces four juvenile phases and adults. Juvenile phases grow in resin of diseased trees. It feeds firstly in cells and in future over fungi which enter the dying or lifeless tree. Nematode then yields fourth stage spreading juveniles adapted to live in respiratory system of the cerambycidae *Monochamus carolinensis* as well as *M. alternatus* so by them are spread to vigorous pine trees. Pinewood nematode overwinters in wood of diseased and dead trees. These have larvae of vectors (beetles) of nematode. In spring, larvae excavate minor holes in wood within which these pupate. As adults (beetles) emerge from pupae late in season of spring, great numbers of 4<sup>th</sup> stage juveniles nematodes go in beetles and nearly fill their tracheae in respiratory system of insects with around 15,000 to 20,000 juvenile. These beetles having nematodes appear and fly to new branch tips of fit trees of pines where these feed themselves some weeks. These beetles from bark reach cambium, the juveniles begin from insect and go in the tree by wound. Juveniles in tree at that time go through last molt and yield nematode adults. The nematode adults travel to resin canals. They feed on cells and source their death. From there and they travel in xylem and cortex. Here these reproduce rapidly and construct vast residents of nematodes and kill whole trees.

## TOPIC 5. INSECT TRANSMISSION OF PLANT PATHOGENIC FUNGI

### Fungal Diseases

As with bacteria, many insects transmit many fungi from unhealthy to well plants. Insects are involved in diseases as these break epidermis and defensive tissues in plants with mouthparts, ovipositor through fungus enters. Many insect broadcasts of fungi are unintentional which means it is because insects develop outwardly or inside polluted with fungus and spores. As soon as these go to diseased plants and have spores to the plants. In a few cases, insect broadcast of fungus

happens as insect goes to buds in pollination, while others cases, it happens however wounding plants during oviposition. In maximum cases, spread happens though wounding plants in feeding. In comparatively fewer cases, insect and fungus these spread cultivate a symbiotic association within which individually value from its association with the other.

### **Stalk or stem-infecting fungi**

Numerous fungi contaminating stems or stalks including *Gibberella*, *Fusarium*, *Diplodia* in corn are seemingly assisted by numerous insects for example the extensive European corn borer.

### **Trunk and branch canker-causing fungi**

Many species of *Neofabrea*, *Leucostoma*, *Nectria*, *Ceratocystis*, and *Leptosphaeria* which cause tree cankers, are seemingly frequently linked with and aided by insects in the beginning and growth of cankers. Insects vary with the specific host as well as fungus. For example, *Neofabrea perennans* fungus, which is cause of perennial canker in apple is spread due to wooly apple aphid *Eriosoma lanigerum*. These aphids forage on bark at base of stem and they source creation of galls in which they increase. In the early spring, galls eruption takes place, then aphids arise out and fungus assaults wounded tissue and then from it progresses into fit tissue and yields canker. In summer, the apples tree yields callus tissues and covers off fungus and range of the canker ends. Aphids on the other hand develop in the callus tissue and a new gall is formed, and the procedure is constant.

In malformation mango disease, seemingly which is by fungus named *Fusarium moniliforme*, fungus is spread by mite *Aceria mangifera*, although additional fungi appear to be passed in digestive region of different termites.

### **Sooty molds**

These black colore fungi and cultivate on faces of leaves of plants, especially in the tropics or subtropics. This fungi do not enter and contaminate plants but source infection by obstructing the light from getting to leaves. These do not parasitize host plants but feed on honeydew delivered

by insects which include mealybugs, aphids, whiteflies and scales etc. This fungi is dispersed by their spores being propelled about due to wind. Nevertheless, they are blowout by the insects producing honeydew and also by numerous other ways of insects such as wasps, flies, bees and ladybugs all of them pursue honeydew as basis of food and by this way develop with them fungus spores which they transfer.

### **Wood rots**

Wood rotting is due to chiefly by basidiomycete fungus. Shelf or conk shape fruiting bodies of several of the fungi are routinely visited by numerous types of insects and it is supposed that numerous of these insects action as vectors of wood rotting fungus. Insects and mites have been associated with blowout of pine rust infections although at least three scolytid beetles are shown to have association in the broadcast of scleroderis called as canker of spruce and pine.

### **Vascular wilts**

Numerous vascular wilts affect trees and even cause extensive death of trees as fungus accountable for disease is spread from unhealthy to healthy trees due to specific insects. These are (A). Persimmon wilt caused due to fungus *Cephalosporium diospyri* which enters through all kinds of wounds but is also transmitted by the powder-post beetle *Xylobiops basilaris* and the twig girdler beetle *Oncider cingulatus*,

(B) Mango wilts, due to fungus *Diplodia recifensis*. It is spread by the beetle *Xyleborus affinis*, and another caused by the fungus *Ceratocystis fimbriata* and transmitted by the scolytid beetle *Hypocryphalus mangiferae*.

(C) Vascular wilts are oak wilt

(D) Dutch elm disease and will be discussed in some detail below.

Only one is discussed

## **Dutch elm disease**

It is due to fungus (*Ophiostoma ulmi*) or *Ophiostoma novo-ulmi*, which is now substituting the previous species. It eradicates elm tree branches and entire trees by blockage of xylem vessels and obstructing movement by water from roots to above parts. This disease is particularly devastating in US where the native elm tree is very vulnerable to the pathogen. The disease has destroyed all trees, particularly elm trees established along streets as well as parks. These trees in forests have also been destroyed but numerous of them have absconded infection so far.

The fungus of this disease is transmitted from unhealthy to well trees by European bark beetle named as *Scolytus multistriatus* and native beetle *Hylurgopinus rufipes* and also by normal root grafts. This fungus overwinters in bark of dead or dying elm trees and also logs in the form of mycelium as well as spores. These beetles lay eggs in galleries which they create in the surface amid bark and wood in debilitated or dead trees. If a tree is previously diseased with this disease and if insects transfer with them spores of fungus, the fungus cultivates and yields novel spores in tunnels. After eggs hatch, the larvae create tunnels vertical to those made by the adults. The adults appear and have thousands of spores of fungi on body. The emergent adults choose to feed on new twigs and small branches. As beetles tunnel into bark and wood to have sap, spores taken on their body are placed in wounded humid tissues of tree. There spores propagate and cultivate in the wounded bark and wood and the fungus spreads the xylem vessels of tree where it cultivates making mycelium as well as spores. Latter are taken ascending by the sap watercourse where these begin novel infections. Shoots afar the diseased areas go brown, wilt and die and as numbers increase, the tree displays additional more wilted branches. Finally large parts or whole trees wilt as well as die while fungus remains to produce and blowout in dead tree. These trees then are visited by adult beetles which lay eggs there and the cycle is constant.

## **Foliar diseases**

Numerous foliar infections are possibly spread by many insects going to or walking around leaves that are showing contaminations by spore making fungus. Spores of various fungi are adhesive and adhere to legs and other body portions of some insects and are passed to other plant life they

travel next. A few instances of foliar infections due to insect broadcast of the fungus pathogens occurring are detailed below

### **Powdery mildews**

These infections affect maximum yearly and year after year plants. They are categorized as having superficial mycelial growth white in color as well as sporulation due to small set of fungi which source symptoms on shoots, leaves, blossoms as well as fruit of the hosts. This disease is source of food for numerous mycophagous fungus and yield great amounts of spores attached loosely. These spores convert to, and are dispersed by insects with which they come in interaction. Examples comprise feeding by thrips in and broadcast of spores from the fungus *Sphaerotheca panosa* as well as *Uncinula necator* which causes powdery mildew in roses and grapes, correspondingly. Though these fungi are dispersed by wind, it is likely that broadcast is assisted by insects.

### **Rust diseases**

Maximum rust infections yield numerous types of surface spores on their hosts in powdery mildews, are simply dispersed by air but are similarly visited, eaten as well as transferred by a wide-ranging insects. Additionally, numerous rust fungi yield spermatia as well as receptive hyphae in the similar spermagonium nonetheless these are self-sterile. Various insects, when come across these spermagonia get dirty with gluey spermatia. When the insects come across continuous spermagonia, they transfer to the receptive hyphae spermatia from the opposite, compatible type. These spermatia fertilize receptive hypha which then yields dikaryotic mycelium as well as spores which have 2 nuclei. These dikaryotic spores exhibit completely changed properties. Like, these can contaminate a totally changed host from plant on which these were created. Participation by insects in rust infections is, thus, vital in both the spreading of spores to novel hosts and further notably, in the fertilization of fungus and, thus, in growing the potential of fungus to yield novel and perhaps new strong races.

## **Ergot of cereals and grasses**

This disease is due to fungi *Claviceps purpurea* that grows in flowers and yields spores that are confined in a sugary and gluey material. The material is attractive for various insects, mainly flies as well as beetles. Insects feed on spores and get smeared with them outwardly and transfer them, on the outside and through their feces, to strong flowers. While major contaminations due to ergot fungi are chiefly from ascospores created from sclerotia overwintering on earth and carried through air streams, insect broadcast of conidia is significant for secondary broadcast of infection and for broadcast over extended spaces. Some beetles though feed on ergot sclerotia on earth and can transfer mycelium and ascospores on their bodies to fit plants and through them can cause main contaminations.

## **Rots of fleshy fruits**

### **Fig rots**

Endosepsis of figs: This disease is caused by fungi *Fusarium moniliforme* and it outcomes in the form of whole fruit contents into a pulp. This fungus is spread among the fruits by fig wasp namely *Blastophaga psenes* that similarly shows a vital character in pollination process for figs. Fig trees these being dioecious, include male trees which yield staminate flowers about opening and gall flowers in cavity and female trees which yield pistillate flowers. Fig wasps lay eggs in ovules of gall flowers in male plants that are thus stimulated to cultivate. Eggs hatch and larvae parasitize galls till these go to pupation. Adults arise from pupae and females get fertilized though still in male fig. Once these come out of fig, females brush alongside staminate flowers surrounding the opening and turn into smeared with the pollen. Female wasps transfer pollen to male as well as female flowers they then visit for oviposition. In female flowers, though, for the reason that of length of styles, oviposition flops but pollination is however effective and fruit grow into eatable figs. If, still, as female wasp stays around diseased figs it develops smeared with spores of fungus and it spreads spores to male and female figs it stays, and fungus then sources endosepsis in female figs.

## Gray mold of grapes

This disease is caused by fungus namely *Botrytis cinerea*. Fungal spores usually blowout due to air streams. Infiltration of grapes as well as shoots, nevertheless, appear to be enlarged by injuries made on them by larvae of lepidopteran like *Argyrotaenia pulchellana* as well as *Lobesia botrana*.

## Boll rots of cotton

These are caused by some fungi comprising *Fusarium moniliforme*, *Aspergillus flavus*, *Rhizopus nigricans* and *Alternaria tenuis*. Numerous insects seemingly are involved in the spread for these fungi and they appear to use diverse mechanisms of spread. Therefore, in boll rot because of *Fusarium* as well as *Alternaria*, fungi enter cotton bolls by feeding and oviposition injuries prepared by boll weevil namely *Anthonomus grandis*, the cotton bollworm namely *Heliothis zea* and tarnished bug namely *Lygus lineolaris*, or these are transported to and enter by the nectarines by nectar nourishing flies like *Drosophila* and cabbage looper namely *Trichoplusia ni*. In boll rots due to *Aspergillus flavus* as well as additional aflatoxin creating species, fungus is chiefly wind dispersed but it is similarly carried inside and outwardly by insects, for example the lygus bug namely *Lygus hesperus* and stink bug namely *Chlorochroa sayi*, which often go through cotton bolls. Latter fungus, nevertheless, appears to rest on for arrival on the occurrence of big injuries like large exit holes created by developed larvae of pink bollworm namely *Pectinophora gossypiella*. Instead, boll rots due to *Rhizopus stolonifer* happen when injuries prepared by bollworm namely *Earias insulana* as well as by pink bollworm are existing. In lint rot of cotton, due to fungus namely *Nigrospora oryzae* fungus is spread very powerfully by mite named *Siteroptes reniformis*. Stigmatomycosis or internal boll disease which are caused by the fungus named *Nematospora gossypol*, cotton fibers get stained due to lack of outside symptoms. This disease is related to feeding of some species of plant bugs chiefly of the genus *Dysdercus*, frequently denoted as cotton stainers. These insects bring fungus spores outwardly on mouthparts and inside in deep stylet pouches and present through their proboscis through the wall of new cotton bolls.

## **Molds and decays of grains and legumes**

Many decays as well as molds attack many grains and legumes although still in field and their incidence and severity rise as the amount of insects infesting the crops, and feeding on the seeds, increases. In corn for example seed rots can be due to species of fungi like *Fusarium*, *Trichoderma*, *Cephalosporium*, *Physalospora*, *Gibberella*, *Diplodia*, *Nigrospora*, *Cladosporium*, *Penicillium*, *Aspergillus*, *Rhizopus*, and others. Insects most usually spreading and enabling contamination of corn kernels due to this fungi are corn earworm, (*Heliothis zea*), and European corn borer namely *Pyrausta nubilalis* nonetheless other borers and insects likewise play significant roles of vectors and notably, as helpers of contamination by this fungus by making injuries which let the mold enter the kernels. In seed contaminations by *Aspergillus* and *Fusarium* the additional hostile effect of creation of unbearable mycotoxins are also there. Like, while not as much of considered conditions are described for rice contaminations by fungus for example *Nematospora corylii*, spread and assisted by injuries through due to rice stinkbug namely *Oebalus pugnax*, wheat as well corn contaminations due to fungus namely *Nigrospora* sp. As well as *Fusarium poae*, spread from large amounts of *Pelliculopsis* mites nourishing on and carrying spores of the fungus.

## **TOPIC 6. INSECT TRANSMISSION OF PLANT PATHOGENIC BACTERIA**

### **Bacterial Diseases**

#### **Soft rot of Potatoes**

Seed Corn maggot (*Delia platura*) (Diptera: Anthomyiidae) described to play an essential part in spreading and expansion of bacterial soft rot in vegetables like potatoes both in field and storage. Soft rot bacteria typically bring together in a field of potato on diseased or adulterated kernel quantities nonetheless they can similarly live in all phases of insect, comprising pupae and in that case they might endure cold or dry climate environments. Insect larvae turn out to be polluted with bacteria as these feed in or creep about on those diseased seed fragments. They also bring bacteria to fit plants and in that case they submit them into injuries they make. Even as soon as the plants or storing organs are resistant against soft rot bacteria besides can usually discontinue



the development of the bacteria by evolving a hurdle of cork coatings, maggots abolish the cork coatings as fast as these are shaped and soft rot remains to blowout.

Some other associated flies for instance like the maggot *Delia florilega*, *Drosophila busckii* and perhaps others appear to have similar connection to soft rot of potato and additional fleshy tissues. It has exposed that some flies have parallel connections to soft rot bacteria and host plants on whom they wish to feed. Such associations, for instance, occur amid the cabbage maggot and soft rot in the Brassicaceae, *Delia antiqua*, *Tritoxa flexa*, the seedcorn maggot, and the onion bulb fly, *Eumerus strigatus*, the soft rot of onion and the iris borer, *Macronoctua onusta* (Grote) (Lepidoptera: Noctuidae) and soft rot of iris.

There is slight uncertainty, though, that insect broadcast of soft rot bacteria occurs in a way that insects aid make known to the bacteria into injuries they open, and the occurrence of pests in soft-rotting tissues hinders the resistance response of plants against bacteria. Insects by transporting the soft rot bacteria inside their bodies, aid bacteria endure hostile ecological circumstances. Instead, bacteria appear to aid their insect vectors by making for them a further nutritious substrate through half-done softening of host tissues.

### **Bacterial wilts of plants**

In some bacterial infections in plants, bacteria go in the xylem of plants and move, reproduce and block those vessels. Blocking xylem vessels is enlarged due to materials coming through cell walls due to enzymes of bacteria and this affects the water translocation over the shoots of the plant. Consequently due to scarce water, leaves and shoots drop turgor, wilt and finally go brown and die as well. In roughly bacterial wilts, the bacteria abolish and soften parts of the xylem walls and travel in the neighboring tissues where they make pockets full of bacteria from which bacteria exude out on plant surface through fissures or normal openings. In other bacterial wilts, bacteria stay restricted in xylem and do not grasp plant surface till plant is destroyed by infection.

Wilt producing bacteria hibernate in plant debris in soil, in seed, in somatic propagative material, and some cases include in insect vector. These go in plants from injuries, and these blowout from plant to plant from soil, from tools and direct handling of plants, or from insect vectors. Most significant bacterial wilts within which insects show a major role in the broadcast of bacteria from plant to plant are termed briefly below.

## **Bacterial wilt of cucurbits**

Bacterial wilt of cucurbits is described from maximum established nations but it possibly happens all over world. It disturbs numerous classes of cucurbits, comprising cucumber, squash, pumpkin and muskmelon. Watermelon is hardy or resistant to bacterial droop/wilt. Sick plants change a rapid wilting of leaves and climbers and finally die. Unhealthy squash fruit changes a slimy rot in storing. Losses as of bacterial wilt differ from an rare wilted plant to damage of 75 to 95% of crop. Bacterial wilt of cucurbits is due to bacterium namely *Erwinia tracheiphila*. Bacterium endures inside diseased plant remains for a small number of weeks but it lives over season in intestines of two insect vectors, striped cucumber beetle and the spotted cucumber beetle. The bacterium depends on these two vectors for its broadcast to and injection of new plants. In spring some striped cucumber beetles as well as to a smaller degree, spotted cucumber beetles, which bring bacteria, forage and source injuries on leaves of cucurbits. Insects submit bacteria in injuries by their feces and bacteria go in the injured xylem where these reproduce quickly and by which they move to different portions of plant. In xylem, bacteria defecate polysaccharides, discharge enzymes which disrupt cell wall materials, and make xylem parenchyma cells to yield tyloses in xylem. All of these collected procedure gums or gels that block vessels, particularly at end walls, thus dropping upward movement of water in xylem by equal to eighty % and producing leaves and vines go wilt. Beetles feed on diseased cucurbits pick bacteria in mouthparts and as they feed on fit plants they submit bacteria in new injuries they made. Therefore, bacteria begin new contamination. Each polluted beetle can contaminate numerous fit plants afterward one feeding on a diseased plant. It appears that a relatively small portion of insects change transporters of bacteria over the winter. Spotted cucumber beetles spread wilt bacteria somewhat late in season, so they are measured less significant vectors of this infection compared to lined cucumber beetles.

## **Fire blight of pears, apples and other rosaceous plants**

The disease is caused due to bacterium *Erwinia amylovora*. It is the most overwhelming disease affecting rosaceous plants. Symptoms involve diseased flowers and new shoots which become discolored and water soaked, then killed quickly and seeming brown to black as though scorched by fire. The disease spreads quickly into bigger branches, which it also kills, and parts of or complete trees may be killed. At the base of branch or twig infections, cankers grow at margins

of which the bacteria hibernate. Fruit too convert diseased and exude bacterial droplets. Bacteria kill and soften contents of chiefly parenchyma cells on flowers and in bark of new shoots, but as they abolish these cells they leave mass in the bark. Bacteria also go in phloem and xylem by which they could move over comparatively short spaces.

The fire blight bacteria hibernate at margins of cankers of plant parts. In spring, the bacteria about cankers increase and their derivatives absorb water and build interior pressure. This outcomes in drops of fluid holding masses of bacteria discharge out of the cankers. Bacteria in ooze are dispersed by splashing shower and likewise by insects, some of which are concerned to bacterial discharge, and their legs, mouthparts and bodies converted smeared with bacteria. More than two hundred species from numerous insect groups, together with aphids, psyllids, beetles, flies, leafhoppers and ants, have been revealed to go to oozing cankers fit blossoms, though bees and wasps appear not to visit oozing cankers regularly. Insects which get smeared with bacteria coming out at cankers bring bacteria to new shoots somewhere they submit them in present injuries or in fresh wounds they make upon feeding, or in the nectar of the flowers. Once the fire blight bacteria are transmitted to blossoms by rain or insects, they enter the flower tissues through nectarhodes or injuries, grow quickly in them, and exude out of them and commingle with the nectar in the flower. Same kinds of insects seemingly can spread fire blight bacteria from diseased to fit flowers but flower to flower spread of fire blight bacteria is done so much proficiently by pollinating insects, namely bees that the involvement of other insects to that kind of spread appears to be comparatively unimportant. As honeybees, bumblebees, wild bees, wasps and other insects visit apple, pear and other flowers diseased with fire blight bacteria, their legs, mouthparts and other body parts converted smeared with bacteria in nectar. Insects at that time bring bacteria and submit them in nectar of fit flowers they visit and there bacteria begin new contaminations. Bacteria, though, do not endure on or in the insects for more than a few days and do not seem to hibernate in link with the insects.

### **Citrus greening disease**

Citrus greening is damaging infection of all kinds of citrus. It happens in maximum citrus generating zones of Asia, together with the Arabian Peninsula, and in Africa. The disease is due to bacterium *Liberobacter asiaticum* in Asia and *L. africanum* in Africa. Both bacteria are

restricted to phloem of host plants and have yet to be cultured. The infection first seems as chlorosis and leaf mottling on one shoot or branch, due to which it has the name “huanglongbing”, or “yellow shoot”, in Chinese. Later on, whole trees become chlorotic as though they are suffering from zinc deficiency, their twigs die back, and the trees decline rapidly and become non-productive. Fruit on diseased trees is small, lopsided, and does not color uniformly as it ripens but large parts of it remain green even when mature, thereby the “greening” name of the disease. Diseased fruit is also quite bitter.

Citrus greening is spread by vegetative propagation with buds and grafts, and by at least two citrus psyllids: *Diaphorina citri* Kuwayama, which is the principal vector of the more severe and more destructive Asian form of the citrus greening bacterium that occurs at higher temperatures (30 to 35°C), commonly found at lower elevations; and *Trioza erytreae* Del Guercio, which is the principal vector of the milder, less severe, lower temperature (27°C) African form of the bacterium, which is normally found at higher elevations. Both vectors, however, can transmit both forms of the bacterium. Asian psyllids acquire the bacterium within 30 minutes of feeding while African psyllids require 24 hours. The bacterium apparently multiplies in the vector and can be transmitted within 8 to 12 days from acquisition.

Infected plants and vectors have been introduced into several citrus-producing countries but in most cases it was eradicated before it could become established. The vector of the greening bacterium *Diaphorina citri* was introduced in Brazil in the early 1980s and in Florida in 1998 but, so far, the causal bacteria apparently have not been introduced and no trees have been found in either place to be infected with citrus greening.

### **Cucurbit yellow vine disease**

Yellow vine disease affects watermelon, melon, squash, and pumpkin. It was first reported in the Texas-Oklahoma area and has since been found in Massachusetts, New York, and Tennessee. Affected plants show vines with yellow leaves, the phloem of leaves and vines becomes discolored, and the leaves and vines collapse and die. The disease has been severe in the Texas and Oklahoma areas where it annually destroys thousands of acres of cucurbits costing millions of dollars.

Cucurbit yellow vine disease is caused by a phloem-limited bacterium that has been placed in the species *Serratia marcescens* and its properties are still being characterized. The bacterium is most

probably transmitted by insect vectors. The squash bug, *Anasa tristis*, is considered to be a vector of this bacterium, but its involvement in transmitting this bacterium has been questioned.

## **TOPIC 7. INSECT TRANSMISSION OF PLANT PATHOGENIC MOLLICUTES**

### **Insect transmission of plant diseases caused by mollicutes**

Mollicutes are prokaryotes (bacteria) that lack cell walls. In nature, plant pathogenic mollicutes are limited to the phloem of their host plants. All mollicutes, that is, phytoplasmas and spiroplasmas, are spread from plant to plant through vegetative propagation and, in nature, these pathogens depend for their transmission on phloem-feeding, sap-sucking insects, mainly leafhoppers, planthoppers, and psyllids. These insects can acquire the pathogen after feeding on appropriate infected plants for several hours or days, or if they are artificially injected with extracts from infected plants or insects. More insects become vectors when they feed on young leaves and stems of infected plants than on older ones. The insect vector cannot transmit the pathogen immediately after feeding on the infected plant but it begins to transmit it after an incubation period of 10 to 45 days, depending on the temperature. The quickest transmission (10 days) occurs at about 30C, while the slowest (45 days) takes place at about 10C.

### **Aster yellows**

Aster yellows is caused by phytoplasmas and occurs worldwide. It affects numerous annual crops, mostly vegetables and ornamentals, for example, tomato, lettuce, carrot, onion, potato, chrysanthemum, aster, and many others, on which it causes severe symptoms and serious losses, in some crops amounting to 10-25% of the crop and occasionally up to 80-90% of the crop. Plants infected with aster yellows develop general chlorosis (yellowing) and dwarfing of the whole plant, abnormal production of shoots and, sometimes, roots, sterility of flowers, malformation of organs, and a general reduction in the quantity and quality of yield. The aster yellows phytoplasma is transmitted by several leafhoppers, one of which is the aster leafhopper *Macrostelus fascifrons*. The various leafhopper vectors have a wide host range, as does the aster yellows phytoplasma. The phytoplasmas survive in perennial ornamental, vegetable, and weed plants. The vector leafhopper acquires the phytoplasmas while feeding by inserting its stylet into the phloem of infected plants and withdrawing the phytoplasmas with the plant sap. After an incubation period, when the insect feeds on healthy plants it injects the phytoplasmas through the stylet into the phloem of the healthy

plants where they establish a new infection and multiply. The phytoplasmas move out of the leaf and spread throughout the plant causing the symptoms characteristic of the host plant.

### **Tomato big bud**

The disease occurs in many parts of the world but except for a few areas, it is of little economic importance. It affects most Solanaceous vegetables and lettuce. The symptoms include small, distorted, yellowish green leaves and production of numerous thickened, stiff, and erect apical stems that have short internodes. The flower buds are excessively big, green, and abnormal looking, and fail to set fruit. Fruit present when infection takes place becomes deformed.

Tomato big bud is caused by a phytoplasma that is transmitted by several leafhoppers, the main one of which is the common brown leafhopper *Orosius argentatus*. The insect feeds and breeds on infected weed hosts and when they become undesirable the insects move into tomato or other crops bringing with them the big bud phytoplasmas.

## **TOPIC 8. MANAGEMENT OF INSECT-VECTORED PATHOGENS OF PLANTS**

This section is organized according to the concept of integrated pest management, including chemical control, biological control, host plant resistance, cultural control, and regulatory measures. Each of these categories will be considered in terms of the vectors and the pathogens.

### **Chemical control**

Chemical control of vectors will limit spread of vectored plant pathogens if two conditions are met. First, the vector being controlled must be a colonizer of the crop being treated. Some plant viruses are transmitted by passing flying insects. In these cases, chemical control has a poor record. Second, most of the spread must come from within the treated area (secondary spread).

For the most part, insecticides simply prevent the buildup of populations of insects within a treated area. Most pesticides will not kill an immigrant insect before it has a chance to transmit a pathogen.

Thus, if there is an infected field nearby with a high population of vectors, pesticide treatment may not be very effective in preventing infection from primary spread. Pesticide treatment has a good track record for control of certain luteovirus diseases such as potato leafroll and barley yellow dwarf, spread by colonizing aphids. Even if the above conditions are met, dependence on chemical control of vectored pathogens in perennial crops may be risky, because a single missed spray application or insecticide failure could result in infection and demise of the crop.

Another variation on chemical control is the use of stylet oil. Certain horticultural oils can reduce transmission of viruses by aphids. This methodology can reduce transmission of pathogens carried by transient insects, something that is not usually possible with standard insecticides. There is also potential for use of insect repellents for vector management. Some work has been done with visual repellents (mulches, paints, colored netting), but little has been done with chemical repellents.

There is no known chemical control of plant viruses, but some fastidious bacteria in perennial tree crops can be controlled with injected antibiotics. Remission of symptoms usually is temporary.

### **Biological control**

The use of biological control for management of vectored pathogens has a mixed review. Obviously, fewer vectors is better; however, sometimes the economic threshold for vectors is so low that it is not achievable through biological control. Sometimes, the presence of natural enemies evokes scatter responses in vector prey. This can actually cause an increase in pathogen transmission.

Total dependence on biological control for a vectored pathogen in a perennial crop is risky. Biological control inherently fluctuates between high populations of pests and high populations of natural enemies. If pests are not vectors, the crop often can stand temporary high pest populations, but if the main concern is pathogen transmission, the entire crop can be lost if populations fluctuate in favor of the pest. Similarly, often a grower will have to spray for another pest. The pesticide may kill the natural enemies of the vector, resulting in an increase in transmission of the pathogen and destruction of the crop.

Biological control in simpler island ecosystems may have a better chance to work than in more complex settings. For example, the introduction of psyllid parasitoids into Reunion Island dramatically reduced the transmission of the bacteria that cause citrus greening disease. However,

the same parasitoids are present in Viet Nam, and citrus greening disease is a major limiting factor in citrus production there.

Sometimes disruption of existing biological control can result in an increase in transmission of a plant pathogen. Increased spraying for late blight (caused by a fungus) in potatoes was linked to a major increase in numbers of green peach aphids. Evidently, populations had been controlled by an aphid pathogenic fungus that was killed by the fungicide applications for late blight. The result was a big increase in incidence of potato leafroll virus (PLRV), transmitted by the aphids.

Biological control of the pathogen is another option. Usually this is done by means of cross protection - infecting a crop with a mild strain of a pathogen that prevents symptom expression of a more severe strain that may be transmitted by an insect later on. Cross protection has worked very well for control of citrus tristeza virus, an aphid transmitted closterovirus, in several parts of the world.

### **Host plant resistance**

The efficacy of plant resistance to vector depends on the means of resistance and the mode of transmission. Resistance that prevents feeding or repels the insects can prevent transmission of pathogens spread by feeding. If the resistance merely prevents or slows population growth, it cannot prevent primary spread. It can, however, have some effect on secondary spread.

Resistance to the pathogen probably is the most effective means of controlling vectored plant pathogens. If plant resistance to the pathogen is available, it should be the first line of defense. Complications with host plant resistance can occur if the pathogen evolves strains that can break down the resistance. Also, some crops are notoriously difficult to breed due to difficult genetics or longevity.

Resistance to the pathogen may be the only means of management in some cases. An example is sugarcane mosaic virus. Sugar cane is propagated vegetatively, so the virus is propagated with the crop. Once planted, a crop remains in the same field for several years of production, and planting is staggered throughout the production area. Thus, even if clean planting stock could be found, there is no possibility for an area-wide crop-free (and virus-free) period. The virus is transmitted by transient winged aphids, including species that do not necessarily colonize the sugar cane crop, so pesticide application is not effective. Fortunately, there has been success with mosaic resistant cultivars.



## **Cultural control**

Many of the effective management practices for diseases caused by vectored plant pathogens involve some sort of cultural control such as adjusted planting date, pruning, roguing, and removal of volunteer crop plants and other non-crop reservoir hosts of vectors or pathogens.

Adjusting planting dates can minimize crop exposure to vectored pathogens. In the Pacific Northwest, several vectored pathogens, transmitted by eriophyid mites and aphids, can damage early-planted fall cereals. The mite transmitted viruses are particularly serious because they cause severe yield loss, resulting in total crop failure in some cases. These viruses infect newly seeded winter wheat that is planted near a maturing crop from the previous season, or near infected volunteer grain. These viruses are most troublesome in years when the old crop is slow to mature, and there is overlap between late-maturing cereal crops from the previous season and emergence of newly sown winter wheat. The infective mites are blown from the old crop to the new one. After landing on the new crop, they apparently stay put, because there is little secondary spread in the fall. The mites cannot survive very long off a host plant, so any break in the “green bridge” between old and new crops will prevent infection. As little as 10 days delay in planting can make the difference between crop failure and negligible incidence. Similarly, the aphid-transmitted barley yellow dwarf virus complex largely can be prevented by delay in planting. Other viruses are managed on an area-wide basis by maintaining a crop-free period at some time during the year. For such a policy to work, all growers in the area must cooperate in field clean-up and coordination of planting dates.

Pruning and roguing often are used as a means of management of vectored plant pathogens, especially in perennial crops. The efficacy depends on whether latent infections remain, and if so, whether plants with latent infections are suitable source plants for vectors. In the early stages of an epidemic, there usually will be more plants with latent infections than with visible symptoms. If plants with latent infections can serve as source plants for vectors to acquire the pathogens, and vectors are present, pruning and roguing will not be very effective.

In some cases, pruning actually can eliminate disease. Pierce’s disease of grape vines is caused by a xylem limited bacteria called *Xylella fastidiosa*. It moves slowly in the plants. If Pierce’s disease is transmitted by small leafhoppers that feed on twigs, winter

## **Regulatory measures**

Regulatory measures for control of vectored plant pathogens are a very important aspect of management, especially for those pathogens that are transmitted through propagation. Strict sanitation measures for propagative material are common. Other kinds of regulatory measures include crop-free periods, quarantines, and required virus testing.

One of the best ways to prevent vector-borne disease in plants is to keep the disease and the vector out. Many plant diseases and vectors that occur elsewhere in the world do not occur in the United States. Some of these are listed as quarantine or actionable pests/ diseases. If they are found at U.S. ports of entry, the shipment is rejected. If a pest or disease has a limited distribution in the U.S., there may be a state quarantine for certain items. For example, if a pest is found in Florida but not in California, California may reject shipments from Florida that contain the pest.

Production of healthy propagation materials involves regulatory agreements. Potato tubers grown for seed are subject to winter testing for a variety of vector-borne viruses. Standards are much stricter for early generation seed. Citrus trees are propagated vegetatively by grafting, in order to ensure varietal uniformity. Citrus trees used for budwood in Florida are required to be tested for citrus tristeza virus every year. Those found to contain severe strains are no longer allowed to be used for propagation purposes. Lettuce mosaic virus and bean common mosaic virus are transmitted by seed as well as by aphid vectors. Some states have regulations in place requiring that seed used commercially in the state be tested and meet standards for virus incidence.

In warm climates where crops are grown year around, control of diseases caused by vectored pathogens can be particularly challenging. Sometimes an agreement is made to adhere to a crop-free period to break the disease cycle. State regulatory agencies may be involved to ensure that there are consequences for any growers that do not comply.

Other regulations are tailor-made for a given situation. Green peach aphids (*Myzus persicae* (Sulzer)) transmit potato leafroll virus (PLRV), which causes an important disease in potatoes. The aphids overwinter in peach and apricot trees. In the spring, they leave the trees and infest potatoes and other plants. In parts of Idaho where seed potatoes are grown, it is illegal to grow peach and apricot trees. If the trees are found, the state can require their removal. Guy Bishop, University of Idaho, discovered that another source of green peach aphids was greenhouse grown bedding plants. These infested seedlings were purchased by home gardeners, who often also grew potatoes. Frequently, the home grown potatoes either were grown from seed saved from previous years, or

from unregistered seed. Thus, the PLRV infection rate in home gardens was high. Bishop found that the closer a seed potato field was to a town with home gardens, the more likely it was that the seed farmer had unacceptably high levels of PLRV. Regulations were made to prevent sale of infested bedding plants. Additionally, the local growers provided home gardeners with clean potato seed tubers. Incidence of PLRV in seed potatoes decreased dramatically in the region after the regulations were implemented.

### **Integrated management**

Most often control of vectored pathogens of plants will involve an integrated approach. A good example is potato production. Some popular varieties of potatoes develop internal discoloration as a result of infection with PLRV that results in rejection by potato processors. In order to prevent PLRV infection there are regulatory measures to ensure clean propagation material. Additionally, commercial ware potato growers employ scouts to survey for green peach aphids. If numbers reach an economic threshold the crop is treated with insecticide to prevent secondary spread of PLRV. Cultural controls include removal of volunteer potatoes, and in some cases, removal of peach and apricot trees that are overwintering sites for vectors. Department of Agriculture inspectors make the rounds of retail vendors of bedding plants, preventing sale of infested ones. Finally, there is work at the federal and state levels to breed potato varieties that are more tolerant to PLRV infection, but that also retain the taste, baking and processing qualities of the popular susceptible cultivars. Epidemiology of vectored pathogens affecting crops is a complex and very interesting field of study. Many more puzzles remain to be solved that will make even more effective management a possibility.

## **TOPIC 8. STUDY OF CAUSAL ORGANISMS, IDENTIFICATION, DISEASE CYCLE, SIGNS, SYMPTOMS**

**Please see next pages in slides where all the relevant information is available**





# Introduction to Plant Pathology



# What is a plant disease?

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- A plant disease is any abnormal condition that alters the appearance or function of a plant. It is a physiological process that affects some or all plant functions. Disease may also reduce yield and quality of harvested product.
- Disease is a process or a change that occurs over time. It does not occur instantly like injury.



# What is a plant disease?

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- Visible effects of disease on plants are called **symptoms**. Any detectable changes in color, shape, and/or functions of the plant in response to a pathogen or disease-causing agent is a symptom.
- **Signs** of plant disease are physical evidence of the pathogen, for example, fungal fruiting bodies, bacterial ooze, or nematode cysts. Signs also can help with plant disease identification.

# What causes plant disease?

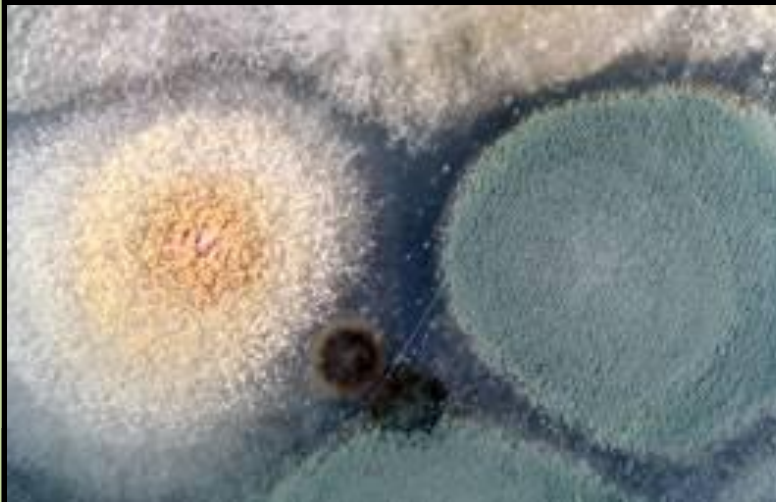
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- Infectious plant diseases are caused by living organisms that attack and obtain their nutrition from the plant they infect. The parasitic organism that causes a disease is a **pathogen**. Numerous fungi, bacteria, viruses, and nematodes are pathogens of corn and soybean in Iowa.
- The plant invaded by the pathogen and serving as its food source is referred to as a **host**.

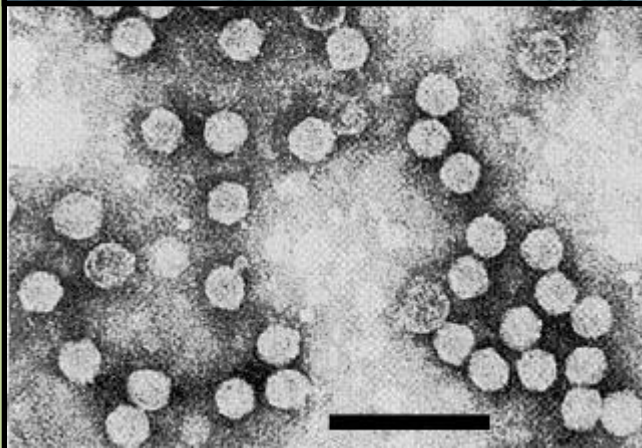
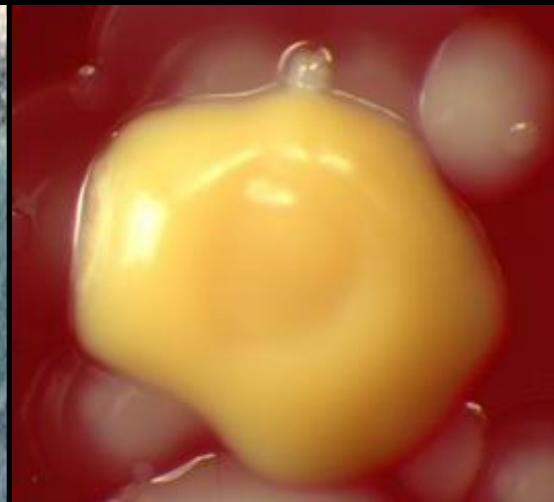
# Types of pathogens

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Fungi



Bacteria



Viruses



Nematodes



# Role of the environment

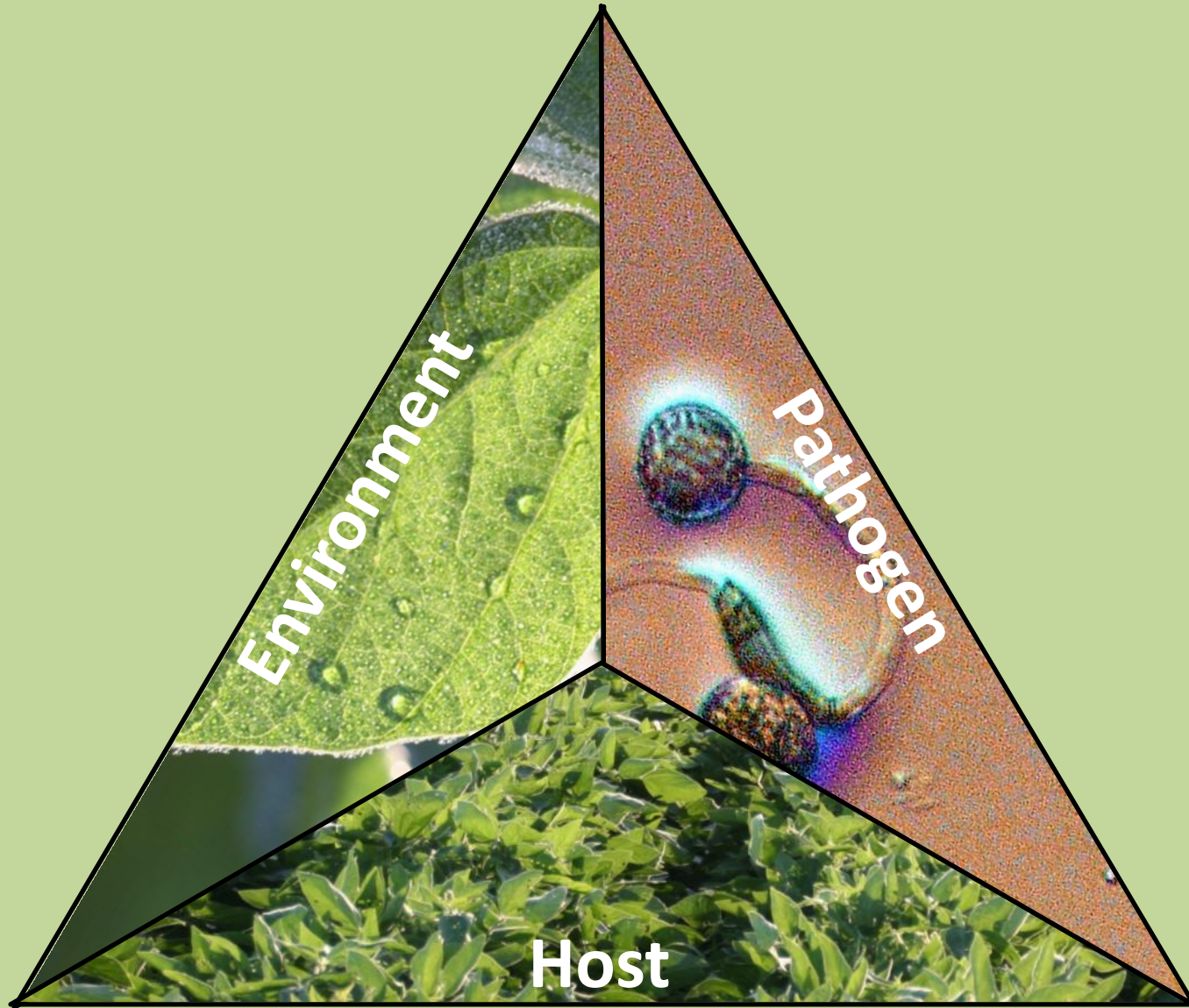
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- A **favorable environment** is critically important for disease development – even the most susceptible plants exposed to huge amounts of a pathogen will not develop disease unless environmental conditions are favorable.



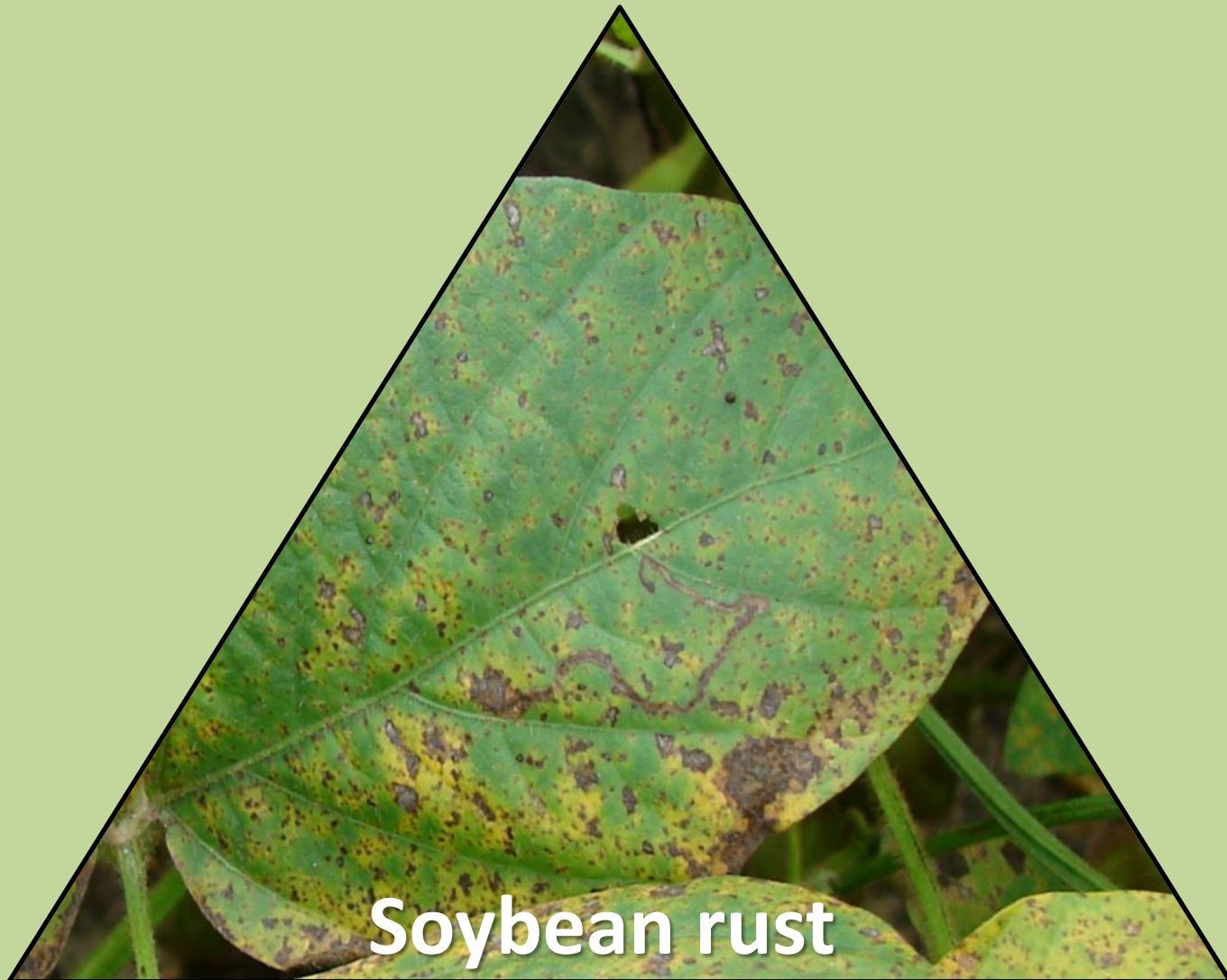
# The Disease Triangle

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# The Disease Triangle

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Soybean rust



# Groups of plant pathogens - fungi

- Vast majority are beneficial
- Can cause plant, human, and livestock diseases
- Most cannot be seen without a microscope
- Lack chlorophyll
- Composed of growing structure of delicate, threadlike filaments called hyphae
- Reproduce by forming spores



# Groups of plant pathogens - bacteria

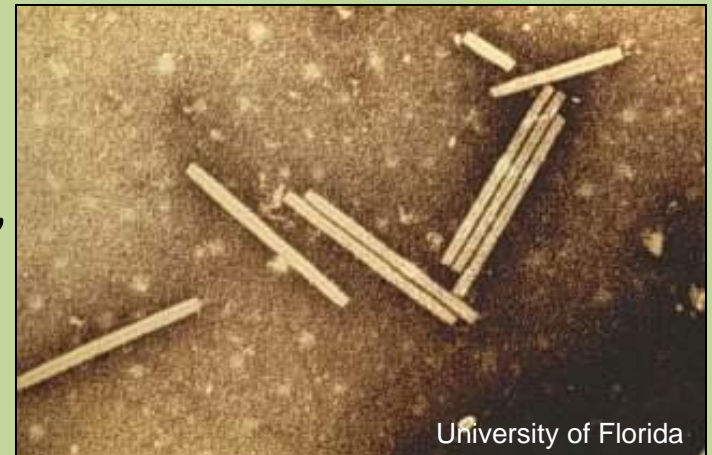
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- Extremely small organism requiring microscope to be seen
- Bacteria population can increase in number in short time period
- Cells clump together in masses called colonies
- Obtain food from dead or decaying organic matter or living tissue
- Spread plant to plant by wind-driven rain
- Gain entrance through natural plant openings or injuries



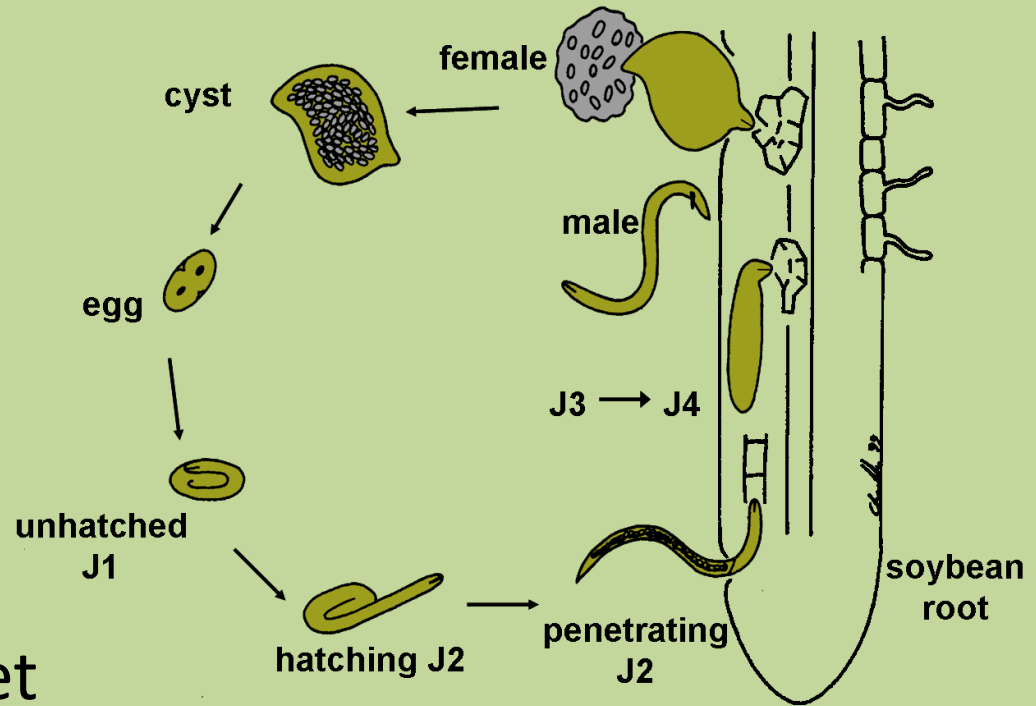
# Groups of plant pathogens - viruses

- Most familiar because they cause human and animal diseases such as influenza, polio, rabies, smallpox, and warts
- Cause some destructive plant diseases
- Measure only about one-millionth of an inch in size
- Are not complete living systems
- Survive only in living cells
- Transmitted by insects which are called vectors



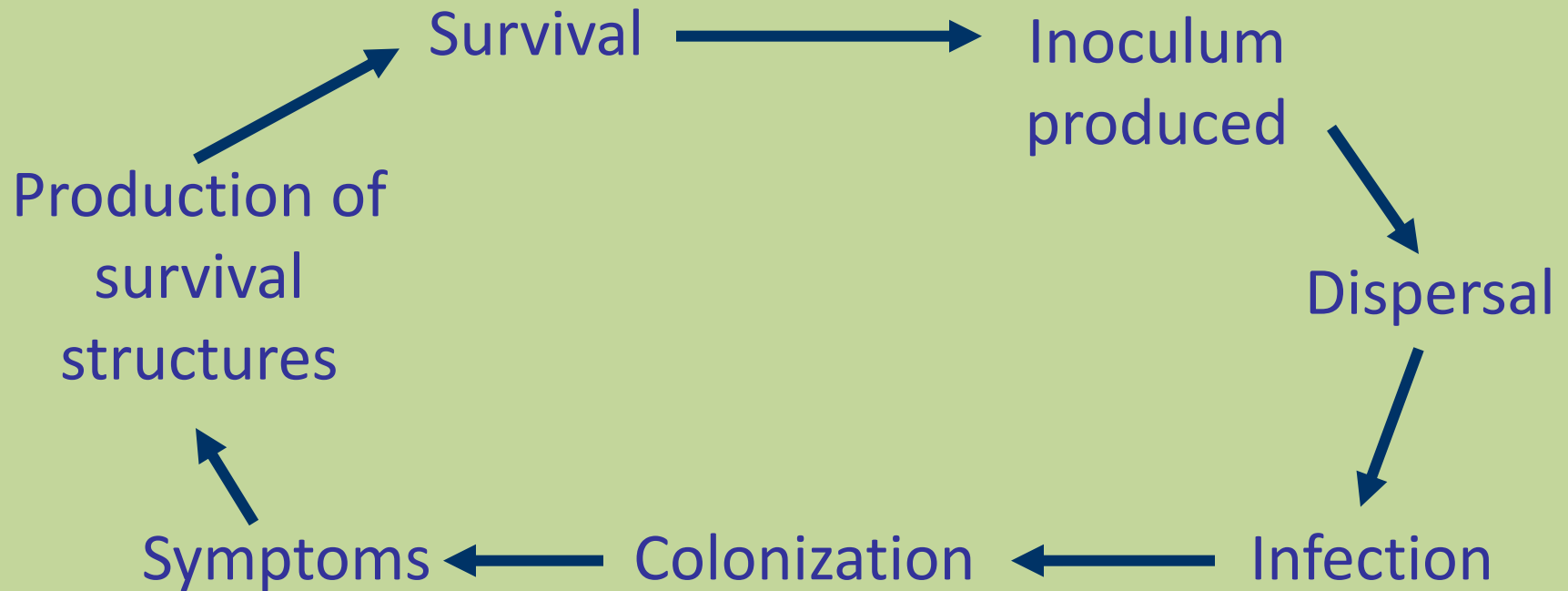
# Groups of plant pathogens - nematodes

- Round, slender, threadlike worms
- Some are parasites on animals, insects, fungi, other nematodes, and plants
- Plant-parasitic nematodes have a stylet
- Most live in the soil and feed in or on plant roots



# Disease cycle

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# Comparison of disease cycles

	Fungi	Bacteria	Viruses	Nematodes
Survival	Crop residue	Crop residue	-	Crop residue
	Soil	Soil	-	Soil
	Alt. hosts	Alt. hosts	Alt. hosts	-
	-	Insect vectors	Insect vectors	-
Dispersal	Wind	Wind	-	Tillage
	Rain	Rain	-	Equipment
	Insects	Insects	Insects	Water run-off
Infection	Directly	-	-	Directly
	Wounds	Wounds	-	-
	Insect feeding	Insect feeding	Insect feeding	-

# Inoculum

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## Source of inoculum varies for each disease

- May be produced on residues left in the field
- Present in the soil
- Present in weeds or other crops in the area
- Present in or on the seed
- Present in soil sticking to equipment or tools
- Carried by wind or water
- Carried by insect vectors
- Carried in by animals, birds, and people

# Spread of inoculum

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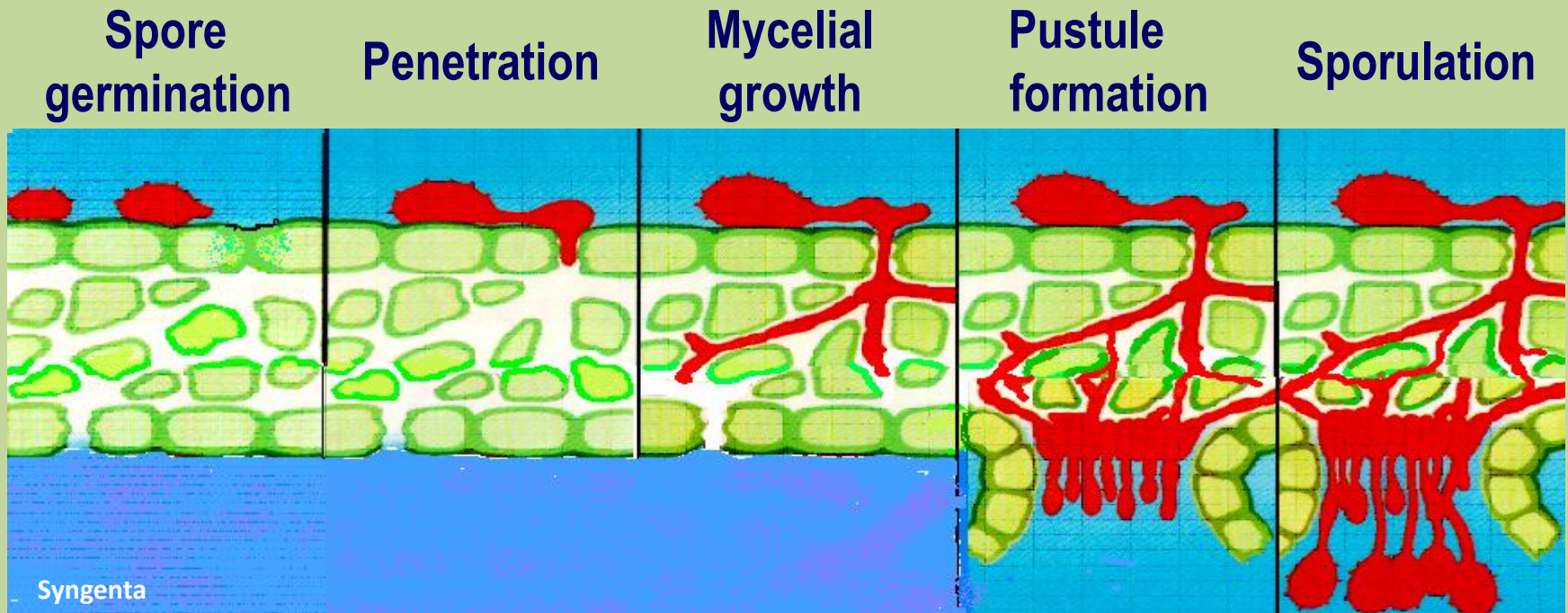
## Two ways

1. Plant placed in soil that contains a pathogen
2. Inoculum moves from its source to host plant



# Penetration of inoculum and infection

- **Infection** occurs when a pathogen successfully enters a plant and grows, reproduces, and spreads within the plant
- Pathogens enter a host through natural openings, wounds on plant surfaces, or by penetrating directly into the plant





# Secondary cycles

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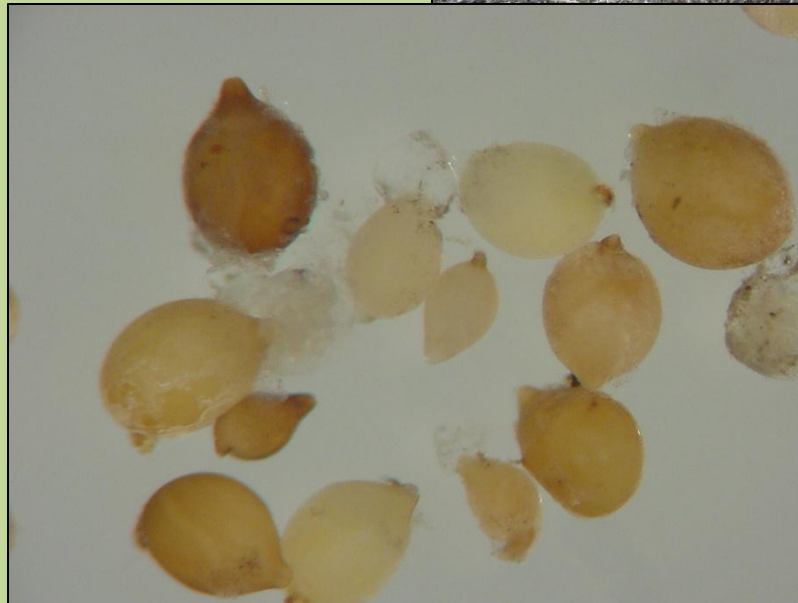
- Some diseases have only one cycle during the growing season (often root rots)
- Some diseases develop secondary or repeating cycles during the growing season (often foliar diseases)
- Number of cycles depends on the pathogen, susceptibility of the host, and environmental conditions



# Pathogen survival

## Pathogens survive season to season in:

- Soil
- Crop residue
- Weed or noncrop hosts
- Seed or vegetative plant parts
- Insects
- Mild climates



# Summary

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- Understanding the difference between a sign and a symptom is key in identifying a plant disease
- A plant disease cannot develop if a susceptible host, pathogen, and favorable environment do not occur simultaneously
- The major plant pathogens responsible for disease development in plants are fungi, bacteria, viruses, and nematodes
- The disease cycle describes the interaction of the pathogen with the host