LECTURE NOTE

PLANT – PATHOGEN INTERACTION

The mode of attack of plant by plant pathogens

Plants exist in a world filled with microorganism. They continue to grow in the same location throughout the growing season or for many years, as in the case of perennial plants. Their surfaces are constantly exposed to bacteria, fungi, nematodes, and possibly parasitic plants. They may be inoculated with viruses during feeding by insects or by other vectors. Plant pathogens have made many adaptations to enable them to invade plants, overcome plant defense mechanisms, and colonize plant tissues for growth, survival, and reproduction. Once established inside the plant, they have at least temporarily escaped the intense competition from saprophytic organisms on plant surfaces and in the soil. Therefore, for a pathogen to infect a plant, it must be able to make its way in to and through the plant, obtain nutrients from the plant, and neutralize the defense reactions of the plant.

Pathogens accomplish these activities mostly through secretions of chemical substances that affect certain components or metabolic mechanisms of their hosts. Penetration and invasion, however, seem to be aided by or in some cases be entirely the result of, the mechanical force exerted by certain pathogens on the cell walls of the plant.

(a) Mechanical forces exerted on host tissues by pathogens

Viruses are usually introduced directly through the plant cells by insects, therefore they do not exert mechanical forces. Many fungi are known to apply mechanical forces on the plant they are about to attack. When fungus lands on a plant surface, and contact is established, diameter of the tip of the hypha or radical in contact with the host increases and forms the flattened, bulb-like structure called the APPRESSORIUM. This increases the area of adherence between the two organisms and securely fastens the pathogen to the plant. From the appressorium, a fine growing point, called the PENETRATION PEG arises and advances into and through the cuticle and the cell wall.

(b) Chemical weapons of pathogens

Although some pathogens may use mechanical force to penetrate plant tissues, the activities of pathogens in plant are largely chemical in nature. Therefore, the effects caused by pathogens on plants are almost entirely the result of biochemical reactions taking place between substances secreted by the pathogen and those present in or produced by the plant.

The main groups of substances secreted by pathogens in plants that seem to be involved in production of disease, either directly or indirectly, are enzymes, toxins, growth regulators and polysaccharides (plugging substances). These substances vary greatly as to their importance in pathogenicity, and their relative importance may different from one disease to another. In general, plant pathogenic enzymes disintegrate the structural components of host cells, breakdown inert food substances in the cell, or affect components of its membranes and the protoplast directly, thereby interfering with its functioning systems. Toxins seem to act directly on protoplast components and interfere with the permeability of its membrane and with its functions. Growth regulators exert a hormonal effect on the cells and their increase or decrease their ability to divide and enlarge. Polysaccharides seem to play a role only in the vascular diseases, in which they interfere passively with the translocation of water in the plants.

(i) Enzymes

(a) Cutinases: Cutin is the main component of the cuticle. The upper part of the cuticle is admixed with waxes, whereas its lower part in the region where it merges into the outer walls of epidermal cells, is admixed with pectin and cellulose. Cutinases break down cutin molecules and release monomers as well as oligomers of the component fatty acid derivatives from the insoluble cutin polymer e.g. *Fusarium* spp and *Botrytis cinerea.*

(b) Pectinases: Pectin substances constitute the main components of the middle lamella i.e. the intercellular cement that holds in place the cells of plant tissues. Several enzymes degrade pectic substances and are known as pectinases or pectolylic enzymes. The first pectic enzymes is pectin methyl esterases, which removes small branches off the pectin chains. The second pectic enzymes is a chain splitting pectinases called polygalacturonases. It split the pectic chain by adding a molecule of water and breaking the linkage between two galacturonan molecules. Pectin lyases (3^{rd} pectic enzymes) split the chain by removing a molecule of water frm the linkage, therby breaking it and releasing products with an unsaturated double bond. Examples of pathogens include *Ralstonia solanacearum, Didymella bryoniae.*

(c) Cellulases: Cellulose is also a polysaccharide, but it consists of chains of glucose (1-4) β-D-glucan molecules. The glucose chains are held to one another by a large number of hydrogen bonds. Glucose is produced by a series of enzymatic reactions carried out by several cellulases and other enzymes. One cellulase (C_1) attacks native cellulose by cleaving croos-linkages between chains. A second cellulose (C_2) also attacks native cellulose and breaks it into shorter chains. These are then attacked by a third group of cellulases (Cx) whch degrade them to the disaccharide cellobiose. Finally, cellobiose is degraded by the enzyme β- glucosidae into glucose.

Saprophytes fungi, mainly certain groups of basidiomycetes, and to a lesser degree, saprophytic bacteria cause the breakdown of most of the cellulose decomposed in nature.

In living plant tissues, however, cellulolytic enzymes secreted by pathogens play a role in the softening and disintegration of cell wall materia**l.**

(ii) Microbial toxins

Toxins are metabolites that are produced by invading microorganisms and act directly on living host protoplast, seriously damaging or killing the cells of the plant. Some toxins act as ageneral protoplasmic poisons and affect many species of plant representing different families. Others are toxic to only a few plant species or varieties and are completely harmless to others. Many toxins exist in multiple forms that have different potency.

(a) Non-host specific toxin or non host-selective toxins

Several toxic substances produced by phytopathogenic microorganisms have been shown to produce all or part of the disease syndrome not only on the host plant, but also on other species of plants that are not normally attacked by the pathogen in nature.

(1) Tabtoxin- is produced by the bacterium *Pseudomonas syringae* pv *tabaci* which causes the wildfire disease of tobacco, by strain of pv *tabaci* occurring on other hosts such as bean and soybean and by other pathovars of *P. syringae* such as those occurring on oats maize and coffee.
(2) Phaseolotoxin- is produced by the bacterium *Pseudomonas syringae* pv *phaseolicola,* the cause of halo blight of bean and some other legumes.
(3) Tentoxin- is produced by the fungus *Alternaria alternata* which causes spots and chlorosis in plants by many species.
(4) Cercosporin- is produced by the fungus *Cercospora* and by several other fungi. It causes damaging leaf spot and blight diseases of many crop plants such as *Cercospora* leaf spot of Zinnia and gray leaf spot of corn.

(b) Host specific or host-selective toxins
This is a substance produced by a pathogenic microorganism that physiological concentrations, is toxic only to the hosts of that pathogen and shows little or no toxicity against non-susceptible plants.
(1) Victorin or HV toxin – is produced by the fungus *Cochliobolus Victoriae.* This fungus infects the basal portions of susceptible oat plants and produces a toxin that is carried to the leaves,causes a leaf blight and destroys the entire plant.
(2) T-toxin- is produced by race T of *Cochliobolus heterostrophus,* the cause of southern corn leaf blight. Race T is indistinguishable from other all other *C. heterostrophus* races except for its ability to produce the T toxin.

(3) HC-toxin- is produced by Race 1 of *C. carbonum* causing northern leaf spot and ear rot disease in maize.

(iii) Growth Regulators

Plant growth is regulated by a small number of groups of naturally occurring compounds that act as hormones and are generally called growth regulators. The most important growth regulators are auxins, gibberellins, and cytokinins, but other compounds, such as ethylene and growth inhibitors, play important regulatory roles in the life of the plant. Plant pathogens may produce more of the same growth regulators as those produced by the plant or more of the same inhibitors of the growth regulators as those produced by the plant. Pathogens often cause an imbalance in the hormonal system of the plant and bring about growth responses incompatible with the healthy development of the plant.

(a) Auxins- It occurs naturally in plants as indole-3-acetic acid (IAA). It is required for cell elongation and differentiation, and absorption of IAA to the cell membrane also affects the permeability of the membrane. Increased IAA levels occur in many plants infected by fungi, bacteria, viruses, nematodes and mollicutes, although some pathogens seem to lower the auxin level of the host e.g *Exobasidium azalea* causing azalea and flower gall, *Ustilago maydis* causative organism of corn smut.

(b) Gibberellins- These are normal constituents of green plants with a striking growth promoting effects. They speed the elongation of dwarf varieties to normal sizes and promote flowering, stem and root elongation and growth of fruits. The foolish seedling diseases of rice, in which rice seedlings infected with the fungus *Gibberella fujikuroi* grow rapidly and become much taller than healthy plants is apparently the result, to a considerable extent at least, of the gibberellins secreted by the pathogen.

(c) Cytokinins- These are potent growth factors necessary for cell growth and differentiation, and also inhibit the breakdown of proteins and nucleic acids, thereby causing the inhibition of senescence, and have the capacity to direct the flow of amino acids and other nutrients through the point of high cytokinin concentration. Cytokinin activity increases in clubroot galls, in smut and rust – infected bean leaves.it is partly responsible for several bacterial galls of leafy gall diseae of sweet pea caused by bacterium *Rhodococus fasciens*.

(d) Ethylene – Produced naturally by plants and exerts a variety of effects on plants, including chlorosis, leaf abscission, epinasty, stimulation of adventitious roots and fruit ripening. In the fruit of banana infected with *Ralstonia solanacearum*, the ethylene content increases proportionately with the (premature) yellowing of the fruits, whereas no ethylene can be detected in the healthy fruits.

(iv) Polysaccharides

Fungi, bacteria, nematodes and possibly other pathogens constantly release varying amounts of mucilaginous substances that coat their bodies and provide the interface between the outer surface of the microorganism and its environment. The role of the slimy polysaccharides in plant disease appears to be particularly important in wilt diseases caused by pathogens that invade the vascular system of the plant. Large polysaccharide molecules released by the pathogen in the xylem may be sufficient to cause a mechanical lockage of vascular bundles and thus initiate wilting.

Plants response to pathogens attack

Plants defend themselves against pathogens by a combination of weapons from two arsenals: (1) structural characteristics that act as physical barriers and inhibit the pathogen from gaining entrance and spreading through the plant and (2) biochemical reactions that take place in the cells and tissues of the plant and produce substances that are either toxic to the pathogen or create conditions that inhibit growth of the pathogen in the plant. The combination of structural characteristics and biochemical reactions employed in the defense of plants are different host-pathogen systems. However, whatever the kind of defense or resistance a host plant employs against a pathogen or against an abiotic agent, it is ultimately controlled, directly or indirectly by the genetic material (genes) of the host plant and of the pathogen.

(1) Structural defenses

(a) Preexisting structural defenses

This structural characteristic may already be present in plant even before the pathogen comes in contact with the plant.

(i) Wax and cuticle that cover the epidermal cells

Waxes on leaf and fruit surfaces forms a water-repellant surface, thereby preventing the formation of a film of water on which pathogens might be deposited and germinate (fungi) or multiply (bacteria).

(ii) Tough and thick epidermal cells

These are important factors in the resistance of some plants to certain pathogens by making direct penetration fungal pathogens difficult or impossible.

(iii)Nature of natural opening

Many pathogenic fungi and bacteria enter plants only through stomata. The structure of stomata e.g. a very narrow entrance and broad, elevated guard cells, may confer resistance to some varieties against certain bacterial pathogens.

(iv)Internal structural barriers

The cell walls of the tissue invaded vary in thickness and toughness and may sometimes inhibit the advance of the pathogen.

(b) Induced structural defenses

These are usually not present in the plants but are produce in response to invading pathogens.

(i) Cork layers formation

Infection by fungi or bacteria and even by some viruses and nematodes frequently induces plants to form several layers of cork cells beyond the point of infection, apparently as a result of stimulation of the host cells by substances secreted by the pathogen.

(ii) Formation of abscission layers

An abscission layer consists of gap formed between two circular layesrs of leaf cells surroding the locus of infection. Upon infection, the middle lamella between these two layers of cells is dissolved throughout the thickness of the leaf, completely cutting off the central area of infection from the rest of the leaf.

(iii) Formation of tyloses

Tyloses are overgrowths of the protoplast of adjacent living parenchymatous cells which protrude into xylem vessels through pits. Tyloses form an impenetrable barrier to the movement of water and nutrients.

(iv) Deposition of gums

The defensive role of gums stems from the fact that they are deposited quickly in the intercellular spaces and within the cells surrounding the locus of infection, thus forming an impenetrable barrier that completely encloses the pathogen.

(2) Biochemical defenses

(a) Preexisting biochemical defenses

(i) Inhibitors released by the plant in its environment

Plants exude a variety of substances through the surface of their aboveground parts as well as through the surface of their roots. Some of these exudates seem to have an inhibitory action against certain pathogens.

(ii) Inhibitors present in plant cells before infection

Some plants are resistant to diseases caused by certain pathogens because of one or more inhibitory antimicrobial compounds known as phytoanticipins, which are present in the cell the cell before infection.

(iii)Defense through deficiency in nutrients essential for pathogen

Species or varieties of plants that for some reason do not produce one of the substances essential for the survival of an obligate or for development of infection by any parasite, would be resistant to the pathogen that requires it.

(b) Induced biochemical defense

(i) Inhibitors produced by plants

Many plants produce substances in response to microorganism or to mechanical and chemical injuries e.g phenolics and phytoalexins

(ii) Defense through production of substances that inhibit effect of enzymes produced by pathogens

The production of substances which inhibit the effect of extra cellular enzymes produced by phytopathogenic organism has been known to contribute to resistance e.g. production of polyvalent cations such as Ca++ around developing *Rhizoctonia* lesions, restrict further tissue maceration by polygalacturonase produced by invading pathogen.

(iii)Defense through detoxification of pathogen toxins

In some plants, resistance to the pathogens is the same as resistance to the toxin as shown by the correlation between toxins production and pathogenicity.

Plant Resistance to Pathogen

What is resistance?

Resistance is any heritable characteristics of a plant which enables it to overcome, either completely or in-part, the effect of a pathogen or other damaging factor. Disease resistance that is controlled genetically by the presence of one, a few, or many genes for resistance in the plant is known as **true resistance.**

In true resistance, the host and the pathogen are more or less incompatible with one another, either because of lack of chemical recognition between the host and the pathogen or because the host can defend itself against the pathogen. There are 2 kinds of true resistance;

(i) Partial, quantitative, polygenic or horizontal resistance

All plants have certain but not always the same level of possibly unspecific resistance that is effective against each of their pathogens. Partial resistance is probably controlled by several genes, thereby the name polygenic or multigene resistance. Partial resistance is usually less effective than the vertical resistance.

(ii) R - gene resistance, race – specific, monogenic or vertical resistance

Many plant varieties are quite resistant to some races of a pathogen while they are susceptible to other races of the same pathogen. In other words, depending on the race of the pathogen used to infect a variety, the variety may appear strongly resistant to one pathogen race and susceptible to another race (race specific) under a variety of environmental conditions.

Apparent Resistance

Under certain conditions or circumstances some very susceptible plants or varieties of crops may remain free from infection or symptoms and thus appear resistant. The apparent resistance to disease of plants known to be susceptible is generally a result of disease escape or tolerance to disease.

(i) Tolerance to disease

Tolerance to disease is the ability of plants to produce a good crop even when they are infected with a pathogen. In other words, tolerance is inherent or acquired capacity to endure disease. For instance, in tolerant plants, the yield may not be affected despite the presence of pathogen.

(ii) Disease escape

Disease escape occurs whenever genetically susceptible plants do not become infected because the three factors necessary for disease do not coincide and interact at the proper time or for sufficient duration. Thus disease escape is due to incomplete synchronization between the life cycles of the pathogen and the plant and it is not due to any mechanism of resistance.

Gene – for Gene Concept

In nature, the coexistence of host plants and their pathogens side by side in nature indicates that the two have been evolving together. The stepwise evolution of virulence and resistance can be explained by the gene-for-gene concept, according to which for each gene that confers virulence to the pathogen, there is a corresponding gene in the host that confers resistance to the host and vice versa. Generally, but not always, in the host the genes for resistance are dominant (R), whereas genes for susceptibility i.e. lack of resistance are recessive (r). In the pathogen, however, genes for avirulence i.e. inability to infect, are usually dominant (A) whereas genes for virulence are recessive (a). The modern interpretation is that resistant plants have dominant resistance genes (R genes) that match dominant avirulence genes (avr genes) in the pathogen. A pathogen's *avr* gene

is a gene that produces **elicitor** of plant defenses. R genes of plants are associated with **receptors** that recognize the elicitor. It is this elicitor-receptor interaction that gives many host-pathogen interactions their genetic specificity. The **incompatible reaction of R gene receptors** in the plant and the **avr gene elicitors** in the pathogen leads to resistance.

It is thought that genes for resistance appear and accumulate first in hosts through evolution and that coexist with nonspecific genes for pathogenicity which evolve in pathogens. Genes for pathogenicity exist in pathogens against all host plants that lack specific resistance.

Plant Disease Epidemiology

What is an epidemic?

When a pathogen spreads to and affects many individual within a population over a relatively large area and within a short time, the phenomenon is called an **epidemic**. Therefore, an **epidemic** is disease increase in a population over time. The study of disease increases in populations, including factors that influence their initiation, development, and spread is called **epidemiology**. The more specific term **epiphytotic** is sometimes used to describe disease increase in a plant population.

Plant disease epidemics have two models: (i) monocyclic pathogens and (ii) polycyclic pathogens.

Monocyclic pathogens complete only one generation during a single growing season commonly referred to as simple interest disease. Primary inoculum (or initial inoculum) initiates the disease, and no new inoculum is produced until the end of the growing season. Many, but not all, monocyclic pathogens are soilborne. Mangement of monocyclic pathogens focuses on reducing the amount or the efficacy of the primary inoculums.

Polycyclic pathogens in contrast, may complete several to many generations during the growing season and is termed compound interest disease. After the primary inoculum initiates disease, secondary inoculum is produced and causes additional infections. Infections may be may be on the same plant or on neighboring plants that are susceptible to the disease. They are often airborne or vectorborne. Management of polycyclic pathogens focuses on reducing primary inoculum and the rate of infection.

(a) **Host factors that affect the development of epidemics**

Several internal and external factors of particular host plants play an important role in the development of epidemics involving hosts.

(i) Level of genetic resistance or susceptibility of the host

Host plants carrying race-specific (vertical) resistance do not allow a pathogen to become established in them, and thus no epidemic can develop. Host plants carrying partial (horizontal) resistance will probably become infected, but the rate at which the disease and the epidemic will develop depends on the level of resistance and the environmental conditions. Therefore, in the presence of a virulent pathogen and a favourable environment, susceptible hosts favour the development of disease epidemics.

(ii) Degree uniformity of host plants

When genetically uniform host plants, particularly with regard to the genes associated with disease resistance, are grown over large areas, a greater likelihood exists that a new pathogen race will appear that can attack their genome and result in an epidemic.

(iii)Type of crop

In diseases of annual crops epidemics generally develop much more rapidly (usually in a few weeks) than those do in diseases of branches and stems of perennial woody crops such as fruit and forest trees.

(iv) Age of host plants

Plants change in their reaction (susceptibility or resistance) to disease with age. The change of resistance with age is known as ontogenic resistance.

(b) Pathogen factors that affect development of epidemics

(i) Levels of virulence

Virulence pathogens capable of infecting the host rapidly ensure a faster production of larger amounts of inoculums, and thereby disease, than pathogens of lesser virulence.

(ii) Quantity of inoculums near hosts

The greater the number of pathogen propagules within or near fields of host plants, the more inoculums reaches the hosts and at an earlier time, thereby increasing the chances of an epidemic greatly.

(iii) Type of reproduction of the pathogen

Some plant pathogenic fungi, bacteria and viruses have short reproduction cycles and therefore are polycyclic i.e they can produce many generations in a single growing season. Several pathogens such as the smuts and several short-cycle rusts, require an entire year to complete a life cycle (monocyclic pathogens) and can therefore cause only

one series of infections per year. In such monocyclic diseases the inoculums builds up from one year to the next and the epidemic is usually polyetic i.e. it develops over several years.

(iv) Ecology of the pathogen

Some pathogens such as most fungi and all parasitic higher plants, produce their inoculums on the surface of the aerial parts of the host. From there, inoculums can be dispersed with ease over a range of distances and can cause widespread epidemics where spread of pathogen is rare or impossible in vascular and soil borne pathogens.

(v) Mode of spread of the pathogen

The spores of many plant pathogen fungi such as those causing rusts, mildews and leaf spots are released into the air and can be dispersed by air breezes or strong wind over distances varying from a few centimeters up to several kilometers. Likewise inoculums carried by airborne vectors and windblown rain are almost annually responsible for severe but somewhat epidemics.

(c) Environmental factors that affect development of epidemics

(i) Moisture

Abundant, prolonged or repeated high moisture, whether in the form of rain, dew or high humidity, is the dominant factor in the development of most epidemics of diseases caused by oomycetes and fungi, bacteria and nematodes. Moisture not only promotes new succulent and susceptible growth in the host, but more importantly, it increases sporulation of fungi and multiplication of bacteria.

(ii) Temperature

Epidemics are sometimes favoured by temperatures higher or lower than the optimum for the plant because they reduce the plant's level of partial resistance. The most common effect of temperature on epidemics however, is its effect on the pathogen during the different stages of pathogenesis i.e. spore germination, host penetration, pathogen growth or reproduction, invasion of the host and sporulation.

(d) Effect of human cultural practices and control measures

(i) Site selection and preparation

Low lying and poorly drained and aerated fields, especially if near other infected fields, tend to favour the appearance and development of epidemics.

(ii) Selection of propagative material

The use of seed, nursery stock, and other propagative material that carries various pathogens increases the amount of initial inoculum within the crop and favours the development of epidemics greatly.

(iii) Cultural practices

Continuous monoculture, large acreages planted to the same variety of crop, high levels of nitrogen fertilization, injury by herbicide application and poor sanitation all increases the possibility and severity of epidemics.

(iv) Disease control measures

Chemical sprays, cultural practices (such as sanitation and crop rotation), biological controls and other control measures reduce or eliminate the possibility of epidemics.

Types of Epiphytotics

(a) Destructive epiphytotics which occur on a continental scale are said to be **pandemic** e.g. ACMV which is found wherever cassava is grown in West Africa. Also, the sigatoka leafspot of banana, and plantain which is found in all musa growing areas of the world such as East and West Africa, Central and latin America and also in Island covering the pacific ocean.

(b) Sometimes epiphytotics can be caused by pathogens which are long established in an area, such epiphytotics are said to be **endemic** e.g. tomato wilt caused by *Ralstonia solanacearum* is restricted to the fadama around Zaria and Jos.

Epiphytotics are also said to develop slowly or rapidly.

Slow epiphytotics

These are usually associated with perennial plants and the pathogens are systemic to varying extent and it may take several years for the disease to reach its full potential. The infected plants may remain in the field for several years before dying e.g. the cocoa swollen shoot virus and citrus histeza virus

Rapid epiphytotics

These are caused by non-systemic pathogens in annual crops. The pathogens have fairly short generations and high multiplication rate. They are characterized by a rapid buildup of disease to a fairly distinct peak followed by a rapid decline as environmental conditions becomes unfavourable e.g. *Xanthomonas malvacearum* causing angular leafspot in cotton and *Ralstonia solanacearum* causing wilt in tomato.

Measurement of Plant Disease and of Yield Loss

(a) Incidence: The incidence of the disease i.e. the number or proportion of plant units that are diseased (i.e. the number or proportion of plants, leaves, stems and fruit that show any symptoms) in relation to the total number of units examined.
(b) Severity: The proportion of area or amount of plant tissue that is diseased and is usually expressed as a percentage or proportion of plant area or fruit volume destroyed by a pathogen.
(c) Yield loss: The proportion of the yield that the grower will not be able to harvest because the disease destroyed it directly or prevented the plants from producing it (the yield loss is the difference between attainable yield and actual yield)