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E-CONTENT
Prepared for UG (Botany Hons. Courses under CBCS)

Course Name	Course Code	Credits	System	Syllabus
CORE COURSE- 2 MYCOLOGY AND PHYTO-PATHOLOGY	BOT-A-CC-1-2-TH	THEORETICAL (Credits 4, Lectures 60)	CBCS	University of Calcutta

CORE COURSE 2

MYCOLOGY AND PHYTO-PATHOLOGY (BOT-A-CC-1-2-TH)

THEORETICAL (Credits 4, Lectures 60)

PHYTO-PATHOLOGY

1. Terms and Definitions : 1.1. Disease concept, 1.2. Symptoms, 1.3. Etiology & causal complex, 1.4. Primary and secondary inocula, 1.5. Infection, 1.6. Pathogenecity and pathogenesis, 1.7. Necrotroph and Biotroph, 1.8. Koch's Postulates, 1.9. Endemic, Epidemic, Pandemic and Sporadic disease, 1.10. Disease triangle, 1.11. Disease cycle (monocyclic, polycyclic and polyetic).

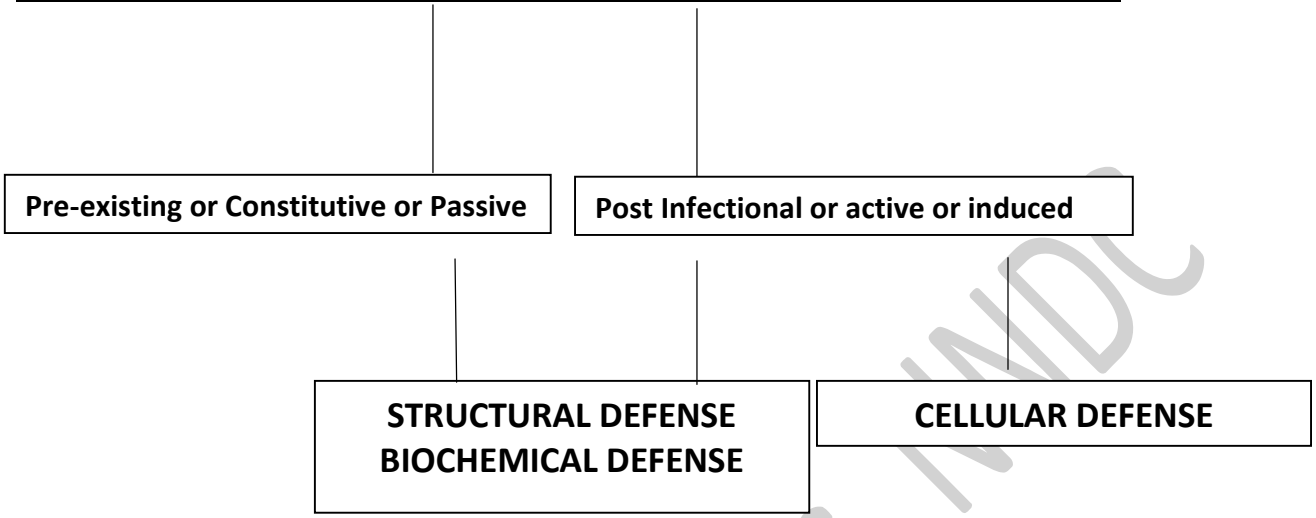
2. Host – Parasite Interaction: 2.1. Mechanism of infection (Brief idea about Pre-penetration, Penetration and Post-penetration), 2.2. Pathotoxin (Definition,criteria and example), 2.3. Defense mechanism with special reference to Phytoalexin, 2.4. Resistance- Systemic acquired and Induced systemic. lectures

3. Plant Disease Management : 3.1. Quarantine, 3.2. Chemical, 3.3. Biological, 3.4. Integrated. l

4. Symptoms , Causal organism, Disease cycle and Control measures of: 4.1. Late blight of Potato, 4.2. Brown spot of rice, 4.3. Black stem rust of wheat, 4.4. Stem rot of jute. …….10 lecture lectures 4. Symptoms , Causal organism, Disease cycle and Control measures of: 4.1. Late blight of Potato, 4.2. Brown spot of rice, 4.3. Black stem rust of wheat, 4.4. Stem rot of jute.

DEFENSE MECHANISM IN PLANTS

Defense Mechanism in plants or Mechanism of plant disease resistance



STRUCTURAL DEFENSE MECHANISM

STRUCTURAL PRE-EXISTING:

1. Waxes
2. Hairs or trichomes
3. Glabrous surface
4. Thick layers of cuticles
5. Structure of epidermal cell wall (motor cells of *Puccinia graminis*, Mandarin orange resistance against *Xanthomonas citri*)

STRUCTURAL POST-EXISTING:

1. Cork layer (Resistant against *Rhizoctonia solani*)
2. Abscission layer (*Pyrus* sp *Psidum guasava*)
3. Formation of Tylose (sweet potato; *Fusarium oxysporum*)
4. Gum deposition/tannin deposition

BIOCHEMICAL DEFENSE MECHANISM

PRE-EXISTING BIOCHEMICAL REACTIONS:

1. Prohibitions or phenolic compounds. Eg. Catechuic acid, nonanol, allicin
2. Defence through lack of essential factors-
   i. Nutrients
   ii. Recognition site
   iii. Toxin receptors
   iv. Presence of pre existing chemical tannin
3. pH -osmotic pressure, temperature
4. lack of antigen

POST-INFECTIONAL BIOCHEMICAL REACTIONS:

1. synthesis of inhibitors (chlorogenic acid, caffeic acid, ferulic acid)
2. PR protein
3. Phytoalexins
4. Active oxygen radicals, superoxide
5. Host cell wall
6. Hypersensivity response

CELLULAR DEFENSE MECHANISM:

A. i. The infected cell swells, and swelling is accompanied by production of fibrillar material that trap the pathogen.
   ii. Cell wall thickens
   iii. Callose papillae
B. Cytoplasmic defence reaction
C. Hypersensitivity reaction

Each plant species is affected by approximately 100 different kinds of fungi, bacteria, mycoplasma, viruses and nemtatodes. Plants have resistant factors, either inborne or procured which help to ward off the pathogen. Disease resistant appears to be a rule rather than an exception in nature. The capacity or ability of plant to defend itself against a pathogen is governed by its genetic constitution and the environmental conditions under which the genes operate. The attributes of the host that reduce the chances of infection or the development of pathogen are considered to be defense mechanism.

In general, plant defends themselves against pathogen by a combination of weapon from 2 arsenals.

1. Structural characteristics:
   That act as physical barriers and inhibit the pathogen from gaining entrance and spreading through the plant.
2. Bio-chemical reactions:
   That makes place in the cells and tissues of the plants and produce substances that either are toxic to pathogen or create conditions that inhibit growth of the pathogen in the plant.
   The combination of structural characteristics and bio-chemical reaction employ in defense of plant are different in different host pathogen system. In addition, even within the same host and pathogen, the combination vary with the age of the plant, the kind of plant organ and tissue attacked the nutritional condition of the plant and also with the weather condition.

STRUCTURAL DEFENSE:

A. Pre-existing defense structure:

The first line of defense against pathogen is the surface barrier which a pathogen must penetrate before it can cause infection. The entry of the pathogen may either be through the epidermal cell wall directly or through natural openings like stomata, lenticels etc or through wounds. Certain structural features of epidermis may greatly effect the ability of pathogen to penetrate or to invade a host plant.

They are as follows:

1. Waxes- On leaf and fruit surfaces, waxes play a defense role by forming a hydrophobic surface which acts as a water repellant and thereby prevents retention of water on which pathogens may be deposited and germinate to further invade the host.
2. A thick mat of hairs or trichomes on plant surfaces may also exert a similar water repelling effect and may reduce infection.
3. A thick layer of cuticle may increase resistance to infection in diseases where the pathogen enters its host through direct penetration only. *Berberis graminis*, disease resistance has been attributed to the outer epidermal cell wall with a thicker cuticle.
4. Structure of epidermal cell wall: Thickness and toughness of the outer wall of epidermal cells are important factors in disease resistance. Cell walls which are lignified or silicified are more adapted to resistance than others. In case of Rice Blast fungus *Pyricularia oryzae*, outer walls of all epidermal cells of leaves of resistant plants are lignified. Entry of the pathogen in susceptible host is through some thin-walled motor cells which lack lignin deposition.
5. Structure of natural opening- Most pathogen enters plant through natural openings. Majority of pathogens can force their way through close stomata yet some, like *Puccinia graminis* var *tritici* can enter only when the stomata remain open. In resistant wheat varieties, stomata open late in the morning, so the spores get desiccated and dry.

   Xanthomonas citri is responsible for citrus canker. Mandarin orange variety *Szinkum* is resistant to it since it possesses a broad cuticular ridge projecting over the stomata cavity so that rain drops containing fungal spores and bacteria rarely reach the mesophyll of the leaf.
6. Internal structural barrier to pathogen invasion- The cell wall varies in thickness and toughness and may sometimes inhibit the advancement of pathogen. The presence of bundles or extended areas of sclerenchyma patches as is found in the stem of many aerial crops, may stop the further spread of pathogens like the stem rust fungi.

B. Post-infectional defence structure:

Inspite of preformed defence structures most pathogens manage to penetrate their hosts and to produce various degrees of infection. In response to infection caused by the pathogen, a new set of defense structure is formed by the host. These are explained below:

i. Histological/Structural defense structure:

a. Formation of cork layers: Infection by fungi or bacteria frequently induce plants to form several layers of cork cells beyond the point of infection as a result of stimulation of the host cells by substance secreted by the pathogens. The cork layers inhibit the further invasion by the pathogen beyond the initial lessions and block the spread of any toxic substances secreted by the pathogens. Further more, cork layers stop the flow of nutrients and water from the healthy to the infected area and deprive the pathogen of nourishment resulting in its death. In *Rhizoctonia* disease of potato tubers cork layers are formed.

b. Abscission layers: As a result of infection by several fungi, bacteria or viruses, abscission layers are formed in the young active leaves of stone fruits like *Pyrus, Psidum guajava*. Due to the formation of a layer, there is a swelling of the 2 layers of cells surrounding the infected spot. Th cells become thin walled while the pectin materials of the middle lamella get dissolved. The leaf area drops off which results in the formation of shot holes or a gap throughout the leaves extending from upper to lower epidermis. This causes removal of pathogen from leaves.

c. Formation of tyloses- Tyloses forms in xylem vessels of most plants under various condition of stress and during invasion by most of the vascular pathogens. They are the outgrowths of the protoplast of adjacent living parenchyma cells which protrude into xylem vessel through half bordered pits. Tyloses are usually considered to be one of the factors which causes wilt. The time and rapidity of tyloses formation, determines whether their role will be defensive or whether they

themselves be one of the factors for the cause of the disease. In sweet potatoes wilt caused by *Fusarium oxysporum*, in resistant variety of sweet potato, the tyloses are formed abundantly and quickly before the pathogen and thus brings about resistance as this prevents the further spread of pathogen.

d. Gum deposition- Various types of gums are produced by many plants around lessions after infection by pathogens or injury. Gum secretion is more common in stone fruit trees but also occurs in other plants. Gum deposition along the borders often serve as a protective demarcation and constitutes a type of mechanical resistance. In rice variety, resistant to blast or *Helminthosporium* leaf spot, gum deposits are formed in the intercellular space that helps in restricting the fungus to the area of primary invasion. This impenetrable barrier completely encloses the pathogen. The pathogen then becomes isolated starved and sooner or later dies.

CELLULAR DEFENSE STRUCTURE:

i. Cellular defense structure involves morphological change in the cell wall being invaded by the pathogen. Three main type of defense mechanism is observed.

a. Cell wall of parenchymatous cells in contact with incompatible bacteria swells, and this swelling is accompanied by production of an amorphous, fibrillar materials that surrounds and traps the bacteria.
b. Cell walls thicken in response to viral and fungal pathogens.
c. Callose papillae are deposited on the inner side of cell walls in response to invasion by fungal pathogens. Papillae seem to be produced by cells within minutes after wounding although main function of papillae appears to be repair of cellular damage, sometimes they also seem to prevent the pathogen

from subsequently penetrating the cell. In some cases, hyphae penetrating cells are sheathed by cellulosic materials.

ii. Cytoplasmic defense reaction:

In a few cases of slowly growing, weakly pathogenic fungi that induce chronic diseases or nearly symbiotic conditions the cytoplasm surrounds the clump of hyphae and the nucleus, is stretched to the point where it breaks in two. In some of the invaded cell the cytoplasm and nucleus enlarge. The cytoplasm becomes granular and dense and various particles or structure appear in it. Finally mycelium of the pathogen disintegrates and the invasion stops.

iii. Defense through hypersensitivity:

In many host pathogen combinations, the pathogen may penetrate the cell wall but as soon as it establishes contact with protoplast of the cell, the nucleus moves towards the invading pathogen and soon disintegrates, and brown resin like granules form in the cytoplasm, first around the pathogen and then throughout the cytoplasm. As the browning discolouration of the cytoplasm of the plant cell continues and death sets in, the invading hyphae begins to degenerate. In most cases, the hypha does not grow out of such cells and further invasion is stopped. In bacterial infections of leaves, the hypersensitive reaction results in destruction of all cellular membranes of cells in contact with bacteria and that is followed by dessication and necrosis of the leaf tissues invaded by the bacteria. The necrotic tissue isolates the obligate parasite from the living substance on which it depends absolutely for its nutrition and therefore, results in its starvation and death.

BIOCHEMICAL DEFENSE:

Although structural characteristics may provide a plant with varying degrees of defense against attacking pathogens, it is clear that resistance of a plant against pathogen attacks depends not so much on its structural barriers as on the substances produced in the cells before or after infection. This type of defense which is of a chemical nature is called biochemical defense. These may be preexisting or post infectional.

A. Pre-existing defense mechanisms:

a. Inhibitors released by the plant in its environment:
   Plants exude a variety of substances through their underground and above aerial ground parts. Some of these compounds seem to have inhibitory action on certain pathogens. Fungitoxic exudates on leaves of tomato and sugarbeet inhibit germination of spores of *Botrytis* and *Cercospora* respectively. Work on onion smudge disease caused by *Colletotrichum circinans* show the red scaled variety contains catechol and protocatechuic acid which are phenolic compounds, diffuse out of the red scales and prevent germination of spores.

b. Defense through lack of essential factors:
   i. Lack of recognition between host and pathogen:
      Plants of a species may not become infected by a pathogen if their surface cells lack specific recognition factors that can be recognized by the pathogen.
   ii. Lack of host receptors and sensitive sites for toxins:
      In host -pathogen combinations, the pathogen produces a host specific toxin. The toxin gets attached to and reacts with specific receptors or sensitive sites in the cell. Only plants that have such receptors or sites get diseased.

iii. Lack of essential Nutrients for the pathogen:

Species or varieties that do not produce one of the essential substances necessary for survival of the pathogen do not get infected. For *Rhizoctonia* to infect a plant, the plant need not have a substance necessary for formation of a hyphal cushion from which the fungus sends its penetration hyphae.

iv. Inhibitors present in plant cell before infection:

Plant cells contain tannins and phenolic compounds prior to infection. These chemicals are present in high concentration in young fruits, buds, flowers etc. They are supposed to be fungitoxic. Thus, young parts are supposed to be more resistant to disease than older parts. As young tissues grow older, their inhibitor content and resistance decrease sharply. Tomatin and Avenacin in tomato and *Avena* respectively are two such inhibitors. The high concentration of cholorogenic acid in root of certain potato varieties has been considered the main mechanism of defense against *Verticillium* (now called *Lecanicillium* sp) like pathogen.

c. pH:

pH of plant tissues influence many physiological process in both plants and parasites, notably enzyme activities. There are a few convincing cases of disease resistance directly attributable to pH. Resistance to pH sensitive pathogens is related to unsuitable pH of the host.

d. Osmotic Pressure and permeability Effects:

Cells of parasitic fungi usually have an unusually higher OP than host cells. Resistance to the pathogen may be induced in plants where host cells have OP higher than OP of the pathogen. Further, resistance can be modified by attaining the mineral nutrition of the plant and shaded leaves.

e. Defense through absence of common antigens:

A concept was developed that antigens shared by the host and the pathogen may determine the nature of disease reactions. Susceptibility depended on the presence in the host of a protein serologically similar to one possessed by the pathogen. Pathogenic races are thus always found to have more antigens in common to the host than non pathogenic races.

B. Post-infectional disease resistance:

Host pathogen interaction induce this kind of defense.

a. Biochemical inhibitors produced in plants in response to injury which may be mechanical or chemical or pathogenic through a series of biochemical reactions that seem to be armed at isolating the irritant and at healing the wound. This reaction causes formation of fungitoxic substances, mostly phenolic compounds such as chlorogenic and caffeic acid, oxidation products of phenolic compounds and also phytoalexins.

b. Defense through Hypersensitive reactions (HR):

Upto the stage of penetration, no difference exists between resistant and susceptible plants. After infection cells in resistant varieties rapidly lose turgor, turn brown and die while cells in susceptible varieties survive. This is due to

i. Loss of permeability of cell membranes.
ii. Increased rate of respiration.
iii. Accumulation and oxidation of phenolic compounds.
iv. Production of phytoalexins.

c. Formation of substrates resisting the enzymes of the pathogen:

The substrates are usually complexed between pectin, protein and polyvalent ions like Ca^{+2} and Mg^{+2}. These complexes cannot be broken down by the enzymes of the pathogen resulting in starvation of the pathogen.

d. Degradation of pathogenic enzymes:
   Phenolic compounds and the oxidative products have inhibitory action on the pectolytic and other enzymes of the pathogen rather than on the pathogen itself. *Diplodia* contains polygalacturonase (PG) which is inhibited by proteins secreted from the rind of orange.
e. Production of Antimicrobial substances in host cell:
   i. Pathogenesis related proteins (PR)
      These are called PR proteins and toxic to invading fungal pathogen. They are synthesized in higher concentration following pathogen attack or stress intracellularly or intracellularly. Different plant organs such as seed, leaf and root may produce different types of PR proteins. The better-known PR proteins are PR 1, β -1-3 glucanases, chitinases, lysozymes, PR 4, osmotin like protein, glycine and cysteine rich proteins, protinases and peroxidases.
   ii. Phytoalexins