Defense Mechanism in Plants

Plants Response Towards Pathogenic Interaction

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Need for Defense in Plants

- **Adjustment** is probably, one of the most important advantage of a system that ensures it survival
- **Resistance to Exploitation** as a Biological System forced by co-existence with pathogen, has led to development of defense mechanism in plants
- **Survival of plants** in spite of living amongst some of the potentiality disturbing pathogens in addition to abiotic stresses

❖**Thus, defense means resistance against any 'damaging act' or response of plant system**

Host – Pathogen Relationship

- **Biotrophs:** Pathogens that keep their host alive and feed on living plant tissues

Ex. Powdery mildew fungus *Blumeria graminis* and the bacterial rice pathogen *Xanthomonas oryzae*

- **Necrotrophs:** Pathogens often produce toxins or tissue-degrading enzymes that beat plant defenses and promote the quick release of nutrients

Ex. Gray mold fungus *Botrytis cinerea* and the bacterial soft-rot pathogen *Erwinia carotovora*

- **Hemibiotrophs:** Pathogens are biotrophic during the early stages of infection but become necrotrophic during the latter stages of disease

Ex. Fungus *Magnaporthe grisea*, the causative agent of rice blast disease

Outcome of Host- Pathogen Relationship

1. Compatible response: Results in disease

2. Incompatible response: Results in little or no disease at all

The response depends upon Disease resistance of individual plant species that exists as a range of responses

a. **Immunity** (the complete lack of any disease symptoms)
b. **Highly resistant** (some disease symptoms)
c. **Highly susceptible** (significant disease symptoms)

Plant Resistance

- **Basal Resistance (Innate Immunity): Some Plants have surveillance mechanisms that recognize potentially dangerous pathogens and rapidly respond before those organisms have a chance to cause serious damage**

Ex. Plant cells recognize microbe-associated molecular patterns like specific proteins, lipopolysaccharides, and cell wall components commonly found in microbes

- **Hypersensitive Response (HR): Plant cells deliberately induce suicidal response at the site of infection**

Ex. Plant cell recognize the presence of specific disease-causing effector molecules introduced by the Bacteria, fungi, viruses, and nematodes

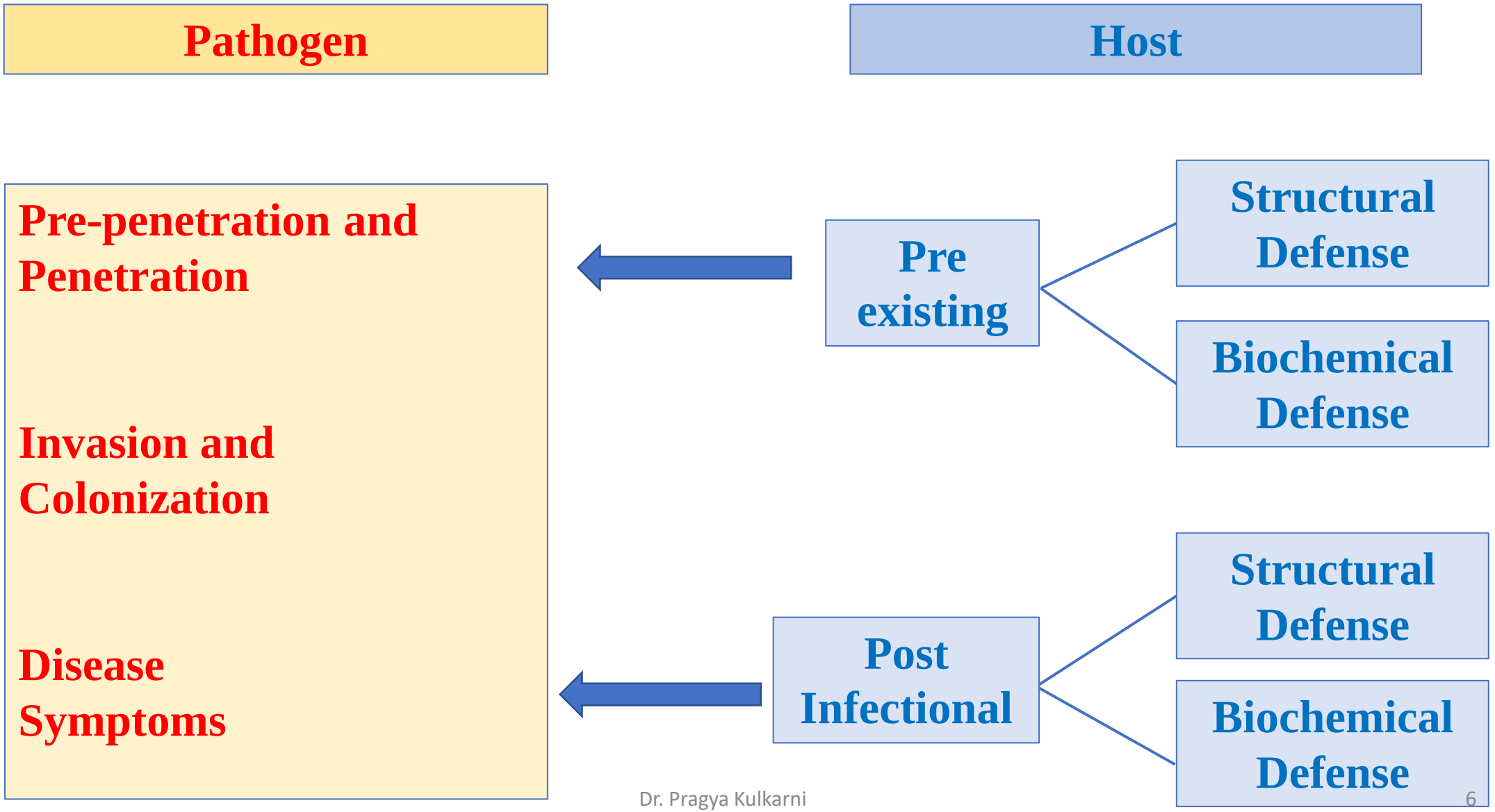
- **Systemic Acquired Resistance (SAR): Plant tissues become highly resistant to a broad range of pathogens for an extended period of time by producing plant activators**

Ex. These substances are much less toxic to humans and wildlife and their protective effects can last much longer

- **RNA Silencing: Plants can defend themselves by a refined genetic system through recognizing foreign molecules(dsRNA or DNA) and respond by digesting the genetic strands into useless fragments**

Ex. Enhancement of resistance to biotic stress caused by Viral, Bacterial and Fungal diseases

Plant Defense



I. Structural Defense

Pre Existing Defense Structures	Induced Defense Structures
• Cuticular Wax • Thickness of Cuticle • Structure of Epidermal Cell Wall • Structure of Natural openings	• Cytoplasmic defense reaction • Cellular defense structure • Histological defense structures • Necrotic defense reaction through HR

(A) Pre Existing Defense Structures

(i) Cutin and Wax:

- Cutin is composed of fatty acids and Wax is a mixtures of long chain aliphatic compounds get deposited on the cuticular surface of plants causing a hydrophobic surface where water is repelled and the pathogen does not get sufficient water to germinate or multiply
- Due to the presence of fatty acids a negative charge develops on the leaf surface which prevents/reduces the chance of infection by many pathogens

Ex. Infection against many Bacteria and Fungi

(ii) Thickness of Cuticle:

- The thickness of cuticle obstructs the path of pathogen and also checks the exit of the pathogen from inside the host, thus reducing the secondary infection

Ex. Linseed cuticle act as a barrier against *Melampsora lini*

(iii) Structure of Epidermal Cell Walls:

- Tough and thick outer walls of epidermal cells
- The presence or absence of lignin and silicic acid in the cell walls
- Most outer walls of epidermal cells of many plants are lignified

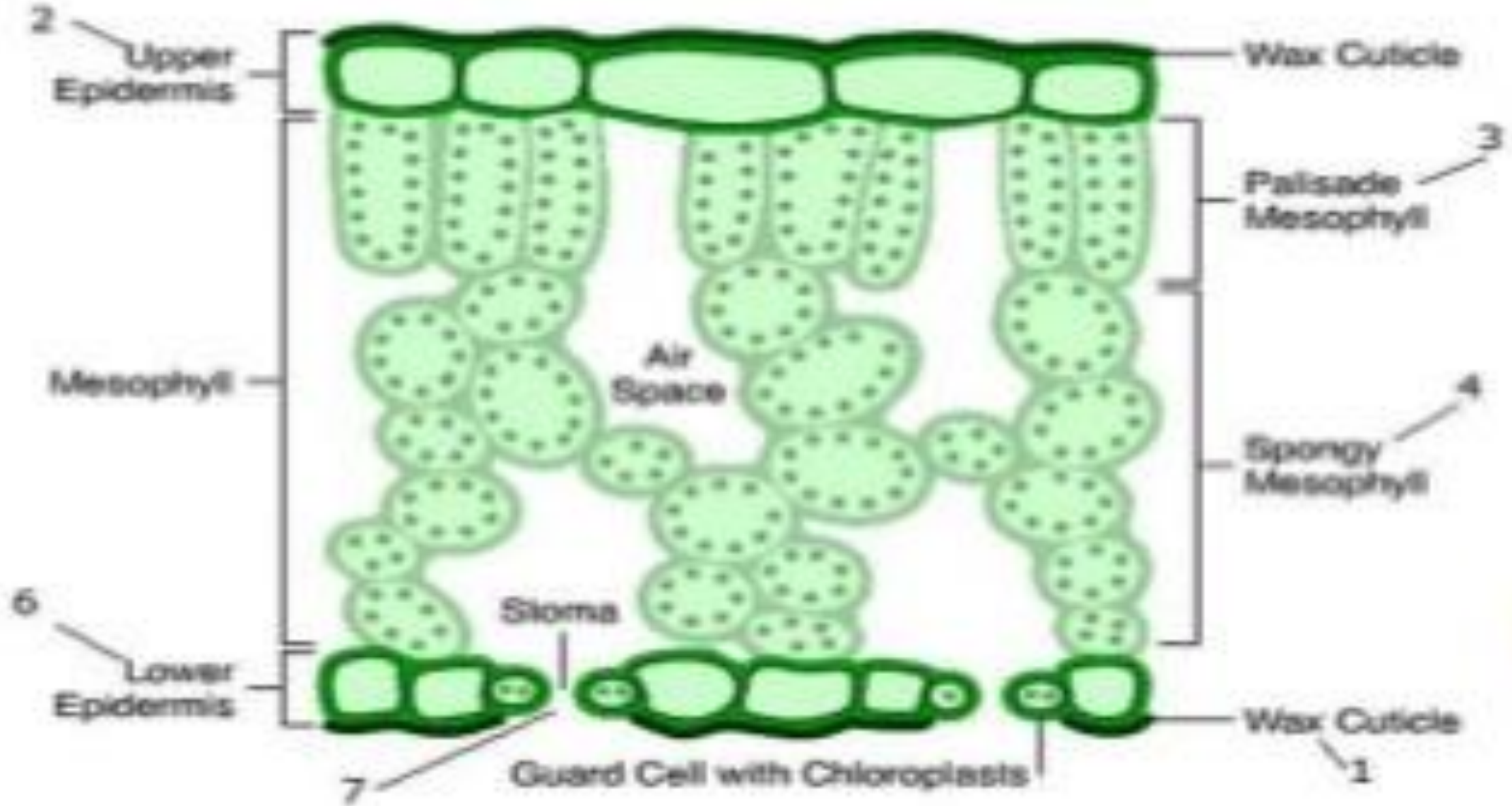
Ex. The resistant varieties of potato tubers the epidermal cells contain higher fiber contents

Ex. Rice leaves against *Pyricularia oryzae* causing Rice blast

(iv) Structure of Natural openings:

- The size and shape of stomata surrounded by broad lipped raised structures prevent entry of water drops containing pathogens
- The small lenticels in many plants may play a defensive role against the pathogens
- Nectaries provide openings in the epidermis and may play a defensive role due to high osmotic concentration of the nectar

Ex. Resistant varieties of Citrus for Citrus canker bacteria *X. citri*


2
Upper Epidermis
Wax Cuticle
3
Palisade Mesophyll
Mesophyll
Air Space
4
Spongy Mesophyll
6
Stoma
Lower Epidermis
Wax Cuticle
1
7
Guard Cell with Chloroplasts

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

(i) Defense Reactions in the Cytoplasm:

- The cytoplasm of the invaded cell surrounds the hyphae of the pathogen and the nucleus of the host cell gets stretched and enlarge
- The cytoplasm becomes granular and dense resulting the disintegration of the pathogen mycelium and thus the further invasion of pathogen stops

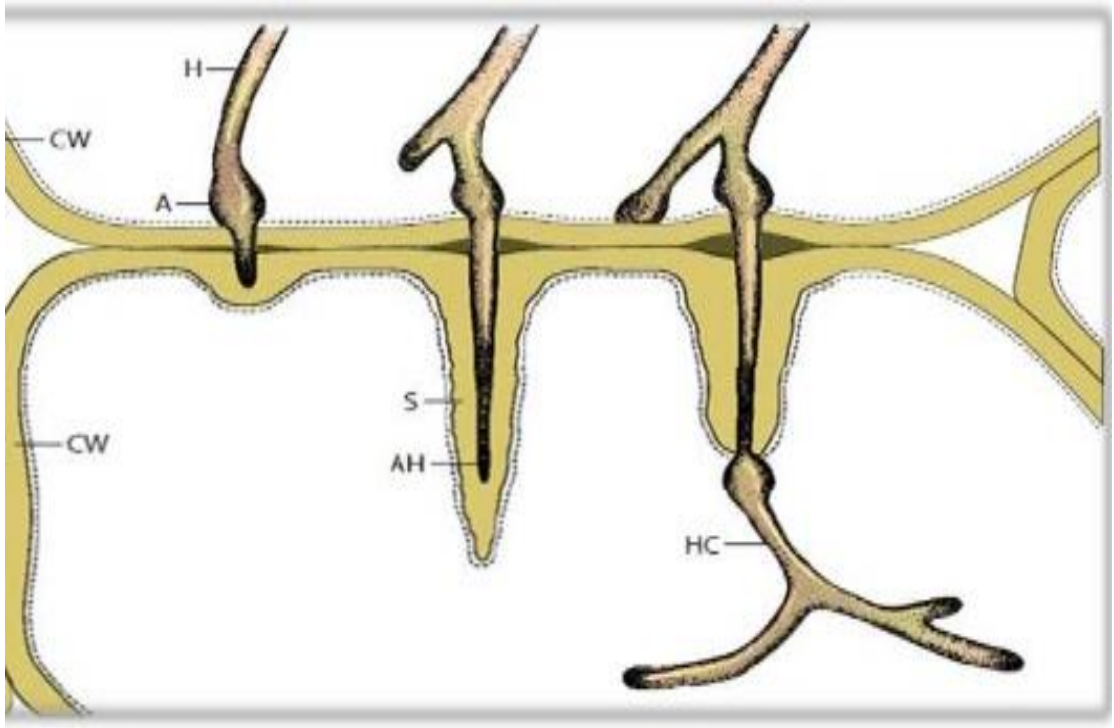
(ii) Cell Wall Defense Structures:

Three types of cell wall defense structures are generally observed –

(i) Cell walls thicken in response to the pathogen by producing a cellulose material

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

(iii) Callose papillae get deposited on the inner layers of the cell walls



Ex. Hyphal sheathing by F. oxysporum

(iii) Defense Structures Developed by the Tissues:

(a) Gum Deposition:

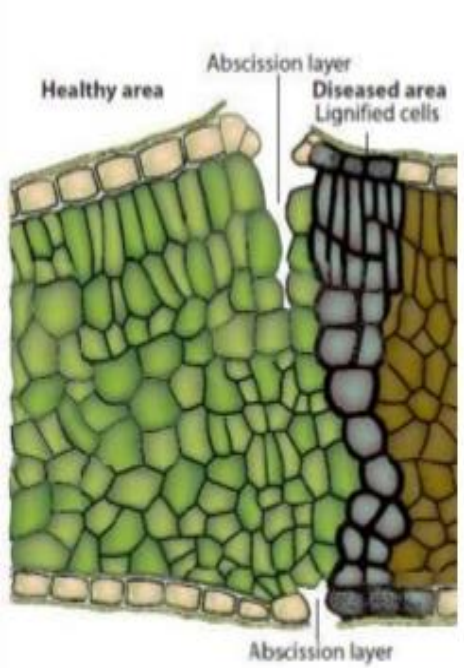
- Plants produce a variety of gummy substances around lesions or spots as a result of infection

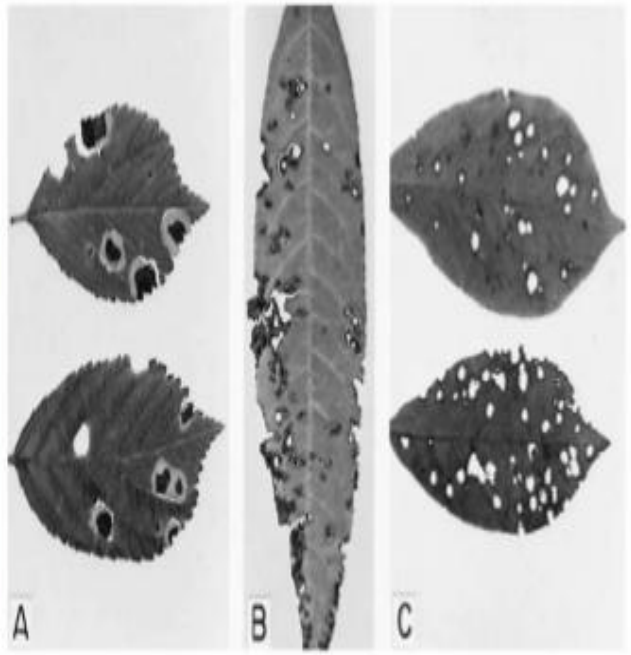
Ex. In resistant Rice plants against *Helminthosporium* infection

(b) Abscission Layers:

- These layers develop in their young leaves in response to infection by several fungi, bacteria or viruses resulting formation of a gap between two circular layers of cells surrounding the point of infection
- Abscission layer formation protects the healthy leaf tissue from the attack of the pathogen

Ex. By *Xanthomonas pruni* on Peach leaves

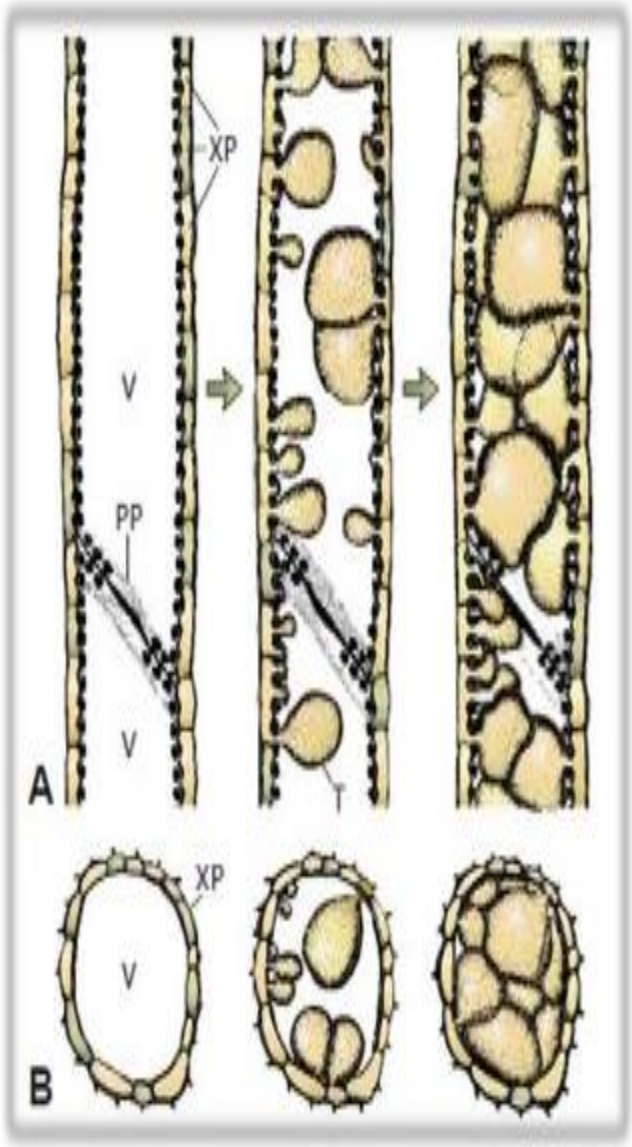




(c) Tyloses:

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

Ex. In most of the plants against vascular wilt pathogen

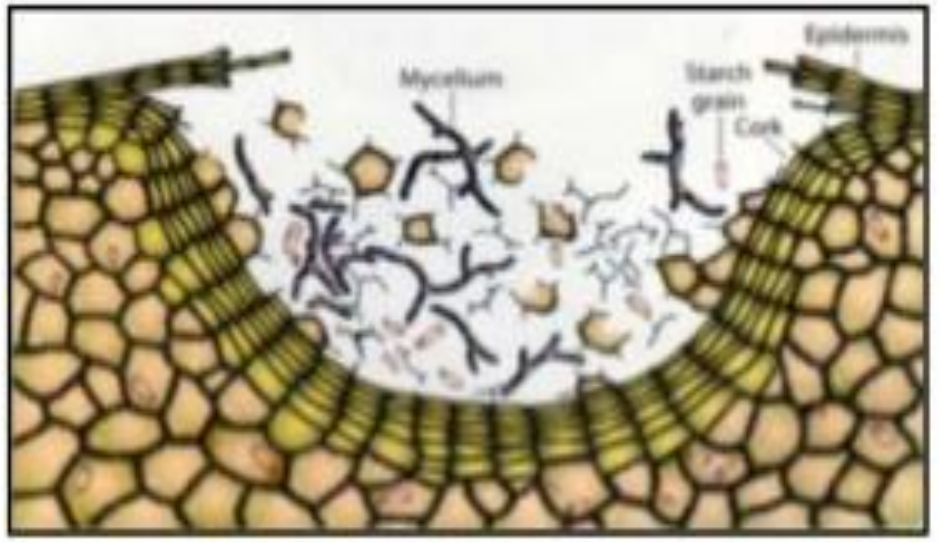


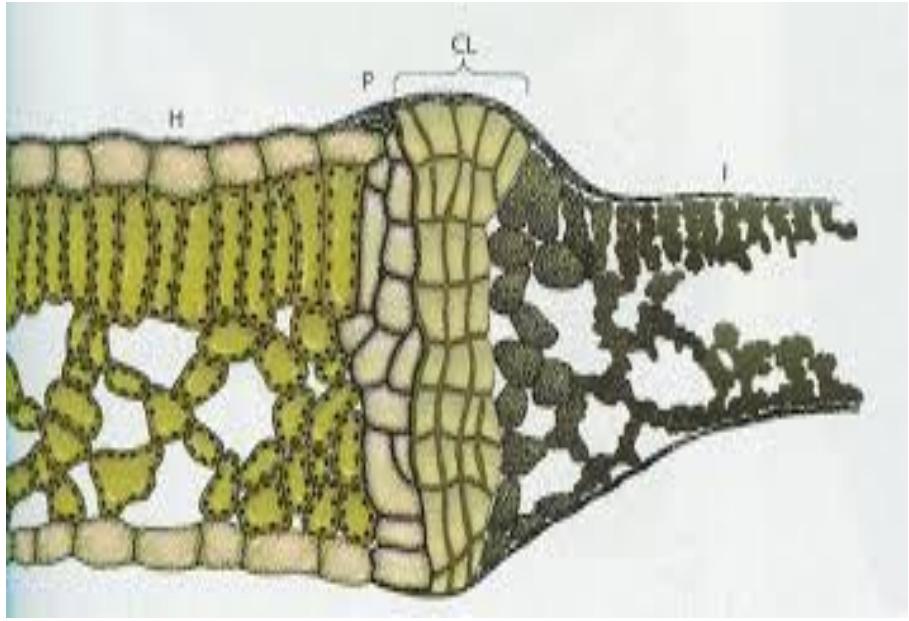
Tyloses formation in Vessels

(d) Formation of Cork Layers:

- Some pathogens like certain bacteria, some fungi and even some viruses and nematodes stimulate the host to form multilayered cork cells in response to infection
- These layers inhibit the further invasion by the pathogen and also block the flow of toxic substances secreted by the pathogen
- Cork layers also stop the flow of nutrients of the host thus also depriving the pathogen of the nutrients

Ex. Potato tuber infected with *Rhizoctonia sp.*

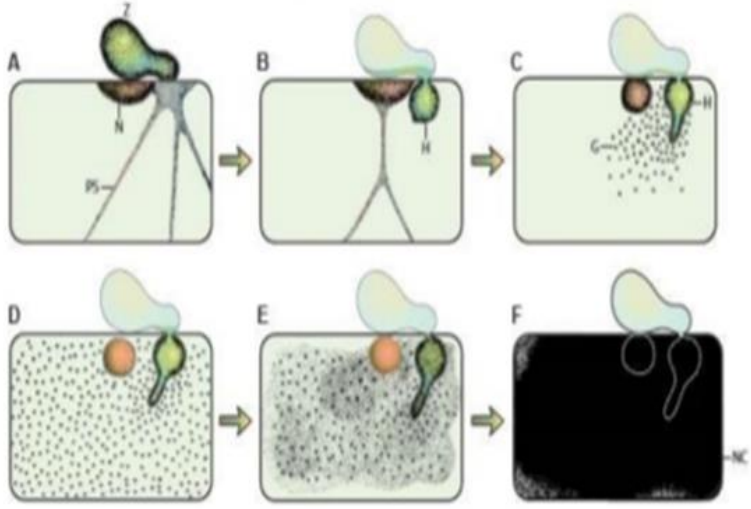




(iv) Necrosis or Hypersensitive Type of Defense:

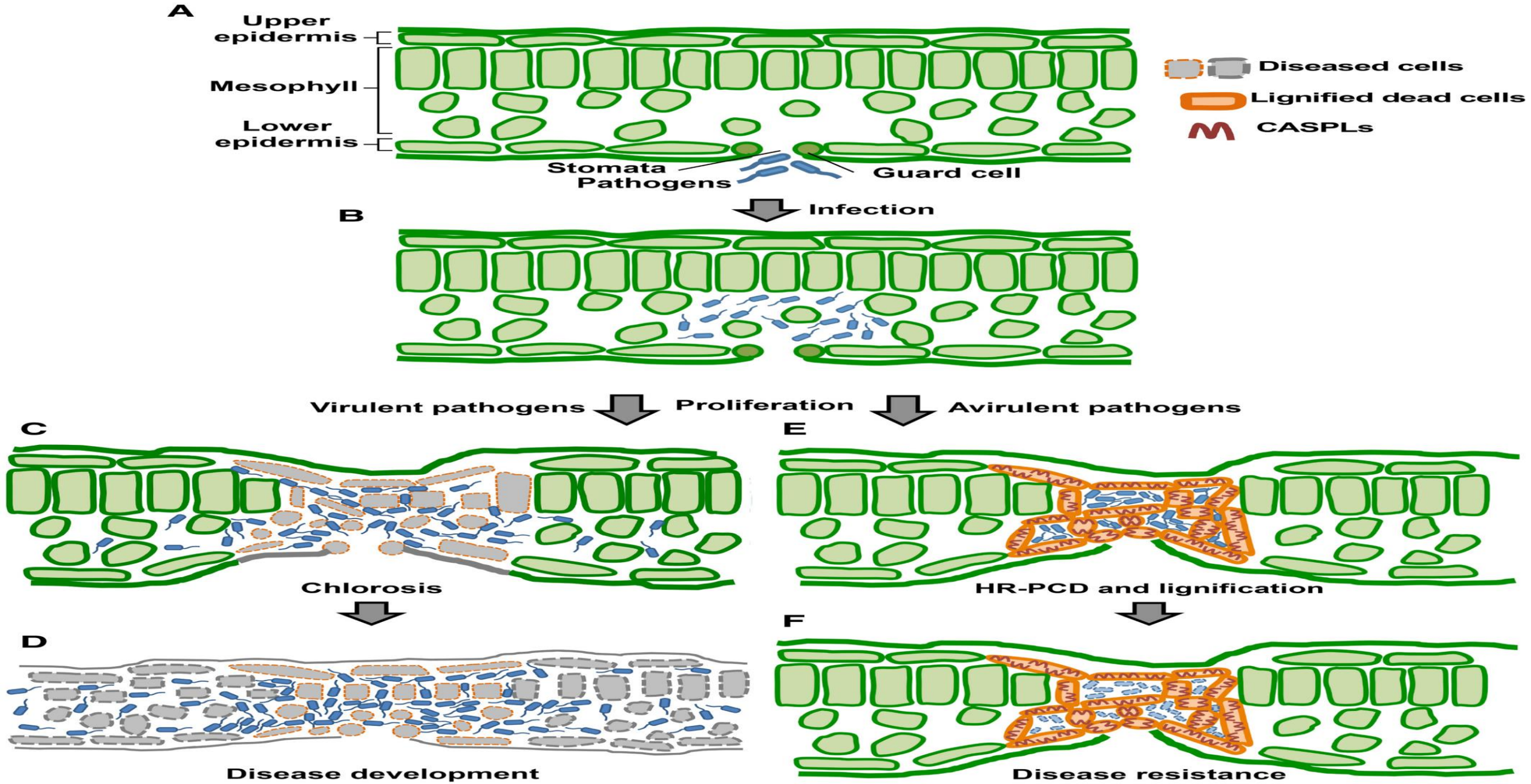
- In this case, the host nucleus moves toward the pathogen and soon disintegrates into brown granules
- Soon the cell membrane swells and finally the cell bursts and dies
- These cause the pathogen nucleus to disintegrate into a homogenous mass and its cytoplasm dense resulting the pathogen fails to grow beyond the necrotic or dead cells and the further growth of the pathogen is stopped

Ex. *Synchytrium endobioticum* causing wart disease of potato, *Phytophthora infestans* causing late blight disease of potato and *Pyricularia oryzae* causing blast of rice



N, nucleus; PS, protoplasmic strands; Z, zoospore; H, hypha; G, granular material; NC, necrotic cell

(Tomiyama *et al.*, 1999)


A
Upper epidermis
Mesophyll
Lower epidermis
Stomata
Pathogens
Guard cell
Diseased cells
Lignified dead cells
CASPLs
Infection
B
Virulent pathogens
Proliferation
Avirulent pathogens
C
Chlorosis
E
HR-PCD and lignification
D
Disease development
F
Disease resistance

II. Biochemical Defense

Pre Existing Biochemical Defense	Induced Biochemical Defense
• Inhibitor released by the plants in environment • Inhibitors in plant cell before infection • Lack of essential factors	• HR • Disruption of Host cell membrane • Host cell resistance • Release of antimicrobials • Detoxification of pathogens toxin • Acquired resistance • Plantibodies • Genetically induced disease resistance

(A) Preexisting Biochemical Defense

(i) Inhibitors Released at the Pre penetration Stage:

- Plant generally exudes organic substance through above ground parts (phyllosphere) and roots (rhizosphere)
- Some of the compounds released by some plants are known to have an inhibitory effect on certain pathogens during the prepenetration stage

Ex. Fungistatic chemicals released by tomato and sugar beet to prevent the germination of *Botrytis* and *Cercospora*

Ex. Presence of phenolics like protocatechuic acid and catechol in scales of red onion variety inhibit the germination of conidia of *Colletotrichum circinans*

(ii) Inhibitors in plant cell before infection:

- Presence of several phenolics, tannins and some fatty acid like compounds in cells

Ex. The tubers of resistance vars of potato against potato scab disease contain higher concentrations of chlorogenic acid around the lenticels and tubers

Ex. Several other compounds like saponin tomatin in tomato and avinacin in oats have antifungal activity

Ex. Some enzymes like glucanases and chitinases present in cells of some plants may break down the cell wall components of pathogens

(iii) Lack of essential factors:

a. Lack of recognition between host and pathogen like oligo and polysaccharides, proteins or glycoproteins

Ex. Varieties of linseed which have an antigen common to their pathogen are susceptible to the disease rust of linseed caused by *Melampsora lini*

Ex. Leaf spot disease of cotton caused by *Xanthomonas campestris pv. malvacearum*

b. Lack of host receptor and sensitive sites for toxin

c. Lack of nutrients essential for pathogen

Ex. Rhizoctonia infection in only those plants having substances help to formation of hyphal cushion

(B) Post-Infection-Biochemical Defense

- These substances are generally produced around the site of infection or injury with the main aim at overcoming the problem

(i) Hypersensitive Responses (HR):

- ✓Localized induced cell death in the host at the site of infection
- ✓Rapid burst of oxidative reactions
- ✓Increased ion movement (K^+, H^+) through cell membrane
- ✓Disruption of cell membrane and loss of compartmentalization
- ✓Crosslinking of phenolics with cell components
- ✓Production of phytoalexins
- ✓Production of PR Proteins

(ii)Disruption of Host cell membrane:

- Structural and permeability changes in cell membrane due to infection
- Production of signal transducers like Protein kinases, calcium ions, phosphorylases, phospholipases, ATPases, H_2O_2 and ethylene
- Release of Systemic signal transducers like salicylic acid, jasmonic acid, systemin, fatty acids and oligogalacturonides
- Release and accumulation of reactive oxygen radicals and lipoxygenase enzyme
- Activation of phenol oxidases and oxidation of phenolics
- Rapid generation of superoxidase (O_2-) and Hydroxyl radicals (OH)

(iii) Host cell resistance:

- Accumulation of defense related substances near the cell wall forming insoluble structures

Ex. Callose, Glycoprotein rich in hydroxyproline, lignin, suberin and silicon and calcium like minerals

(iv) Release of antimicrobials in attacked cells:

a. Common Phenolics:

- Production and accumulation after infection in resistant varieties of plants showing combined effects on pathogens
- The synthesis of phenolic compounds takes place either through “acetic acid pathway” or “Shikimic acid pathway”
- These are Chlorogenic acid, Caffeic acid, Ferulic acid

b. Phytoalexins:

- Phytoalexins are toxic antimicrobial substances synthesized ‘de novo’ in the plants in response to injury and infection
- Muller and Borger (1940) used this term for fungi static compounds produced by plants in response to mechanical or chemical injury or infection
- Phytoalexins are lipophilic compounds believed to be synthesized in living cells leading to necrosis
- Phytoalexins were first detected after a study of late blight of potato caused by *Phytophthora infestans*
- It is a metabolite of the host plant interacts with specific receptor on the pathogen’s membrane resulting in the secretion of “phytoalexin elicitor”

Phytoalexin	Host	Pathogen
Ipomeamarone	***Ipomoea batata***	***Ceratocystis fimbriata***
Orchinol	***Orchis milliteris***	***Rhizoctonia ripens***
Pistatin	***Pisum sativum***	***Sclerotinia fructicola***
Phaseolin	***Phaseolus vulgaris***	***Sclerotinia fructicola***
Medicarpin	***Medicago sativa***	***Helminthosporium turcicum***
Rishitin	***Potato tubers***	***Phytophthora infestance***
Isocoumarin	***Daucas carota***	***Ceratocystis fimbriata***
Gossypol	***Gossypium hirsutum***	***Verticillium alboatrum***
Cicerin	***Cicer areitinum***	***Ascochyta rabiel***
Glyceolin	***Soybean, Alfalfa, Clover***	***Glomerella cingulata***
Capisidiol	***Pepper***	***Pernospora trifoliorum***
Trifolirhizin	***Trifolium pratense***	***Helminthosporium turcicum***

c. Phenolics from non toxic glycosides:

- Plant glycosides converted into phenolics through enzyme glycosidase hydrolyses

d. Polyphenol oxidase:

- Production of phenol oxidizing enzymes to convert phenols to more toxic quinines
- Enzyme peroxidases liberate H_2O_2

e. Pathogen Related Protein (PR Proteins):

- Present in plant cells in trace amount, but induced in greater amount after pathogens entry
- These are highly acidic and extremely basic and also high soluble and reactive
- Classified on the basis of function, serological relationship, amino acid sequence and molecular weight
- Inhibit spore release, germination and strengthens the host cell wall

PR Protein	Type Member	Properties
PR 1	Tobacco PR 1a	AntiOomycetes, Antifungal
PR 2	Tobacco PR 2	B 1-3 glucanase
PR 3	Tobacco P, Q	Chitinase type I, II, III, IV, V VI, VII
PR 4	Tobacco R	Chitinase type I, II
PR 5	Tobacco S	Thaumatin like
PR 6	Tomato inhibitor I	Proteinase inhibitor
PR 7	Tomato P_{69}	Endoproteinase
. . .		
PR 14	Barley LTP 4	Lipid transfer Protein

(v) Detoxification of pathogens toxin:

- Resistant varieties of plants metabolise the pathogen toxins or combined with less toxic compounds

Ex. Detoxification of HC-Toxin and Pyricularin by resistant varieties of maize and rice

(vi) Acquired resistance:

- This reduces penetration of pathogen
- Local acquired resistance (LAR) in the beginning
- Systemic acquired resistance (SAR)

Ex. Salicylic acid, arachidonic acid, 2,6- dicholoroisonicotinic acid in resistant plants are applied as spray or injection to induce LAR and SAR in plants

(vii) Plantibodies:

- This is about immunization of plants through transgenics
- Antibodies encoded by animal genes but produced in and by plants

Ex. Transgenic plants producing plantibodies against coat protein of viruses have been produced

(viii) Genetically induced disease resistance:

a. Pant derived genes:

- Plant derived R gene into plants capable to produce resistance

Ex. Hm1 gene in corn in 1992 for detoxification of HC toxin produced by *Choclibolus carbonum*

Ex. Tobacco plants introduced with Chitinase R gene become resistant for *Rhizoctonia solani*

b. Pathogen derived gene:

- Incorporation of pathogen derived genes in plants

Ex. Tobacco transformation to express coat protein gene of TMV

Summary

- Plants protect themselves at pre germination and germination stages of pathogen's entry with structural defense (Pre existing structures)
- Biochemical defense activated after plant fails to protect by structural defense
- Signal transduction takes place when pathogen's elicitors reaches to host's receptors for induced structural and biochemical defense
- Plant defense is not a single mechanism, but is a cumulative effect of more than one mechanisms
- All these responses exhibited by plants are governed by it genetic constitution and can be manipulated

References

- **An Overview of Plant Defenses against Pathogens and Herbivores**
- **Brian C. Freeman and Gwyn A. Beattie**
- **Iowa State University**
- **Freeman, B.C. and G.A. Beattie. 2008. An Overview of Plant Defenses against Pathogens and Herbivores. The Plant Health Instructor. DOI: 10.1094/PHI-I-2008-0226-01**

Thank You…..