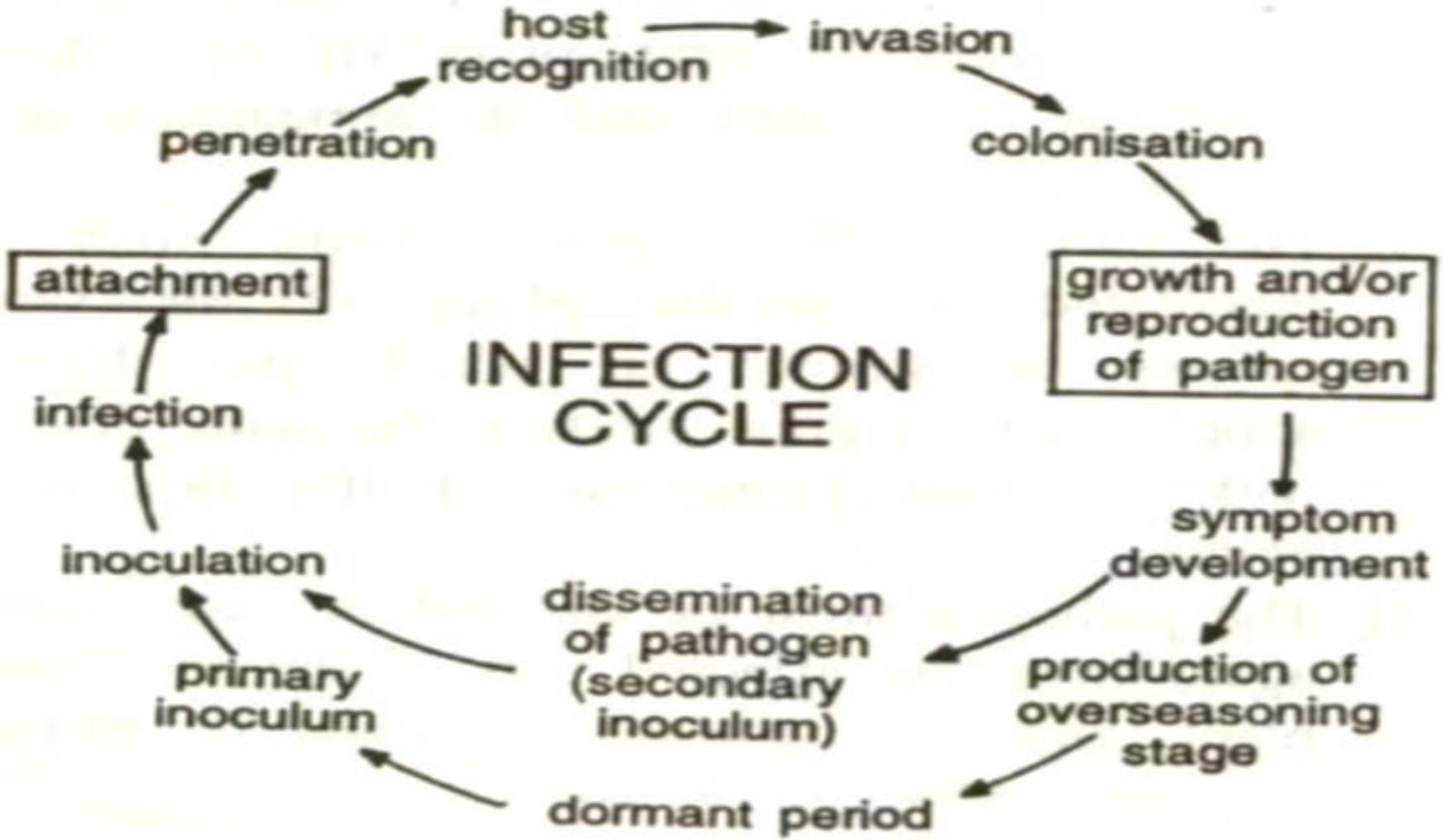
MECHANISM OF INFECTION

Penetration or PHENOMENON OF INFECTION

(Pre-penetration,Penetration,Post-penetration)

- Infection process means establishment of pathogen in the host plant.
- Entry and colonization of pathogen in the host tissues is known as establishment and
- the infective propagules coming in contact with the host are known as inoculum.

Title: PHENOMENON OF INFECTION



Stages in the development of infection or disease cycle

1. PRE-PENETRATION

Depending upon the plant pathogen activity, the plant pathogens are classified in to 2 categories.

ACTIVE INVADERS	PASSIVE INVADERS
Pathogens which make an aggressive effort to gain entry into intact host cells	No aggressive effort
They do not require help of any external agency to gain entry into host cells	Require help of external agencies like insect vectors or wounds caused by agricultural implements.
Eg. Phyto-pathogenic fungi Phanerogamic parasites	Eg. Plant viruses Phyto-pathogenic bacteria

1.Pre-penetration

- Dormant period
- Primary Incoculum
- Inoculation
- Infection
- Attachment

"Primary Inoculum"

- The inoculum that initiates the epidemic is called primary inoculum
- Primary inoculum may be:
- Sclerosium,
- Dormant Sexual spore like OOSopre,
- Clamydospore,
- Fungal Mycellium,
- Bacterial Endospre(Gram Positive Bacteria)
- Rizomorph (root like structures) etc.

"Infection"

- Infection is the establishment of parasitic relationship between two organisms,
- following entry or penetration (or) the establishment of a parasite within a host plant.

PENETRATION

Pathogens penetrate plant surfaces by direct penetration or indirectly through wounds or natural openings.

- INDIRECT PENETRATION – caused by wounds,natural opening
- DIRECT PENETRATION – Most fungi, nematodes and parasitic higher plants are capable of penetrating the host surface directly. However, the plants are provided with different mechanisms of defense which include structural features of the host, presence of chemical coverings on the cell walls, and anti-infection biochemical nature of the protoplasm. Hence, the pathogen should have mechanisms to overcome these barriers for direct penetration

1 - Break Down Of Physical Barriers

2 -Break Down of Chemical Barriers

2.Penetration

- Host recognition

“Host recognition”

- Pathogens penetrate plant surfaces by direct penetration or
- indirectly through wounds or natural openings.
- Bacteria enter plants mostly through wounds and less frequently through natural openings.
- Viruses, viroids, mollicutes, fastidious bacteria enter through wounds made by vectors.
- Fungi, nematodes and parasitic higher plants enter through direct penetration and less frequently through natural openings and wounds

3.Post Penetration

- Invasion
- Colonization
- Incubation period

"Invasion & Colonization"

- A parasitic relationship is formed between host cytoplasm and parasite cytoplasm.
- During infection,
- pathogens grow and multiply within the plant tissues.
- Invasion of plant tissues by the pathogen, and
- growth and reproduction of the pathogen (colonization) are two concurrent stages of disease development.

"Symptoms development"

- Incubation period:
- The time interval between inoculation and appearance of disease symptoms is called the incubation period.

- Secondry Inoculum:
- The inoculum which develops from the primary symptom is called secondary inoculum.
- Secondry Inoculum may be Air borne Asexual spores like Conidia, Zoospres, Infected Mycellium

“Production of Over seasoning Stage or Exit of Pathogen from Host”.

- After invasion and colonization of the host, the pathogens come out of the host
- to maintain the continuity of the infection chain or disease cycle and escape death due to overcrowding.

Over seasoning stage

- Viruses can exist only with the living protoplasm and hence
- disseminated through their animate vectors like insects, fungi, nematodes, etc.
- The bacteria ooze out in the form of slime on the host surface from where
- they can be disseminated through water and insects.
- However, the fungi have the most elaborate system of exit through secondary Inoculum Or make Oospre, Sclerocia, Clamydospre(Hard or thick form of Mycellium)

PLANT DEFENCE SYSTEMS

INTRODUCTION

- Plants represent a rich source of nutrients for many organisms including fungi, bacteria, virus, nematodes, insects, and vertebrates.
- Plant lacking an immune system comparable to animals.

- Plants have developed a stunning array of structural, chemical, and protein-based defenses designed to detect invading organisms and stop them before they are able to cause extensive damage.

In general, 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.

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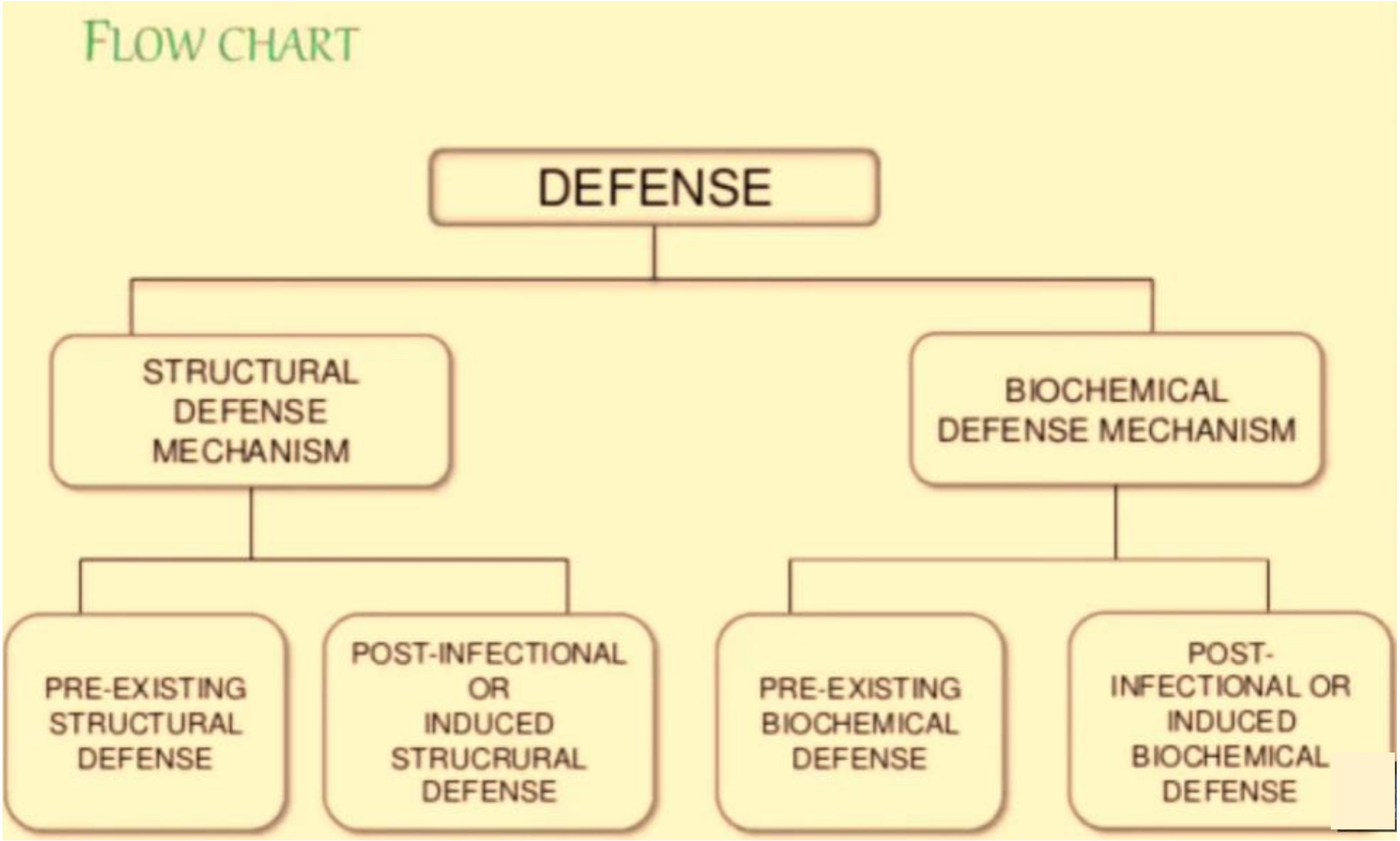
DEFENSE
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(A) PRE-EXISTING DEFENSE STRUCTURES

- ✓ The external and internal structural barriers existing before attack by pathogen are called Pre-existing defense structures.
- ✓ The surface of the host plant forms the first line of defense against pathogen.
- ✓ Several characters of the plants surface function as barriers to penetration which pathogen must breach to enter the host.

(A) PRE-EXISTING DEFENSE STRUCTURES

- ✓ The external and internal structural barriers existing before attack by pathogen are called Pre-existing defense structures.
- ✓ The surface of the host plant forms the first line of defense against pathogen.
- ✓ Several characters of the plants surface function as barriers to penetration which pathogen must breach to enter the host.
- ✓ Important pre-existing defense structures are-

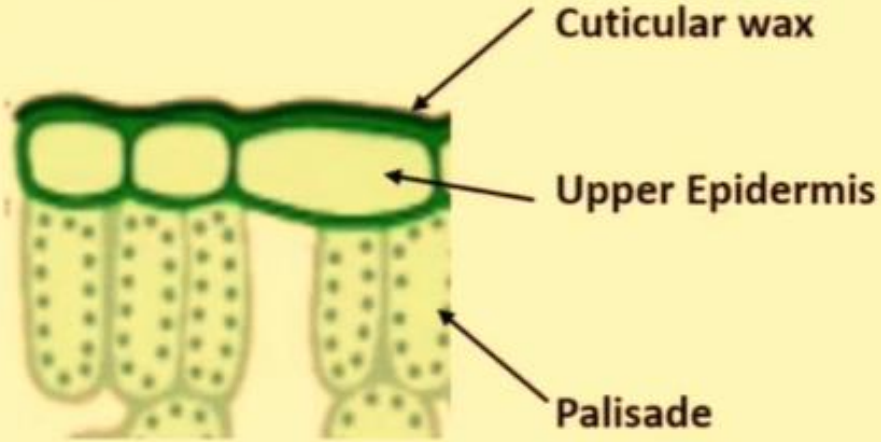
1. **Cuticular wax**
2. **Cuticle**
3. **Epidermal cell Walls**
4. **Natural openings**
5. **Internal defense structures**

(A) Pre EXISTING DEFENCE STRUCTURE :-

(i) CUTICULAR WAX :

✓ Wax :

1. Mixtures of long chain aliphatic and nonpolar compound.
2. Synthesized by epidermis of some plants.
3. Forms a protective coating.
4. Extremely hydrophobic



✓ Forms a hydrophobic surface where water is repelled.

✓ Pathogen does not get sufficient water to germinate and multiply.

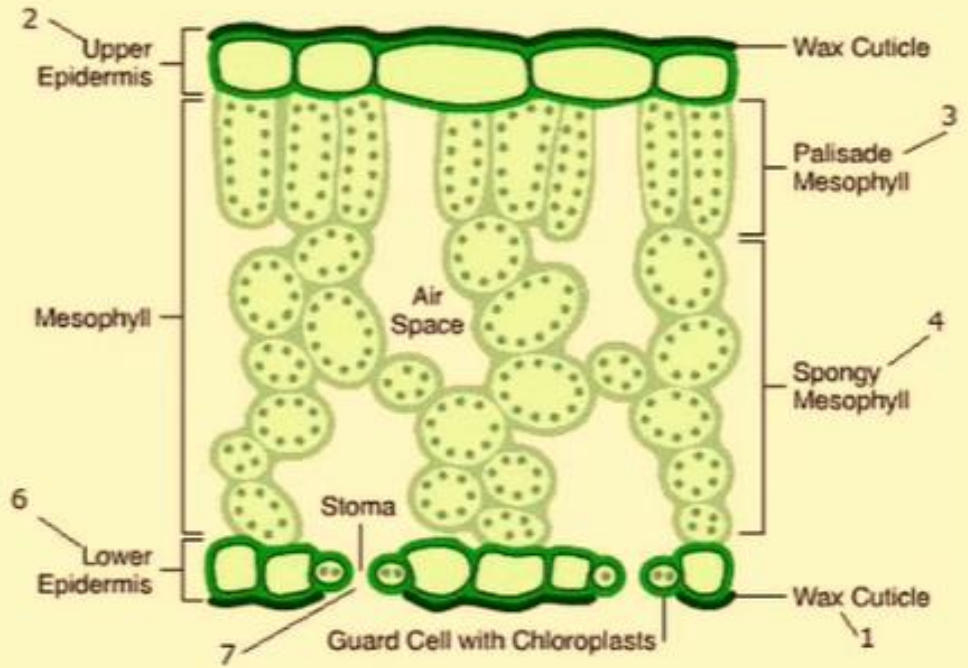
✓ In addition, a negative charge develops on the leaf surface due to the presence of fatty acids.

✓ Negative charge prevents/reduces the chance of infection by many pathogens.

(A) Pre EXISTING DEFENCE STRUCTURE :-

(III) EPIDERMIS :

- ✓ Lignification and Silicification of epidermal cell walls.
- ✓ It increases toughness and thickness of outer walls of epidermal cells.
- ✓ These structures directly prevents penetration of the pathogen.

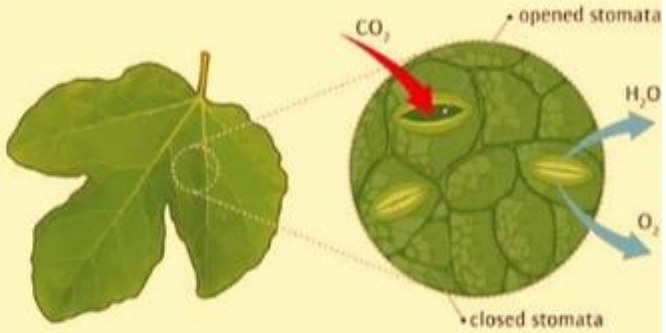


(A) Pre EXISTING DEFENCE STRUCTURE :-

(IV) NATURAL OPENINGS AND ITS STRUCTURE :

✓ Most pathogen enter plants through natural openings like Stomata, Lenticels and Nectaries.

Example : *Puccinia graminis tritici* (Stem rust of wheat) can enters into its host only when stomata are open.



✓ Structure of these natural openings provide resistance to penetration by certain pathogens.

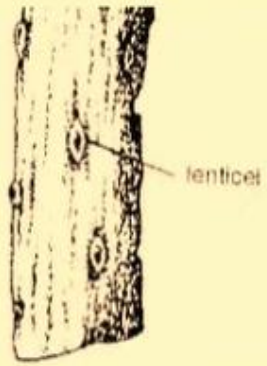
(a) STOMATA

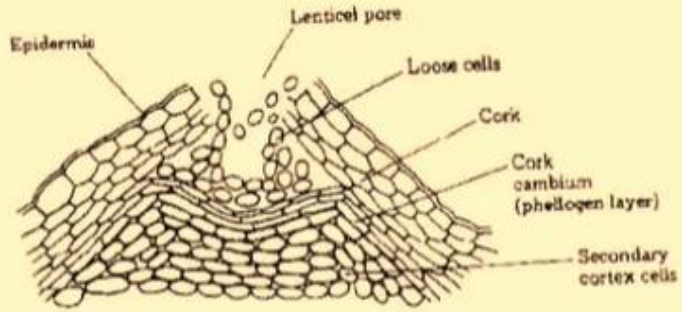
✓ Structure of stomata provides resistance to penetration by certain pathogens.

Example : In Szincum variety of citrus, the stomata are smaller in size and having very narrow openings surrounded by Broad lipped raised guards cells which prevent entry of water drops containing citrus canker bacterium (*Xanthomonas citri*).

(b) LENTICELS

- ✓ Lenticels are the openings found on stem, fruits and tubers.
- ✓ Size, shape and internal structures of lenticels can increase or decrease the incidence of penetration of the pathogen.





(c) NECTARIES

- ✓ Nectaries are found in the epidermis of various parts of the plant body.
- ✓ May play a defensive role due to high osmotic concentration of the nectar and presence of hairs.

(B) POST-INFECTIONAL OR INDUCED STRUCTURAL DEFENSE MECHANISMS

- ✓ After successfully managing pre-existing defense structures of the host, pathogen invades the cells and tissues of the host.

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- ✓ After successfully managing pre-existing defense structures of the host, pathogen invades the cells and tissues of the host.
- ✓ In order to check the further invasion by the pathogen, the host plants develop some structures/mechanisms to defend the act of pathogen.
- ✓ Even after the establishment of infection, the host defense system tries to create barriers for further colonization of tissues.
- ✓ This may be at two levels:
  - (a) Cellular defense structures (hyphal sheathing)
  - (b) Histological defense barriers

(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 –

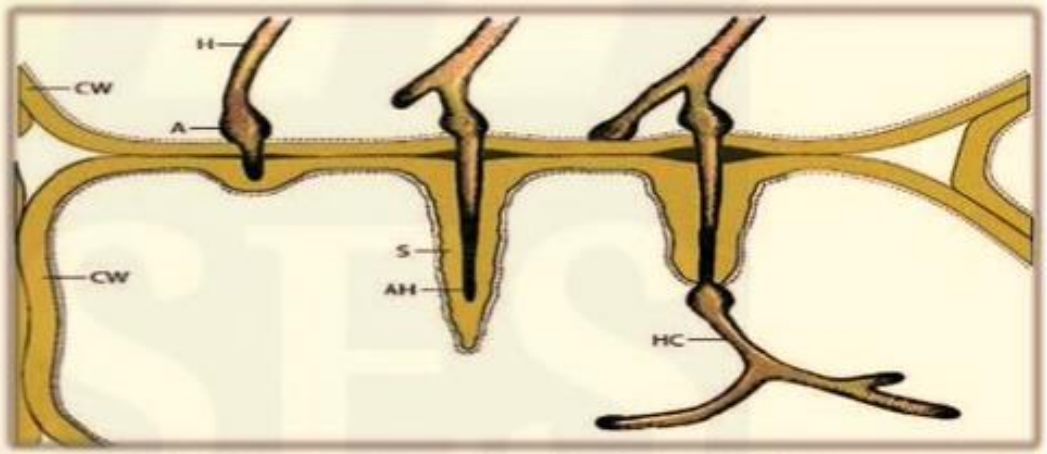
(i) Cell walls thicken in response to the pathogen by producing a cellulose material

(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

2 CELLULAR DEFENSE STRUCTURE

- **Hyphal sheathing:-**



Hyphal sheathing is observed in flax infected with *Fusarium oxysporum* f.sp. *lini*.

2. DEFENSE STRUCTURES DEVELOPED BY THE TISSUES :

Four different types of defense structures may be developed in tissues in response to penetration by pathogen : These are

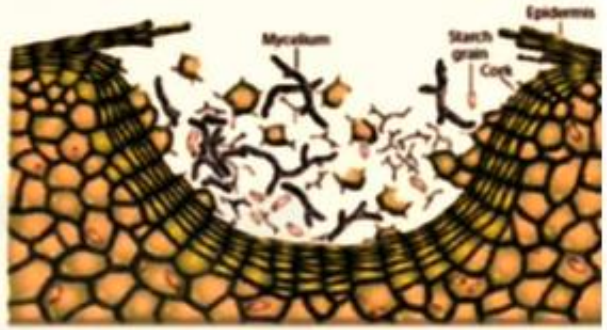
(i) Suberization and formation of cork layers

(ii) Formation of abscission layers

(iii) Formation of tyloses

(iv) Gum deposition

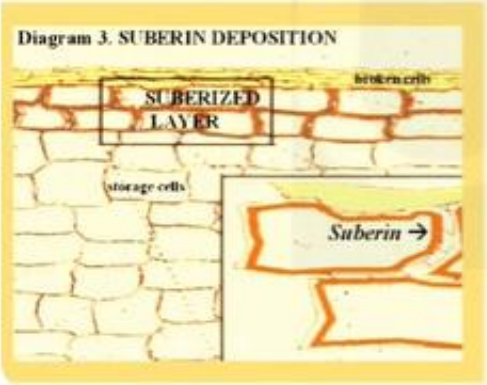
SUBERIZATION AND FORMATION OF CORK LAYERS :



Ex- Potato tubers infected by *Rhizoctonia*

- ✓ Some pathogens like certain bacteria, fungi, some viruses and nematodes stimulate -
  - (a) Suberization of the host cells and formation of cork layer and
  - (b) Formation of multilayered cork cells in response to infection.
- ✓ Cork layers checks :
- ✓ The further invasion by the pathogen ,

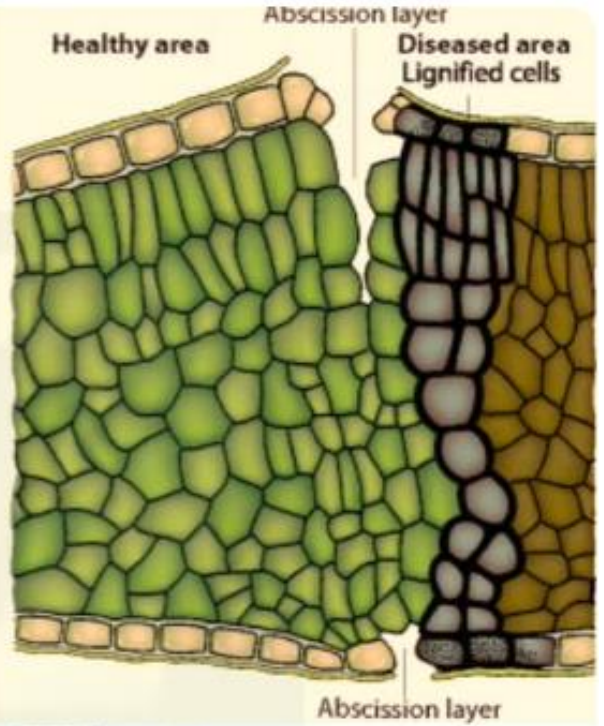
- ✓ Example : 1. Potato tuber disease caused by *Rhizoctonia* sp.,

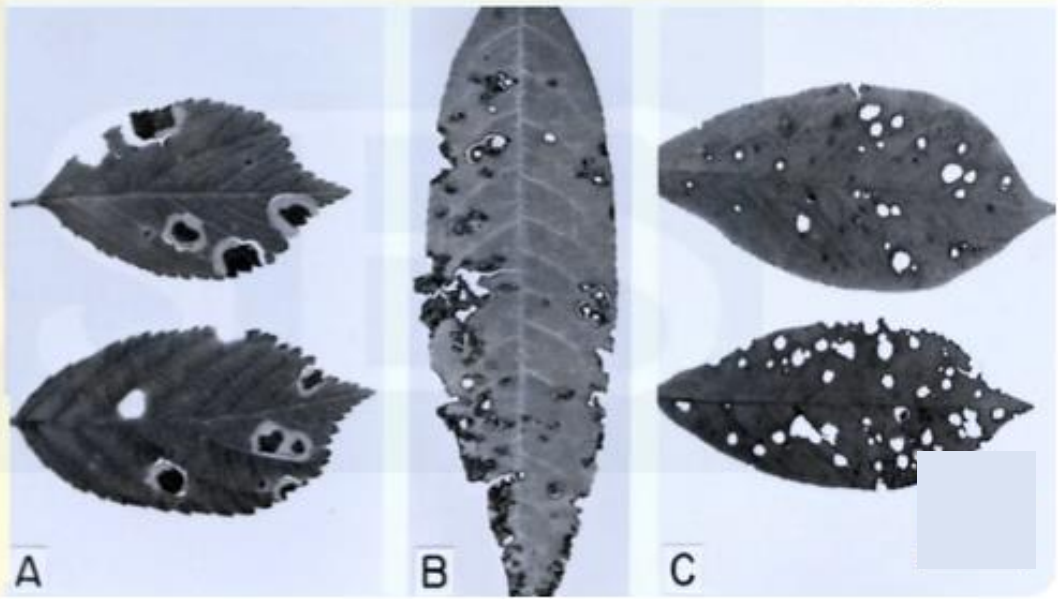


2. Scab of potato caused by *Streptomyces scabies*.

(b) FORMATION OF ABSCISSION LAYERS :

- ✓ Abscission layers are naturally formed in plants as a device for dropping off older leaves and mature fruits from the plant.
- ✓ But in some plants, abscission layers develop in their young leaves in response to infection by Pathogen.
- ✓ This layer helps in dropping-off infected or invaded plant tissue or parts, along with pathogen from the plant body.
- ✓ An abscission layer a gap is formed between two layers of cells surrounding the point of infection.





Ex: *Xanthomonas pruni,* and *Closterosporium carpophylum* on peach leaves.

(c) FORMATION OF 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.
- ✓ The size and number of tyloses physically block the vessel.
- ✓ Example : Sweet potato caused by *Fusarium oxysporum*.

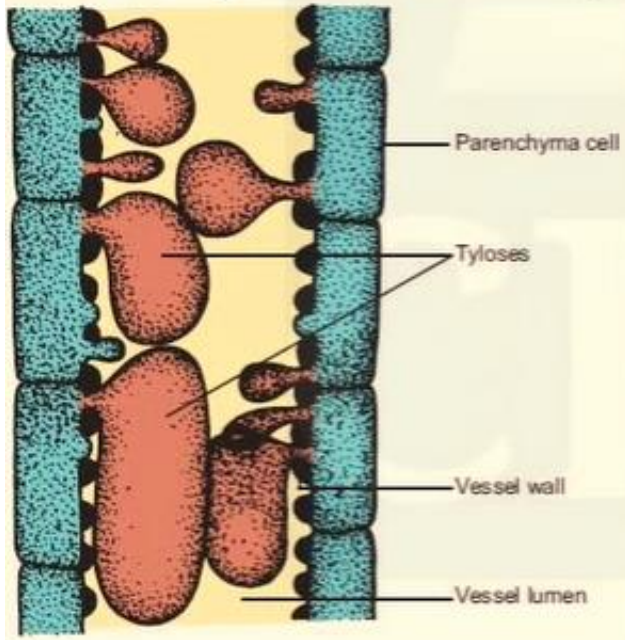
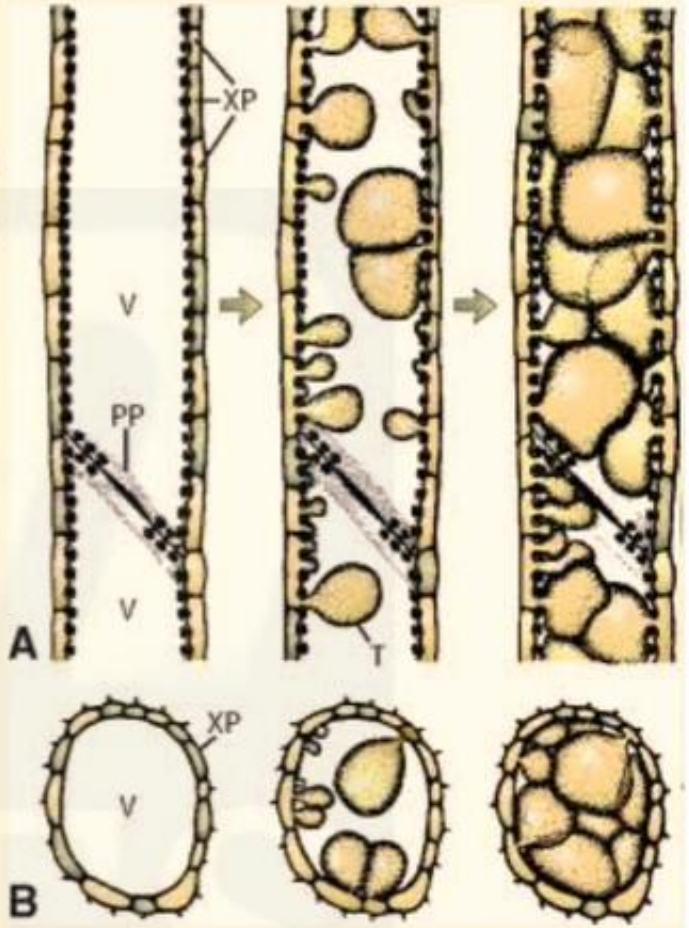


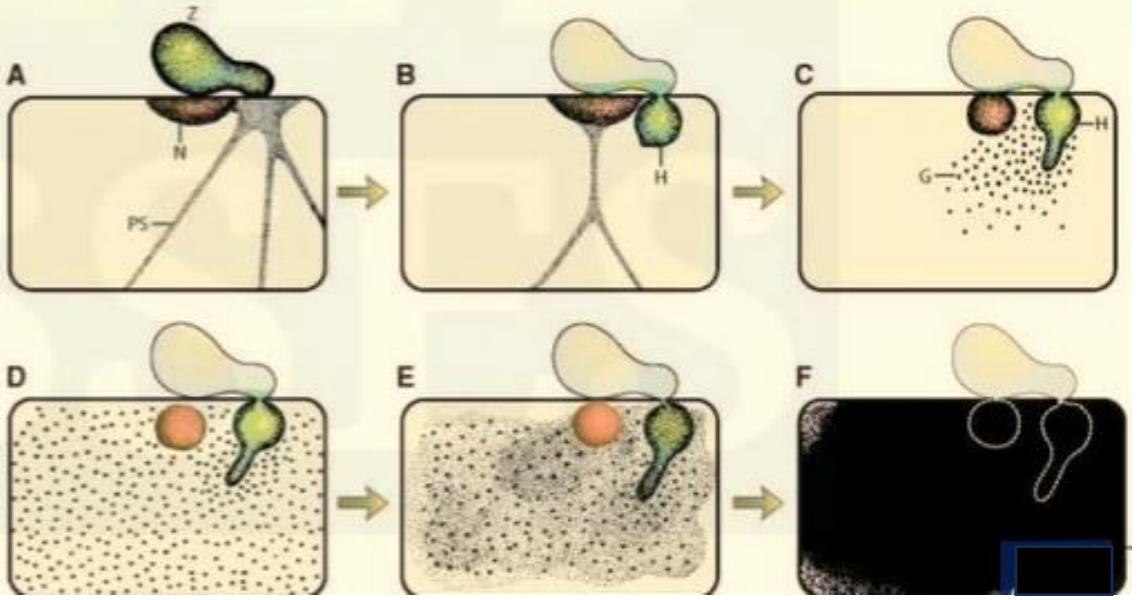
Figure 10.9: Structure of tyloses



(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



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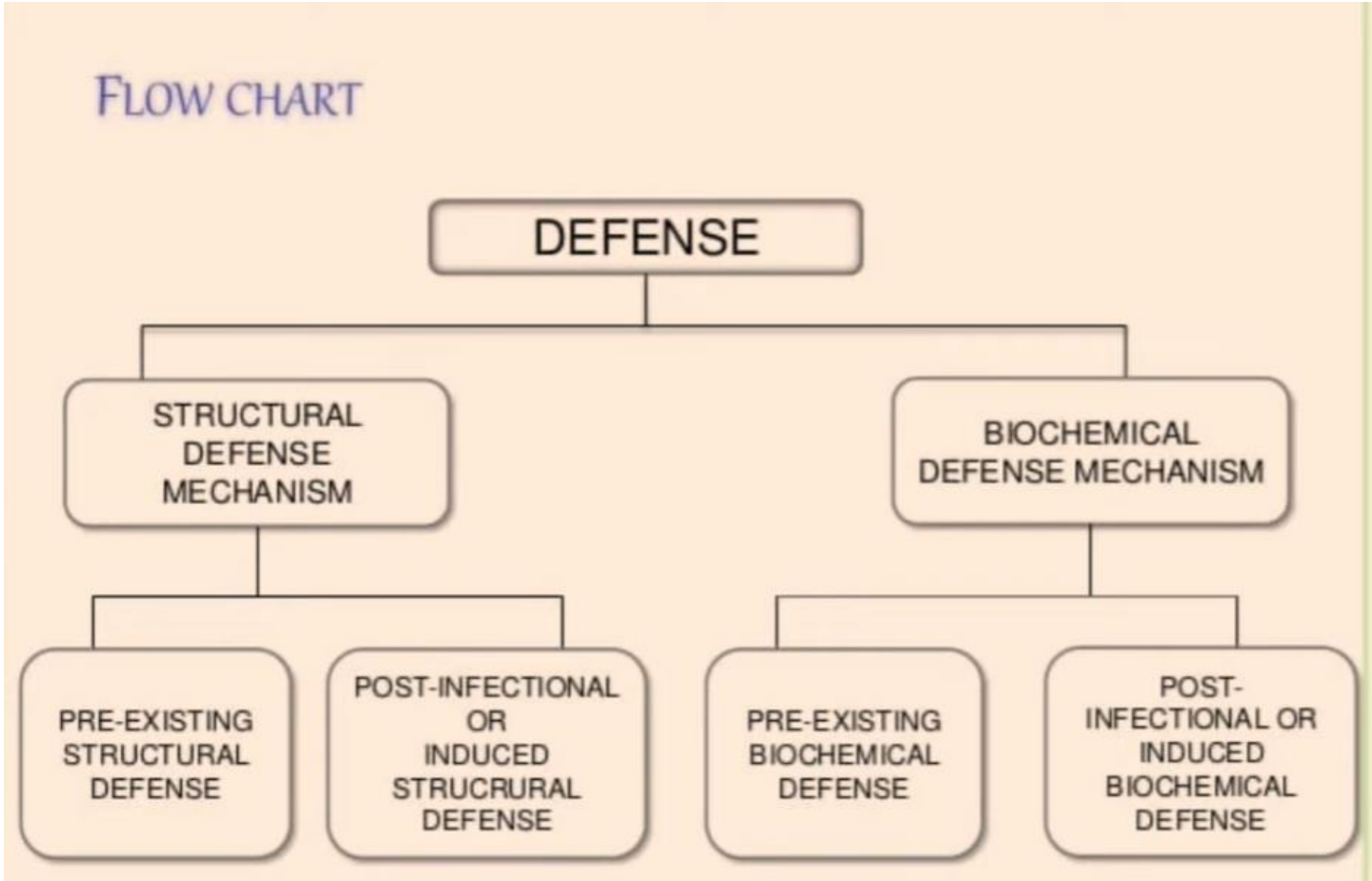
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II. Biochemical Defense

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 resis e

II. BIOCHEMICAL DEFENSE

A) Preexisting Biochemical Defence:

i) Inhibitors released in prepenetration stage

ii) Lack of nutrients essential for the pathogen

iii) Absence of common antigen in host plant

B) Post-Infection Biochemical Defence:

i) Phenolic compounds

ii) Phytoalexins

iii) Substance produced in host to resist enzymes produced by pathogen

iv) Detoxification of pathogen toxins and enzymes

v) Biochemical alterations

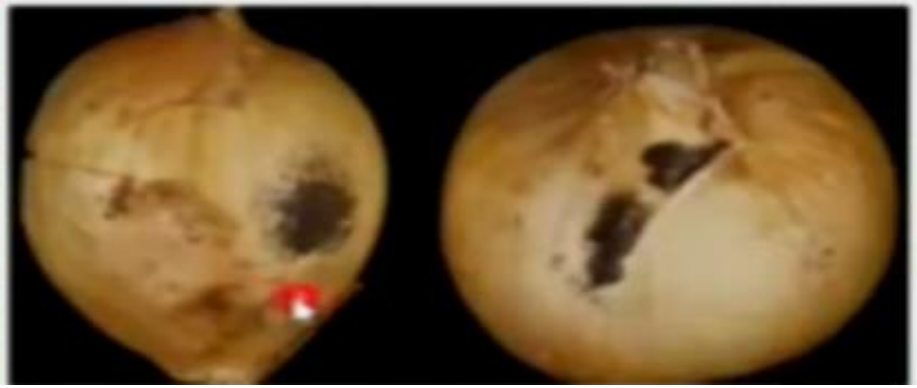
(A) Preexisting Biochemical Defense

(i) Inhibitors Released at the Pre penetration Stage:

- Plant generally exudes organic substance through above (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:

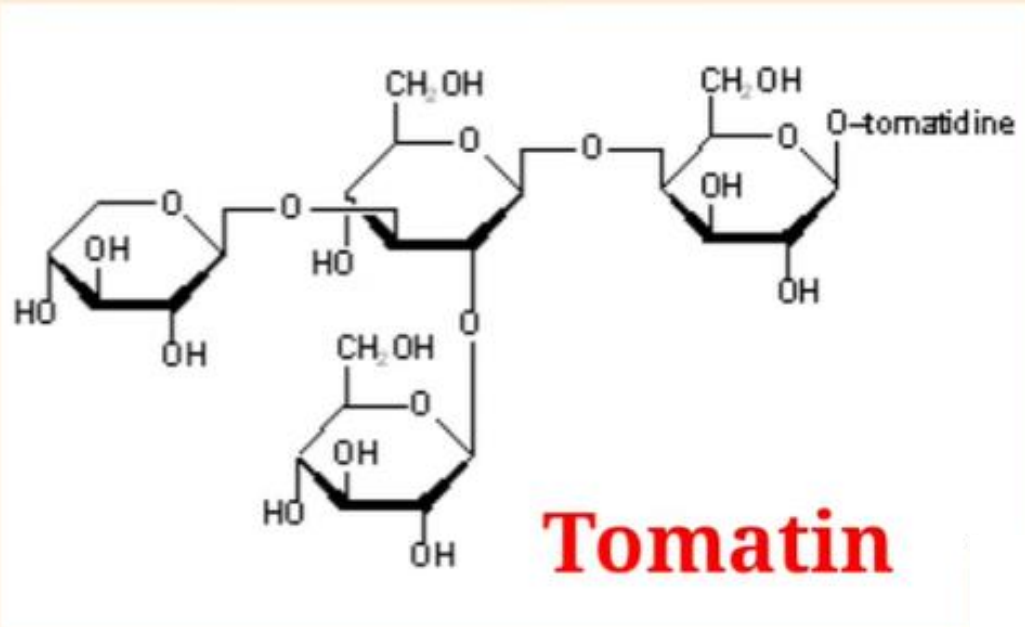
- Presence of several phenolics, tannins and some fatty acid like compounds in cells

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

Saponin



Tomatin

(iii) Lack of essential factors:

a. Lack of recognition between host and pathogen like 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 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, 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	Chemical Nature of the phytoalexin
1. Ipomeamarone	*Ipomoea batata*	*Ceratocystis fimbriata*	Furanosesquiterepene ketone.
2. Orchinol	*Orchid malitaris*	*Rhizoctonia repens*	Phenanthrene
3. Pistatin	*Pisum sativum*	*Ascochyta pisi*	Kievitone
4. Phaseolin	*Phaseolus* sp.	*Monilia fruticola*	
5. Medicaripin	*Medicago lupulina*	*Colletotrichum phomoides*	Dimethyl-homoptero-carpin
6. Rishitin	*Solanum tuberosum* var. *rishiri*	*Phytophthora infestans*	Nonsequiter-pene
7. Isocoumarin	*Daucus carrota*	*Fusarium oxysporum f. lycopersici*	-
		Rhizopus solonifer	
8. *Cicerin*	*Cicer arietinum*	*Ascochyta rabie*	
9. Gossypol	*Gossypium* sp.	*Verticillium alboatrum*	

C. 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

The families of pathogenesis-related proteins

Sr No.	Family	Type /Member	Properties
1.	PR-1	Tobacco PR-1a	Unknown
2.	PR-2	Tobacco PR-2	β-1,3-glucanase
3.	PR-3	Tobacco P,Q	Chitinase type I,II,IV,V,VI,VII
4.	PR-4	Tobacco R	Chitinase type I,II
5.	PR-5	Tobacco S	Thaumatin like protein
6.	PR-6	Tomato Inhibitor-I	Protease inhibitor
7.	PR-7	Tomato P69	Endoproteinase
8.	PR-8	Cucumber chitinase	Chitinase type III
9.	PR-9	Tobacco lignin forming peroxidase	Peroxidase
10.	PR-10	Parsley PR-1	Ribosome inactivating protein
11.	PR-11	Tobacco class V chitinase	Chitinase type I
12.	PR-12	Radish R5 AFP-3	Defensins

(v) Detoxification of pathogens toxin:

- **Resistant varieties of plants metabolise the pathogen toxin 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

THANK YOU