DEPARTMENT OF BOTANY
Guru Ghasidas Vishwavidyalaya, Bilaspur
M. Sc. III Semester
LBT 303: Plant Pathology

Section A

Time: 3 hours **Maximum marks – 60**

Define the following terms **2 × 10 = 20**

1. **Incubation period:** The period of time (or time lapse) between penetration of a host by a pathogen and the first appearance of symptoms on the host. It varies with pathogens, hosts and environmental conditions.
2. **Hyperplasia:** In some diseases, the plant organs become increased in size by the action of the parasites. This increase in size of one or all plant organs brought about by either or both of the two processes hyperplasia and hypertrophy. Gall formation, curling, intumescences and hairy root formation are the example of hyperplasia.
3. **Sterilization** is achieved by exposing materials to lethal agents which may be chemical, physical or ionic in nature or in case of liquids, physical elimination of cells from medium.
4. **Disease Triangle:** The interactions of three components of disease, i.e., the host, pathogen and environment, can be visualized as a disease triangle. The length of each side is proportional to the sum total of the characteristics of each component that favour disease. The interaction of susceptible host plant, virulent pathogen and favourable environmental conditions leads to the development of the disease.

5. **Phytoalexins** (*Phyton* = plant; *alexin* = to ward off) **Muller** and **Borger** (1940) first used the term phytoalexins for fungistatic compounds produced by plants in response to injury (mechanical or chemical) or infection. Phytoalexins are toxic antimicrobial substances produced in appreciable amounts in plants only after stimulation by phytopathogenic micro-organisms or by chemical or mechanical injury.
6. **SAR: Induction of plant defenses by artificial inoculation with microbes or by treatment with chemicals**. Plants develop a generalized resistance in response to infection by a pathogen or treatment with certain natural or synthetic chemical compounds ***i.e. Jasmonic acid, salicylic acid***, ***Probenazole***, ***Riboflavin***.
7. **Phaseolotoxin:** is produced by the bacterium ***Pseudomonas syringae pv. Phaseolicola*** the cause of halo blight of bean. Phaseolotoxin is a modified **ornithine-alanine-arginine** tripeptide carrying a phosphosulfinyl group. It inhibits pyrimidine biosynthesis, reduce the activity of ribosome, interfere with lipid synthesis, change the permeability of membranes and result in the accumulation of large starch grains in the chloroplasts.
8. **Invasion:** of plant tissues by the pathogen, and growth and reproduction of the pathogen (**colonization**) are two concurrent stages of disease development. During establishment,

pathogen produces different substances which include enzymes, toxins, growth hormones and polysaccharides which will help in colonization of the host.

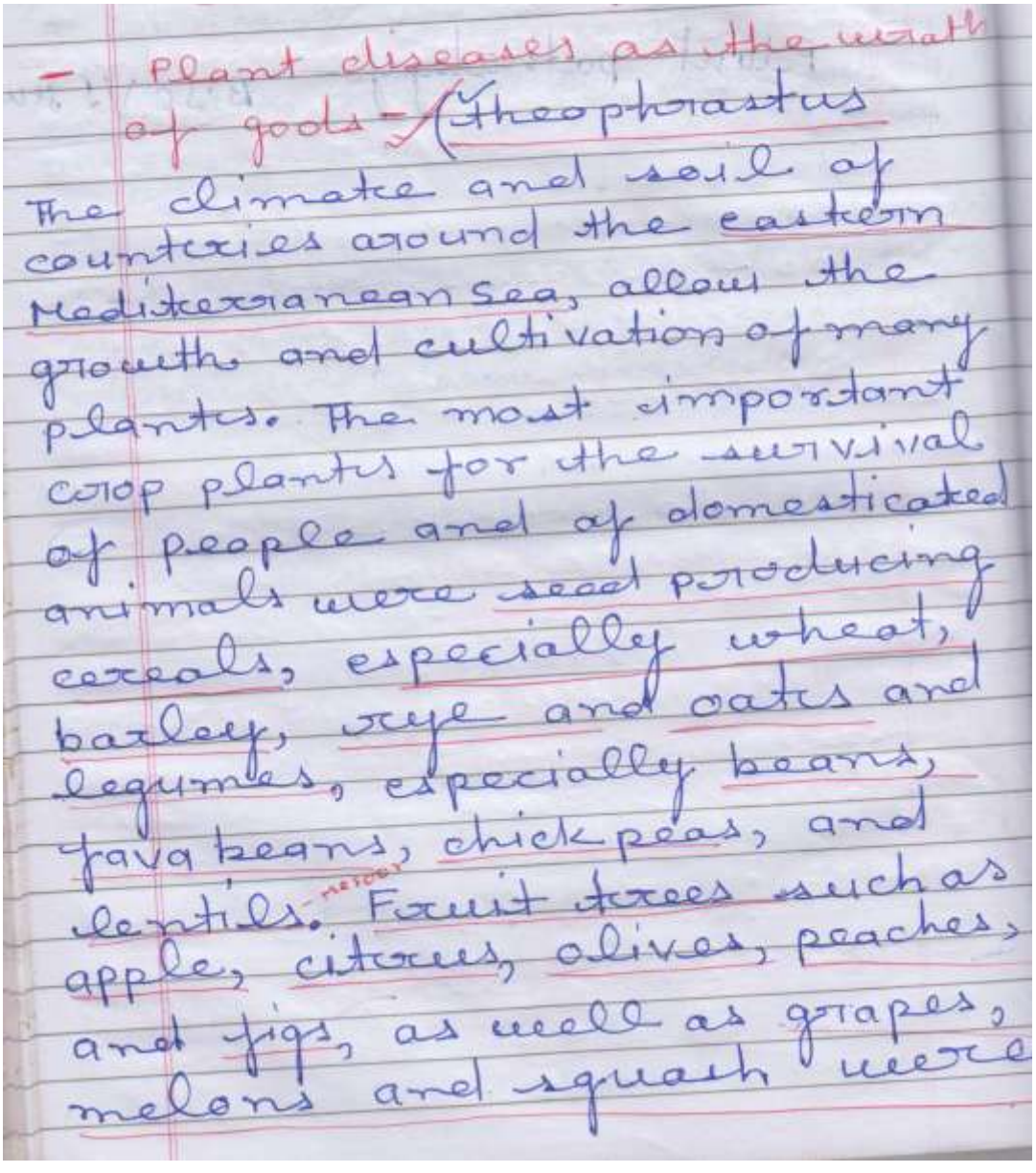
9. **Surface sterilization:** Seeds may be sterilized and germinated *in vitro* to provide clean material. Covering growing shoots for several days or weeks prior to harvesting tissue for culture may supply cleaner material. Explants or material from which material will be cut can be washed in soapy water and then placed under running water for 1 to 2 hours.
10. **Inoculum potential:** The energy of growth of a parasite available for infection of a host at the surface of the host organ to be infected or the resultant of the action of environment, the vigour of the pathogen to establish an infection, the susceptibility of the host and the amount of inoculum present.

Section B

Descriptive answer type questions (attempts any four) **10 × 4 = 40**

1. Write short notes on:

(i) Views of Theophratus



cultivated. All of these crops suffered losses annually due to drought, insects, diseases and weeds. Because most families grew their own crops and depended on their produce for survival, until the next crop produced the following year, losses of any amount caused serious hunger and and survival problem of them. Occurrence of mildew blasts and blights on cereals and legumes.

Mistletoe recognized as the first plant pathogen

Mistletoes are plants that live as parasites on branches of trees but, for various reasons, they have caught the fancy of people in various cultures have made a name for themselves.

(ii) ANTON De BARY (Germany):

- He was the father and founder of modern Mycology.
- He was the founder of modern experimental plant pathology.
- In 1863, he studied the epidemics of late blight and renamed the casual organism as *Phytophthora infestans.*
- He discovered ***heteroecious nature of rust fungi*** (1865).
- He gave detailed account on life cycles of downy mildew genera.
- He studied about vegetable rotting fungi and damping off fungi.
- He wrote a book named "***Morphology and Physiology of fungi, lichens and Myxomycetes***" (1866).
- He reported the role of enzymes and toxins in tissue disintegration caused by *Sclerotinia sclerotiorum*

3. What are phytopathological techniques? Discuss briefly methods of isolation and inoculation.

Phytopathological Techniques

Requirements for isolation of microorganisms: To isolate microorganisms from diseased plant tissues the following things are required:

1. Culture Media

(i) Natural media (ii) Semi-synthetic media (iii) Synthetic media

2. Sterilization

(i) Red heat (ii) Hot air (oven)
(iii) Steam under pressure (Autocalving) (iv) Gas Sterilization
(v) Filtration (vi) Surface sterilization

3. Isolation

a). Isolation of fungi

(i) Soil plate dilution method (ii) Soil plate method
(iii) Soil desiccation method (iv) Direct inoculation method
(v) Isolation by baiting

4. Inoculation technique

(i) Soil infestation (ii) Inoculation with stem or leaves with fungi or bacteria

5. Plant disease assessment method

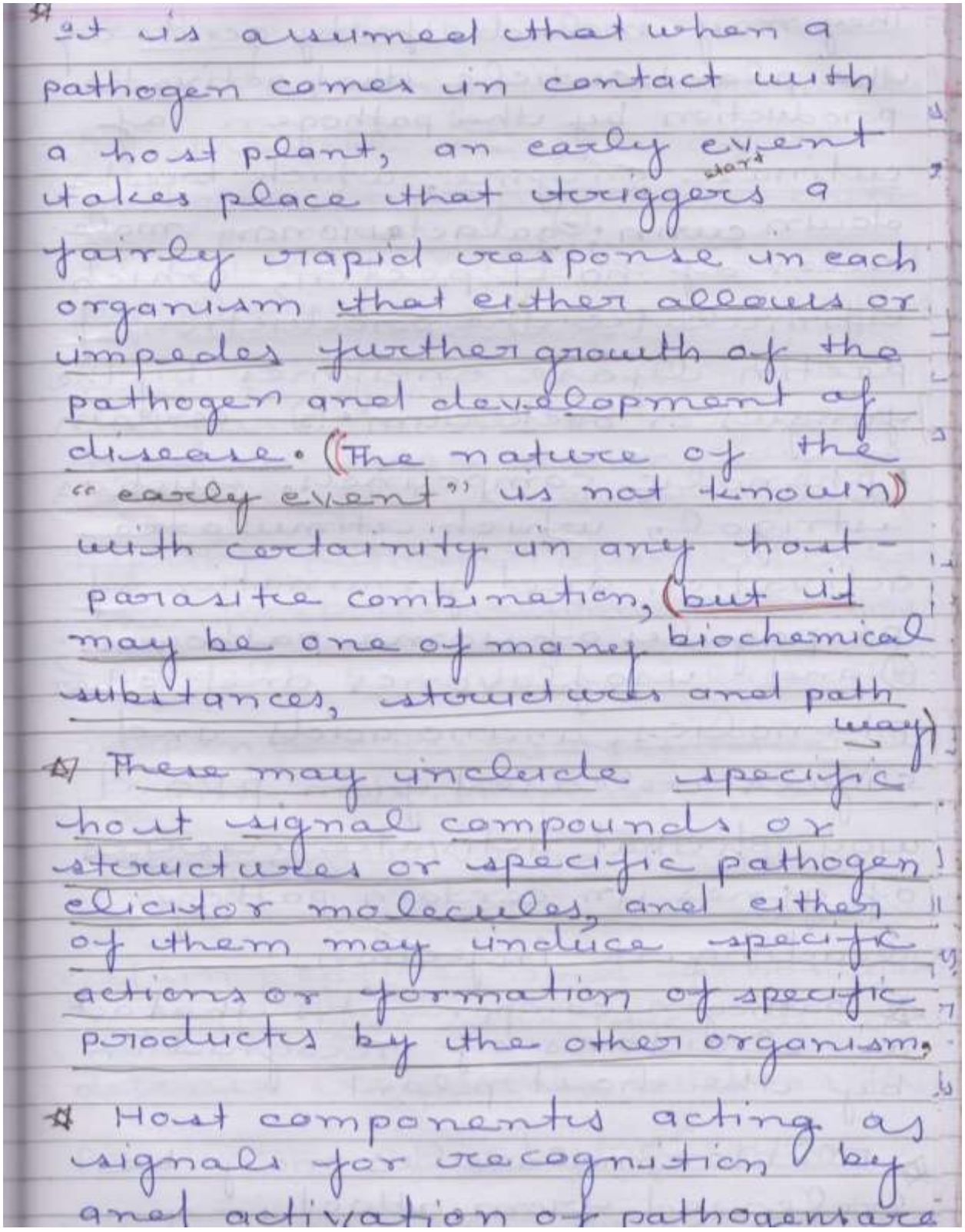
(i) Percentage of diseased plant or organs (ii) Visual method in the field
(iii) Disease incidence (iv) Disease severity

6. Model for estimating yield losses

(i) Critical point model (ii) Multiple point model

4. What is the latest concept of recognition between host and pathogen with suitable examples.

Recognition between host and pathogen



They may include fatty acids of the plant cuticle that activate production by the pathogen of cutinase enzyme, which breaks down cutin; (2) galacturonan molecules of host pectin, which stimulate the production of pectin lyase enzymes by the fungus or bacterium; (3) certain phenolic compounds, such as strigol, which stimulate activation and germination of propagules of some pathogens; (4) and isoflavones and other phenolics, amino acids and sugars released from plant wounds that activate a series of genes in certain pathogens leading to infection.

* pathogen components that act as elicitors of recognition by the host plant

* Elicitor molecules may be released from attacking

entry unto the host. Some elicitors may be components of the cell surface of the pathogen (e.g. - β glucans, chitin or chitosan) that are released by the action of host enzymes (e.g. β-glucanase and/or chitinase) and have broad host ranges; some may be synthesized and released by the pathogen after it enters the host as response to host signals. Elicitors include the harpin proteins of bacteria that induce development of hypersensitive response, certain hydroxylipids, and certain peptides and carbohydrates that induce specific host defense responses such as the production of phytoalexins.

Elicitors are considered as

avirulence
determinants of pathogen avirulence, as by their presence they elicit the hypersensitive (resistance) response for host and initiation of transcription of the plant genes that encode the various components of the defense response. These defense measures by the host plant; in turn result in the pathogen appearing as avirulent.

When the initial recognition (in case of host develop no defense, disruption of attacking signal transduction pathway) received by the pathogen favors growth and development, disease may be induced; if the signal suppresses pathogen growth and activity, disease may be aborted. However, if the initial recognition elicitor received by the host triggers a defense reaction, pathogen growth and activity may be slowed or stopped and disease may not develop.

5. What is penetration and describe different methods of penetration by fungi with suitable diagrams.

Penetration

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.

A. Indirect Penetration

Wounds: Wounds caused by farm operations, hail storms, or insect punctures, etc., will help in the entry of different plant pathogens into the host cells. Organisms which cause storage diseases and ripe rots will enter through the wounds caused by farm operations. Ex. ***Rhizopus***, ***Gloeosporium***, ***Aspergillus***, ***Penicilium***, ***Colletotrichum***, ***Diplodia***, etc. Weak parasites enter through the wounds caused by hail storms and freezing. Ex. ***Macrophomina phaseolina***. Pathogen causing brown rot of fruits (*Sclerotinia fructicola*) enters through the wounds caused by insect punctures. Similarly, causal organism of **Dutch elm disease (*Ceratostomella ulmi*)** enters through the wounds caused by elm bark beetle.

Natural openings

Stomata: There is variation in the behaviour of germ tube at the time of penetration through the stomata. In ***Puccinia graminis tritici***, the uredospore germinates and forms a germ tube which on approaching stoma swells at the tip to form an appressorium in the stomatal aperture. From the appressorium a blade like wedge grows through the stomatal slits and swells inside to form a sub-stomatal vesicle from which the haustoria penetrating the cells are produced. In ***Peronospora destructor*** infecting onion leaves, the germ tube continues to grow after the formation of first appressorium. In ***Pseudoperonospora cubensis***, the hyphae penetrate the stomatal aperture and swell to form a sub-stomatal vesicle from which in turn other hyphae grow to form haustoria in the adjacent cells of the leaves.

Mycosphaerella musicola forms a small structure called **stomatopodium** over the pore of the stoma after growing for few days on the surface of the leaf. A hypha then arises from it which grows into the sub-stomatal chamber and swells to form a vesicle, which in turn gives rise to hyphae which invade palaside tissues.Other examples: *Xanthomonas* ***campestris*** **pv. *malvacearum* (Black arm of cotton)**, ***Xanthomonas phaseoli*** **(Bacterial leaf spot of green gram)**, ***Phytophthora infestans*** **(Late blight of potato)**, ***Albugo candida*** **(White rust of crucifers)** and **uredospores of *Puccinia graminis tritici* (Black stem rust of wheat)**.

Lenticels: *Sclerotinia fructicola* (Brown rot of fruits), ***Streptomyces scabies*** (Scab of potato), *Phytophthora arecae* (Mahali disease of arecanut)

Hydathodes: ***Xanthomonas campestris pv. campestris*** (Black rot of crucifers)

B. 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.

Breakdown of physical barriers: Viruses have no physical force or enzyme system of their own to overcome structural or chemical barriers of the host and therefore come in contact with the host protoplasm only through wounds. Bacteria are mostly weak parasites and cannot employ force to effect penetration. Fungi and nematodes are the only group of plant pathogens that employ force for direct penetration of the host. Fungi penetrate host plants directly through a fine hypha produced directly by the spore or mycelium or through a penetration peg produced by an appressorium. These structures exert pressure on the surface which results in stretching of the epidermis which becomes thin. Then the infection peg punctures it and effects its entry.

Breakdown of chemical barriers: The host is provided with defense mechanisms against invasion which include **i)** presence of cuticular layer on the epidermis, **ii)** lack of suitable nutrients for the pathogen in the host cells, **iii)** presence of inhibitory or toxic substances in the host cells, iv) exudation of substances toxic to pathogen or stimulatory to antagonists of the pathogen. Ex: The glands in leaf hairs of begalgram contain maleic acid which is antifungal and provide resistance to infection by the rust fungus (Uromyces ciceris arietini). Similarly, protocatecheuic acid and catechol in the red scales of onion provide resistance to onion smudge pathogen, *Colletotrichum circinans*. To overcome nthese physical and chemical barriers, the fungi produce various enzymes, toxins organic acids and growth regulators.

Through non-cutinized surfaces:

a) Seedlings: Grain smut of jowar (*Sphacelotheca sorghi*), Loose smut of jowar (*Sphacelotheca cruenta*), Downy mildew of jowar and bajra (*Sclerospora graminicola*), Wheat bunt disease (*Tilletia caries, Tilletia foetida*)

b) Root hairs: Wilt causing fungi (*Fusarium* sp.), Club root of cabbage (*Plasmodiophora brassicae*), Root rot of cotton (*Phymatotrichum omnivorum*)

c) *Buds*: Pea rust fungi (*Uromyces pisi*), Witches broom of cherries (*Taphrina cerasi*)

d) Flowers: Loose smut of wheat (*Ustilago nuda tritici*), Long smut of jowar (*Tolyposporium ehrenbergi*), Bunt of rice (*Neovossia horrida*), **Ergot of rye** (*Claviceps purpurea*)

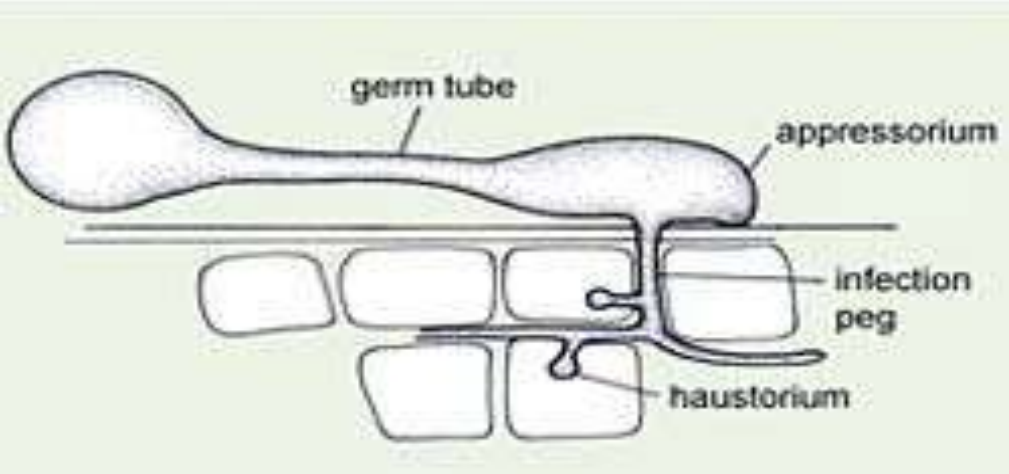
e) Leaves: Basidiospores of white pine blister rust fungus (*Cronartium ribicola*) germinate and grow down into branches and leaves, where aecia are produced.

d) Nectaries: **Fire blight of apple** (*Erwinia amylovora*)

e) Stalk ends: *Penicillium italicum, Theilaviopsis paradoxa* (Post harvest disease fungi)

Through cutinized surfaces:

a) Cuticle: Leaf spot of spinach (***Cercospora beticola***), early blight of solanaceous plants (*Alternaria solani*), Tikka disease of groundnut (***Cercospora personata***)



6. How will you classify cell wall degrading enzymes on the basis of their substrate, utilization and mode of action?

Role of enzymes in pathogenesis

Enzymes are large protein molecules which catalyze all inter-related reactions in the living cell. Most pathogens derive energy principally from enzymatic break down of food materials from host tissue.

Composition of the cell wall: Functionally cell wall is divided into 3 regions, middle lamella (made of pectins), primary wall (cellulose, pectic substances) and secondary cell wall (entirely cellulose). Middle lamella acts as intercellular cement which binds the cells together as intercellular cement which binds the cells together in tissue system.

Pectin or pectic substances are major chemical constituents of wall layers and entire middle lamella, where as in other layers, cellulose is found in good amounts. Besides these two major components, other components such as hemicelluloses, lignin and some amount of protein is also

present. Main components of cell wall are pectic substances, cellulose, hemicelluloses, lignin and small quantity of protein. The epidermis of plants is covered by cuticle, whose major chemical substance is cutin in addition to cuticular wax.

Cuticular wax: Plant waxes are found as granular or rod like projections or as a continuous layer outside / within the cuticle. Wax formation is a continuous process and it is not a terminal phase in the development of leaf. Cuticular waxes are made up of long chain molecules of paraffin, hydrocarbons, alcohols, ketones and acids. Most of the fungi and parasitic higher plants penetrate wax layers by means of mechanical force alone.

Cutin: It is an insoluble polyester of unbranched derivatives of **C_{16} and C_{18} hydroxy fatty acids**. Cutin is admixed with waxes on upper side and with pectin and cellulose on the lower side. **Cutinases** break cutin molecules and release monomers as well as oligomers from insoluble cutin polymer. Cutinases reaches its highest concentration at penetrating point of the germ tube and at infection peg of appressorium forming fungi **.**

Ex: *Colletotrichum gloeosporioides, Sphaerotheca pannosa, Venturia inaequalis, Helminthosporium victoriae*.

Pectic substances: These are major components of middle lamella (intercellular cement **that** holds in place the cells of plant tissues). They also make up a large portion of primary cell wall in which they form an amorphous gel filling the spaces between cellulose microfibrils. Pectic substances are polysaccharides consisting mostly of **dgalactouronic acid** units with **α-1,4-glycosidic bonds**. These chains are esterified with **methyl** groups or linked with other carboxyl groups in calcium and magnesium salt bridges. Pectic substances are of three types, namely, **pectic acid** (non methylated units), **pectinic acid** (<75% methylated galacturonan units) and **pectin**. Term **protopectin** is used to denote substances which are soluble in water and upon restricted hydrolysis yields pectinic acid. The enzymes that degrade pectic substances are known as **pectinases** or **pectolytic** enzymes. Pectinases and pectolytic enzymes are pectin methyl esterases (PME's), polygalactouronases (PG's) and pectin lyases (PL's).

1. **Pectin methyl esterases**: Breaks ester bonds and removes methyl groups from pectin leading to the formation of **pectic acid** and **methanol** (CH_3OH).

2. Polygalacturonases: Split pectin chain by adding a molecule of water and breaks the linkage between two galacturonan units. These enzymes catalyze reactions that break α- 1,4-glycosidic bonds.

3. Pectin lyases: Split pectin chain by removing a molecule of water from the linkage, there by breaking it and releasing products with unsaturated double bonds. These pectin enzymes can be **exopectinases** (break only terminal linkage) or **endopectinases** (break pectin chain to random sites). Pectin degradation results in liquefaction of the pectic substances and weakening of cell walls, leading to tissue maceration. Ex: Soft rot bacterium, *Erwinia caratovora* subsp. *caratovora* and other fungi like *Botrytis cinerea, Sclerotium rolfsii*, etc.

Cellulose: Cellulose is a polysaccharide, made of chains of **β-D-glucopyranose** units (where C_1 is linked to C_4). Glucose chains are held by **hydrogen** bonds. Cellulose occurs in all higher plants as the skeletal substance of cell walls in the form of microfibrils. Primary and secondary wall consists of a matrix in which a large number of microfibrils are embedded. These microfibrils are like bundles of iron bars in a reinforced concrete building. In some parts of microfibrils the chains are arranged in an orderly fashion attaining crystalline form, when arranged in less orderly fashion, it attains amorphous form. If the proportion of crystalline portion is more, the resistance of the host to pathogen is more. The space between microfibrils and between micelles or cellulose chains is filled with pectins, hemicelluloses and also lignin at maturity. Cellulose is insoluble in **crystalline** form (native form), and soluble in **amorphous** form (modified cellulose). The enzymatic breakdown of cellulose results in final production of glucose molecules. Cellulose is degraded by **cellulases**. Cellulase one (**C_1**) attacks native cellulose by cleaving cross-linkages between chains. A second cellulase (**C_2**) also attacks native cellulose and breaks into shorter chains. These shorter chains are then attacked by **C_x** enzyme, which degrade them into disaccharide, **cellobiose**. Finally cellobiose is degraded by the enzyme, **β-glucosidase** into glucose. **Cellulase** degrading enzymes play a role in softening and degradation of cell wall material and facilitate easy penetration and spread of pathogen in the host. Ex: Basidiomycetes fungi

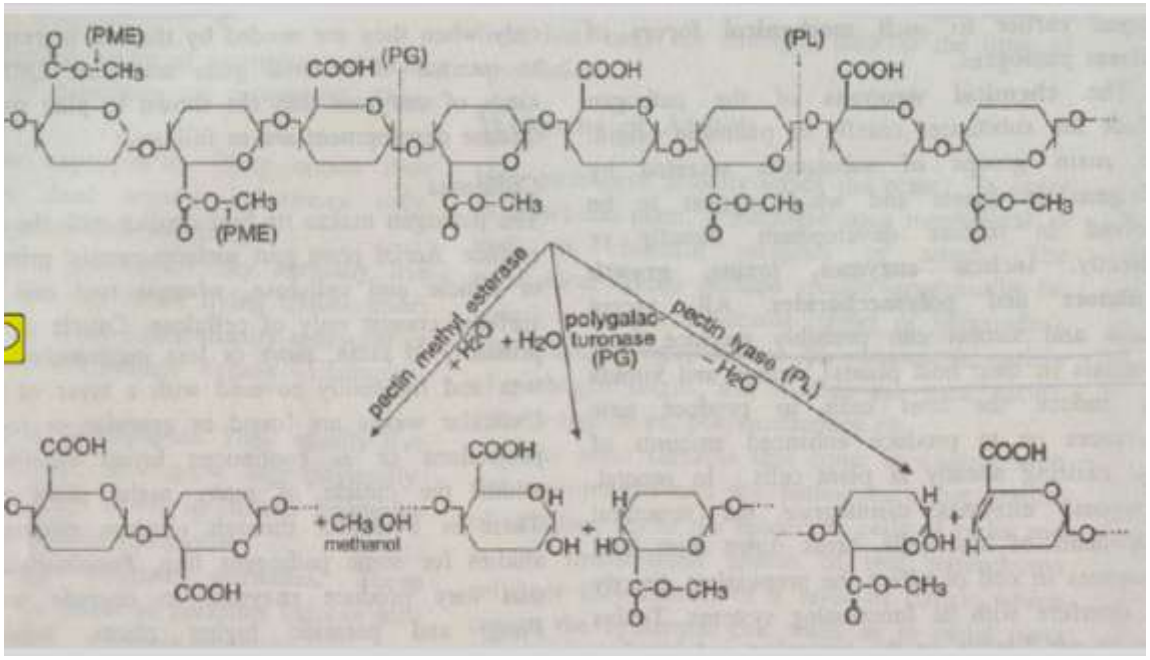
Hemicellulose: These are the major constituents of **primary cell wall** and also seen in middle lamella and secondary cell wall. The hemicellulose polymers include primarily **xyloglucan** but also glucomannans, galactomannans, arabinogalactans, etc. Hemicelluloses link the ends of pectic polysaccharides and various points of the cellulose microfibrils. Hemicellulases degrade hemicelluloses and depending on the monomer released from polymer on which they act, they are termed as xylanase, galactanase, glucanase, arabinase, mannose, and so on. Ex: *Sclerotinia sclerotiorum, Sclerotinia fructigena.*

Lignin: Lignin is found in the **middle lamella**, as well as in the secondary cell wall of xylem vessels and the fibres that strengthen plants. It is an amorphous, three-dimensional polymer made up of basic structural unit, **phenylpropanoid**. Lignin forms by oxidative condensation (C-C and C-O bond formation) between phenylpropanoid units or substituted **cinnamyl alcohols** (p-coumaryl alcohol, coniferyl alcohol and sinapyl alcohol). **White rot fungi** (Basidiomecetes) secrete one or more ligninases which enable them to utilize lignin. Ex: *Xylaria, Chaetomium, Alternaria, Cephalosporium*, etc.

Cell wall proteins: Cell wall proteins are similar to other proteins, except that they are rich in aminoacid, **hydroxy proline**. Five classes of structural proteins are found in cell walls: extensins, proline-rich proteins (PRP's), glycine-rich proteins (GRP's), Solanaceous lectins and arabinogalactan proteins (AGP's). Proteins are degraded by means of enzymes, **proteases** or **proteinases** or **peptidases**.

Lipids: Various types of lipids occur in all plant cells. The most important ones are **phospholipids** and **glycolipids**. These lipids contain fatty acids, which may be saturated or unsaturated. Lipolytic enzymes, called **lipases** (phospholipases, glycolipases) hydrolyze lipids and release fatty acids.

Starch: Starch is the main reserve polysaccharide found in plant cells. It is a glucose polymer and exists in two forms: **amylose**, a linear molecule, and **amylopectin**, a highly branched molecule. Starch is degraded by enzyme, amylases.



7. Write short notes on the following:

(i) Wilt pathogens and their symptoms

- Vascular wilt diseases are caused by pathogenic fungi or bacteria that enter the water-conducting xylem vessels of a plant, then proliferate within the vessels, causing water blockage.
- The typical symptoms include wilting and death of the leaves, followed often by death or serious impairment of the whole plant. As a group, therefore, the vascular wilts are among the most devastating plant diseases.
- There are several interesting features of these diseases.
- For example, the blockage of the xylem vessels is not necessarily caused by the pathogens themselves but often by the host reactions to invasion - the production of gel-like materials which serve as potential barriers to spread of the pathogens in the vessels.

FUNGAL WILT DISEASES

Wilt diseases caused by fungi are in either of the genera *Fusarium* or *Verticillium*.

Fusarium wilts can affect many plants (hosts) but primarily occur in annual vegetables and flowers. These include:

- Solanaceous crops (tomato, potato, pepper, eggplant)
- Crucifers (radish, cauliflower, cabbage)
- Cucurbits (cucumber, squash, melon)
- Beans and peas
- Strawberries
- Herbaceous ornamentals (carnation, chrysanthemum, gladiolus, crocus, lily, daffodil, tulip)

- *Verticillium* wilts can affect over 200 species of plants; in the home garden, solanaceous plants are particularly susceptible, but strawberries and raspberries may also become infected.
- All vascular wilts, regardless of the pathogen, have some symptoms in common. The leaves of infected plants or of parts of infected plants lose turgidity, turn lighter green to greenish-yellow, droop and wilt, turn yellow then brown, and die. In cross sections of infected stems, discoloured brown areas appear as a complete or interrupted ring of discoloured vascular tissues.

BACTERIAL WILT DISEASES

- Wilt diseases in the home garden may also be caused by bacterial pathogens. These belong primarily to three genera: *Clavibacter*, *Erwinia*, and *Xanthomonas*.
- **Bacterial Wilt and Canker of Tomato :** Bacterial wilt and canker of tomato, incited by *Clavibacter michiganense* has symptoms that include single leaflets that curl upward and inward followed by whole leaves shrinking, drying and turning brown. Symptoms may be restricted to one side of the plant. Also, spotting may occur during wet periods on leaves, stem and fruit.
- **Bacterial Wilt of Cucurbits:** Bacterial wilt of cucurbits, caused by *Erwinia tracheiphila* is most severe on cucumbers and muskmelon, and is less severe on squash and pumpkin.
- **Bacterial Wilt of Corn:** Bacterial wilt of corn caused by *Erwinia stewartii* rarely occurs. Where it does, it is more of a problem on sweet corn than field corn. The symptoms appear as rapid wilting of the plant, which is either killed or remains stunted and produces no ears.
- **Black Rot of Crucifers:** Black rot of crucifers caused by *Xanthomonas campestris* first appears as V-shaped yellowed spots at the margins of the leaves. These progress toward the midrib of the leaf while some of the veins on the affected area turn black.
- **Other causes of wilt:** Fungi, such as *Sclerotinia* and *Botrytis*, which attack and girdle the stem, also can cause wilting. Nematodes (microscopic worms which feed on or in plant roots) or root rot fungi, both of which reduce the ability of roots to absorb water, can cause wilt symptoms. Boring insects may break the continuity of the vascular system resulting in wilting. Finally, toxins produced by walnut trees may cause wilting of many garden plants, especially tomatoes.

(ii) Mosaic of sugarcane

- Mosaic disease of sugarcane has been known for long in many countries. It is continuously observed on the widely grown variety Co740 in Maharashtra although it is not known to have caused any serious damage to yields, due probably to the absence of virulent strains of viruses and tolerant nature of the varieties.

Causal Organism: Sugarcane mosaic virus

Symptoms

- The characteristic symptom of the disease appears more prominently on the basal portion of younger foliage than the older ones. Generally, chlorotic or yellowish strips alternate with the normal green portions of the leaf giving the mosaic pattern.
- When young affected leaves held against bright light we observe yellowish spots of uneven stripes.
- In severe infections, the chlorotic area considerably increases over the normal green and symptoms also appear on the leaf sheath.
- Sometimes necrotic lesions are regularly produced in the parenchymatous tissues of the internodes and entire plant becomes stunted and chlorotic control.

Control measures

- Roguing of the affected stools.
- Control of insect pests.
- Control of weeds.
- Avoid multiple ratooning of the affected crop

8. Comment on the followings:

(i) Sandle spike

- Sandal (*Santalum album.* L), a semi-root parasitic tree is the source of the East Indian sandalwood and oil.
- Spike disease caused by *Mycoplasma* (phytoplasma) is the major disease of sandalwood.
- The disease is noticed in all major sandal-growing states of India.
- Spike disease is characterized by extreme reduction in leaf size accompanied by stiffening and reduction of internode length.
- In advanced stage, the entire shoot gives the appearance of a spike inflorescence. Spiked trees die within 1–2 years after the appearance of visible symptoms.

Control measures

- Roguing of the affected stools.
- Control of insect pests.
- Control of weeds.
- Avoid multiple ratooning of the affected crop

(ii) Bacterial blight of paddy

- Bacterial blight of rice has high epidemic potential and is destructive to high-yielding cultivars in both temperate and tropical regions especially in Asia. Its occurrence in the 70s in Africa and the Americas has led to concerns about its transmission and dissemination.

Symptoms

- Symptoms appear on the leaves of young plants as pale-green to grey-green, water-soaked streaks near the leaf tip and margins. These lesions coalesce and become yellowish-white with wavy edges.
- The whole leaf may eventually be affected, becoming whitish or greyish and then dying. Leaf sheaths and culms of more susceptible cultivars may be attacked.
- Systemic infection results in wilting, desiccation of leaves and death, particularly of young transplanted plants.
- In older plants, the leaves become yellow and then die. In its advanced stages, the disease is difficult to distinguish from leaf blight caused by *Xanthomonas oryzae* pv. *oryzicola*, but lesion margins are wavy rather than linear as for the former.
- Damage is often associated with lepidopteran leaf rollers, leaf-folders and hispa beetles, since bacteria readily enter the damaged tissue caused by insect infestation.

Management

- Management of bacterial leaf blight is most commonly done by planting disease resistant rice plants.
- The applications of copper compounds or antibiotics are largely ineffective in the control of bacterial leaf blight.
- Biological control methods are relatively recent developments which are not currently in common use.
- Use of resistance varieties.