SEMESTER I (HONOURS)

Q) What is Plant pathology?

A: Plant pathology (Gr. '*pathos'* means suffering; *logos* means knowledge) is a branch of botany which deals with the study of the nature, development and control of plant disease or the study of the suffering plants.

Q) What is disease?

A: Disease is an interaction among the host, parasite, and environment.

Q) What is non-parasitic and parasitic disease?

A: The disease of a plant may be caused by environmental factor or factors, called non-parasitic disease and the disease may be caused by microorganisms such as fungi, bacteria, nematodes etc., called parasitic disease.

Q) What is host and pathogen?

A: A host is an organism that is harbouring a parasite or pathogen from which it obtains its nutrients. A pathogen is an organism which causes a disease.

Q) What is symptom?

A: The manifestation of the disease is called symptoms.

Q) What is syndrome?

A: When a number of symptoms are available for a particular disease, they are collectively known as syndrome.

Q) What is sign?

A: When the disese is identified by seeing the pathogen externally on the host surface, it is known as sign, e.g., powdery mildew of cucurbits.

Q) What is parasite? Mention its two types.

A: The organism which grow on living tissue are called parasites. It has mainly two types-

(a) Obligate parasites : The organisms which grow only on living tissue and not in axenic cultures are called obligate parasite, e.g., *Puccinia graminis tritici* (Black stem rust of Wheat).

(b) Facultative parasites : The organisms which can live as saprophytes for long period in pure culture or otherwise, but under certain conditions they becaome pathogenic are called facultative parasites, e.g., Fusarium udum (wilt of pigeon pea)

Q) What is disease cycle? What is monocyclic, polycyclic, polyetic plant disease?

A: The disease cycle is the life cycle of a pathogen including the series of events starting from the time of infection to disease expression on a host.

When pathogens are able to complete only one or part of disease cycle in one year, they are called monocyclic pathogens and the disease as **monocyclic disease**, e.g., wilt of pigeon pea.

When pathogens are able to complete more than one generation in a growing season, they are called polycyclic pathogens and disease as **polycyclic disease**, e.g., late blight of potato.

When pathogens are not able to complete a disease cycle within one year and take several years before the inoculum is developed on plant and can be disseminated and may initiate new infection, they are called polyetic pathogen and the disease as **polyetic disease**, e.g., dutch elm disease.

Q) What are pathogenicity and pathogenesis?

A: The efficiency of a pathogen to cause disease is the pathogenicity and pathogenesis is the process or chain of events of disease development.

Q) Define casual complex.

A: In addition to pathogen, other environmental factors such as atmospheric humidity, temperature, wind speed, light etc. are essential for the development of disease on a host. Pathogen along with other environmental factors are collectively called casual complex. Casual complex = casual organism + other environmental factors.

Q) What is penetration?

A: The initial invasion of the pathogen inside the host tissue is called penetration.

Q) Define infection?

A: The establishment of the pathogen of the pathogen inside the host tissue is called infection.

Q) What is inoculum?

A: The portion of the pathogen responsible for infection is called inoculum.

Q) What is primary and secondary inoculum?

A: The inoculum which comes from the neighbouring field is the primary inoculum and the inoculum which develops from the primary symptom is called secondary inoculum.

Q) What is etiology?

A: The determination vis-a-vis study of the cause of a disease is called etiology.

Q) What is necrotrophy and biotroph?

A: The microorganisms which feed only on dead organic tissues, e.g., *Rhizopus* etc. known as necrotrophy and the organism that can live and multiply only on another living organism, e.g., Vasicular-arbascular mycorrhizae (VAM) known as biotroph.

Q) Define disease triangle?

A: The interactions of the three components of a disease (host, pathogen and environment) have often been visualised as a triangle, commonly referred to disease triangle.

Q) What is spot?

A: When necrotic region develops on the host surface, formed by killing of tissue in a limited area, it is called spot, e.g., Brown spot of rice.

Q) What is blight?

A: Blight can be defined as the rapid killing of different plant parts, such as leaves, blossoms etc., and the killed tissues may become slimy and often emit pungent odour, e.g., Late blight of potato.

Q) What is rot?

A: Rot is a type of necrosis, where the dead tissues are in a more or less advanced stage of disintegration in the infected region, e.g., Stem rot of jute.

Q) What is rust?

A: The symptoms of rust appear as small pustules on the epidermis of leaf and stem of the host, e.g., Black stem rust of wheat.

Q) What is IDM?

A: The management programme should cover the integration of all methods; those favour the host, discourage the pathogen and modify the environment. This type of management programme to control plant diseases is called Integrated Disease Management (IDM) or Integrated Pest Management (IPM).

Q) What are endemic and epidemic diseases?

A: When diseases are constantly present year after year in moderate to serve form in a particular area, they are called as endemic species and when a disease occur widely, but periodically in severe form, they are called epiphytotic or epidemic species.

Q) What are sporadic and pandemic diseases?

A: When diseases occur in small areas and at very irregular intervals, they are known as sporadic diseases, e.g., angular leaf spot of cucumber and when diseases occur in vast and much extensive areas, they are known as pandemic disease, e.g., late blight of potato.

Q) Pathogen of Brown spot of rice.

A: *Helminthosporium oryzae*.

Q) Pathogen of Late blight of potato.

A: *Phytopthora infestans*.

Q) Pathogen of Black stem rust of wheat.

A: *Puccinia graminis tritici*.

Q) Pathogen of Stem rot of jute.

A: *Macrophomina phaseolina*.

Q) Scientific name of rice.

A: *Oryza sativa.*

Q) Scientific name of wheat.

A: *Triticum aestivum.*

Q) Scientific name of potato.

A: *Solanum tuberosum*

Q) Scientific name of jute.

A: *Corchorus capsularis* (Tita pat)

Corchorus olitorius (Mitha pat)

Q. Brown Spot of Rice

Answer:

Host : *Oryza sativa* L. (Family-Gramineae i.e., Poaceae).

Pathogen : *Helminthosporium oryzae*

Symptoms : The pathogens usually attack all parts of the rice plant at all stages of development, excepting roots. The symptoms occur on the coleoptile, leaf blade, leaf sheath, and also on the inilorescence.

1. **On coleoptile**: Symptoms on the coleoptile appear as spots. The spots are brown, small, pinhead to oval in shape. Rarely the spot takes the form of long streaks. In severe cases, the coleoptile becomes blighted and causes death of the seedling.

2. **On leaf blade**: Symptoms on leaf blade appear as discrete, dark brown, elliptical to eye-spots.

3. **On leaf sheath**: Symptom on leaf sheath is very to the symptoms present on leaf blade.

4. **On inflorescence**: If the inflorescence becomes infected at an early stage it doesn’t develop any grain.

Disease cycle : Brown spot of rice is a localised disease i.e., the pathogen is restricted to the infected zone and cannot pass long distance through the inner tissue. The built up inoculum from the infected region disseminates further and causes infection. During dormant phase, the pathogen is prevalent on seed (both externally and internally) and also on infected plant debris.

1. From seed: During germination of infected seeds, the coleoptile gets infected and then gradually it affects the seedling, showing blight symptom.

The infected regions of the seedling i.e., coleoptile, leaves, etc., then produce a large number of conidia. These conidia, after dissemination by wind, fall on the different regions of the same seedling or on different seedlings and cause infection. This is the secondary cycle, which repeats several times in the growing season and enhances progress of the disease.

Towards the end of the season, the inoculum from the infected leat may go to the inflorescence and develops infected seeds; where the pathogen may present as mycelium (inside the seed) and/or conidia (on seed).

2. From plant debris. Previous year's infected plant debris also act as source of primary inoculum. Thus, through infected seeds and infected plant debris, disease may appear in the next season and repeat the cycle. The disease may also progress from year to year through collateral hosts, like *Echinochloa colonum and Setaria* sp.

Disease management:

The disease can be controlled or reduced by the following procedures:

1. **Eradication of collateral hosts**: Collateral hosts like *Leersia hexandra, Echinochloa colonum and Setaria sp*. should be eradicated, where the pathogen can survive out of season.

2. **Sanitation**: Cleaning and burning of the previous year's infected plant debris are effective to reduce the source of

primary inoculum.

3. **Irrigation water**: During irrigation, the water should not pass from infested field to the field without disease.

4. **Leaching of metals**: Leaching down of metallic elements like Fe, Mn and K from soil during rain should be

checked, ptherwise the plants become more susceptible to the disease.

5. **Application of metals:** Addition of metals like K in the field during growth period, reduce the severity of the disease.

6. **Spacing**: Proper spacing during transplanting also reduces the disease incidence.

7. **Nitrogen fertiliser**: Optimum nitrogen fertiliser (Urea) application, transplanting at optimum dates proved to be

helpful lor the reduction of disease.

8. **Sunlight**: Soaking the seeds for 24 hrs in water and then drying (52-54 C) in bright sunlight reduces the source of primary inoculum by killing the activated mycelium.

9. **Foliage treatment**: Spraying or dusting of fungicide, two to three times at regular intervals, gives effective control of the disease. Dithane Z-78 (0.2%), Dithane M45 (0.3%), Hinosan (0.1%), Blitox (0.3%), Benlate (0.2%) and Bordeaux mixtute (5:5: 50) are used to control the disease.

10. **Disease tolerant varieties**: A number of disease tolerant varieties have been developed in India, particularly in C.R.R.l. (Central Rice Research Institute), Cuttack, Orissa. These are CH 13, CH 20,CH 45, T 141, T 2114, IET 13238, CR 84-30, JB 83 etc.

Q. Pre-Penetration:

Answer: During pre-penetration stage the pathogen (inoculum) on arrival on the host surface interacts sharply with the surrounding environment and host itself. The environment which is an aggregate of all external conditions including temperature, moisture (relative humidity), light and the competing microorganisms; affects the life and form of the pathogen of the inoculum.

The optimum environmental condition for ideal growth of a pathogen again varies on the nature of the pathogen and the host surface. For example, the development and abstraction of conidia are favoured by high air temperature and humidity in downy mildew.

Whereas in powdery mildew both the number of spores and their germination are greater in bright sunlight. In cereal rusts uredospore’s germinate at low temperature, but the infection

process is delayed at this temperature. Again, soil pH plays a very vital role in the growth of bacterial plant pathogen in the rhizosphere (area of soil immediately surrounding the roots).

Whether a pathogen will survive and grow on the host surface also depends on its behaviour with the exudates of, the host surface and the microbial population present on it. The exudates of the host surface may encourage or inhibit the growth of the pathogen. The root exudates mainly sugars and amino acids are nutrients for the growth of fungi and bacteria.

But root exudates like hydrocyanic acid, various organic acids and antibiotics are antifungal and antibacterial. For example, spores of *Rhizopus* germinate only in presence of proline (amino acid) present in the rhizosphere region; whereas exudates of root of onion varieties inhibit spore germination of *Colletotrichum*.

Leaves also exude substances which may go in favour or against the growth of the pathogen.

Protocatechuic acid, an exudate of onion skin is also antifungal. The pathogen has to neutralize these exudates or has to be resistant to them for survival. Besides these, the rhizosphere region contains microbial population which is antagonistic to the growth of the pathogen. As such, the pathogen has to overcome the above barriers during pre-penetration stage before it can survive for host penetration.

Once a favourable relationship is established with the host surface, multiplication or growth of the pathogen begins. Rapid proliferation of cells of bacterial pathogen results in a relatively short time. The bacterial cells so formed being delicate structures may be easily killed by unfavourable conditions.

Hence, they survive under layers of slime. Again, development of fungal phytopathogen usually includes spore germination by germ tube which grows producing infection hypha as a result of hyphal tip growth or may give rise to an aspersorium which anchors the fungus to the host surface.

Penetration peg is produced from the aspersorium with which the pathogen causes host penetration. But multiplication of viruses takes place only in the living host cells.

Q. Phytoalexins:

Answer: Phytoalexins are toxic antimicrobial substances synthesized ‘de novo’ in the plants in response to injury, infectious agents or their products and physiological stimuli. The term phytoalexin was first used by the two phytopathologists Muller and Borger (1940) for fungi static compounds produced by plants in response to mechanical or chemical injury or infection.

All phytoalexins are lipophilic compounds and were first detected after a study of late blight of potato caused by *Phytophthora infestans*. Phytoalexins are believed to be synthesized in living cells but surprisingly necrosis follows very quickly.

According to Bill (1981), peak concentration of phytoalexins almost always coincides with necrosis. Although the exact mechanism of production of phytoalexin has not been properly understood, it is considered that a metabolite of the host plant interacts with specific receptor on the pathogen’s membrane resulting in the secretion of “phytoalexin elicitor” which enters the host plant cells and stimulates the phytoalexin synthesis.

Phytoalexins are considered to stop the growth of pathogens by altering the plasma membrane and inhibiting the oxidative phosphorylation.

Phytoalexins have been identified in a wide variety of species of plants such as Soyabean, Potato, sweet potato, barley, carrot, cotton etc. are being investigated. Some common phytoalexins are Ipomeamarone, Orchinol, Pistatin, Phaseolin, Medicarpin, Rishitin, Isocoumarin, ‘Gossypol’ Cicerin, Glyceolin, Capisidiol etc.