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B. Sc. II Semester -IV

A TEXTBOOK OF PLANT PROTECTION

Paper IV (DSC ID 46)

CROP DISEASES, THEIR MANAGEMENT AND PATHOPHYSIOLOGICAL SKILLS




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PREFACE

Forthcoming challenges to the foreign Universities, University Grant Commission, New Delhi has adopted new education policies to maintain the standard of higher education. UGC recommended the advanced curricula in Indian Universities.

Shivaji University, Kolhapur has implemented UGC, CBCS syllabus for all courses in science and technology. We are most gratified to place the B.Sc. II plant protection textbook in the precious hands of all teachers, students and farmers because this is the first text of the plant protection of B. Sc. II for the undergraduate students.

According to the semester pattern, A text book of plant protection paper Number IV (DSC ID-46) for Sem IV entitled: ***Crop Diseases, Their Management and Pathophysiological Skills*** *has been written strictly in accordance with Shivaji University, Kolhapur CBCS syllabus. For knowing the concepts of weed management clearly this book is written in very simple language. At the end of each unit descriptive, short and objective questions have been given to the students to upgrade their knowledge with practice.*

We hope that this book will provide fundamental information regarding the weeds and their management.

Authors are extremely thankful to Dr. V. M. Patil, Principal, The New College, Kolhapur; Dr. Sarjerao Gholap, Principal, S. B. R. College, Mhaswad, Dr. Balwant, Principal, Dahiwadi College, Dahiwadi and Dr. B. S. Padwal I/C Principal , M.H. Shinde Mahavidyalaya Tisangi We are also very much thankful to Prof. Dr. V. D. Jadhav, Head Department of Botany, Shivaji University, Kolhapur and all teaching Faculties of the Botany department of Shivaji University and affiliated colleges. We are thankful to BOS Chairman and all the BOS members of the Botany, Shivaji University, Kolhapur for their valuable suggestions and guidance.

We will whole heartily accept the criticism and constructive suggestions for the improvement of future editions.

- Authors

SEMESTER IV PAPER – IV

PAPER III (DSC ID 46)

CROP DISEASES, THEIR MANAGEMENT AND PATHOPHYSIOLOGICAL SKILLS

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LECTURE PERIOD: 3 PER WEEK, LECTURE HOURS: 2.4 PER WEEK

MARKS: 50

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Unit 1 Crop Diseases

1.1 Disease definition and concept:

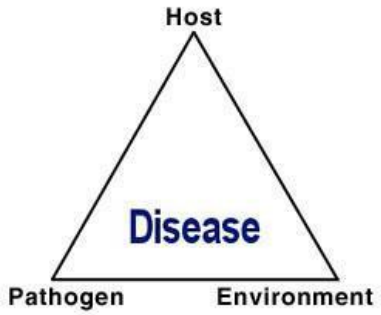
Food is the most basic need of the human being. Plants are the primary producers of food. So crops are sown in the fields for getting this food. When these crops have been sown other bio-organisms are also trying to get their food from these crop plants. The bio-organisms may include fungi, bacteria, viruses, MLO, and insects and animals, birds etc. This leads to the competition between humans and rest of all other bio-organisms. This competition develops the branch Plant pathology which includes study of different pathogens attacking on crop plants.

Plant pathology is a Greek word, Pathos means "to Suffer" and logy means to "Study'. So in Plant Pathology we are studying the plants which are suffered means which are unhealthy or the plants which are affected. It is also known as phytopathology. Plant pathology or phytopathology is the science, which deals with the plant diseases. It is concerned with health and productivity of growing plants. Phytopathology (Greek Phyton = plant + pathos - disease, ailments + logos = discourse, knowledge) is the branch of agricultural, botanical or biological science which deals with the cause, etiology (aetiology), resulting in losses and management methods of plant diseases. Plant pathology can also be defined as the study of the nature, cause and prevention of plant diseases.

When the plant is suffering i. e. not developing and functioning in the expected manner it is said to be diseased. Plant diseases are recognized by the symptoms (external or internal) produced by them or by sick appearance of the plant. The term plant disease signifies the condition of the plant due to disease or cause of the disease. Plant disease is mainly defined in terms of the damage caused to the plant or to its organ. Plant diseases are defined by various workers in different ways. Some of the important definitions are given below:

1. Julius Kuhn in 1858 had defined plant diseases as "abnormal changes in physiological processes which disturb the normal activity of the plant."
2. In 1918, E. J. Butler, who is said to be the Father of Plant Pathology of India, defined the disease as "variation from normal physiological activity which is sufficiently permanent."
3. Disease is a malfunctioning process that is caused by continuous irritation, which results in some suffering producing symptoms. This definition is accepted by both the American Phytopathological Society and the British Mycological Society.

4. Disease is an alteration in one or more of the ordered sequential series of physiological processes culminating in a loss of coordination of energy utilization in a plant as a result of the continuous irritation from the presence or absence of some factor or agent.
5. A plant is said to be "diseased" when there is a harmful deviation from normal functioning of physiological process (Federation of British Plant Pathologists, 1973).
6. The disease can also be defined as 'any disturbance brought about by a living entity or non-living agents or environmental factors which interfere with manufacture, translocation or utilization of food, mineral nutrients and water in such a way that the affected plant changes in appearance with or without much loss in yield than that of a normal healthy plant of the same variety.
7. In general disease is an interaction among the host, parasite and the environment. It is the triangle between Host, Pathogen and Environment. Each of these factors must be present then and then only disease will appear in the plants.



8. Horsfall and Diamond (1959) defined disease as "malfunctioning process in the plant body due to continuous irritation which results in suffering."
9. According to the opinion of Singh *et al*. (1989) disease is " a sum total of the altered and induced biochemical reactions in a system of the plant or plant part brought about by any biotic or abiotic factor or virus leading to malfunctioning of the plant body or its part.
10. From the above opinion, diseases can also be defined as serious deviations of the plant or plant part which leads to decrease the market value of the plant or plant part.
11. Diseases can also be defined as "the abnormal behavior of the plant with or without symptoms." Sometimes there is no visiblesymptom, but then the market value has decreased so this condition is also called as diseased.

Concept of the Disease:

Biotic diseases can be represented by a table having four legs. Each leg of the four-legged table represents one of the essential components for biotic disease development. Where the three components of virulent pathogen, susceptible host and conducive environment coincide for a sufficient period of time, disease will occur. In the simplest interpretation, lacking any

one of the components will preclude disease development. Abiotic disease development (disease caused by a non-living agent) can be similarly represented by a three-legged table. Where the two components of a susceptible plant (no longer a host in the strict sense) and harmful environment coincide for a sufficient period of time, such conditions will support the development of abiotic disease. Inherent plant defects or genetic disorders which result in disease symptoms may be expressed under stable, innocuous environmental conditions, or may require some normally harmless abiotic or biotic stimulus for symptom expression. A few cases of disease induced by exo pathogens (pathogens not in intimate contact with a susceptible plant) are known, and may have been mistakenly attributed to inherent plant defects or abiotic agents in the past. Additionally, there are examples of seemingly symptomless plant diseases that are not uncovered until yield losses are compiled, or the true healthy appearance of the plant is revealed (Fox, 1993).

It is important to remember that even though a disease may appear to be static, as a rule diseases are dynamic, and the disease syndrome will change over time. Symptoms are usually not reversible, though with some abiotic causal agents such as drought stress or nutrient deficiency, symptoms (wilting, chlorosis) can be reversible. Of course, healing of the damage done by biotic and abiotic causal agents, or even by inherent defects in the host plant can occur, though some evidence of the disease usually remains.

Plant is healthy, or normal, when it can carry out its physiological functions to the best of its genetic potential. The kinds of cells and tissues that become affected determine the type of physiological function that will be. For example, infection of roots may cause roots to rot and make them unable to absorb water and nutrients from the soil; infection of xylem vessels, interferes with the translocation of water and minerals to the crown of the plant; infection of the foliage, (leaf spots, blights, rusts, mildews, mosaics etc.), interferes with photosynthesis.

There are many ways in which plant disease pathogens can affect plants

- By utilizing host cell contents
- By killing the host or by interfering with its metabolic processes through their enzymes, toxins etc.
- By weakening the host due to continuous loss of the nutrients.
- By interfering with the translocation of the food, minerals and water.
- They can suppress the chlorophyll content.
- They can reduce the leaf area.
- They can curb the movement of solutes and water through the stems.
- They sometimes reduce the water-absorbing capacity of the roots.
- They suppress the translocation of photosynthates away from the leaves.

- They sometimes promote wasteful use of the products of photosynthesis as in the formation of galls.

Important Terminologies in Plant Pathology:

Host: A host is an organism (e. g. a plant) that is harboring a parasite or pathogen from which it obtains its nutrients. The host range refers to the various kinds of host plants that a given pathogen may parasitize.

Pathogen: Pathogen is any agent which causes damage. Here agent means living organism that is fungi, bacteria, viruses etc.

Pathogenicity: It is the quality or characteristics of pathogen of being very able to cause disease.

Pathogenesis: It is the sequence of progress in disease development from the initial contact between the pathogen and its host to the completion of syndrome.

Symptoms: The internal (physiological) or external (morphological) reactions or changes as a result of disease are referred to as symptoms.

Infection: It implies the establishment of pathogens inside the host following penetration in which a parasitic relationship between the two organisms is established.

Incubation period: The time interval between the penetration of the host by a pathogen and the first appearance of symptoms on the host is known as incubation period.

Disease cycle: A series of events involved in disease development, including the stages of development of the pathogen and the effect of the disease on the host, is called the disease cycle.

Susceptibility: It is the inability of a plant to resist the effect of a pathogen of any other damaging factor. In other words susceptibility is the permission to enter the pathogen in the host body.

Immunity: This implies exemption from infection by pathogens. This is the ultimate power of the host to withdraw the attack of the pathogen.

Resistance: It is the inherent ability of a plant to prevent or restrict establishment and subsequent activities of a potential pathogen.

Inoculum : Inoculum is the infectious material that can cause disease and it is that portion of the individual pathogens that is brought into contact with the host.

Penetration: Penetration is the first step in the contact of the inoculum with the host. It refers to the initial invasion of the host by an organism.

Infection: It implies the establishment of pathogens inside the host following penetration in which a parasitic relationship between the two organisms is established.

Etiology: It is the determination and study of the cause of disease. A pathogen can be living or nonliving, but usually refers to a live agent.

Sub-Unit 1.2

Classification of Plant Diseases Based on

a) Pathogens

b) Symptoms

c) Severity of Disease

d) Transmission of pathogens

The classification of diseases may be grouped based on different criteria depending upon the way pathogen attacks on the host plant. When a pathogen attacks on the host body through the air then the disease is said to be air borne. Similarly when a disease is transmitted through the seed, the disease is said to be seed borne. All these transmission methods of disease have to be classified under the category transmission methods. The disease classification is important to understand the nature of the pathogen which will add to the application of some methods of management. However the disease is not air borne or soil borne but the actual pathogen is transmitted through air or soil. The symptoms or signs which appear on the affected parts or the entire plant also form the basis of grouping the plant diseases. In all these cases the name of the disease is derived from the most conspicuous symptom of the disease appearing on the host body.

There are various methods or criteria of disease classification out of which some have been discussed as below:

a) Classification of Diseases based on Pathogens:

This type of disease classification is based on the type of the pathogen affecting the host plant. They have following two subtypes:

1. Non- infectious disease\non parasitic disease:

- This type of disease is caused by non-living organisms.
- Could not be spread to others.
- They are induced by unfavorable environmental conditions of soil or air such as mineral deficiencies or excesses in the soil, low or high temperatures, improper water, oxygen and light relations.
- May also be caused by air pollution, nutrient deficiency, mineral toxicity, etc.
- These diseases may also be caused by mechanical injuries.

- Example- blossom and rot of potatoes, black heart of potatoes scald of apples.

2. Infectious disease\Parasitic disease:

- These are caused by the attack of some living agents called pathogens. The causal agents may be a plant or an animal or a virus.
- It can spread from diseased to healthy plants.
- In the case of a disease caused by a parasitic organism the diseased plant is called a host.
- The pathogen may subsist in whole or in part upon the living tissue of the host.
- Biotic factors like fungi, bacteria, algae, nematodes, viruses etc. are the causing agents.

b) On the basis of symptoms:

Pathogens when attacked on the plant body, different symptoms will appear on the host plant. These symptoms may be according to the nature of the pathogen and the host plant. Some of the important symptoms given here:

- **Rust-** caused by Basidiomycetes of the order Uredinales. e. g. stem rust of wheat, caused by *Puccinia graminis tritici.*
- **Smuts-** caused by a fungus of the order Ustilaginales e. g. loose smut of wheat, caused by *Ustilago nuda tritici* and Grain smut of Jowar caused by *Sphacelotheca sorghi*
- **Rots-** disease that infect the underground part, caused by bacteria, fungi, or nematodes. e. g. *Rhizoctonia* is root rot.
- **Blight-** It is a rapid and complete chlorosis and death of plant tissue. e. g. late blight disease of potato by *Phytopthora infestans*.
- **Leaf spot-** caused by fungi and bacteria. Early and late blight of groundnut or Tikka disease caused by *Cercospora personata* and *Cercospora arachidicola.*
- **Canker-** It is a dead area, e.g. citrus canker commonly caused by *Xanthomonas campestris*.
- **Wilt-** It affects the vascular system of plants, e. g. Bacterial wilt of cucurbits caused by *Erwinia tracheiphila*.
- **Powdery mildew**- causes foliage of stems, flowers, and fruits, e. g. grapes, cucumber, etc.
- **Downy mildew**- caused by the family Peronosporaceae. E.g. Rose and lettuce.

c) On the basis of Severity of Disease:

Study of plant diseases in relation to their occurrence (interaction of populations of plants, pathogens and environment) is known as epidemiology. There may be infectious disease and contagious disease. A disease which spreads slowly and is incited by a transmissible pathogen is referred to as infectious disease, and that which spreads rapidly is a contagious disease.

According to these criteria diseases may be classified as discussed below:

1) **Endemic disease:** A disease which is regularly present in a certain region or a part of a region (district) in a moderate to severe form is referred to as endemic. These diseases may be found throughout the year in any form irrespective of season or environmental conditions.

e. g. Wart disease of potato caused by *Synchytrium endobioticum* is an endemic disease in India. Greeneria fruit rot of grape (*Vitis vinifera*) is an endemic disease in India caused by *Greeneria uvicola*.

2) **Epidemic [Epiphytotic] disease:** Epiphytotic is term applied to the infectious plant disease which spreads widely but occurs periodically. The causal agent may be regularly present in the locality but the environment favorable for its rapid development occurs only periodically. The epiphytotic diseases are thus very responsive to variation in the environment.

e. g. Rust of wheat caused by *Puccinia graminis tritici* found only in rabbi season, late blight, mildews etc. These diseases occur periodically based on environmental conditions.

3) **Sporadic disease:** These are the plant diseases which occur at irregular intervals and in relatively few instances. A given disease may be endemic in one region and epidemic in another. There is no prediction about these types of diseases.

e. g. Powdery mildew of cucurbits caused by *Erysiphe cichoracearum*, leaf blight, wilt etc.

4) **Pandemic diseases:** A disease may be endemic in one region and epidemic in another. When epiphytotics become prevalent throughout a country, continent or the world, the disease may be termed as pandemic.

d) On the basis of Transmission of pathogens:

These are the diseases which are having their primary inoculum transmitted through different agencies like air, seed and soil. Here one should understand that actually disease is not transmitting but the spores or primary inoculums of the pathogen is transmitted.

1] Soil-borne diseases-Pathogens survive in soil or on infested plant debris present on the soil either as their resting spores or as mycelial strands and rhizomorphs. e. g. Root rot, wilt and seedling blight.

2] **Seed-borne diseases-** The microorganisms are carried along with seeds from one generation to the next. Majority of the seeds are contaminated from outer surfaces of the seeds.

e. g. damping off.

3] Airborne diseases- The microorganisms are spread through air. The spores of different pathogens are very tiny and light in weights which are easily transmitted through air current.

e. g. the spores of blight, rust, powdery mildew etc.

Sub Unit 1.3:

Methods of Studying Plant Pathogens

Plants are fulfilling basic needsof humans. Likewise other microorganisms are also depending on plants to mitigate their food requirement. This leads to competition between humans and other microorganisms. These pathogens create serious problems in agriculture and food stuff. In addition phytopathogens produce mycotoxins, which are harmful to humans and livestock. Phytopathogens cause a number of different diseases such as rusts, smuts, rots, powdery mildews, downy mildews etc which are responsible for reducing agricultural produce and their economic value. Considering these facts, the management of all such phytopathogens needs to be studied, and for that the study of pathogens in relation to their nature, identification, methods of survival, reproduction, hosts range etc has to be done. Thus study of the pathogen is an important task for the implementation of management.

In 1890 the German physician and bacteriologist Robert Koch gave certain criteria for judging whether a pathogen is the cause of a given disease. Koch's criteria brought some much-needed scientific clarity to study a pathogen. These criteria later became popular as Koch's postulates. These are as follows:

1. The microorganism must be found in abundance in all organisms suffering from the disease, but should not be found in healthy organisms.
2. The microorganism must be isolated from a diseased organism and grown in pure culture.
3. The cultured microorganism should cause disease when introduced into a healthy organism.
4. The microorganism must be reisolated from the inoculated, diseased experimental host and identified as being identical to the original specific causative agent.

Koch's postulates were developed in the 19^{th} century as general guidelines to identify pathogens that could be isolated with the techniques of the day. Even in Koch's time, it was recognized that some infectious agents were clearly responsible for disease even though they did not fulfill all of the postulates. Attempts to rigidly apply Koch's postulates to the diagnosis of viral diseases in the late 19^{th} century, at a time when viruses could not be seen or isolated in culture, may have impeded the early development of the field of virology. Currently, a number of infectious agents are accepted as the cause of disease despite their not fulfilling all of Koch's postulates. Therefore, while Koch's postulates retain historical importance and continue to inform the approach to microbiologic diagnosis, fulfillment of all four postulates is not required to demonstrate causality.

The study of plant pathogens include three steps

a) Isolation

b) Inoculation

c) Incubation.

These steps have to be followed while studying the pathogen.

a) Isolation:

Isolation is the term used for separation of pathogens from its host. "*To isolate*" means to separate. It is the process of separating a single species of microorganism from its natural habitat and growing it by itself, without interference from other organisms, on a sterile substratum, i.e. in culture media. The pathogen before studying in the laboratory must be isolated in its pure form for establishing on culture media. This separation of pathogen form its host for developing it on suitable culture media is called isolation. Microorganisms occur in natural environments like soil. They are mixed with several other forms of life. Many microbes are pathogenic. They cause a number of diseases with a variety of symptoms, depending on how they interact with the patient. The isolation and growth of suspected microbes in pure culture is essential for the identification and control of the infectious agent.

The primary culture from a natural source will normally be a mixed culture containing microbes of different kinds. But in the laboratory, the various species may be isolated from one another. A culture which contains just one species of microorganism is called a pure culture. The process of obtaining a pure culture by separating one species of microbe from a mixture of other species, is known as isolation of the organisms.

The pathogen can easily be isolated in artificial culture media for identification and subsequent characterization.

The isolation of different pathogens associated with different forms can be isolated using some following methods:

i) Leaf cut Method:

1. An infected plant or leaf of rice was brought to the laboratory from the field for isolation of pathogens.
2. Initially the symptoms were examined under a microscope or by hand lens. The infected part was removed by a knife and quickly transferred to the sterilizing solution.
3. Surface sterilization of the infected plant part was done by transferring the excised infected leaf segment into a petri dish containing mercuric chloride solution (1: 1000) and kept for 2 – 3 min.

4. Then the leaf segment was transferred to a series of petri dishes containing sterile distilled water for washing of mercuric chloride.
5. Finally the excised leaf segment was placed aseptically into the slant for culture of pathogen.
6. The slant was then incubated at a required temperature for 3 – 5 days after proper leveling.
7. Finally, the culture thus developed was examined microscopically.

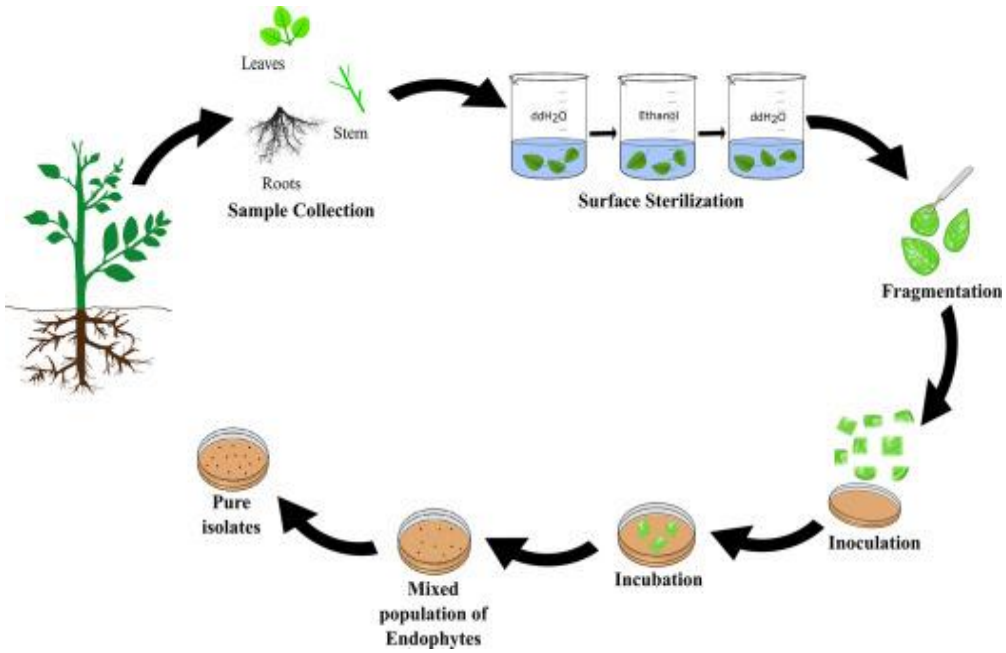


Figure 1.2: Steps for Isolation of Pathogens by leaf cut method

ii) Isolation of Phyto-pathogenic bacteria from diseased plants:

1. The method normally differs from that used for fungi.
2. Cut diseased tissue from the advancing lesion using sterile razor blade in a drop of sterile water and after several minutes, examine under microscope. If bacterial ooze is seen, proceed for isolation.
3. After surface sterilization of sections of diseased tissues as in case of fungal isolations.
4. Immerse the disinfested cut portions in I ml of sterile water taken in a clean sterilized test tube.
5. Crush the cut portions of the leaf with a sterile glass rod. Allow it to stand for 5 minutes to allow the bacteria to diffuse out of the cut tissue and into the water and then part of this homogenate is diluted serially.
6. Finally, plates containing nutrient agar are streaked with a loop dipped in each of the different serial dilutions
7. Incubate the Petri dishes in an inverted position at 28°C and examine for 3-5 days.

iii) Serial dilution Method:

1. Serial dilution, as the name suggests, is a series of sequential dilutions that are performed to convert a dense solution into a more usable concentration.
2. The objective of the serial dilution method is to estimate the concentration (number of organisms, bacteria, viruses, or colonies) of an unknown sample by enumeration of the number of colonies cultured from serial dilutions of the sample.
3. In serial dilution, the density of cells is reduced in each step so that it is easier to calculate the concentration of the cells in the original solution by calculating the total dilution over the entire series.
4. Serial dilutions are commonly performed to avoid having to pipette very small volumes (1-10 µl) to make a dilution of a solution.
5. By diluting a sample in a controlled way, it is possible to obtain incubated culture plates with an easily countable number of colonies (around 30–100) and calculate the number of microbes present in the sample.
6. The sample/culture is taken in a test tube and six test tubes, each with 9 ml of sterile diluents, which can either be distilled water or 0.9% saline, are taken.
7. A sterile pipette is taken.
8. 1 ml of properly mixed sample/culture is drawn into the pipette.
9. The sample is then added to the first tube to make the total volume of 10 ml. This provides an initial dilution of 10^{-1}.
10. The dilution is thoroughly mixed by emptying and filling the pipette several times.
11. The pipette tip is discarded, and a new pipette tip is attached to the pipette.
12. Now, 1 ml of mixture is taken from the 10^{-1} dilution and is emptied into the second tube. The second tube now has a total dilution factor of 10^{-2}.
13. The same process is then repeated for the remaining tube, taking 1 ml from the previous tube and adding it to the next 9 ml diluents.
14. As six tubes are used, the final dilution for the bacteria/cells will be 10^{-6} (1 in 1,000,000).

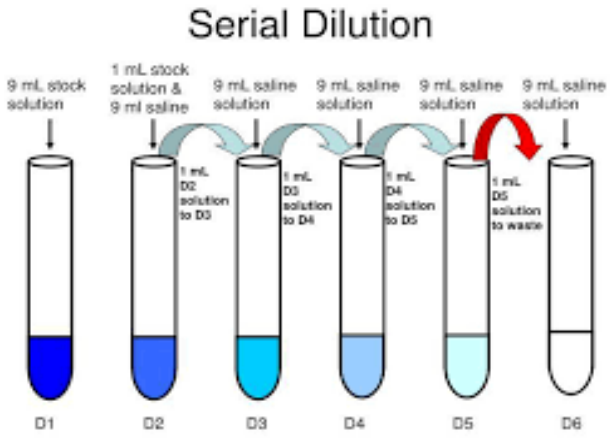


Figure 1.3: Serial dilution method

iv) Single Spore Isolation:

1. Another way to isolate a pure culture of a particular species is to remove individual spores from the species of interest.
2. This procedure can only be used if the fungus sporulates in culture or in plant, otherwise, hyphal tipping is the best option.
3. There are two ways to isolate single spores. When working with plant material, it is sometimes possible to observe individual spores from an infection lesion on the plant using a dissecting microscope.
4. Dip the needle in sterile distilled water to wet the end and try to remove single spore from the infected tissue. Place the spore onto an agar medium.
5. If the spores are small, are difficult to manipulate, or there are other contaminating fungi, it may be necessary to remove several spores in bulk and dilute them in sterile distilled water.
6. Even though there may be other contaminating fungi along with your fungus of interest, the idea is to dilute them away from each other.
7. It will then be possible to isolate the fungi from each other either by hyphal tipping or by single spore isolation without worry of nearby contaminants.
8. Plant materials and contaminated plates will be used so that you can practice these techniques.

Using different isolation methods as discussed above, isolation of pathogens can be used for further studies.

b) Inoculation:

Introduction of plant pathogens to the suitable host or culture media is known as inoculation. Here we are considering hosts as suitable culture media as we are trying to develop the pathogen artificially in the laboratory. Different pathogen groups required different inoculation methods.

Spread method:

Ten microliters from appropriately diluted samples are taken with a micropipette and placed on the surface of the agar medium. The sample is then evenly coated on the agar surface using a sterile glass spreader (triangle rod and L-shaped rod). At the appropriate dilution, each bacterial cell from the specimen should form a single colony after incubation.

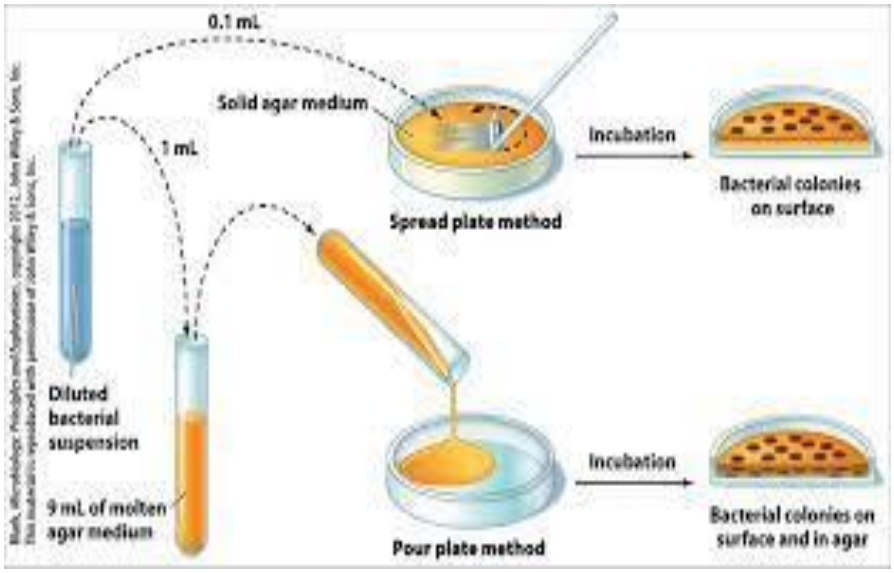


Figure 1.4: A) Spread method; B) Drop Method

2. **Drop method:**

Twenty-five or 50 µl samples at the appropriate dilution are dropped onto the surface of the agar using a micropipette. Then, the plates are placed directly into the dry incubator.

3. **Spiral plater method:**

The spiral plater is the most advanced method for inoculating bacteria for the purpose of counting colony-forming units. The sample liquid is automatically diluted and inoculated using a needle tip on the surface of the agar plate by the instrument, and the bacteria colonies grow uniformly along the spiral trajectory after incubation. As a result, sample counting and pathogen colony observation are more accurate and reproducible.

All these methods require very hygienic conditions and sterilized areas as well as the equipment used during the experiment. The contamination with some other pathogens from the air or already present on the hands or equipment may affect the results. The sterilization may be achieved as per the need of the material. Majority in the laboratory inoculation chamber, laminar flow etc is used during inoculation. Sometimes flame (spirit lamps), chemicals like alcohol, sodium chloride may be used for surface sterilization. For sterilizing large areas some rays like UV-rays have also been used. Some of the sterilization techniques discussed below:

Sterilization Techniques:

For isolating, identifying, and multiplying inoculum, and biological and physiological studies of pathogens require pure cultures. A microbe-free environment is therefore essential. Sterilization of utensils, equipment, and work areas means that all living cells (microorganisms) must be eliminated or inactivated. This is done by physical or chemical

means that irreversibly destroy the protoplasmic structure of cells. Selection of the sterilization method depends upon efficiency and safety requirements, taking into account toxicity, availability, cost, and the effects of the sterilizing agent on the physical and chemical properties of the object to be sterilized. The most common physical methods are heat, ionic radiation, and ultrafiltration (for some liquids). Heat sterilization is used when materials to be sterilized are not damaged by high temperatures under dry or moist conditions.

Dry heat:

Used to sterilize utensils made of glass, metal, and certain kinds of plastic. This type of sterilization requires higher temperatures and longer exposure than moist heat processes, and also hot air ovens that produce uniform heat. The time of exposure is inversely proportional to the temperature; for example, 1 hr at 180°C, 2 hr at 170°C, 4 hr at 150°C, and 12-16 hr at 120°C. Start counting sterilization time when maximum temperature is reached. Glassware should be completely dry, otherwise it may break. Graduated materials should not be sterilized by this method because their dimensions may change. To avoid breakage, sterilized materials should be left in the oven until they reach room temperature.

Moist heat:

The most convenient method for sterilizing most materials, moist heat is quick and has greater penetrating power at lower temperatures in a shorter period of time. It requires using water vapor pressure inside an autoclave. For most purposes, exposure is 20 minutes at 121°C or 30 minutes at 115°C. It is important to expel all the air from the chamber before closing the valve; if not, temperature will not rise. Exposure is calculated starting from the time desired temperature and pressure are reached (15 Lb/in3 or 1.4 kg/cm2). Moist heat can be used on liquids, plastics (although not on all of them), soil, sand, and vermiculite.

Ultraviolet light:

Ultraviolet light is used for sterilization because it kills most microorganisms. While it is recommended for plastic materials, it cannot penetrate glass. Ultraviolet systems must be installed in a closed chamber to avoid exposure, since it is harmful to the eyes.

Ultrafiltration:

Used when proteins and sugars (which break down easily if exposed to high temperatures) must be kept intact. Filters act as barriers for microorganisms. Their size and consistency vary depending on the microorganisms to be filtered.

c) Incubation:

The pathogens introduced on suitable culture media then kept under aseptic conditions for the growth of the colonies of the pathogens. This has been observed after every 24 hrs. for any

growth of the colony and the observations are noted further studies. Incubation has been done maintaining moisture, air flow and temperature required for the growth of the pathogen cultured. The conditions may be different as per the pathogen requirements. Then the colonies are counted and the growth has been detected under microscope.

Incubator:

- An incubator is a device that is used in the laboratories for the growth and maintenance of microorganisms and cultures.
- Incubatorsprovide an optimal temperature, pressure, moisture, among other things required for the growth of microorganisms.

Working:

- The incubator is based on the principle of maintaining a proper atmosphere for the growth of microorganisms.
- Incubators have a heating system that allows for the temperature within the incubator to be adjusted according to the type of organism cultivated inside.
- Similarly, they are provided with adjustments for maintaining the concentration of CO2 to balance the pH and humidity required for the growth of the organisms.
- Variation of the incubator like a shaking incubator is also available, which allows for the continuous movement of the culture required for cell aeration and solubility studies.

Uses:

- Incubators have a wide range of applications including cell culture, pharmaceutical studies, hematological studies, and biochemical studies.
- Incubators can also be used in the stem cell research area.

Unit 2 Mechanism of Plant Infection

2.1 Mechanism of Infection:

Infection is the establishment of a parasite inside the host cell or tissue. Successful infections usually result in the appearance of disease symptoms. Infection process means establishment of pathogens in the host plant. Colonization of a host results from the establishment, growth and reproduction of the pathogen on or in an infected plant. Infestation refers to the establishment of pathogens on the surface of a host. This occurs when the pathogen invades the plant tissue and establishes a parasitic relationship between itself and the plant. Viruses, bacteria, and phytoplasmas are not able to actively penetrate or enter plant host tissues. Therefore they must rely on other methods to infect plant tissues and cells. Associations with insect vectors have been established by these pathogens to aid inoculation and dispersal.

Entry and colonization of pathogens in the host tissues is known as establishment and the infective propagules coming in contact with the host are known as inoculum.

Inoculum potential: It is the inoculum needed for successful infection. It is a function of inoculum density and their capacity.

Definition:

It is defined as the resultant of the action of environment, the vigor of pathogen to establish an infection, susceptibility of the host and amount of inoculums present (Diamond and Horsfall, 1960). Or It is defined as the energy of growth of a parasite available for infection of a host at the surface of the host organ to be infected (Garret, 1960). In the case of specialized pathogens such as rusts and powdery mildews, very few or even one spore is capable of causing infection successfully. In case of non-specialized pathogens such as *Pythium, Phytophthora, Rhizoctonia* and *Sclerotium* require high density of inoculum on the surface of susceptible host for successful infection.

The success of process of infection depends on

1. Host factors

- Susceptibility of host: It is genetically controlled by DNA and it is an inheritable character which is transmitted from parents to offsprings.

- Disease proneness of the host: It is decided by the external factors such as host nutrition, i.e., more nitrogen application makes the host more susceptible and more potash application leads to less susceptibility.

2. Pathogen factors

- Virulence / aggressiveness of the pathogen: It is determined by genetic material which is inheritable.
- High multiplication rate of the pathogen: Chances of infection increases with high rate of multiplication. High birth rate and low death rate is highly essential for successful infection.
- Proper inoculum potential: In case of specialized pathogens very few or even one spore is capable of causing infection successfully, whereas, non-specialized pathogens require high density of inoculum on the surface of susceptible host for successful infection.

3. Environmental factors:

- Environmental conditions such as temperature, relative humidity, moisture, etc., are very important for survival, dissemination and infection process.

Process of infection can be grouped into three stages, i.e., pre-penetration, penetration and post-penetration.

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 two categories

1. Active invaders and 2.Passive invaders

1. Pathogens which make an aggressive effort to gain entry into intact host cells.

2. They do not require help from any external agency to gain entry into host cells.

3. e. g. Phyto-pathogenic fungi Phanerogamic parasites

1. No aggressive effort

2. Require help of external agencies like insect vectors or wounds caused by agricultural implements.

3. e. g. Plant viruses

Phyto-pathogenic bacteria

Plant viruses are particulate in nature and they do not have any capacity to enter the host cell so they do not make any aggressive effort for entry, but depend on different insect vectors for their entry into the host cell. Bacteria have no dormant structures; hence no pre- penetration activity except for multiplication in infection drops on the natural openings.

However, nematodes show some orientation towards the root surface before actual penetration. In fungal pathogens, pre-penetration includes spore germination and growth of the resulting germ tube on the surface of the host plant. Germination is essentially the change from low metabolic rate to a high metabolic rate and involves a change from near dormancy to intense activity; for this an energy source is needed such as a carbohydrate or fat reserve in the propagule. Fungal invasion is chiefly by germ tubes or structures derived from them. In some fungi like *Rhizoctonia solani* and *Armillariella mellea*, the hypha act in a concerted way to achieve the penetration. In *Rhizoctonia solani*, the fungus on coming in contact with root surface, first forms infection cushions and appressoria and from these multiple infections takes place by means of infection pegs. In *Armillariella mellea*, the fungus hyphae form the rhizomorphs (aggregation of hyphae into rope-like strands) and only these can cause infection.

2. 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, and 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

1. 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, Penicillium, 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 organisms of Dutch elm disease (*Ceratostomella ulmi*) enter through the wounds caused by elm bark beetles.

2. Natural openings

a) Stomata: There is variation in the behavior of the 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*, a destructor infecting onion leaves, the germ tube continues to grow after the formation of the 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 a few days on the surface of the leaf. A hypha then arises from it which grows into the substomatal chamber and swells to form a vesicle, which in turn gives rise to hyphae which invade palisade 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).

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

c) 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.

a) 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 affects its entry.

b) 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 Bengal gram contain maleic acid which is antifungal and provide resistance to infection by the rust fungus (*Uromyces ciceris arietini*). Similarly, protocatechuic acid and catechol in the red scales of onion provide resistance to onion smudge pathogen, *Colletotrichum circinans*. To overcome these physical and chemical barriers, the fungi produce various enzymes, toxins, organic acids and growth regulators.

Through non-cutinized surfaces:

The pathogen can enter through some non-cutinized surfaces on the plants. They have been illustrated as below:

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 ehrenbergii*), 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, Thielaviopsis 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*)

3. Post Penetration

Invasion and colonization: Infection is the process by which pathogens establish contact with the susceptible cells or tissues of the host and derive nutrients from them. 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.

Fungi spread into all parts of host organs, either by growing directly through the cells as an intracellular mycelium or by growing between the cells as an intercellular mycelium.

During establishment, pathogensproduce different substances which include enzymes, toxins, growth hormones and polysaccharides which will help in colonization of the host.

In ectoparasites the main body of the pathogen lies on the surface of the host with only feeding organs (haustoria) penetrating the tissues Ex: Most of the powdery mildew fungi.

Some fungal parasites develop both external and internal mycelium Ex: *Rhizoctonia solani*. The endophytic parasites or endoparasites grow subcuticularly (*Diplocarpon rosae*, black spot of rose), in parenchyma tissues (most fungal and bacterial pathogens as well as many nematodes) or in vascular tissues (vascular wilt parasites). Some pathogens are endobiotic, i.e., mycelium is not produced and the thallus is entirely present within a host cell Ex: *Synchytrium endobioticum*.

Bacteria invade tissues intracellularly, but also grow intracellularly when parts of the cell walls dissolve. Viruses, viroids, mollicutes and fastidious bacteria invade tissues by moving from cell to cell intracellularly.

Infection caused by microbes may be local (involve single cells or few cells or small area) or systemic (pathogen spreads and invades most or all susceptible cells and tissues throughout the plant Ex: *Sclerospora graminicola*). The time interval between inoculation and appearance of disease symptoms is called the incubation period.

Exit of the pathogen

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. Once the pathogens exit from the host, they survive and are disseminated to other hosts and continue the infection cycle.

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. Most plant pathogenic fungi grow out on the host surface and produce repeating spores (secondary inoculum), usually asexually, under favorable conditions. The spores thus formed are disseminated through wind, water, soil, seed, vegetative propagating material, agricultural implements, etc.

Sub-unit 3.1:

Factors affecting mechanism of infection-

Important environmental factors that may affect development of plant diseases and determine whether they become epiphytotic include temperature, relative humidity, soil moisture, soil pH, soil type, and soil fertility.

Temperature:

Each pathogen requires an optimum temperature for growth. In addition, different growth stages of fungi, such as the production of spores (reproductive units), their germination, and the growth of the mycelium (the filamentous main fungus body), may have slightly different optimum temperatures. Storage temperatures for certain fruits, vegetables, and nursery stock are manipulated to control fungi and bacteria that cause storage decay, provided the temperature does not change the quality of the products. Little, except limited frost protection, can be done to control air temperature in fields, but greenhouse temperatures can be regulated to check disease development.

Knowledge of optimum temperatures, usually combined with optimum moisture conditions, permits forecasting, with a high degree of accuracy, the development of such diseases as blue mold of tobacco (*Peronospora tabacina*), downy mildews of vine crops (*Pseudoperonospora cubensis*) and lima beans (*Phytophthora phaseoli*), late blight of potato and tomato (*Phytophthora infestans*), leaf spot of sugar beets (*Cercospora beticola*), and leaf rust of wheat (*Puccinia recondita tritici*). Effects of temperature may hide symptoms of certain viral and mycoplasmal diseases, however, making them more difficult to detect.

Relative humidity:

Relative humidity is very critical in fungal spore germination and the development of storage rots. *Rhizopus* soft rot of sweet potato (*Rhizopus stolonifer*) is an example of a storage disease that does not develop if relative humidity is maintained at 85 to 90 percent, even if the storage temperature is optimum for growth of the pathogen. Under these conditions, the sweet potato root produces suberized (corky) tissues that wall off the *Rhizopus* fungus.

High humidity favors development of the great majority of leaf and fruit diseases caused by fungi, water molds, and bacteria. Moisture is generally needed for spore germination, the multiplication and penetration of bacteria, and the initiation of infection. Germination of powdery mildew spores occurs best at 90 to 95 percent relative humidity. Diseases in greenhouse crops such as leaf mold of tomato (*Cladosporium fulvum*) and decay of flowers, leaves, stems, and seedlings

of flowering plants, caused by *Botrytis* species are controlled by lowering air humidity or by avoiding spraying plants with water.

Soil moisture:

High or low soil moisture may be a limiting factor in the development of certain root rot diseases. High soil-moisture levels favor development of destructive water mold fungi, such as species of *Aphanomyces*, *Pythium*, and *Phytophthora*. Excessive watering of houseplants is a common problem. Overwatering, by decreasing oxygen and raising carbon dioxide levels in the soil, makes roots more susceptible to root-rotting organisms.

Diseases such as take-all of cereals (*Ophiobolus graminis*); charcoal rot of corn, sorghum, and soybean (*Macrophomina phaseoli*); common scab of potato (*Streptomyces scabies*); and onion white rot (*Sclerotium cepivorum*) are most severe under low soil-moisture levels.

Soil pH:

Soil pH, a measure of acidity or alkalinity, markedly influences a few diseases, such as common scab of potato and clubroot of crucifers (*Plasmodiophora brassicae*). Growth of the potato scab organism is suppressed at a pH of 5.2 or slightly below (pH 7 is neutral; numbers below 7 indicate acidity, and those above 7 indicate alkalinity). Scab is not normally a problem when the natural soil pH is about 5.2. Some farmers add sulfur to their potato soil to keep the pH about 5.0. Clubroot of crucifers (members of the mustard family, including cabbage, cauliflower, and turnips), on the other hand, can usually be controlled by thoroughly mixing lime into the soil until the pH becomes 7.2 or higher.

Soil type:

Certain pathogens are favored by loam soils and others by clay soils. *Phymatotrichum* root rot attacks cotton and some 2,000 other plants in the southwestern United States. This fungus is serious only in black alkaline soils—pH 7.3 or above—that are low in organic matter. *Fusarium* wilt disease, which attacks a wide range of cultivated plants, causes more damage in lighter and higher (topographically) soils. Nematodes are also most damaging in lighter soils that warm up quickly.

Soil fertility:

Greenhouse and field experiments have shown that raising or lowering the levels of certain nutrient elements required by plants frequently influences the development of some infectious diseases e. g. fire blight of apple and pear, stalk rots of corn and sorghum, *Botrytis* blights, *Septoria* diseases, powdery mildew of wheat, and northern leaf blight of corn. These diseases and many others are more destructive after application of excessive amounts of nitrogen fertilizer. This condition can often be counteracted by adding adequate amounts of potash, a fertilizer containing potassium.

Unit 3 Diseases

Sub Unit 3.1

Disease caused by Phytoplasma

Little leaf of Brinjal:

Name of the Host:*Solanum melongena* L.

Causal Organism: Mycoplasma Like Organism(MLO)/ Phytoplasma

Little leaf of brinjal is known to cause heavy economic losses in India. As the name indicates, symptoms of the disease include shortening of the petioles and production of leaves which are much smaller in size. Petioles are so short that leaves seem to be glued to the stem.

Fig. 3.1 Healthy Brinjal Leaves

Infected leaves of Brinjal

Causal Organism:

Mycoplasma-like organism (MLO).

Phytoplasmas/ Mycoplasmas are obligate bacterial parasites of plant phloem tissue and of the insect vectors that are involved in their plant to plant transmission. Phytoplasmas were discovered in 1967 by Japanese scientists who termed them Mycoplasma Like Organisms (MLOs).

Symptoms of Little Leaf Disease:

1) The main symptom of the disease is the production of very short leaves by affected plants.
2) The petioles are so much reduced in size that leaves appear sticking to the stem. Such leaves are narrow, soft, smooth and yellowish in color.
3) Newly formed leaves are further reduced in size.
4) The internodes are shortened and at the same time a large number of axillary buds are stimulated to grow into short branches with small leaves.
5) This gives the whole plant a bushy appearance.

6) Usually such plants are unable to form flowers. Fruiting is very rare.

Disease Cycle:

The disease is transmitted through the vector (*Cestius phycitis*) and by a leafhopper (*Hishimonus phycitis*). Artificially the disease has been transmitted successfully to tomato, potato and tobacco. Probably during the season of Brinjal crop, the causal agent survives on weed hosts and from there it is transmitted to the main crop by its insect vector.

Management / Control Measures of Little Leaf Disease:

i. Since no effective control measure is found it is better to eradicate the weed host and remove the diseased Brinjal plants.
ii. Tetra-cycline has been reported to control the disease.
iii. Seeds are treated with Thiram @2g/kg of seeds before sowing.
iv. Continuous raising of nurseries in the same plot should be avoided.

Sub Unit 3.2

Disease caused by Virus

Yellow Vein Mosaic of Bhendi or Okra:

Host: *Abelmoschus esculentus*or *Hibiscus esculentus*

Causal Organism: Bhendi Yellow Vein Mosaic Virus

It was first found in 1924 in Bombay of India and Sri Lanka. It is the major limitation of the production of okra. This disease is transmitted by the whitefly.

Fig. 3.2 Healthy Leaf of Okra

Infected Leaf of Okra

Symptoms:

1) Yellowing of the entire network of veins in the leaf blade is the characteristic symptom.
2) In severe infections the younger leaves turn yellow, become reduced in size and the plant is highly stunted.

3) The veins of the leaves will be cleared by the virus and internal area becomes completely yellow or white.
4) In a field, most of the plants may be diseased and the infection may start at any stage of plant growth.
5) Infection restricts flowering and fruits, if formed, may be smaller and harder.
6) The affected plants produce fruits with yellow or white color and they are not fit for marketing.
7) The virus is spread by whiteflies.

Causal Organism

Bhendi yellow vein mosaic (BYVMV) or yellow vein mosaic of okra (OYVMV) is a viral disease caused by *Monopartite Begomovirus*, Bhendi yellow vein mosaic virus (BYVMV, family: Geminiviridae) affecting Okra plants. (*Bhendi virus-1 and Hibiscus virus -1*.)

Disease Cycle

The virus particles are 16 – 18 nm in diameter. The virus is transmitted by - white fly (*Bemisia tabaci*) Leaf hopper (*Empoasea devastans*). The weeds like *Croton sparsiflorus, Malvastrum tricuspidatum, Ageratum spp* are behaving as collateral hosts. This disease is not at all seed borne. Warm humid weather favors theinsect population.

Management:

i. The virus is transmitted by the whitefly (Bemisia tabaci,) so using some insecticides to control this insect will reduce the disease transmission.
ii. For sowing during the summer season, when the whitefly activity is high, the susceptible varieties should be avoided.
iii. Spraying monocrotophos 1.5 ml/liter of water can restrict the disease spread.
iv. Synthetic pyrethroids should not be used because it will aggravate the situation.
v. It can be controlled by application of Chlorpyriphos 2.5 ml + neem oil 2 ml lit of water.
vi. By selecting varieties resistant to yellow vein mosaic like Parbhani Kranti, Arka Abhay, Arka Anamika, Janardhan, Haritha and Varsha Uphar, the incidence of the disease can be minimized.

Sub Unit 3.3

Disease caused by Bacteria

Citrus Canker:

Citrus Canker is a bacterial disease of worldwide distribution occurring wherever citrus is grown. It is a serious menace to our most valued citrus orchards causing objectionable blemishes on the fruit. The disease causes serious damage in India, China, Japan and Java.

The pathogen incites severe canker disease in a number of citrus species on stems, leaves and fruits. The disease attacks most of the species/varieties of citrus. The most susceptible species are the acid lime plants, the sweet orange and the grape fruit.

Name of the Host: *Citrus limon*

Causal Organism: *Xanthomonas citri*

Symptoms:

Citrus canker is mostly a leaf-spotting and fruit-ring-blemishing disease, but when conditions are highly favorable for infection, infections cause defoliation, shoot dieback, and fruit drop.

1) **Leaf Lesions:** Citrus canker lesions start as pinpoint spots and attain a maximum size of 2 to 10 mm diameter.
2) The eventual size of the lesions depends mainly on the age of the host tissue at the time of infection and on the citrus cultivar.
3) Lesions become visible about 7 to 10 days after infection on the underside of leaves and soon thereafter on the upper surface.
4) The young lesions are raised or ‘pustular’ on both surfaces of the leaf, but particularly on the lower leaf surface. The pustules eventually become corky and with a raised margin and sunken center.
5) A characteristic symptom of the disease on leaves is the yellow halo that surrounds lesions.
6) A more reliable diagnostic symptom of citrus canker is the water-soaked margin that develops around the necrotic tissue, which is easily detected with transmitted light.
7) The trees are, however, not commonly killed.
8) The lesions on the foliage, at first, appear on the lower surface as small round raised spots.
9) These are translucent and of yellowish brown color. Later the spots turn white or grayish and finally rupture.
10) The older lesions are corky and brown, sometimes purplish.
11) The necrotic brownish canker regions are surrounded by a yellowish brown to green raised margin and distinct watery yellow halo region.

12) The yellow halo region is free from the pathogen. The cankerous lesions contain the pathogen in millions.
13) The lesions on the twigs are usually irregular in form. The lesions on the fruit are similar to those on the leaves but lack the yellow halo.

Fig. 3.3 Diseased leaves and fruits of *Citrus*

Causal Organism:

Citrus canker, caused by the bacterium *Xanthomonas citri* subsp. *citri*, is a rod-shaped, gram-negative, and has a single polar flagellum. Colonies on laboratory media are usually yellow due to 'xanthomonadin' pigment production. When glucose or other sugars are added to the culture medium, colonies become very mucoid due to the production of an exopolysaccharide slime. A semi-selective medium can be prepared by adding an antibiotic, kasugamycin, which inhibits many contaminants but not xanthomonads. The maximum and optimum temperature ranges for growth are 39°C (95 to 102°F) and 28 to 30°C (82 to 86°F), respectively.

Disease Cycle

Citrus canker can be a serious disease where rainfall and warm temperatures are frequent during periods of shoot emergence and early fruit development.

Most spread of canker by wind and rain is for short distances, i.e., within trees or to neighboring trees. Canker develops more severely on the side of the tree exposed to wind-driven rain. Spread over longer distances, up to several miles, can result from severe meteorological events such as tropical storms, hurricanes, and tornadoes.

Bacteria propagate in lesions in leaves, stems, and fruit. When there is free moisture on the lesions, the bacteria ooze out and can be dispersed to infect new growth. Wind-driven rain is the main dispersal agent that helps in the penetration of bacteria through the stomatal pores (Drawing of the disease cycle) or wounds made by thorns, insects (leaf miner), and blowing sand. Pruning causes severe wounding and can lead to infection. Multiplication of bacteria occurs mostly while the lesions are still expanding and numbers of bacteria produced per lesion is related to general host susceptibility

Control Measures of Citrus Canker Disease:

1. Eradication:

i. The disease is controlled by the eradication of diseased trees. This is accomplished by removing the trees with advanced infection and burning them.

2. Pruning:

i. The infected trees may be cured by removing the diseased foliage and branches with pruning scissors and then spraying the trees with one percent Bordeaux mixture at regular intervals.
ii. The use of disease free nursery stock for planting is the best method of controlling the disease.
iii. The fallen infected leaves and twigs should be collected and burnt.

3. Spraying:

i. Spraying with Bordeaux mixture and lime sulfur is a useful measure to protect the fruit. It should be done during the first three months after the beginning of fruit formation. Spraying should commence before the onset of rains and repeated during the rainy season.
ii. Citrus nurseries should be raised in places away from the regions of heavy and protracted rainfall. There should be no **"khatti"** hedge around the nurseries.
iii. Rangaswamy (1957) reported that the use of antibiotic sprays is useful in controlling the disease. Streptomycin sulfate and Phonomycin have been found to be effective. Vaheeddudin (1959) found that spraying with neem-cake is effective in controlling citrus canker.

Sub Unit 3.4

Diseases caused by Fungi

I) Rust of Sugarcane

Name of the Host: *Sachharum officinarum*

Causal Organism: ***Puccinia melanocephala, Puccinia erianthi***

Sugarcane is one of the plants with the highest bioconversion efficiency. This crop is able to efficiently fix solar energy, yielding some 55t of DM/ha annually. It's the world's largest crop by production quantity (1.9 billion t). Cane accounts for 79% of sugar produced. Molasses, Bagasse are some other products obtained from this crop.

There are 3 Rust species (types) that infect Sugarcane:

Brown rust: ***Puccinia melanocephala***

Orange rust: ***Puccinia kuehnii***

Tawny rust: ***Macruropyxis fulva sp.***

Symptoms:

1) The earliest symptoms are small, elongated yellowish spots that are visible on both leaf surfaces.
2) The spots increase in length, turn brown to orange-brown or red-brown in color, which coalesce and form large, irregular necrotic areas, thus it shows the rusty appearance of leaves.
3) This eventually resulted in premature death of the leaves.

Fig. 3.4. Sugarcane rust

Causal Organism/Pathogen: ***Puccinia erianthi*** Padw. and Khan

Uredinia are elongate, reddish-brown, with capitate, hyaline to light brown paraphyses. Urediniospores are thick-walled, orange-brown, obovoid, measuring 26-34 x 16-20 µm. The urediniospore surface is echinulate with 4-5 equatorial pores. Teliospores are dark brown and measure 30-43 x 17-23 µm, clavate, two-celled and slightly constricted at the septum.

Disease Cycle:

Fig. 3.5. Rust of sugarcane Urediniospores

Fig. 3.6. Rust of sugarcane showing Teliospores

Sugarcane rust is autoecious rust. Uredospores are produced from the pustules that break through the epidermis on the underside of the leaves. Then the spores disperse from pustules via wind or rain onto the leaves of new host sugarcane. The uredospores then germinate on the sugarcane leaves, develop appresoria, infecting new hosts via penetration on the plant's stomata. This cycle can be repeated throughout the year because sugarcane growing areas are conducive for the growth of this pathogen. P. melanocephala infection is non-systemic.

Management

Cultural method

i. The best means of control for sugarcane rust is to grow resistant varieties Use resistant varieties like Co 91010 (Dhanush), Co 87025 (Kalyani).
ii. Affected leaves should be removed and burned immediately.
iii. Sugarcane grown in fields receiving recent applications of mill mud is typically very prone to rust.

Chemical method

i. Spray Tridemorph 1.0 liters or Mancozeb 2.0 kg/ha.
ii. Use Dithane M 45 @ 2 g/lit for one spraying.
iii. Application of triazole or strobilurin or pyraclostrobin fungicide @ 3 g/ lit of water.

II) Rust of Soybean

Soybean rust is one of the most important soybean diseases worldwide. Soybean, a major crop both in the US and the world, is high in vegetable oil and protein (approximately 20 and 40%, respectively) and provides 57% of the vegetable oil consumed worldwide and 68% of the vegetable protein.

Name of the Host: *Glycine max***.**

Causal Organism: *Phakopsora pachyrhizi*

Soybean rust caused by Phakopsora pachyrhizi has been a serious disease in Asia for many decades. It appeared in Africa in 1997, and in the Americas in 2001. Before it was first found in the continental USA in late 2004, probably brought in by a hurricane, it was considered such a threat that it was listed as a possible weapon of bioterrorism. Soybean rust cannot overwinter in areas with freezing temperatures, but it can spread by wind rapidly over such large distances, its development can be so explosive, and it can cause such rapid loss of leaves that it is now one of the most feared diseases in the world's soybean-growing areas.

Symptoms:

Symptoms of soybean rust are usually first observed on the lower leaves on a soybean plant as water-soaked spots which progress to tan or reddish brown lesions that contain rust pustules. These pustules are most pronounced and numerous in the underside of leaves. Infected

foliage turns bronze/yellow and these patches can clearly be seen in disease fields. Waling into severely infected patches can release viable clouds of rust spores. Premature defoliation occurs as a result of infection and this affects the number of pods and the seed weight.

Fig. 3.7. Pustules with many spores

Red brown lesionswithout pustules or spores

Causal Organism/ Pathogen:

Phakopsora pachyrhizi

Environmental conditions that promote healthy growth and high yield in a soybean crop are also the conditions most suitable for the development of soybean rust.

Disease Cycle:

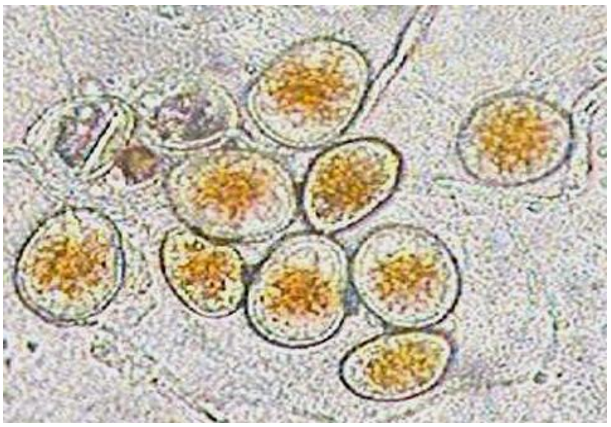


Fig. 3.8: Soybean rust spores under microscope

Phakopsora pachyrhizi, like all rust pathogens, requires green living tissue at all times. To survive winter in the absence of a soybean crop, the soybean rust pathogen must find another living host. Thus, the soybean rust pathogen will survive where freezing temperatures do not occur. Kudzu, a common perennial legume, is thought to be a potential overwintering host in these areas.

Spores of the soybean rust pathogen are transported readily by air currents and can be disseminated rapidly hundreds of miles in 2 to 3 days. Weather conditions will determine when and where the spores travel from south to north.

Infection under favorable temperature and moisture conditions takes place within 6 hours and uredinia can develop in lesions 5 to 8 days afterwards. The first urediniospores can be produced as early as 9 days after infection, and spore production can continue on a uredinium for up to 3 weeks.

Spores can remain viable for almost two months if humidity is high and ultraviolet radiation exposure is low. New uredinia can grow with a pustule and as a result, spores may be continually released from a pustule for up to 15 weeks as long as moisture and moderate temperatures are present. The fungus infects over 95 species of plants including many wild and edible legumes. These alternative hosts can serve as reservoirs for the pathogen when the soybean is not present, where inoculum may build up. Roughly 300 million spores may be produced per plant. During the height of an infection a soybean field can release ca. 1000 trillion spores/ha/day.

Disease Management:

There are three basic management tactics that can play a role in reducing soybean rust epidemics: fungicides, genetic resistance, and cultural practices.

Cultural practice:

Changing planting and harvest dates may avoid disease. Planting early with an early maturing cultivar may avoid the rust until the crop has either been harvested or is so far along that the disease will have little impact on yield. Planting dates may also be delayed so that the vulnerable reproductive period occurs during dry conditions that do not favor rust.

Chemical method:

Several fungicides are registered in the US for soybean rust control, and most can be classified into three groups: chloronitriles, strobilurins, and triazoles.

i. Chlorothalonil is the one chloronitrile fungicide registered for soybean rust control. Its protective mode of action affects many biochemical pathways in the pathogen, but it is not taken up by the plant, not even by the cuticle. As a result, it is more subject to weathering than the strobilurins or the triazoles and complete coverage of the leaf surface is critical. To be effective, chlorothalonil may need to be reapplied several times if new growth or weathering occurs.

ii. Strobilurin fungicides are modeled after a natural antifungal compound.

iii. Triazoles inhibit sterol production, which disrupts cell membrane function in the pathogen. Triazoles are absorbed and translocated upward in the plant.

iv. The key to effective control of soybean rust with fungicides is application timing. This is especially important in areas of the US where the soybean rust pathogen must be reintroduced each year. The introduction or reintroduction will probably occur at different times in different years or not at all in some years.

Genetic resistance:

i. Soybean plants respond to infection by P. pachyrhizi by producing either tan, red-brown, or no lesions at all. Tan lesions produce many pustules with many spores.

III) Rust of Wheat

Name of the Host: *Triticum aestivum*

Causal Organism: *Puccinia graminis tritici*.

Rust diseases represent the most economically significant fungal diseases in wheat and other cereal crops worldwide, and they are widely distributed across wheat growing regions. With the capacity to form new strains of fungus, rusts can attack even previously resistant varieties. Rust diseases possess the ability to spread and travel long distances by dispersal of windborne spores and can rapidly develop under optimal weather conditions.

Fig. 3.9. Rust of wheat

Symptoms:

1) Stem rust, also known as black rust, is caused by the fungus *Puccinia graminis* f. sp.*tritici.* It infects wheat and other cereals across all wheat growing regions.
2) Stem rust occurs when raised spots (pustules) form on stems and leaf sheaths, although occasionally they may form on awns, glumes and seeds.
3) Stem rust spots appear elliptical.
4) The spots form on both lower and upper leaf surfaces and look orange to dark-red in color. These are urediniospores.
5) Telia develop later in the same sorus as uredia.
6) The margins on stem rust spots are ragged. Young pustules release numerous spores. Later in the growing season, spores transform and become dark colored, hence the common name of black rust.

Disease Cycle:

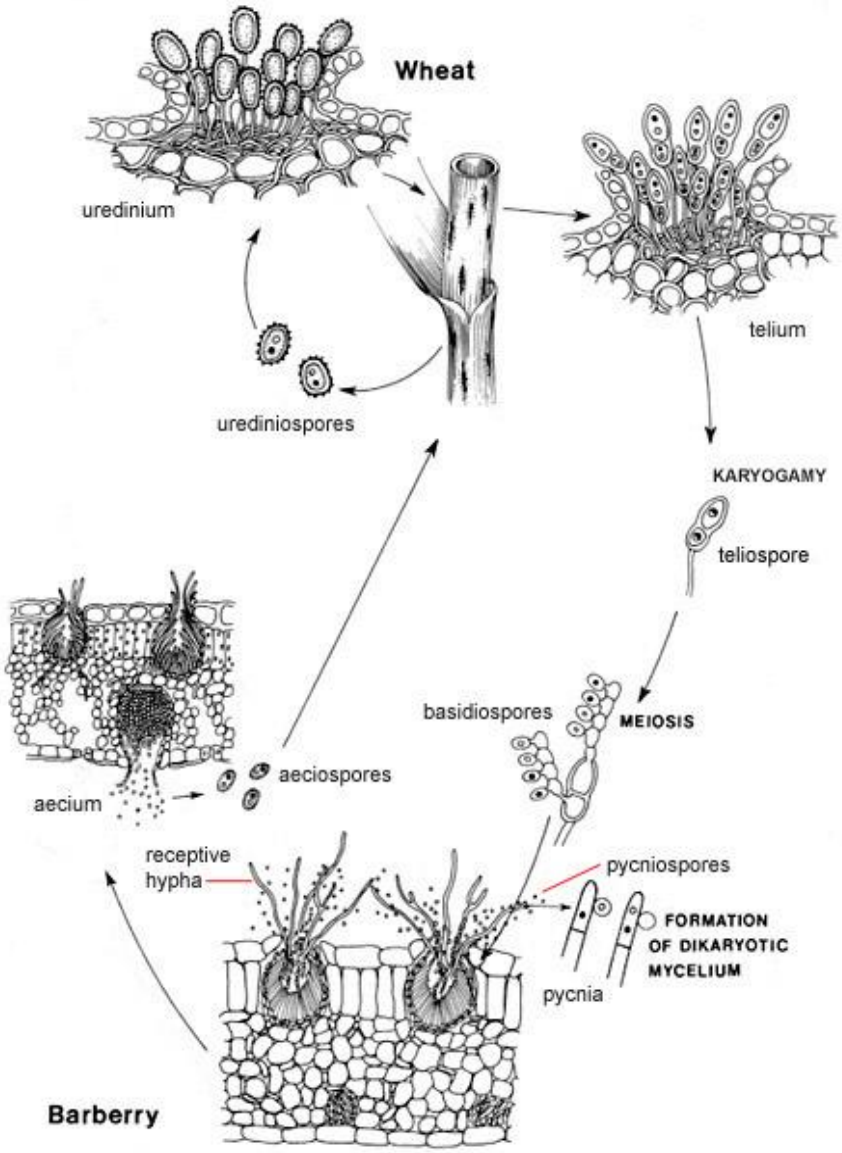


Fig. 3.10. Wheat rust disease cycle

Stem rust development requires the warmest temperatures of the three wheat rusts –ideally 59 to 84 degrees F and six to eight hours of moisture on the leaf surface. With wet weather and optimal temperatures, new lesions are formed in seven to 10 days. Stem rust disease also spreads spores through wind dispersal and splashing water. Spores are produced in multiple cycles during the growing season.

Overwintering spores that develop in the previous year's wheat crop late in the season survive to produce additional spores, which spread via wind to infect wheat. Thus, another lifecycle is completed. In most U.S. growing regions, the primary source of stem rust infection in wheat is repeated spore production.

The rust has five different stages viz. pycniospores, aeciospores, urediniospores, teliospores and basidiospores. Out of these five stages pycnial and aecial stages are completed on alternate host barberry plant. Uredial and telial stages are observed on wheat plants. Thus this disease can not complete its life without two hosts.

Crop Damage

The disease spreads rapidly and can travel long distances by wind or other means of transportation such as farm equipment or plant materials. With severe infections, the disease can turn a healthy crop into a tangle of black stems only weeks away from harvest, resulting in shriveled grain. According to the United Nations Food and Agriculture Organization (FAO), stem rust can result in 70 percent or more loss in wheat yield.

While stem rust has historically been the most damaging disease of wheat, it's not as prevalent today thanks to resistant varieties.

Management of Wheat Rusts:

Farmers have several options to manage wheat rusts, ranging from seed treatments, variety selection, scouting, cultural practices and fungicide applications.

Seed treatments

Using the correct fungicide seed treatment and rate is one way to provide effective and economical disease control in wheat production. Seed treatments protect seed and young plants from disease and other threats to plant health and yield. Areas commonly infected with rust also will need a foliar fungicide treatment to provide protection beyond the seedling growth stage.

Variety selection

Where available, use rust-resistant varieties for best protection against leaf rusts. Every commercially available wheat variety has a unique disease package, and excellent disease resistance is not available to manage all disease threats in high-yielding varieties. It's best to select two or three high-yielding varieties that offer the best resistance to common diseases found on your individual farm.

Scouting

When scouting for weeds and insects, check for the presence of wheat rusts and other diseases. Monitor reports of wheat rust development occurring in states south of your area. This will allow you to track the progression of rust diseases migrating north from overwintering hosts and will also help you predict the timing and severity of infestations before they might affect your region. Keep a close eye on weather conditions because rust spores spread through wind currents to promote disease infection.

If you suspect leaf rust, stem rust or stripe rust infection, take samples and work with your county Extension agent to confirm a diagnosis. Crop identification guides are also helpful. Scouting

helps determine levels of infection so you can make the best decision about the necessity and rate of fungicide applications.

Cultural practices

Disease-free seed gives seedlings a good start. Good weed control preplant, at planting and throughout the growing season also helps protect against disease and other pests.

Fungicides

Good control of wheat rusts can be achieved with commercially available fungicides and proper application timing. The decision to use fungicides should be based on scouting for symptoms. It's important to assess disease severity from the onset of infection through the various growth stages. Application timing should take into consideration that diseases should be managed before infection reaches the upper leaves. Other factors affecting fungicide application are infection levels in the field, the susceptibility of the variety and the market price for wheat grain.

IV) White Rust of Crucifers/white blisters disease:

Name of the Host:

The disease affects a large number of crucifer crops of economic importance like Mustard, Cress, Rape, Radish, Cabbage, Cauliflower, turnip etc. In India the disease is reported on Mustard, Rape, Eruca sativa, turnip. Cauliflower and Cleome viscosa.

Name of the Causal Organism:

The causal organism *Albugo Candida* or*Cystopus candidus* is an obligate parasite. White rust or white blisters disease is one of the common diseases of crucifer crops. It is worldwide in distribution occurring in all the areas wherever crop is cultivated. Both wild and cultivated varieties are attacked.

Symptoms of White Rust Disease:

1) The disease affects all the aerial parts of the plant, the roots are not attacked.
2) Symptoms may appear as a result of two types of infection: Local and Systemic.
3) In case of local infection, isolated spots or pustules appear on leaves or stems or inflorescence.
4) The pustules are of variable size, measuring 1 -2 mm in diameter and are raised in shiny white areas.
5) These may arise in close proximity and coalesce to form large irregular patches.
6) Usually, the pustules appear in a circular or concentric arrangement with one or two central areas.

7) The host epidermis ruptures exposing white powdery mass consisting of spores of the fungus.
8) Pustules occurring on leaves are usually confined to the lower surface only.
9) In systemic infections, young stems and inflorescence are infected. The fungus becomes systemic in these parts and the affected tissues are stimulated to various types of deformities.
10) The most prominent is Hypertrophy of the affected parts. Due to Hypertrophy and Hyperplasia of floral parts, these show swellings and distortion.
11) The peduncle and pedicel may become enormously thickened upto 12-15 times the normal diameter. Floral parts become fleshy, swollen, green or violet in color, the stamens falling off early.
12) The petal may turn green sepal like and stamens and carpels are also converted to swollen leaf like structures.
13) The ovules are usually atrophied as also the pollen grains resulting in total sterility. Pustules may also appear on these parts. However, the affected parts are full of oospores and starch.
14) When the systemic infection has taken early, the growth of the entire plant is checked, stunted and only small leaves may be formed. The stem and the axis of the inflorescence may get twisted appearing in a zigzag sequence. Normal dormant buds are stimulated and grow into lateral shoots.

Fig. 3.11. White rust of crucifer

Causal Organism/Pathogen:

The causal organism *Albugo Candida* (Lev.) Kunze or *Cystopus candidus* Lev. is an obligate parasite.White rust or white blisters disease is one of the common diseases of crucifer crops. It is worldwide in distribution occurring in all the areas wherever crop is cultivated. Both wild and cultivated varieties are attacked.

The disease affects a large number of crucifer crops of economic importance like Mustard, Cress, Rape, Radish, Cabbage, Cauliflower, turnip etc. In India the disease is reported on Mustard, Rape, Eruca sativa, turnip. Cauliflower and Cleome viscosa.

Disease Cycle:

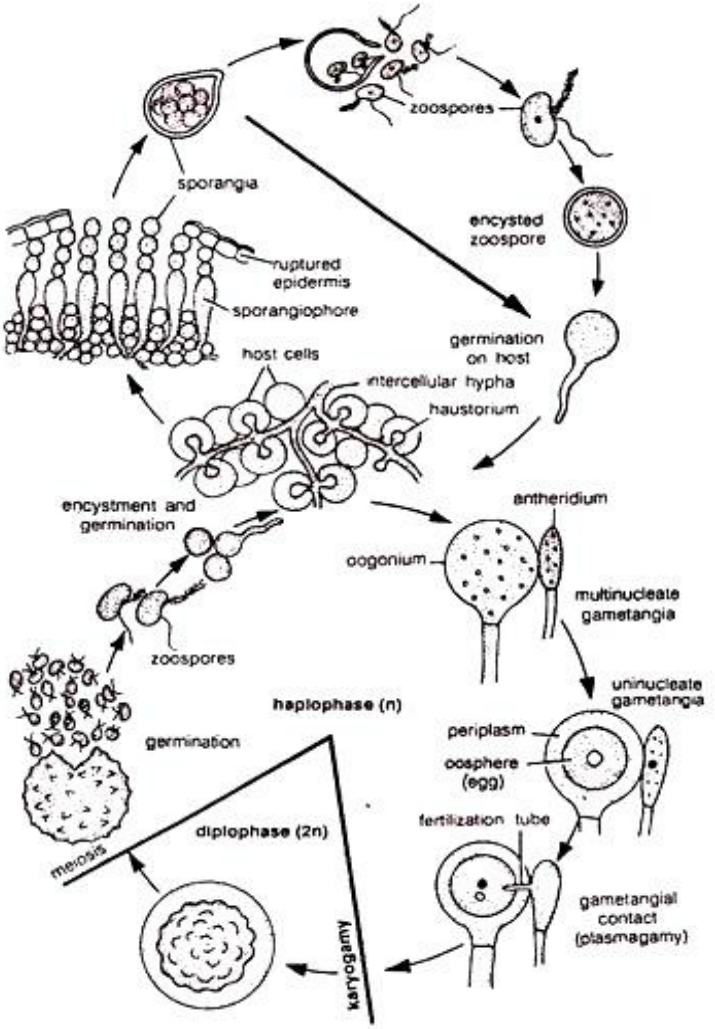


Fig. 3.12. Disease cycle

The primary infection occurs due to oospores perennating in the soil or due to mycelium perennating on perennial hosts. These serve as primary inoculum when the environmental conditions are favorable.

Oospores germinate in presence of water to form a vesicle in which a large number of zoospores are formed. These zoospores swim in a film of water and land on the suitable host, germinate by germ tubes, enter the host and establish infection. The mycelium in the host is intercellular with globose haustoria.

Soon the mycelium after absorbing nutrients and food materials from the host accumulates below the lower epidermis. Conidiophores, which are clavate, and formed at the tip of hyphae,

begin to produce conidia sporangia in basipetal succession. The pressure of these breaks open the lower epidermis and white rust symptoms become apparent on the leaves.

The conidia sporangia produced during the early phase of the growing season cause secondary infection in the host. These are blown away by wind or any other agency, land on the host surface and germinate to form zoospores.

The zoospores germinate by formation of germ tubes which enter the host and cause secondary infection. If the conditions are favorable, this is repeated.

When the conditions become unfavorable or during the later phase of the growing season, the fungus begins sexual reproduction producing oospores. These oospores, being thick-walled, can withstand the unfavorable conditions.

During harvesting of the crop, the diseased hypertrophied portions of the plant are generally left in the field where they perennate waiting for the favorable conditions to return back.

Control Measures of White Rust Disease:

i) Clean cultivation and destruction of weed should be practiced.
ii) Crop rotation will avoid the soil borne primary inoculum.
iii) Spraying with 0.8 percent Bordeaux mixture or Dithane M-45 (0.2%) may be undertaken to check the spread of the disease.
iv) Disease resistant varieties are preferred viz. Chinese cabbage and Pak Choi.

V) Grain Smut of Jowar

Name of the Host: *Sorghum vulgare/bicolor*

Causal Oragnism: *Sporisorium sorghi* (synonym *Sphacelotheca sorghi*)

Grain sorghum is an important feed grain and silage for livestock in the United States but is consumed by humans in many parts of the world. The fungus that causes sorghum smut grows into the ovaries of the plant and replaces the grain with a fungal structure full of spores, called as sori. For this reason, without controls, smuts can be extremely damaging under the proper conditions to a susceptible sorghum crop.

Fig. 3.13. Infected earhead

Symptoms:

1) The first symptom appears when the plant becomes two months old.
2) The ovaries are attacked and the majority of the grains in an ear are converted into smut sori.
3) Generally the ovary is replaced by an oval or conical dirty-gray sac, which is surrounded by the unaltered glumes at the base. Subtending the sac and the glumes of the spikelets, the smut sorus is covered by a tough membrane and remains intact without rupturing early.
4) The interior of the sorus is completely filled with the spore powder except a slender, sometimes curved, central column of hard tissues.
5) The column is called columella and is hollowed into depressions at the surface. Sometimes, the stamens develop normally, but more often they are either involved in the sorus or are absent.
6) Stamens involved in sorus are represented by three conical protrusions from sides of the sorus.
7) In certain sorghum-varieties, the elongated sacs are not formed and the smutted grains appear that of normal shape and size, and are full of smut powder.
8) This condition easily escapes notice hence acts as a dangerous source of contamination of healthy grains during threshing. In such cases the covering of the sorus is generally reddish.

Causal Organism:

Sphacelotheca sorghi

The mycelium is dikaryotic. The smut spores (teleuto-or chlamydospores) are round to shortly oval, dark brown in mass but brownish-olive when seen individually. Each spore is smooth and measures 5 to 9 (generally 6) pm in diameter. The spores may remain viable for more than five years.

On germination, the 4-celled pro-mycelia are formed, on which terminal and lateral sporidia (basidiospores) are produced, which are spindle-shaped. The secondary sporidia may be produced by budding. In some cases, the pro-mycelium directly develops into a branched or unbranched infection hypha.

Disease Cycle:

Spores of S. sorghi are technically called teliospores. The teliospores are seedborne on a diseased sorghum plant and will germinate within the seed if it is used to plant the following season. Seedling plants become infected, but symptoms generally are not apparent until booting or heading. The pathogen grows within the sorghum as the plant matures into the meristem and thus invades floral tissues. The maturing fungal fruiting structures (sori) rupture and release the

teliospores that infect seeds on other plants. Only seed borne spores cause infection. Teliospores adhere to the surface of seeds where they overwinter.

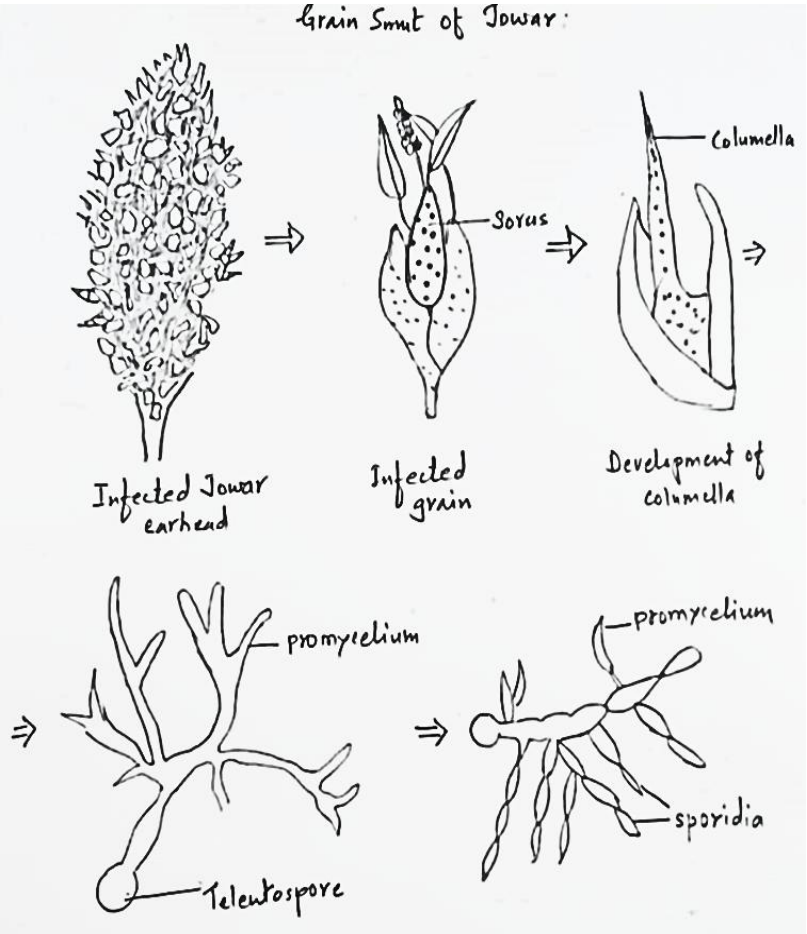


Fig. 3.14. Disease cycle

Management of the disease:

i. Clean seeds selected from cobs free from smut sori should be preferably sown.

ii. Since the disease is externally seed-borne, seed treatment with solar energy and with suitable fungicides are quite effective.

iii. In solar energy treatment, the seeds are soaked in water at ordinary temperature during summer for four hours in the morning and then spread out in the sun or shade to dry. This treatment of seed has been proved effective in Uttar Pradesh.

iv. In fungicidal treatment, steeping the seeds in 0.5-3% formalin for two hours followed by quick drying, in 3% solution of copper sulfate for 15 minutes followed by drying and sowing are very effective recommendations.

v. Dry seed dressing with Agrosan GN (1:500) is simplest and a common practice followed. Sulfur in the form of a fine powder (200 meshes) at 56.5-113.5g/1.6 kg of seed and Thiram

at 2-4g/kg of seed are considerably effective. Other fungicides such as Arasan, Ceresan, Tillex, Capton, Carboxin, and Bavistin have also been used with success.

vi. Resistant varieties such as T 29/1, PJ 7K, PJ 23 K, Nandayal and Bilichigan should be used. Following varieties or sorghum lines have been reported resistant in Maharashtra – CSH-9, SPV-104, SPV-102, SPV-115, SPV-297, SPV-138, SDM-9, RSV-l-R, CHS-7-R, and CHS-5.

vii. A few varieties of Milo, Hegari, Feterita, and Shantung have been found in recent years carrying the genes for resistance. These genes may be exploited by genetic engineers to obtain transgenic plants of sorghum resistant to the disease.

VI) Tikka disease of Groundnut:

Name of the Host: *Arachis hypogea* L.

Causal Organism: The disease is caused by two different pathogens namely *Cercospora arachidicola* and *Cercospora personata.*

Distribution: Widespread in tropical countries where the groundnut crop production is more.

Symptoms:

Cercospora arachidicola cause early leaf spot infection, which includes symptoms like brown-coloured round or oval spots. Yellow halo also appears around the spots in an irregular fashion.Cercospora personata cause late leaf spot infection, which includes symptoms like the appearance of the large and dark black-colored spots on the leaves, stems, seeds and shells of the groundnut.

1. All parts of the host plant above soil level are attacked by the disease.
2. The first visible symptoms appear on the leaflets of lower leaves as dark spots which at a later stage, are surrounded by yellow rings.
3. The spots are circular. They appear in a large number on the leaves.
4. Mature spots are dark-brown to almost black, particularly on the upper surface of the leaflets.
5. Whereas, on the lower surface they are lighter in color.
6. The spots are few on the leaf petioles and stem.
7. Sometimes spots coalesce resulting in defoliation.
8. The shedding of leaves is a characteristic feature of the disease.
9. Due to excessive spotting and consequent leaf fall, smaller and fewer nuts are formed.

10. In cases where young plants are attacked by the disease, nuts fail to develop in them. But the mature plants when attacked by the disease produce immature nuts which are shriveled and become loose in the shell.
11. The total effect is the loss in yield.

Fig. 3.15. Tikka disease of groundnut

Causal Organism/ Pathogen:

The pathogen of Tikka disease of groundnut belongs to the genus Cercospora and typically includes two different species, namely Cercospora arachidicola and Cercospora personata.

- *Cercospora arachidicola* appears dark brown in color and grows intracellular in the plant cell and it lacks haustoria. *Cercospora arachidicola* consists of continuous, unbranched, yellowish-brown, geniculated and septate (1-2 septa present) conidiophore. It is 22-44 µ long and 3-5 µ wide. *Cercospora arachidicola* consists of long, cylindrical, hyaline (Pale yellow in color) conidia. It is 38-108 µ and 6-8 µ wide. The shape of conidia truncates at the base and sub truncates at the apex. Conidia of *Cercospora arachidicola* are septate, and around 1-12 septa are present.
- *Cercospora personata* appears brown-black in color and first grows intercellular and later intracellular and it contains branched haustoria. It consists of continuous, unbranched, light brown in color, geniculated and aseptate conidiophore. It is 25-54 µ long and 5-8 µ wide. It has short, cylindrical conidia. It is 18-60 µ and 6-11 µ wide. The shape of the conidia is rounded at one end and tapered on the other end. The conidia is septate, and around 3-4 septa are present.
- Besides, many differences in the morphology and growth pattern, *Cercospora arachidicola* and *Cercospora personata* also show some similarities.
- Both are pathogenic. The mode of nutrition is saprophytic and parasitic.

Disease Cycle:

The pathogen perennates through conidia on diseased plant debris lying in the soil. The conidia may also remain adhered to the shell. They have also been found to remain associated with

the seeds and are responsible for primary infection. A temperature range of 26°C to 31 °C with high atmospheric humidity is favorable for disease development.

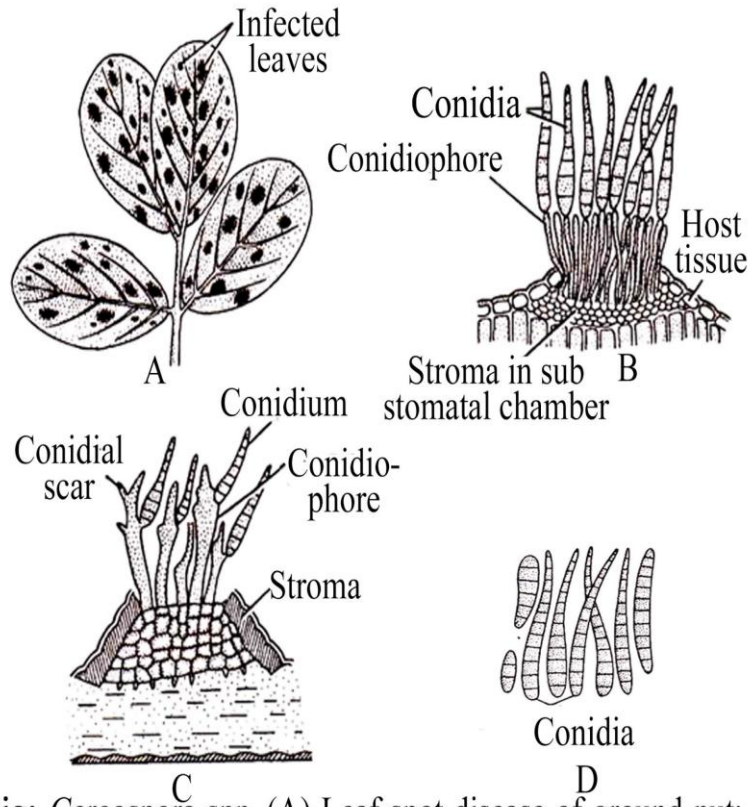


Fig: *Cercospora spp.* (A) Leaf spot disease of ground nut; (B) Conidiophore bearing conidia; (C) LS of acervulus with geniculate conidiophores; (D) Conidia.

Fig. 3.16. Tikka disease of groundnut disease cycle

Prolonged low temperature and dew also favor infection. The entrance of the pathogen in the host tissue takes place either by direct penetration through the epidermal cells or by way of stomata.

The leaf infection is largely through the upper surface of the leaflets. The fungus mycelium ramifies the host tissue in and around the infection court and aggregates underneath the epidermis and forms stroma.

During the development of stroma the epidermis is ruptured by the pressure developed in the host tissue and the conidiophores developed from the stroma emerge out, ultimately conidia are produced on them. These conidia form the secondary inoculum through which secondary infection is induced.

Control Measures/ Management of the disease:

i) As the disease is soil borne, proper crop rotation is important.

ii) Burning of previous year's diseased plant debris will, to a great extent, reduce the source of primary infection.

iii) Avoid late sowing to reduce infection rate.

iv) Growing disease resistant varieties like ICGV-89104, ICGV 91114, ICGV 92093.

v) Seed treatment for half an hour in 0.5 per cent, copper sulfate solution is recommended. Agrosan GN also is an effective disinfectant. Seed dressing with Thiram (1: 350) or Flit 406 (1: 500) before sowing prevents *Aspergillus* seed rot and pre-emergence losses.

vi) Use of carbendazim 0.1 % or mancozeb 0.2 % effectively controls this disease.

vii) Dusting with sulfur (six applications at 10 days interval) or with sulfur containing 3.5 per cent, metallic copper has been found to be very useful for controlling the disease.

viii) Foliar application of aqueous neem leaf extract (2-5%) or 5% neem seed kernel extract at 2 weeks interval after 4 weeks from the planting.

VII) Blight of Marigold:

Name of the Host: *Tagetes erecta* L. and ***Tagetes patula* L.**

Causal Organism: *Botrytis cinerea*.

Symptoms:

1. Flower parts develop water-soaked lesions that become necrotic and die.
2. A gray mass of spores develops on necrotic tissue during wet conditions.
3. Entire petals or flowers may become diseased. Senescent leaves may also be attacked.

Fig. 3.17. Blight of Marigold

Causal organism/ Pathogen:

Botrytis cinerea, a fungus that colonizes dead, dying, and wounded plant parts. From these infections it can attack healthy tissues. A moist, humid environment is ideal for pathogen sporulation and spread. Conidia may come from sources in and outside the greenhouse. It is found everywhere plants are grown and has a wide host range. Flower petals are most susceptible at any stage of development.

Disease cycle:

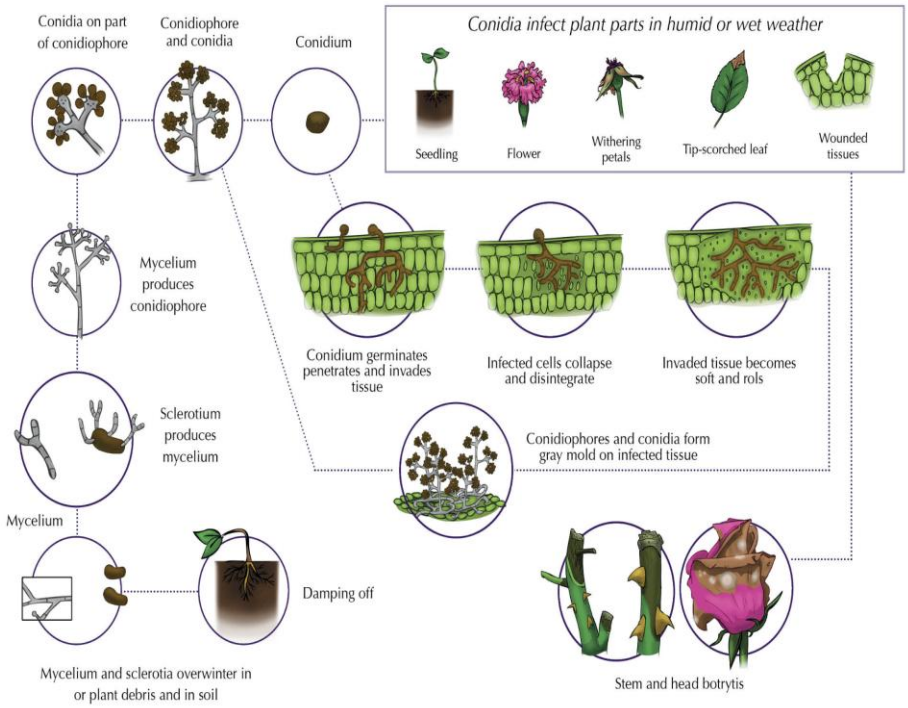


Fig. 3.18. Disease Cycle

Botrytis cinerea persists in the greenhouse year around as (1) mycelium, conidia, or sclerotia on living or dead plants, and as (2) sclerotia or conidia in infested soil. Outdoors, the fungus grows on decayed plant material or in infested soil. In rare cases, a seed lot may be contaminated with sclerotia of the same size as the seed or with bits of plant debris that may carry sclerotia or mycelium. Sclerotia are the main structures for field survival, although conidia may overseason in the field and can survive a temperature range of 40 C to 54 0C. The overwintering stage can be spread by anything that moves soil or plant debris and transports sclerotia, mycelium, or conidia. Upon germination, the sclerotia generally give rise directly to conidia and occasionally to infection hyphae. In some cases, sclerotia of B. cinerea germinate by producing apothecia and ascospores although this type - 4 - of germination is very rare and has been reported to occur from Botrytis isolates on grapes. Greenhouse and field infections commonly result from dispersal of the conidia, which are borne in grapelike clusters (Figure 8). Conidia produced on germinating sclerotia, on infected plants, or on infected plant debris are dispersed in large numbers by air currents to new plants.

Another source of inoculum, with an even higher inoculum potential than conidia, is when diseased flower petals or pieces of Botrytis-infected debris come into contact with healthy tissues. The infected tissue provides a food base, allowing the mycelia of Botrytis to penetrate the underlying green tissue directly upon contact. Botrytis is often considered a cool weather pathogen with best growth, sporulation, spore release, germination, and establishment of infection occurring

at an optimum of 18^0C to 23^0C, however, it is also active at low temperatures and can cause considerable loss of plant material maintained at 0^0C to 10^0C . Once infection occurs, the fungus can grow over a range of 0^0C to 35^0C.

Management of the disease:

Cultural control:

i. Remove all dead and dying plant parts (particularly blossoms) on and around plants.
ii. Avoid overhead irrigation or apply such that plants are not wet for extended periods of time.
iii. Space plants for good air circulation.

Chemical control Best used with cultural controls:

Tank-mix and/or alternate products with different modes of action to prevent the build up of resistant fungi. Limit the use of any one group during crop production.

i. Broadform at 4 to 8 fl oz/100 gal water. Group 7 + 11 fungicide. 12-hr reentry.
ii. Chipco 26019 N/G at 1 to 2.5 lb/100 gal water. Group 2 fungicide. 12-hr reentry.
iii. Copper-Count-N at 1 quart/100 gal water. Group M1 fungicide. 48-hr reentry general or 24-hr reentry for greenhouse.
iv. Decree 50 WDG at 0.75 to 1.5 lb/100 gal water. Group 17 fungicide. 12-hr reentry.
v. Fore 80 WP at 1.5 lb/100 gal water. Do not use French dwarf-double or signet-type marigold seedlings. 24-hr reentry.
vi. Medallion WDG at 2 to 4 oz/100 gal water. Using oils or adjuvants may damage plants. Group 12 fungicide. 12-hr reentry.
vii. Orkestra at 8 fl oz/100 gal water. Group 7 + 11 fungicide. 12-hr reentry.
viii. Pageant at 12 to 18 oz/100 gal water. Group 7 + 11 fungicide. 12-hr reentry.
ix. Phyton 27 at 1.3 to 2 oz/10 gal water. Group M1 fungicide. 48-hr reentry.
x. Spirato GHN at 2 to 4 fl oz/100 gal water. Use with oils or adjuvants may cause plant damage. Group 12 fungicide. 12-hr reentry.
xi. Switch WG at 11 to 14 oz/A. Do not use within 7 days of harvest. Group 9 + 12 fungicide. 12-hr reentry.

Biological control:

i. Prestop (*Gliocladium catenulatum* strain J1446) at 0.33 oz/5 gal water. Do not use it with other products in the tank.

Unit 4 Pathophysiological Skills

Paper Chromatography Technique

Introduction, Principle and Application

Paper chromatography is a technique for separating dissolved chemical substances by taking advantage of their different rates of migration across sheets of paper. It is very powerful analytical tool which requires small quantities of material and so said to be inexpensive technique. It requires a piece of paper or strips serving as an adsorbent in the stationary phase across which a particular solution is allowed to pass. Paper chromatography was discovered by Synge and Martin in the year 1943.

Chromatography is nothing but it is the technique for separating the components, or solutes, of a mixture on the basis of the relative amounts of each solute distributed between a moving fluid stream, called the mobile phase, and a contiguous stationary phase. The mobile phase may be either a liquid or a gas, while the stationary phase is either a solid or a liquid. "Chroma" means color and "Graphy" means arrangement. So one can easily say that chromatography means graphical arrangement of different mixtures with the help of colors.

Principle of Paper Chromatography

Paper chromatography is a form of liquid chromatography where the basic principle involved can be either partition chromatography or adsorption chromatography.

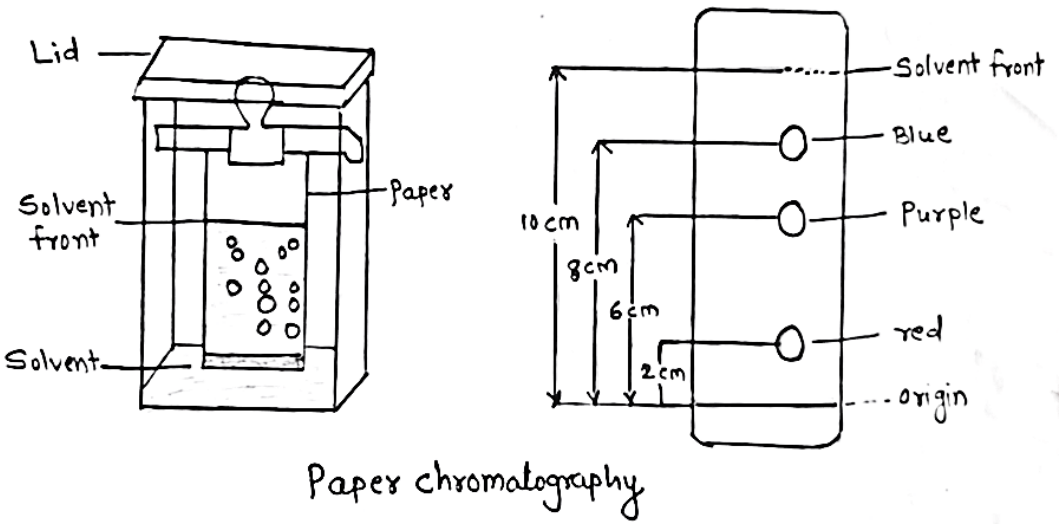


Fig. 4.1 Paper Chromatography

In paper chromatography separation of component is distributed between phases of liquid. Here, one phase of liquid is water that is held amidst the pores of filter paper and the other liquid is the mobile phase that travels along with the filter paper. Separation of the mixture is the result that is obtained from the differences in the affinities towards the water and mobile phase when traveling under capillary action between the pores of the filter paper.

Though in a majority of paper chromatography applications, the principle is based on partition chromatography but sometimes, adsorption chromatography can take place where the stationary phase is the solid surface of the paper and the mobile phase is solvent.

Paper Chromatography Procedure

The procedure is explained to conduct a Paper Chromatography Experiment for easy understanding of students.

1. **Selecting a suitable type of development:** It is decided based on the complexity of the solvent, paper, mixture, etc. Usually ascending type or radial paper chromatography is used as they are easy to perform. Also, it is easy to handle, the chromatogram obtained is faster and the process is less time-consuming.
2. **Selecting a suitable filter paper**: Selection of filter paper is done based on the size of the pores and the sample quality. The paper usually selected for this technique is **Whatman's Filter paper.** Whatman's filter paper is a cellulose paper made by using a high percentage of alpha-cellulose. Its contents are a sign of its consistency and high quality as alpha cellulose is considered the most stable form of cellulose. Not only this, it has the highest degree of polymerization.
3. **Prepare the sample:** Sample preparation includes the dissolution of the sample in a suitable solvent (inert with the sample under analysis) used in making the mobile phase.
4. **Spot the sample on the paper:** Samples should be spotted at a proper position on the paper by using a capillary tube. This step is called spotting or loading of a chromatogram.
5. **Chromatogram development:** Chromatogram development is spotted by immersing the paper in the mobile phase. Due to the capillary action of paper, the mobile phase moves over the sample on the paper. This step is known as running the chromatogram.
6. **Paper drying and compound detection**: Once the chromatogram is developed, the paper is dried using an air drier. Also, detecting solution can be sprayed on the chromatogram developed paper and dried to identify the sample chromatogram spots.

Applications of paper chromatography:

Paper chromatography can be applicable in the different classes of compounds namely:

1) Amino acids and organic acids

2) Alkaloids
3) Polysaccharides
4) Proteins and peptides
5) Natural and artificial pigments
6) Inorganic cations
7) Plant extracts

Paper Chromatography can be applied in different areas which include:

I. **Foods:** Analysis of food colors in synthetic drinks and beverages, ice creams, sweets etc can be done with this technique. Only edible oils are allowed to use in foods that is why identification and quantification of mixed oils can be detected in the foods with the help of this technique.

II. **Isolation and purification:** The purification and isolation of components of mixture can be done with this technique.

III. **Reaction Monitoring:** The reaction progress can be detected by developing the chromatogram over different time intervals by spotting the reactors.

IV. **Pharmaceuticals:** In this sector, the development of new drugs, molecules, reaction completion and progress of manufacturing processes can be detected by using chromatography techniques. The active ingredients present in the drug can be monitored as well as color identification of the drug is possible with the help of paper chromatography.

Types of paper chromatography:

1. Ascending Paper Chromatography – The technique goes with its name as the solvent moves in an upward direction.
2. Descending Paper Chromatography – The movement of the flow of solvent due to gravitational pull and capillary action is downwards, hence the name descending paper chromatography.
3. Ascending – Descending Paper Chromatography – In this version of paper chromatography, movement of solvent occurs in two directions after a particular point. Initially, the solvent travels upwards on the paper which is folded over a rod and after crossing the rod it continues with its travel in the downward direction.
4. Radial or Circular Paper Chromatography – The sample is deposited at the center of the circular filter paper. Once the spot is dried, the filter paper is tied horizontally on a Petri dish which contains the solvent.
5. Two Dimensional Paper Chromatography – Substances which have the same r_f values can be resolved with the help of two-dimensional paper chromatography.

a) Micrometry: Introduction, Principle and Application

Micrometry is the science in which we have some measurement of the dimensions of an object being observed under the microscope. Micrometry is the measurement of microscopic objects (microorganisms) under a microscope which uses two micro- scales known as micrometers.

The method employs some special types of measuring devices which are so oriented that these can well be attached to or put into the microscope and observed. Since microorganisms can be seen only under a microscope, a suitable scale for their measurements should be somewhere in the microscope itself.

There are usually two types of micrometers, i.e. stage micrometer and ocular meter or ocular micrometer. Ocular micrometer is simply a disc of glass upon which etched lines are present. There are usually etched 100 equally spaced divisions, marked 0 to 100 upon an ocular micrometer. However, the scale on the ocular micrometer does not have any standard value. Stage micrometer is simply a microscope glass slide having in its center a known (one millimeter) distance etched into 100 equally spaced divisions. Thus each division of the stage micrometer equals 0.01 mm or 10 µm.

Once we are observing an object under a microscope by the 5X objective and the 10X eyepiece we say that the image that we are able to perceive is 5 × 10 = 50 times of the object. The object, to be measured, is calibrated against these scales.

Principle:

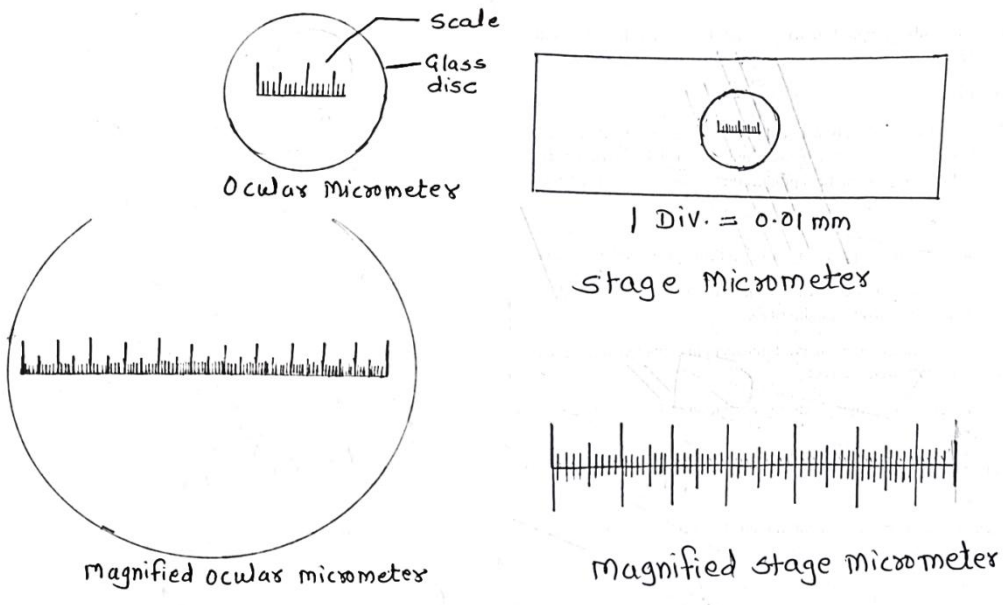


Fig. 4.2: Ocular micrometer and Stage micrometer

The Ocular micrometer is calibrated under different objective lens systems of the microscope by superimposing the graduations of the stage micrometer. By determining how many divisions of an ocular micrometer superimpose a known distance on the stage micrometer, we may find out the exact value of one division of the ocular micrometer in the microscope field. Once calibrated, the ocular micrometer can be used to measure the size of various microbes in terms of length, breadth and diameter.

Procedure:

1. Before starting the experiment, prepare the proper slide of microorganism to be measured for the dimension.
2. Replace the regular ocular lens with an ocular eyepiece lens with an ocular micrometer and observe. There will be seen scale lines of ocular micrometer in sharp focus. These lines and distances will remain unchanged under different objectives.
3. Mount the stage micrometer on the microscope stage and bring its scale in the center of the microscope field under a sharp focus. This is done first with low power objectives and thereafter also with high-power objectives.
4. Adjust the scales of the ocular micrometer and stage micrometer in such a way that the lines of the former superimpose upon those of the latter. If necessary the ocular micrometer may be rotated or stage micrometer be moved on the stage so that lines of ocular micrometer superimpose upon the stage micrometer. The scales of both micrometers are to be adjusted in such a way that the lines of the two coincide at one end of the microscope field. Now count the spaces of each micrometer to a point where the lines of the two micrometers coincide again. In this way we can find out how many divisions of ocular micrometer (unknown scale) are equal to how many divisions of stage micrometer (known scale).
5. Replace the stage micrometer with the slide bearing the microorganisms whose measurements to be taken.
6. Note the divisions occupied by the spore at low power and high power separately and use this reading for further calculations.

Calculation:

Calibration of ocular micrometer for 10 x objective

$$\text{One division of ocular micrometer (C)} = \frac{\textbf{No. of divisions of stage micrometer (A)}}{\textbf{No. of divisions of ocular micrometer (B)}} \textbf{X 10}$$

Readings Taken for calibration:

	A	B	C (C= A/Bx10)
1			
2			
3			
		Mean =	

One division of the ocular micrometer (C) = ------ µm. This is the **factor value** to be used for calculations.

The procedure for calibration of ocular micrometers at low power (10 x) and at high power (45 x) is similar. This procedure is repeated for calibration at high power.

In this way the microscope is calibrated for different combinations of eyepieces and objective lenses and is kept for record. It is to note that this calibration will be just only of the tried lenses on this particular microscope.

Take three readings in this way, and the mean value of these readings will be the actual value of one part of the ocular meter.

Measurement of the Microorganisms:

When the microscope is calibrated, then the object or organism to be measured is kept on the sage of the microscope and is observed through the eyepiece with ocular. The object is measured in the particular magnification by ocular divisions and then is changed into microns by multiplying ocular divisions with the calibrated value of one ocular division in that particular magnification.

After calibration the value obtained is the factor which is to be used in the calculation for measuring dimension of the spore.

Dimension of the spore can be calculated as below:

At low power (10 X) =

	Ocular divisions (A)	Factor (C)	Dimension (A x C)
1			
2			
3			
		Average =	**……..µ**

Similar procedure is applied for the measurement of dimension at high power (45 X) also. Thus, by using the above procedure, the organisms observed under a microscope can be measured.

Sub Unit 4.3

Study of properties, formulation, mode of action and uses of Carbendazim and Benomyl

Carbendazim:

Carbendazim, a systemic benzimidazole fungicide, is applied repeatedly to control plant diseases including soilborne diseases, over a growing season.

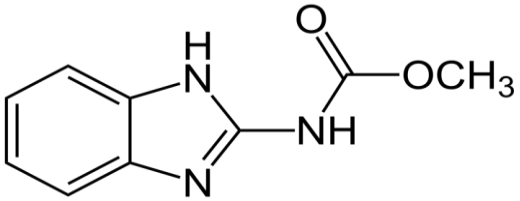
It is also employed as a casting worm control agent in amenity turf situations such as golf greens; tennis courts etc. and in some countries is licensed for that use only.

Carbendazim properties:

Properties	
Chemical formula	$C_9H_9N_3O_2$
Molar mass	191.187 g/mol
Appearance	Light gray powder
Density	1.45 g/cm^3
Melting point	302 to 307 °C (576 to 585 °F; 575 to 580 K) (decomposes)
Solubility in water	8 mg/L Disintegration = 302 -305 degree Temperature of disintegration = 1.5 - 2 hrs
Acidity (pK_a)	4.48

Formulation of Carbendazim:

$C_9H_9N_3O_2$ (Methyl 1 H- benzimidazol- 2 – ylcarbamate)



Carbendazim is a member of the class of benzimidazoles that is 2-aminobenzimidazole in which the primary amino group is substituted by a methoxycarbonyl group. It derives from a 2-aminobenzimidazole.

Mode of action

It is a broad-spectrum systemic fungicide with protective and curative action. It is taken and translocated within the plant as a result of which the latter become fungitoxic.

The exact mechanism of action is unclear; carbendazim appears to bind to an unspecified site on tubulin and suppresses microtubule assembly dynamics. This results in cell cycle arrest at the G2/M phase and an induction of apoptosis.

When applied to the roots, the active ingredient passes Intracellular into the xylem vessels and it is swept along by the sap stream towards the foliage. When applied to the foliage the fungicide gets into the xylem and spreads to the distal parts of the leaf but not in the opposite direction towards the roots.

It mainly acts by inhibiting development of germ tubes, the formation of appressoria and the growth of mycelia.

Uses of Carbendazim:

1. It is used to control plant diseases in cereals and fruit including Citrus, Bananas, Strawberries, Pineapple etc.
2. Mostly used to control *Botrytis*, *Gloeosporium* rots, powdery mildews and apple scab.
3. Carbendazim 50% WP) is a broad spectrum systemic fungicide which effectively controls the diseases of crops.
4. It is used both as curative and preventive for control of diseases in field crops and vegetables.
5. As compared to other fungicides, it is less expensive in the long run with respect to the superior protection and cost per acre.
6. Dhanustin is rapidly absorbed by the plants and translocated in the whole plant. It remains effective even if it rains a few hours after its application.
7. It is also used as

- Foliar Spray
- Seedling Treatment
- Seed Treatment
- Soil Drenching
- Injecting In Trunks
- Post Harvest Treatment

Benomyl:

Benomyl was introduced in 1968 by Dupont. It was synthesized from cyanide and methyl chloroformate. Benomyl is a wide spectrum systemic fungicide It is sold under the trade name Benlate and Tersan. It is Effective against powdery mildew of Cucurbits, Cereals, Pulses. Dose: 0.1 – 0.2 % for foliar spray.

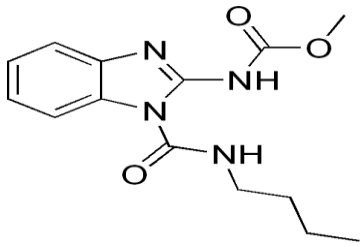
Benomyl is systemic fungicide (Absorbed through the plant). Benomyl is the accepted common name for the fungicide methyl l-(butylcar = bamoyl) -2-benzimidazolecarbamatè. The compound is sold under the trade names of Benlate®, Lignasan® BLP, and Tersan® 1991. Benomyl (MW-290) is a nonvolatile white crystalline solid that is described as insoluble in water and oil (78) "; however, its biological activity implies that it has at least a slight solubility in water.

Properties of Benomyl:

Properties	
Chemical formula	$C_{14}H_{18}N_4O_3$
Molar mass	290.323 g·mol^{-1}
Appearance	white crystalline solid
Odor	acrid
Melting point	290 °C (554 °F; 563 K) decomposes
Solubility in water	0.0004% (20 °C)

Formulation of Benomyl:

The compound has the following structural formula:



Mode of action of Benomyl:

1. Effect on Cell membrane:Fungicides that have cell membranes as their specific site of action. This results in membrane damage and leakage of potassium ions.

2. Effect on enzyme system: The mitochondrial respiration is affected by Oxathiins through inhibition of the necessary enzyme system.
3. Inhibition of protein, RNA & DNA synthesis: Antibiotics are mainly responsible tor inhibition of protein synthesis, metalaxyl and related compounds affects ribosomal RNA of the fungi and interfere with protein synthesis.
4. Nuclear division: The benzimidazole fungicide act as spindle poison binding to the protein subunit of spindle microtubules. It inhibits Mitosis during cell division. E. g. Benomyl.

Uses of Benomyl:

1. Benomyl is the more active compound and is widely applied as a foliar spray, seed dressing or to the soil for control of gray mold (*Botrytis cinerea*), apple scab (*Venturia inaequalis*) canker and powdery mildew (*Podosphaera leucotricha*), leaf spot (*Cercospora beticola*), major fungal diseases of soft fruits and some pathogens of tomato and cucumber.
2. Active against many pathogenic fungi including powdery mildews and soil borne pathogens, *Verticillium albo atrum* on Cotton and black spot-on roses.
3. Benomyl is a benzimidazole compound with a carbamate moiety but has no activity as a cholinesterase inhibitor.
4. It is used in the control of many diseases of fruits, nuts, vegetables, and ornamental plants.

Sub Unit 4.2 Chemical Method

Classification of fungicides based on chemical nature and mode of action.

A fungicide is a chemical that is used for managing or controlling the fungi causing diseases in plants. In other words we can say that " A fungicide is any substance, preparation or organism used for destroying or controlling any fungal species which attacks agricultural plants during production, storage or distribution. The word fungicide originated from two a Latin words; "Fungus" means fungi and "Caedo" means to kill. Thus the fungicide is any agency or chemical which has the ability to kill the fungus. In common usage, the fungicide word is restricted to chemicals which are capable of killing fungi.

Classification of fungicides Based on Chemical Composition/Chemical nature:

The chemical available for plant disease control runs into hundreds, however, all are not equally safe, effective and popular. Major group of fungicides used include salts of toxic metals and organic acids, organic compounds of sulfur and mercury, quinones and heterocyclic nitrogenous compounds. Copper, mercury, zinc, tin and nickel are some of the metals used as base

for inorganic and organic fungicides. The non metal substances include, sulfur, chlorine, phosphorus etc. The fungicides can be broadly grouped as follows and discussed in detail.

Classification based on Chemical nature:

1. Sulfur fungicide:

Inorganic- e.g. powdered sulfur, wettable sulfur, lime sulfur etc.

Organic- e.g. ferbam, ziram, thiram, zineb, maneb, nabam, vapam etc.

2.Copper fungicides:

e.g. Bordeaux mixture, Burgundy mixture, copper oxychloride etc.

3. Mercurial fungicides:

Inorganic- e.g. mercuric and mercurous chloride etc.

Organic- e.g. phenyl mercury acetate, methoxy ethyl mercury chloride etc.

4. Heterocyclic nitrogenous compounds:

e.g. captain, folpet etc.

5. Quinone compounds:

e. g. chloranil, dichlone etc.

6. Oxathiin compounds:

e.g. carboxin, oxycarboxin etc.

7. Benzimidazoles/Carbendazim group:

e. g. benomyl, MBC etc.

8. Miscellaneous group:

e.g. PCNB, dodine, antibiotics etc.

All these different types and subtypes of fungicides have been discussed as below according to their uses and importance:

A) Copper Fungicides

The fungicidal action of copper was mentioned as early as 1807 by Prevost against wheat bunt disease *(Tilletia caries)*, but its large-scale use as a fungicide started in 1885 after the discovery of Bordeaux mixture by Millardet in France. The mixture of copper sulfate and lime was effective in controlling downy mildew of grapevine caused by *Plasmopara viticola* and later, late blight of potato *(Phytophthora infestans).*

Some other copper sulfate preparations later developed were Bordeaux paste, Burgundy mixture and Chestnut compound which are all very effectively used in the control of several plant diseases. In addition, some preparations of copper oxy chloride preparations are also used. These are all insoluble copper compounds very successfully used in managing several leaf diseases and

seeding diseases in nursery. Some of the important diseases controlled by copper fungicides are listed below:

Copper sulfate Preparations

Bordeaux Mixture:

In 1882, Millardet in France (Bordeaux University) accidently observed the effect of the copper sulfate against the downy mildew of grapes caused by *Plasmopara viticola.* When copper sulfate was mixed with lime suspension, it effectively checked the disease incidence. The mixture of copper sulfate and lime was named "Bouillie Bordelaise" (Bordeaux mixture). The original formula developed by Millardet contains 5 lbs of CuSO4 + 5 lbs of lime + 50 gallons of water. The chemistry of Bordeaux mixture is complex and the suggested reaction is:

$CuSO4 + Ca(OH)_2$ $Cu(OH)_2 + CaSO_4$

The ultimate mixture contains a gelatinous precipitate of copper hydroxide and calcium sulfate, which is usually sky blue in color. Cupric hydroxide is the active principle and is toxic to fungal spores. In metric system, to prepare one percent Bordeaux mixture the following procedure is adopted:

One kg of copper sulfate is powdered and dissolved in 50 liters of water. Similarly, 1 kg of lime is powdered and dissolved in another 50 liters of water. Then copper sulfate solution is slowly added to the lime solution with constant stirring or alternatively, both the solutions may be poured simultaneously to a third contained and mixed well.

The ratio of copper sulfate to lime solution determines the pH of the mixture. The mixture prepared in the above said ratio gives neutral or alkaline mixture. If the quality of the used is inferior, the mixture may become acidic. If the mixture is acidic, it contains free copper which is highly phytotoxic resulting in scorching of the plants. Therefore, it is highly essential to test the presence of free copper in the mixture before applying. There are several methods to test the neutrality of the mixture, which are indicated below:

Field Test: Dip a well polished knife or a sickle in the mixture for a few minutes. If a reddish deposit appears on the knife/sickle, it indicates the acidic nature of the mixture.

Litmus paper test: The color of blue litmus paper must not change when dipped in the mixture.

pH paper test: If the paper is dipped in the mixture, it should show neutral pH.

Chemical test: Acid a few drops of the mixture into a test tube containing 5 ml of 10% potassium ferrocyanide. If red precipitate appears, it indicates the acidic nature of the mixture.

If the prepared mixture is in the acidic range, it can be brought to neutral or near alkaline condition by adding some more lime solution into the mixture. Bordeaux mixture preparation is cumbersome and the following precautions are needed during preparation and application.

The solution should be prepared in earthen or wooden or plastic vessels. Avoid using metal containers for the preparation, as it is corrosive to metallic vessels.

i. Always copper sulfate solution should be added to the lime solution, reverse the addition leads to precipitation of copper and resulted suspension is least toxic.
ii. Bordeaux mixture should be prepared fresh every time before spraying. In case, the mixture has to be stored for a short time or a day, jaggery can be added at the rate of 100kg/100 liters of the mixture.
iii. Bordeaux mixture is sometimes phototoxic to apples, peaches, rice varieties like IR8 and maize varieties like Ganga Hybrid 3.

Bordeaux paste

Bordeaux Paste consists of the same constituents as that of Bordeaux mixture, but it is in the form of a paste as the quantity of water used is too little. It is nothing but 10 percent Bordeaux mixture and is prepared by mixing 1 kg of copper sulfate and 1 kg of lime in 10 liters of water. The method of mixing solution is similar to that of Bordeaux mixture. It is a wound dresser and used to protect the wounded portions, cut ends of trees etc., against the infection by fungal pathogens.

Burgundy mixture

It is prepared in the same way as Bordeaux mixture, except the lime is substituted by sodium carbonate. So it is called "Soda Bordeaux". It was developed in Burgundy (France) in 1887 by Mason. The usual formula contains 1 kg of copper sulfate and 1 kg of sodium carbonate in 100 liters of water. It is a good substitute for Bordeaux mixture and used in copper-sensitive crops.

B) **Sulfur fungicides**

Use of sulfur in plant disease control is probably the oldest one and can be classified as inorganic sulfur and organic sulfur. Inorganic sulfur is used in the form of elemental sulfur or as lime sulfur. Elemental sulfur can be either used as dust or wettable sulfur, later being more widely used in plant disease control. Sulphur is best known for its effectiveness against powdery mildew of many plants, but also effective against certain rusts, leaf blights and fruit diseases.

Sulfur fungicides emit sufficient vapor to prevent the growth of the fungal spores at a distance from the area of deposition. This is an added advantage in sulfur fungicides as compared to other fungi toxicants.

Organic compounds of sulfur are now widely used these days. All these compounds, called "carbamate fungicides", are derivatives of Dithiocarbamic acid, Dithiocarbamates are broadly grouped into two, based on the mechanism of action.

Inorganic sulfur:

Elemental Sulfur:It is a dust prepared by fine grinding of the mineral. The effect of fungicide depends upon the particle size which should be within the range of 47-75 microns (200-300 mesh). It is generally used to control powdery mildews of fruits, vegetables, flowers and tobacco. This is also effective against apple scab (Venturia inaequalis) and rusts of field crops.

Wettable sulfur: It can be suspended in water and used as a spray. It is used for control of powdery mildews and rusts in field and garden crops.

Lime Sulfur (Calcium polysulfide): It can be prepared by boiling 9 Kg or rock lime and 6.75Kg of sulfur in 225 liters of water. Lime Sulphur is effective against powdery mildews as a protective fungicide.

Organic Sulfur:

The organic compounds of sulfur are highly effective and popular fungicides. These fungicides are derivatives of dithiocarbamic acids. It is used to protect foliage and fruits of a wide range of crops.

1. Zineb (Zinc ethylene bisdithiocarbamate): It is used against diseases such as early and late blight of potato and tomato, downy mildews and rusts of cereals, blast of rice, fruit rot of chili etc.
2. Maneb: These are protective fungicides used to control many fungal diseases of field crops, fruits, nuts, ornamentals and vegetables, especially blights of potatoes and tomatoes, downy mildews of vines, anthracnose of vegetables and rusts of pulses.
3. Nabam (DSE) (Disodium ethylene bis dithiocarbamate): Nabam is primarily used for foliar application against leaf spot pathogens of fruits and vegetables. Soil applications were also reported to have a systemic action on *Pythium, Fusarium* and *Phytophthora*. It is also used to control algae in paddy fields.
4. Ziram (Zinc dimethyl dithiocarbamate) : Ziram is a protective fungicide for use on fruit and vegetables crops against fungal pathogens including apple scab. It is non phytotoxic except to zinc sensitive plants. It is highly effective against anthracnose of beans, pulses, tobacco and tomato, and also against rusts of beans etc.
5. Thiram (Tetramethylthiuram disulphide): It is used for seed treatment both as dry powder and as slurry. It is a protective fungicide also suitable for application to foliage to control *Botrytis spp.* On lettuces, ornamental, soft fruits and vegetables rust on ornamentals and *Venturia pirina* on pears. It is also effective against soil borne pathogens like *Pythium, Rhizoctonia* and *Fusarium.*

C) Mercury Fungicides

Mercury fungicides can be grouped as inorganic and organic mercury compounds. Both the groups are highly fungitoxic and were extensively used as seed treatment chemicals against seed borne diseases. Ignorance compounds show bactericidal properties also. However, due to their residual toxicity in soil and plants and their extreme toxicity nature to animal and human beings, the use of mercury fungicides is being discouraged. In most of the countries, the use of mercury fungicides is banned and in countries like India, the use of mercury fungicides is restricted only in seed treatment for certain crops. The list of diseases against which mercury fungicides used are listed below

I. Inorganic Mercury

1. Mercuric chloride: It is used for treating potato tubers and propagative materials of other root crops.
2. Mercurous chloride: Mercurous chloride is limited to soil application in crop protection use because of its phytotoxicity.

II. Organomercurials

1. Methoxy ethyl mercury Chloride:
2. Phenyl mercury chloride:
3. Ethyl Mercury Chloride:

These all are used mainly for treatment of seeds and planting materials. These fungicides are used for seed treatment by dry, wet or slurry method. For seed treatment 1% metallic mercury is applied at 0.25% concentration.

D) Heterocyclic Nitrogen Compounds

Heterocyclic nitrogen compounds are mostly used as foliage and fruit protectants. Some compounds are very effectively used as seed dressers. Some of the commonly used fungicides are listed below:

1. Captan (Kittleson‟s Killer) (N-trichloromethyl thio-4- cyclohexence-1,2- dicarboximide): It is a seed dressing fungicide use to control diseases of many fruits, ornamental and vegetable crops against rots and damping off.

2. Folpet (Folpet) [N- (trichloromethyl-thi)] phthalimide: It is also a protective fungicide used mainly for foliage application against leaf spots, downy and powdery mildews of many crops.

E) Benzene compounds

Many aromatic compounds have important anti-microbial properties and have been developed as fungicides. Some important benzene compounds commonly used in plant disease control are listed below:

1. Quintozene (PCNB): It is used for seed and soil treatment. It is effective against *Botrytis*, *Sclerotium*, *Rhizoctonia* and *Sclerotiniaspp.*
2. Dichloran: It is a protective fungicide and very effective against *Botrytis, Rhizopus and Sclerotinia* spp.
3. Dinocap (2,4-dinitro-6- octyl phenyl crotonate): It is a non-systemic acaricide and control fungicide recommended to control powdery mildews on various fruits and ornamentals. It is also used for seed treatment.

F) Quinone Fungicides

Quinone are present naturally in plants and animals and they exhibit antimicrobial activity and some compounds are successfully developed and used in the plant disease control. Quinones are very effectively used for seed treatment and two commonly used fungicides are listed below:

1. Chloranil (2,3,5,6- tetrachloro-1,4-benzoquinone) : Chloronil is mainly used as a seed protectant against smuts of barely and sorghum and bunt of wheat.
2. Dichlone (2,3-dichloro- 1,4- naphthoquinone): Dichlone has been used widely as seed protectant. This is also used as a foliar fungicide, particularly against apple scab and peach leaf curl.

G) Systemic Fungicides:

Since the late 1960s there has been substantial development in systemic fungicides. Any compound capable of being freely translocated after penetrating the plant is called systemic. A systemic fungicide is defined as a fungitoxic compound that controls a fungal pathogen remote from the point of application, and that can be detected and identified. Thus, a systemic fungicide could eradicate established infection and protect the new parts of the plant.

Several systemic fungicides have been used as seed dressing to eliminate seed infection. These chemicals, however, have not been very successful in the cases of trees and shrubs. On the basis of chemical structure, systemic fungicides can be classified as Benzimidazoles, Thiophanates, Oxathilins and related compounds, pyrimidines, morpholines, organo-phosphorus compounds and miscellaneous groups.

H) Oxathilin and related compounds

Oxathalins were the earliest developed compounds. This group of systemic fungicides is also called carboxamides, carboxylic acid anilide, carboxaanillides or simply as anillides which are effective only against the fungi belong to *Basidiomycotina* and *Rhizoctonia solani.* Some of the chemicals developed are (i) Carboxin (DMOC: 5,6 - dihydro-2-methyl-1, 4- oxathin-3-carboxanillide) and (ii) Oxycarboxin (DCMOD- 2,3-dihydro-5-carboxanillido-6- methyl-1, 4 oxathilin-4, 4, dioxide). The diseases controlled by these chemicals are listed below.

1. Carboxin (5,6-dihydro- 2- methyl-1-4-oxanthin-3- carboxylic): It is systemic fungicide used for seed treatment of cereals against bunts and smuts, especially loose smut of wheat.
2. Oxycarboxin (5,6- dihydro-2-methyl- 1,4- oxathiane-3 carboxianilid- 4,4- dioxide): It is a systemic fungicide used for the treatment of rust diseases of cereals, pulses, ornamentals, vegetables and coffee.

I) Benzimidazoles

The chemicals of this group show a very broad-spectrum activity against a variety of fungi. However, they are not effective against bacteria as well as fungi belonging to *Mastigomycotina*. Two types of fungicidal derivatives of benzimidazoles are known. The first type of derivatives includes fungicides such as thiabendazole and fuberidazole. The fungicidal moiety of the second type is methyl-2-benzimidazole carbamate (MBC). The fungicides of this group may be simple MBC such as carbendazim or a complex from such as benomyl, which transforms into MBC in plant system. Some of the important diseases controlled by these compounds are shown below:

Benomyl (Methyl – 10 (butyl carbamoyl)-2 benzimidazole carbamate): It is a protective and eradicative fungicide with systemic activity, effective against a wide range of fungi affecting field crops, fruits and ornamentals.It is very effective against rice blast, apple scab, powdery mildew of cereals, rose, cucurbits and apple and Diseases caused by Verticillium and Rhizoctonia. It is also used as pre-and postharvest sprays of dips for the control of storage rots of fruits and vegetables. Carbendazim is a systemic fungicide controlling a wide range of fungal pathogens of field crops, fruits, ornamentals and vegetables. It is used as spray, seedling dip, seed treatment, soil drench ad as post harvest treatment of fruits. It is very effective against wilt diseases especially, banana wilt. It effectively controls the sigatoka leaf spot of banana, turmeric leaf spot and rust diseases in many crops.

B. Classification based on mode of action:

i. Protectants:

The chemicals that are applied before infection/disease is established are called as proteants. e. g. Sulphur, Captan, Thiram, Zineb, Mancozeb etc.

These fungicides have two sub groups:

a. Contact: The chemicals that target the fungus at rest either before or after presence on the host. Fungicides called eradicants are grouped in contact fungicides. In simple terms, Eradicants are those fungicides that remove pathogens from an infection court. e.g., Copper, lime sulfur etc.

b. Residual: Chemicals that are applied as a stationary layer on the host so as to stop the mobile fungus. E.g. Dithiocarbamate fungicides like Zineb, Maneb.

ii. Therapeutants:

Chemicals that are applied after appearance of infection/disease to cure the disease by killing the pathogen are known as therapeutants.

e.g. Oxathiins, benzimidazoles, Bordeaux mixture, antibiotics etc.

iii. Systemic:

Chemicals that are absorbed by the treated plants and distributed or translocated to the different parts of the plant are systemic fungicides. The sites of action of these fungicides are different from the site of application.

e .g. Oxathin, benzimidazoles, Metalaxyl, thiophanate, Antibiotics etc.

iv. Non-systemic:

These fungicides do not show systemic nature. They are mainly contact and residual and limited to the sprayed parts only.

e .g. Sulfur, copper Quinone, heterocyclic nitrogenous group etc.

Sub Unit 4.1

Mechanical Method: Eradication

Plant diseases have caused severe losses to humans in several ways. The goal of plant disease management is to reduce the economic and related damages caused by plant diseases. Traditionally, this has been called plant disease control, but current social and environmental values changed this word to management due to different views regarding disturbances in food chain and environmental affected parts. Single, often severe, measures, such as pesticide applications, soil fumigation or burning are no longer in common use. Further, disease management procedures are frequently determined by disease forecasting or disease modeling rather than on either a calendar or prescription basis. Disease management might be viewed as proactive whereas disease control is reactive, although it is often difficult to distinguish between the two concepts, especially in the application of specific measures.

Eradication is one of the methods used for disease management without disturbing the environment or food chain. Eradication is the term used to describe the process of removing all infected plant parts after the outbreak of either a new disease in an area or an old disease in a new area. In simple language eradication means the elimination. Elimination of a disease refers to the deliberate effort that leads to the reduction to zero of the incidences of infection caused by a specific agent in a defined geographic area.

This principle aims at eliminating a pathogen after it is introduced into an area but before it has become well established or widely spread. It can be applied to individual plants, seed lots, fields or regions but generally is not effective over large geographic areas.

Eradication of the golden nematode involved removing infested soil, fumigating soil in infested fields and eventually abandoning infested potato fields for housing developments and other uses. Citrus canker eradication involved widespread removal and burning of diseased trees and, in some cases, destruction of entire citrus groves and nurseries. The disease appeared to be contained and the pathogen eradicated, but the disease has reappeared and new attempts at eradication are ongoing.

Following are some of the practices discussed which include eradication of pathogens:

1. Eradication of alternate or collateral host:

Eradication can also be on a more modest scale such as the removal of apple or pear branches infected by the fire blight bacteria (Erwinia amylovora) or pruning to remove blister rust cankers (caused by Cronartium ribicola) on white pine branches. Or, it can be the sorting and removal of diseased flower bulbs, corms or rhizomes. Hot water seed-treatment of cereal seeds to kill smut mycelium in the seed and heat treatment to eliminate viruses from fruit tree budwood for grafting are other examples of pathogen eradication.

Two programs that are actually forms of protection and not pathogen eradication are barberry eradication for reducing stem rust (caused by Puccinia graminis) of wheat and Ribes eradication for preventing white pine blister rust. The strategy is that removing these alternate hosts breaks the disease cycles and prevents infection of the economically more valuable host. These two examples are mentioned here because they are frequently cited as eradication measures. However, stem rust can readily spread from wheat to wheat in many regions by the uredinial stage although elimination of the aecial host, barberry, may deter or diminish the development of pathogenic races of the rust. The white pine blister rust fungus is perennial in the pine host and eradication of the alternate host only protects non-infected trees but does not necessarily eliminate the pathogen from the area.

Eradication may also be accomplished by destroying weeds that are reservoirs of various pathogens or their insect vectors. Elimination of potato cull piles is an effective method of eradicating overwintering inoculums of the late blight pathogen.

2. Eradication of Soil borne pathogens:

Soil fumigation has been a widely used eradication strategy. This technology involves introducing gas-forming chemicals such as carbon disulfide, methyl bromide, or chloropicrin into soil to kill target pathogens. However, undesirable side effects such as killing beneficial

organisms, contamination of groundwater, and toxicity of these chemicals have resulted in less reliance on this approach for disease management. Volatile fumigants like methyl bromide are injected into soil and sealed with a plastic film. Some water-soluble fumigants like metam-sodium can be injected into the soil and the soil simply compacted to form a seal.

3. Eradication through Crop Rotation:

Crop rotation is a frequently used strategy to reduce the quantity of a pathogen, usually soil-borne organisms, in a cropping area. Take-all of wheat (caused by Gaeumannomyces graminis) and soybean cyst nematode (Heterodera glycines) are two examples of soil borne diseases that are easily managed by short rotations of 1 and 2 years, respectively, out of susceptible crops, which may include susceptible weed hosts such as grasses in the case of take-all.

4. Eradication by burning:

Burning is an effective means of eradicating pathogens and is often required by law to dispose of diseased elm trees affected by Dutch elm disease (DED), citrus trees infected by citrus canker or of bean fields infected by halo blight bacteria (Pseudomonas syringae pv. phaseolicola). Propane flaming can effectively destroy Verticillium microsclerotia in mint stems, and flaming potato stems prior to harvest may prevent tuber infection by the late blight pathogen. However, burning agricultural fields is controversial because the smoke creates human health and safety and environmental concerns.

5. Eradication of seed borne inoculum:

Many pathogens are internally or externally seed borne. If the inoculum is allowed to survive it produces the disease in the next season. The fungal spore or inoculums may be attached to the seed externally. Seed dressing can remove such an external infection if done before sowing the seed. Internally seed borne infection can be deep seated in the endosperm or embryo and such a deep-seated infection may be eradicated by hot water treatment at 54 0 C or using solar energy.

6. Eradication through Field Sanitation:

The remaining parts of the plant like stem stubbles, leaves, and flowers after harvest are the best source for the pathogen to survive during off season. Pathogens may remain hidden in the soil on this debris and may infect in the next season. Eradication of such plant debris is called field sanitation. Various measures which can be adopted to destroy these sources are as:

a) Removal of diseased plant debris and its burning.
b) Ploughing to bury the fallen diseased leaves, twigs etc deep in the soil.
c) Use of chemicals to disinfect the fallen plant debris.
d) Hot weather deep ploughing.

Question Bank

Q. 1: Rewrite the following sentences by choosing appropriate alternatives (MCQs). **(1 Mark each)**

1) The establishment of pathogen within the host is called --------

a) Infection b) Inoculation c) Isolation

2) When disease occurs consistently in a particular area, is called as ------- disease.

a) Sporadic b) Epidemic c) Epiphytotic

3) The citrus canker disease is a ------- disease.

a) Viral b) Mycoplasmal c) Bacterial

4) The capacity of the host to withdraw attack of pathogen is known as -----

a) Susceptibility b) Resistance c) Hypersensitivity

5) -------- is the capacity of the pathogen to cause a disease.

a) Pathogenicity b) Pathogenesis c) Incubation period

6) The excessive multiplication of cell due to infection is ------

a) Hypoplasia b) Hypersensitivity c) Hyperplasia

7) Blight of marigold is caused due to ------ pathogen.

a) *Erysiphea cicoracearum* b) *Botrytis cinere* c) *Cercospora arachidicola*

8) Development of black sooty mass in the grains is the symptom observed in ------- type of disease.

a) Rust b) Smut c) Powdery mildew

9) -------- is a phytoplasmal disease.

a) Rust of soybean b) Yellow vein mosaic of bhendi c) Little leaf of brinjal

10) Tikka disease of groundnut is caused by----------

a) *Piccinia graminis triticii* b) *Plasmopara viticola* c) *Cercospora arachidicola*

11) Separation of pathogen from host tissue is called as -------

a) Inoculation b) Isolation c) Incubation

12) Successful invasion of pathogen inside the host body is known as ------

a) Infection b) Pathogenesis c) Etiology

13) The chemical control used against plant disease is based on principle of --------.

a) Resistance b) Exclusion c) Protection

14) The gynoecium part of the flower is converted into sorus in _____ disease symptom.

a) Rusts b) Powdery mildews c) Smuts

15) The _____ pathogen is responsible for causing Citrus canker disease.

a) *Erisiphe cicoracearum* b) *Xanthomonas compestris* c) *Pseudomonas*

16) Little leaf of brinjal is _____ type of disaease.

a) Bacterial b) Viral c) Phytoplasmal

17) *Albugo candidus* is the causal organism of _____ disease.

a) Downey mildew of grapes b) White rust of Crucifer c) Powdery mildew of crucifer

18) _____ is the chain of events followed after entry of pathogen inside the host body.

a) Pathogenesis b) Pathogenicity c) Incubation period

19) The ability of plant to prevent entry and development of pathogen is known as _____

a) Susceptibility b) Resistance c) Immunity

20) _____ is the infectious material which causes disease, when come in contact with suitable material.

a) Inoculum b) Disease cycle c) Pathogen

21) The study of the causal organism is called as _____

a) Etiology b) Pathogenicity c) Immunity

22) Hyperplasia is a _____ multiplication of the cells due to infection.

a) Less b) Excessive c) Normal

23) The time period between penetration and development of first sign of disease symptom is__

a) Inoculum b) Infection c) Incubation

24) The *Xanthomonas* causes infection in Citrus plant is _____

a) Smut b) Rust c) Canker

25) Zineb, Maneb, Nabam are the examples of _____ fungicides.

a) Sulphur b) Copper c) Mercury

26) Use of chemicals in plant disease control is based on principal of _____

a) Protection b) Eradication c) Exclusion

27) _____ is a primary infectious material which causes disease, when come in contact with suitable host.

a) Disease Cycle b) Inoculum c) Pathogen

28) The separation of pathogen from the host is known as _____

a) Incubation b) Isolation c) Inoculation

29) _____ is the capacity of the pathogen to cause a disease.

a) Pathogenesis b) Pathogenicity c) Incubation period

30) _____ is the causal organism of white rust of crucifer disease.

a) *Albugo candidus* b) *Puccinia purpurea* c) *Erysiphae cicoracearum*

31) A successful entry of the pathogen inside the host body is known as _____

a) Etiology b) Pathogenesis c) Infection

32) _____ mixture is a fungicide also called as “Soda Bordeaux”.

a) Bordeaux b) Burgandi c) Benomyl

33) Viral diseases in plants are mostly transmitted by _____

a) Water b) Air c) Insects

34) The symptoms produced due to extra development of tissues is known as _____

a) Hyperplasia b) Hypertrophy c) Hypoplasia

35) Grain smut of jowar is caused by _____

a) *Ustilago scitaminea* b) *Albugo candidus* c) *Puccinia graminis triticii*

36) The spread of the disease in a particular area is known as _____ type of disease.

a)Sporadic b) Epidemic c) Endemic

37) The separation and culture of the pathogen in vitro condition is _____

a) Incubation b) Isolation c) Inversion

38) _____is a systemic, benzimidazole, broad-spectrum, fungicide with protective and curative action.

a) Carbendazim b) benomyle c) 2,4-D

39) Benomyl is wide spectrum systemic fungicides It is sold under the trade name Benlate and Tersan.

a) Benomyl b) carbendazim c) sulphur

40) The capacity of the pathogen to cause a disease in the host body is _____

a) Pathogenecity b) pathogenesis c) inoculum potential

41) The *Xanthomonas* produce disease in citrus is known as _____

a) Rust b) Canker c) Smut

42) Yellow mosaic of bhendi is _____ disease.

a) Bacterial b) Viral c) Mycoplasmal

43) The time period between penetration and development of first sign of disease symptom is __.

a) infection b) Incubation c) inoculum

44) The study of causal organism is called as _____.

a) Immunity b) Etiology c) Pathogenicity

45) Rust of soybean is caused by _____

a) *Puccinia graminis* b) *Phakospora pachyrhiza* c) *Cercospora personata*

46) The pathogens which are transferred through wind are _____

a) Air Borne b) Soil borne c) Seed borne

47) Signs produced after infection of pathogen _____

a) Symptoms b) scratches c) wounds

48) Which one of the following is a phytoplasmal disease?

a) Leaf curl of chilli b) Little leaf of brinjal c) Wilt of potato

49) Major factor of successful infection _____

a) Temperature b) Humidity c) Light

50) In paper chromatography, the basic principle involved is _____ chromatography.

a) partition b) joined c) partial

51) _____is nothing but it is the technique for separating the components, or solutes, of a mixture.

a) Micrometry b) Chromatography c) Microtomy

52) In a disease, _____ is the organism that supports the activities of causal organism.

a) host b) Pathogen c) inoculum

53) Vein clearing is a chief symptom of _____ disease.

a) Viral b) MLO c) Fungal

54) Cleistothecia – sexually reproducing bodies are found in _____ symptom.

a) Downy mildew b) Powdery mildew c) Grain smut

55) Floral parts are modified into minute leaf like structures in ____ symptoms.

a) Little leaf b) Phyllody c) Mildew

56) The paper usually selected for paper chromatography technique is _____

a) Whatman's Filter paper b) blotting paper c) gel filter paper

57) _____ is the science in which we have some measurement of the dimensions of an object being observed under the microscope.

a) Micrometry b) Microtomy c) Microscope

58) _____ is a heterocyclic nitrogen compound used as seed dressing fungicide.

a) Nabam b) Captan c) Ziram

59) A _____fungicide is defined as fungitoxic compound that controls a fungal pathogen remote from the point of application, and that can be detected and identified.

a) systemic b) seed dressing c) surface applied

60) Chemicals that are applied after appearance of infection/disease to cure the disease by killing the pathogen are known as _____

a) therapeutants b) contact c) residual

Long Questions (10 Marks)

1) Describe causal organism, symptoms and methods of management of grain smut of jowar and white rust of crucifers.
2) Describe causal organism, symptoms and methods of management of rust of sugarcane and rust of soybean.
3) Describe causal organism, symptoms and methods of management of rust of wheat and Tikka disease of groundnut.
4) Describe causal organism, symptoms and methods of management of blast of marigold and grain smut of jowar.
5) Describe causal organism, symptoms and methods of management of little leaf of brinjal and yellow vein mosaic of okra.
6) Describe causal organism, symptoms and methods of management of citrus canker and little leaf of brinjal.
7) Define disease? Add a note on symptoms, infection, etiology and immunity.
8) Describe definition and concept of disease.
9) Define disease and explain classification of diseases based on severity of disease.
10) Give classification of diseases based on transmission of pathogen and symptoms, with examples.
11) What is isolation? Give different methods of plant pathogen isolation.
12) Describe mechanism of infection in brief.
13) Define diseases? Add a note on factors affecting infection.
14) In brief explain mechanical method of disease management.
15) What are copper fungicides? Explain any two copper fungicides.
16) What are sulphur fungicides? Explain any two sulphur fungicides.
17) What are mercury fungicides? Explain any two mercury fungicides.
18) Explain mode of action and uses of carbendazime.
19) Explain mode of action and uses of benomyl.
20) Give in short applications of paper chromatography.

Q. 3: Write short notes (5 Marks)

1) Disease concept
2) Pathogen, pathogenecity and pathogenesis
3) Severity of diseases

4) Classification of diseases based on pathogens
5) Isolation
6) Incubation
7) Inoculation
8) Any two factors affecting infection
9) Little leaf of brinjal
10) Yellow vein mosaic of bhendi/Okra
11) Citrus canker
12) Rust of sugarcane
13) Rust of soybean
14) Rust of wheat
15) Blight of marigold
16) Eradication
17) Sulphur fungicides
18) Copper fungicides
19) Mercury fungicides
20) Carbendazime
21) Benomyl
22) Procedure of the paper chromatography
23) Applications of the paper chromatography
24) Principle of Micrometry
25) Applications of Micrometry

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B. Sc. II Semester - IV

A TEXTBOOK OF PLANT PROTECTION: Paper IV (DSC ID 46)

CROP DISEASES, THEIR MANAGEMENT AND PATHOPHYSIOLOGICAL SKILLS

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