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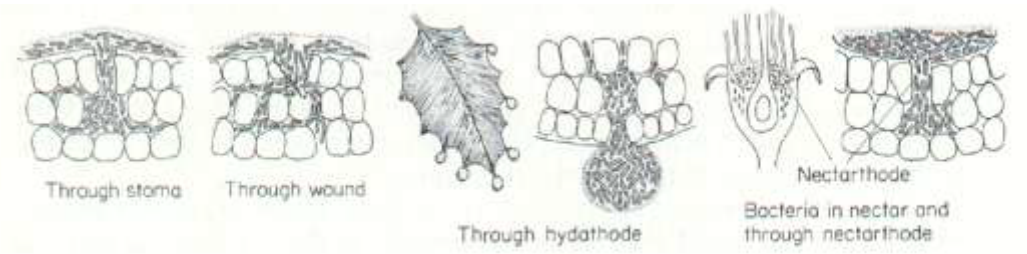

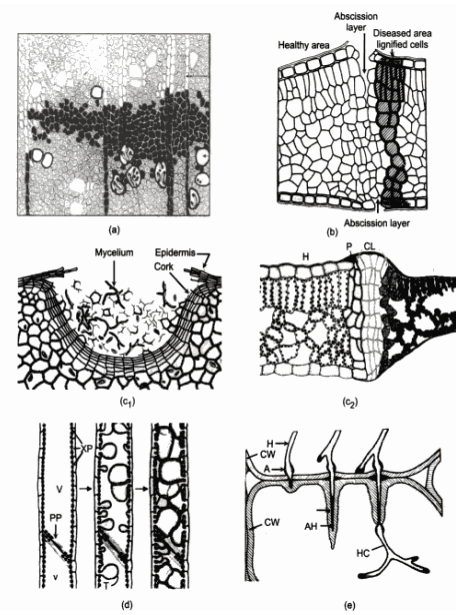

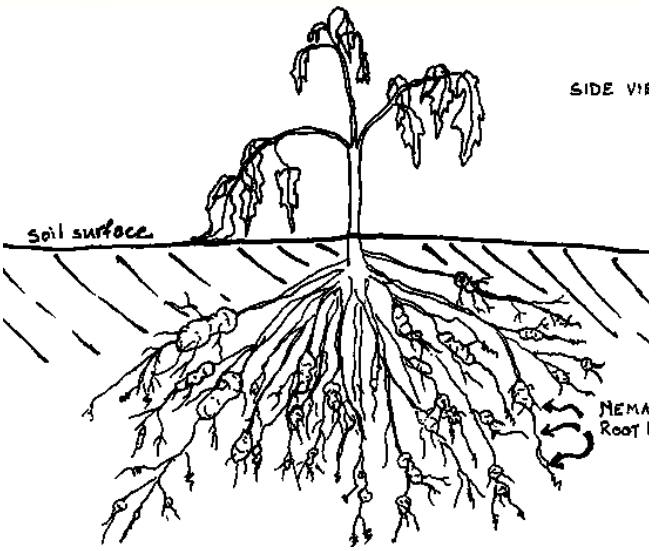

Plant Pathology

MBO-09

Vardhman Mahaveer Open University, Kota

Plant Pathology

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Plant Pathology

Vardhman Mahaveer Open University, Kota

Preface

The present book entitled **"Plant Pathology"** has been designed so as to cover the unit-wise syllabus of MBO-09 course for M.Sc. Botany (Final) students of Vardhman Mahaveer Open University, Kota. The basic principles and theory have been explained in simple, concise and lucid manner. Adequate examples, diagrammes, photographs and self-learning exercises have also been included to enable the students to grasp the subject easily. The unit writers have consulted various standard books on the subject and they are thankful to the authors of these reference books.

Unit - 1

Plant Pathology

Structure of the Unit:

1.0 Objectives

After studying this unit, you will understand the concept of Plant Pathology which is study of plant, their various pathogens, different types of plant diseases and their interactions in terms of-

- Concept and Scope of plant pathology
- Different types of pathogens and diseases caused by them
- The Origin and evolution of parasitism

1.1 Introduction

Plants make up the bulk of the earth's living environment as trees, grasses, flowers, etc. They are the only higher organisms on this planet that can convert energy of the sunlight into stored, usable chemical energy in the form of carbohydrates, fats and proteins. All animals, including humans depend directly or indirectly on these plant substances for living.

The growth of the plants depend on the availability of nutrients and water in the soil where they grow and the normal ranges of environmental factors such as light, temperature, and pH. Plants, however, do get sick. Anything that affects the health of plants may affect their growth and may seriously reduce their usefulness to themselves and to mankind. Plant pathogens, unfavorable weather, and insect pests are the most common causes of reduction or destruction of plant growth and production. Plants suffer from diseases whose causative agents are similar to those affecting animals and man. Although there is no evidence that plants feel pain and discomfort, the development of disease follows the same steps and is usually as complex in plants as it is in animals and man.

Plant pathology is the study of (1) the living entities and the environmental conditions that cause disease in plants; (2) the mechanisms by which these factors produce disease in plants; (3) the interactions between the disease-causing agents and the diseased plant; and (4) the methods of preventing disease, alleviating the damage it causes, or controlling a disease either before or after it develops in a plant.

Plant diseases are mostly caused by biotic (living) organisms. The most common plant diseases are caused by fungi, bacteria, phytoplasmas, viruses and viroids, nematodes and parasitic higher plants. Abiotic disorders are caused by noninfectious factors. The causal agents of disorders are almost unlimited, but the most common disorders usually are caused by factors, such as temperature extremes, moisture extremes, light extremes, nutrient extremes, poor soil (acidity or alkalinity, salt, texture), pesticide toxicity, air pollution, strong winds, hail and improper cultural practices. They also suffer from competition with unwanted plants (weeds) and by the damage caused by insect attacks. However, plant damage caused by insects, men, or other animals does not usually become the part of plant pathological studies.

1.2 The Concept of Disease in Plants

A plant is healthy or normal when it can carry out its physiological functions to the best of its genetic potential. These functions include normal cell division, differentiation, and development; absorption of water and minerals from the soil and their translocation; photosynthesis and translocation of the photosynthetic products throughout the plant; reproduction for sustaining and multiplication and storage of food supplies for overwintering or reproduction. Whenever the ability of the cells of a plant or a plant part to carry out one or

more of these essential functions is interfered by any pathogen or adverse environmental factor, the normal activity of the cells is hampered in some or the other way, then the plant becomes diseased. The primary causes of disease are either pathogens or factors in the physical environment, but the specific mechanisms by which diseases are produced vary considerably with the causal agent and sometimes with the plant. At first the reaction of the plant to the disease-causing agent at the site of affliction, is of a chemical nature, and is invisible. Soon, however, the reaction becomes more widespread and histological changes take place that manifest themselves macroscopically and constitute the symptoms of the disease. Affected cells and tissues of diseased plants are usually weakened or destroyed by the disease-causing agents. The ability of such cells and tissues to perform their normal physiological functions is reduced or completely eliminated; as a result, plant growth is reduced or the plant dies. Thus, Disease in plants can be defined "as the series of invisible and visible responses of plant cells and tissues to a pathogenic microorganism or environmental factor that result in adverse changes in the form, function and integrity of the plant and may lead to partial impairment or death of the plant or its parts."

Plants are considered to have a disease or a disorder when they are not growing up to expected standards. There are two visual keys for identifying "sick" plants—symptoms and signs.

Symptoms are the visual response of the plant to attack by an infectious organism or an abiotic factor. For example, plants exhibiting leaf spots, cholorosis, necrosis, wilting, and stunting are showing symptoms of an abnormality. Symptoms are a very important part of determining the cause of plant problems. However, there are several factors that prevent symptoms from revealing the exact causal agent.

First, symptoms are nonspecific. Many different pathogens and abiotic factors can have the same affect on the plant. For example, any entity that disrupts the movement of water in the plant will cause the plant to wilt. Another factor is that symptoms change over time. Therefore, good symptom descriptions require observing the plant over time and noting the progression of symptom development. Symptoms often develop away from the infection site. For example, above ground symptoms of water and nutrient stress occur when roots are damaged. Additionally, symptoms will vary due to the pathogen's virulence (aggressiveness), the degree of host susceptibility and the environmental conditions.

Signs are the visual presence of some structure formed by the pathogen on the host plant. Examples include fungal mycelium, spores, fruiting bodies and bacterial ooze. While symptoms are nonspecific, signs allow for a more specific determination of the causal agent. In some cases, the sign will reveal the exact cause. For example, white powdery growth on plant surfaces is caused by powdery mildew fungi. In other cases, signs will help to narrow down the potential causal agent. For example, mycelium on the surface of turf indicates a fungal disease problem.

1.2.1 The Disease Triangle

A disease episode requires the interaction of three components: the host, the pathogen, and the environment. This interaction is known as the disease triangle (Fig. 1).

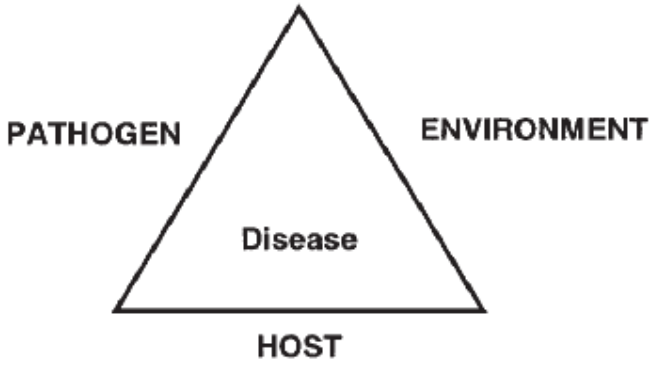


Fig. **1.1 : The Disease Triangle**

In order for a disease to occur, the host plant must be susceptible to the pathogen (disease organism). First, the plant must be susceptible genetically, meaning that the organism present can cause disease on that plant. In some cases, the host must be at a certain physiological state for disease to occur. For example, some organisms attack only young plants, others take advantage of mature or aging plants, and some organisms are able to take advantage of the plant at any growth stage. In most cases, pathogens take advantage of plants that are stressed. Vigorous, strong-growing, non stressed plants are less susceptible to disease than plants growing under stress conditions. Disease only results from the infection by a virulent (aggressive and able to cause disease) pathogen. Most pathogens go through a life cycle in which part of the time the organism is dormant. When a pathogen is dormant, no disease can occur. Although the disease triangle is illustrated by a triangle with all sides equal, the environment really is the most important part of the interaction. The en-

vironment must be conducive (favorable) for disease development. That is the temperature, moisture, nutrients and wind must all favor the pathogen's growth and development. Some diseases (notably those caused by viruses) require a vector (transmitting agent) for infection. In these cases, the vector is an additional component of this interaction. The degree to which these three components interact relates to the disease severity. For example, if the host is highly susceptible, the pathogen is highly virulent, and the environment is highly conducive, then the disease will be very severe. Severity also includes an element of time - the longer the environment remains favorable for disease development, the greater the severity of the disease.

Successful disease management requires the disruption of some part of the disease triangle. Accurate diagnosis of the causal agent is required for effective use of management strategies in general and specifically for effective chemical control. Using an inappropriate chemical will not only be ineffective against the disease agent, but also can lead to additional disease problems by killing beneficial microorganisms in the environment. There are several different strategies for disease management. But, overall, the most successful strategy depends on the integrated use of available control methods.

1.3 History of Plant Pathology

Man became aware of plant diseases in the early times of antiquity. This is evidenced by the inclusion of blasting and mildew in the Old **Testament**. Our ancient religious literature gives informations on plant diseases much before their mention by the Greek philosopher, Theophrastus. *Rigveda, Atharvaveda* (1500-500 B.C.), the *Artha Shashtra* of Kautilya (321-186 B.C.), *Sushruta Samhita* (200-500 A.D.), *Vishnu Puran* (500 A.D.), *Agnipuran* (500-700 A.D.) and *Vishnudharmottar* (500-700 A.D.) are some of the ancient books from India where diseases and other enemies of plants are mentioned. In *Rigveda,* classification of plant diseases and germ theory of disease were discussed. The learned men during Vedic period were aware that the diseases are caused by microbes. The book "*Vraksha Ayurveda*" written by Surapal in ancient India contained information on plant diseases. This is the Indian book, which gave first information on plant diseases. He divided plant diseases into two groups viz., internal and external. Plant diseases like rust, smut, downy mildew, powdery mildew and blight were mentioned in the Bible. The Greek Philosopher, Theophrastus (370-286 B.C.) was the first to study and write about the diseases of trees, cereals and legumes. In his book *'Enquiry into plants*' Theophrastus has recorded his observations, imaginations and experiences but

they were not based on any experiments. He had mentioned that plants of different groups have different diseases, which are autonomous or spontaneous i.e., no external causes were associated with the plant diseases. The history in several aspects of plant pathology is given as below:

- **700 B.C**.- The Romans celebrated the holiday "Robigalia" that involved sacrifices of reddish colored dogs and cattle in an attempt to appease the rust god Robigo.
- **470 B.C**.- Pliny was the first to use a fungicide, amurce of olives, to control blight.
- **1844-1845- The Irish Potato Famine**, caused by the Late Blight fungus *Phytophthora infestans*, struck Ireland and prompted the birth of modern plant pathology. The Famine killed 1.5 million people and forced another 1.5 million to immigrate between 1845-1850. The population of Ireland was 8.5 million in 1840; it decreased to 4 million by 1900.
- **1885-Bordeaux mixture** : Downy Mildew of Grape was a great problem in the growing fields in France but there were no known ways of controlling it. The vineyards were also troubled with pilferers. They began applying a mixture of copper sulfate and lime to the plants along the edges of the fields. It was also observed that these plants held onto their leaves throughout the season.
- **1900- White Pine Blister Rust** caused by *Cronartium ribicola*. The pathogen was introduced on seedlings from European nurseries. White pines, especially young trees and plants belonging to the genus *Ribes* (currants and gooseberries) are susceptible to the disease. Although White Pine Blister Rust is occasionally a severe foliar disease on *Ribes* plants, on white pines it is lethal if allowed spreading from an infected branch into the trunk. This disease caused the first US quarantine in 1912.
- **1904-1940- Chestnut Blight** caused by *Cryphonectria (Endothia) parasitica*. Chestnut seedlings imported from the orient brought with them the pathogen that killed off all the mature chestnuts in eastern North America. The disease devastated the people who relied on the chestnut tree for their livelihood. There are still some sprouts left in the forests, put up by the living root systems, but they too eventually succumbed to the blight.
- **1910- Citrus Canker** caused by *Xanthomonas axonopodis pv. Citri,* was discovered near the Georgia border and was eradicated in 1931. The pathogen was found 400 miles away in Dade County in 1912. The pathogen

spread throughout the Gulf States and as far north as South Carolina. It took more than 20 years to eradicate that outbreak of citrus canker. In 26 counties, over 250,000 grove trees and over 3 million nursery trees were destroyed by burning. Subsequent outbreaks occurred in 1986 and 1995.

- **1930- Dutch Elm Disease** caused by *Ophiostoma ulmi*. This disease devastated mall and roadside planting of American elm trees throughout much of the United States.
- **1941- Golden Nematode**, *Globodera rostochiensis*, was discovered in 1941.It caused a slow decline in potato plants that eventually lead to death. As of 1955, the distribution was believed to be located only in Nassau and Suffolk countries in NYS. After decades of building their population levels, the Golden Nematode was capable of reducing the potato yield up to 70%.
- **1970- Southern Corn Leaf Blight** caused by *Helminthosporium maydis*. Originally considered a minor disease, a change in the genetics of seed corn caused an epidemic. In 1970, the disease was reported in every state east of the Mississippi River, also in several states west of the Mississippi River. In some areas damage caused losses of 50-100%. Nationally losses averaged 20-30%.
- **1995- Sudden Oak Death**, caused by *Phytophthora ramorum,* was discovered in California. A large number of tanoaks were found to be declining with no known cause. It took five years to isolate and identify the causal agent and is also called ramorum blight and ramorum dieback. Although the disease was first observed in the United States in tanoaks, it is also found to infect many other plant species.
- **1999- Southern Wilt/Brown Rot**, caused by *Ralstonia solancearum Race 3 Biovar 2* Southern Wilt is a disease of *Geranium* and Brown Rot is a disease of Potato. *Ralstonia solanacearum Race 3 Biovar 2* has appeared on *Geranium* a few times in recent years but it appears to be confined to greenhouse crops and there is no evidence of spread to potato, tomato, or eggplant.
- **1999- Plum Pox**, caused by *Plum Pox Virus,* is a disease of stone fruits caused by a viral pathogen called the Plum Pox Virus, also known as "*Sharka*". It was first discovered in an Adams County, Pennsylvania Orchard in 1999.
- **2004-Soybean Rust**, is caused by two fungi named *Phakopsora pachyrhizi* and *Phakopsora meibomiae. P. pachyrhizi* appeared in the US in November

2004, apparently entering on winds of Hurricane Ivan. It was found in 9 States shortly thereafter. It was found in Florida early in 2005 on Soybean and new detections for that season remained in the South.

1.4 Scope of Plant Pathology

Plant pathology deals with different aspects of plant diseases and has wide scope than human pathology. The welfare of plants is of particular interest to those most directly concerned with the growth of plants and the manufacture and distribution of plant products and indirectly concerned with every one of us as consumers of plants and of the endless series of products derived from plants.

Plant pathology may call upon the basic techniques and knowledge of botany, mycology, bacteriology, virology, nematology, plant anatomy, plant physiology, genetics, biochemistry, horticulture, soil science, forestry, chemistry, physics, meteorology and many other branches of science. Plant pathology profits from advances in any one of these sciences, and many advances in other sciences have been made in the attempt to solve phytopathological problems. A good knowledge of at least the basic facts of the related sciences is indispensable for efficient performance by any plant pathologist. Although plant pathology as a science attempts to increase our knowledge of the causes and the development of plant diseases, it is also a science with a more practical goal. The purpose is to develop controls for all plant diseases. The goal is to save the produce which today is destroyed by plant diseases and to make it available to the growers who work hard to produce it and to the hungry and ill-clothed millions of our increasingly overpopulated world.

In recent years plant pathologists have begun to specialize in certain fields in which notable advances have been made, which are: Interaction between host and pathogen at chemical, molecular and genetic level, Plant virology, chemistry of fungitoxicity and Disease forecasting, Plant protection chemicals, Breeding for disease resistance, etc. Increased population emphasizes the application of all possible means to meet the food requirements such as expansion of crop area, improved methods of cultivation, increased use of fertilizers, improved varieties, increased irrigation and crop protection.

Table 1.1 : Examples of severe losses caused by Plant Diseases

Disease	Location	Comments
Fungal		
1. Cereal rusts	Worldwide	Frequent severe epidemics; huge annual losses
2. Cereal smuts	Worldwide	Continuous, although lesser, losses on all grains
3. Ergot of rye and wheat	Worldwide	Infrequent, poisonous to humans and animals
4. Late blight of potato	Cool, humid climates	Annual epidemics, e.g., Irish famine (1845–1846)
5. Brown spot of rice	Asia	Epidemics, e.g., the great Bengal famine (1943)
6. Southern corn leaf blight	U.S.	Historical interest, epidemic 1970, $1 billion lost
7. Powdery mildew of grapes	Worldwide	European epidemics (1840s–1850s)
8. Downy mildew of grapes	U.S., Europe	European epidemic (1870s–1880s)
9. Downy mildew of tobacco	U.S., Europe	European epidemic (1950s–1960s); epidemic in North America (1979)
10. Chestnut blight	U.S.	Destroyed almost all American chestnut trees (1904–1940)
11. Dutch elm disease	U.S., Europe	Destroying American elm trees (1918 to present)
12. Pine stem rusts	Worldwide	Causing severe losses in many areas
13. Dwarf mistletoes	Worldwide	Serious losses in many areas
14. Coffee rust	Asia, South America	Destroyed all coffee in southeast Asia (1870s–1880s) since 1970 present in South and Central America
15. Banana leaf spot or Sigatoka disease	Worldwide	Great annual losses
16. Rubber leaf blight	South America	Destroys rubber tree plantations
17. Fusarium scab of wheat	North America	Severe losses in wet years
Viral		
18. Sugar cane mosaic	Worldwide	Great losses on sugar cane and corn
19. Sugar beet yellows	Worldwide	Great losses every year
20. Citrus tristeza (quick decline)	Africa, Americas	Millions of trees being killed
21. Swollen shoot of cacao	Africa	Continuous heavy losses
22. Plum pox or sharka	Europe, North America	Spreading severe epidemic on plums, peaches, apricots
23. Barley yellow dwarf	Worldwide	Important on small grains worldwide
24. Tomato yellow leaf curl	Mediterranean countries, Caribbean Basin, U.S.	Severe losses of tomatoes, beans, etc.
25. Tomato spotted wilt virus	Worldwide	On tomato, tobacco, peanuts, ornamentals, etc.
Bacterial		
26. Citrus canker	Asia, Africa, Brazil, U.S.	Caused eradication of millions of trees in Florida in 1910s and again in the 1980s and 1990s
27. Fire blight of pome fruits	North America, Europe	Kills numerous trees annually
28. Soft rot of vegetables	Worldwide	Huge losses of fleshy vegetables
Phytoplasmal		
29. Peach yellows	Eastern U.S., Russia	Historical, 10 million peach trees killed
30. Pear decline	Pacific coast states and Canada (1960s), Europe	Millions of pear trees killed
Nematode diseases		
31. Root knot	Worldwide	Continuous losses on vegetables and most other plants
32. Sugar beet cyst nematode	Northern Europe, Western U.S.	Continuous severe annual losses on sugar beets
33. Soybean cyst nematode	Asia, North and South America	Continuous serious losses on soybean

1.5 Classification of Plant Diseases

With the vast diversity of plants, there are a large number and kind of diseases that affect them. On an average, each kind of plant can be affected by one hundred or more plant diseases. Each kind of pathogen may affect anywhere from one variety to several dozen or even hundreds of species of plants. To facilitate the study of plant diseases, these can be grouped in some generalized categories. It also becomes essential for further identification and the subsequent control of any given plant disease. There are several criteria that may be used as a basis for classification of plant diseases. Plant diseases are classified according to:-

- Symptoms they cause (root rots, cankers, wilts, leaf spots, scabs, blights, anthracnoses, rusts, smuts)
- The plant organ they affect (root diseases, stem diseases, foliage diseases, fruit diseases)
- The types of plants affected (field crops diseases, vegetable diseases, fruit tree diseases, forest diseases, turf diseases, diseases of ornamental plants)
- The extent to which plant disease is associated with plant:-
  Localized disease: affecting only a part of the plant.
  Systemic disease: affecting the entire plant.
- Mode of natural perpetuation and mode of infection:-
  Soil borne: Inoculums of the diseases causing pathogen remains in soil and penetrate the plant resulting in diseased condition e.g. Root rot, wilt
  Air borne: The micro-organisms are spread through air and attack the plants causing diseases. e.g. Blight, rust, powdery mildew.
  Seed borne: The micro organisms are carried along with seeds and cause diseases when congenial condition occurs. e.g. Damping off.
- Occurrence and distribution of plant disease geographically:-
  Endemic: When a disease is more or less constantly prevalent from year to year in a moderate to severe form in a particular country. e.g., Wart disease of potato is endemic to Darjeeling.
  Epidemic or epiphytotic: A disease occurring periodically but in a severe form involving major area of the crop. It may be constantly present in locality but assume severe form occasionally e.g. Rust, Late blight, Mildews.
  Sporadic: Diseases which occur at very irregular interval and location in a moderate to severe form e.g., leaf blights, wilt.
  Pandemic: Diseases occurring throughout the continent or sub-continent resulting in mass mortality e.g., Late blight of potato.

However, the most commonly used criterion is the type of pathogen that causes the disease. On the basis of **causal factors**, diseases are classified into two groups.

1. **Non parasitic disease:** The causal factors of these are mainly physiological or environmental like freezing injury caused by low temperature, high temperature, unfavourable oxygen or soil moisture, mineral deficiency or

excess mineral etc. Example: Black heart of potato caused due to high temperature, Bark necrosis of red delicious apple caused due to excess mineral, Red leaf of cotton and *Khaira* disease of rice is caused due to mineral deficiency.

2. **Parasitic disease:** The causal factors of the disease are parasitic micro or macroorganisms which need a host plant to survive or to complete the life cycle. Various fungi, bacteria, viruses, mycoplasma, algae and animal parasites such as nematodes etc. parasitize and cause disease in host plants. Example: Bacterial blight of paddy, Club root of crucifer caused by mycoplasma, Tobacco mosaic by virus, ergot, smut and rusts caused by fungi, ear cockle of wheat caused by nematode etc.

On this basis, plant diseases are classified as follows:

I. Infectious Plant Diseases

1. Diseases caused by fungi
2. Diseases caused by bacteria
3. Diseases caused by parasitic higher plants
4. Diseases caused by viruses
5. Diseases caused by nematodes

II. Noninfectious or Abiotic Diseases

1. Nutrient deficiencies
2. Mineral toxicities
3. Lack or excess of soil moisture
4. Too low or too high temperature
5. Air pollution
6. Lack of oxygen
7. Lack or excess of light
8. Soil acidity or alkalinity (pH)

Though Walker (1969) classified plant diseases into three major groups i.e.

(a) Non parasitic disease, (b) Parasitic disease and (c) Mycoplasmal and viral diseases but here Mycolpasmal and viral diseases are kept under parasitic diseases.

Diseases are sometimes classified on the basis of **the kind or type of the characteristic symptoms it produces** on the plants.

(1) Rusts (2) smuts (3) mildews (4) root-rots (5) blights (6) leaf spots (7) wilts

(8) cankers (9) fruit rots etc.

Diseases may also be classified **according to the plant organ they affect**.

Thus there are root diseases, stem diseases, foliage diseases, fruit diseases etc.

1. Root diseases: root-rots *(Verticillium, Colletotrichum, Macrophomina)*, club root disease of crucifer, vascular wilts *(Fusarium* spp., *Verticillium)* and other root diseases caused by *Gauemannomyces graminis, Phymatotrichum omnivorum* and *Armillaria mellea*.
2. Stem diseases: stem rot of jute, Black stem rust of wheat.
3. Foliage diseases:Leaf spot of turmeric,leaf blight of wheat, leaf curl of peach.
4. Fruit diseases: Crown gall of stone fruits, citrus canker.
5. Seedling diseases: seedling blight *(Pythium, Phytophthora, Fusarium, Corticium)*

1.6 Parasitism

When an organism lives on or in some other organism and obtains its food from the latter, then it is a parasite. The relationship between a parasite and its host is called parasitism. A broad definition of parasitism would include all varieties of inter-specific associations in a gradient of interdependence. Therefore, associations defined as commensalism, mutualism and symbiosis are distinct features of a same phenomenon – parasitism.

The parasite-host-environment system is dynamic, with several points of equilibrium. This makes it difficult to trace the thresholds between benefit and damage, and therefore, the definitions of commensalism, mutualism and symbiosis become worthless. Parasitism is essential for life. Life emerged as a consequence of parasitism at the molecular level, and intracellular parasitism created evolutionary events that allowed species to diversify. Leuckart (1879) defined parasites as organisms that find in another organisms their habitat and nourishment. According to Brumpt (1913), parasites are all living beings, plants or animals, in which, at least part of their lives depend upon another organism. Many parasites may be considered as harmless or even necessary for their hosts.

A plant parasite is an organism that becomes intimately associated with a plant and multiplies or grows at the expense of the plant. The removal by the parasite of nutrients and water from the host plant usually leads to reduced efficiency in the normal growth of the plant and becomes detrimental to its further

development and reproduction. Thus, in many cases, parasitism is intimately associated with pathogenicity, since the ability of the parasite to invade and become established in the host generally results in the development of a diseased condition in the host. In some cases of parasitism, as with the root nodule bacteria of legume plants, both the plant and the microorganism are beneficial to the other's development, and this phenomenon is known as symbiosis. Of the large number of groups of living organisms, only a few members of a few groups can parasitize plants: fungi, bacteria, and parasitic higher plants (all three belonging to the plant kingdom), nematodes (of the animal kingdom) and viruses. These parasites, to be successful, must be able to invade a host plant, feed and proliferate in it and withstand the conditions in which the host lives. Some parasites, including viruses, nematodes, and among the fungi, those causing downy mildews powdery mildews and rusts, can grow and reproduce only on living hosts, and they are called obligate parasites. Other parasites (most fungi, bacteria) can live on either living or dead hosts and are, therefore, called non-obligate parasites. Some non-obligate parasites live most of the time or most of their life cycles as parasites but, under certain conditions, may grow saprophytically on dead organic matter (facultative saprophytes), whereas others live most of the time and thrive well on dead organic matter but, under certain circumstances, may attack living plants and may become parasitic (facultative parasites). There is usually no correlation between the degree of parasitism of a pathogen and the severity of disease it can cause, since many diseases caused by weakly parasitic pathogens are much more damaging to the plant than others caused even by obligate parasites.

Moreover, certain fungi, e.g., those causing sooty molds, can cause disease by growing on the surface of the plant and feeding on insect excretions rather than by parasitizing the plant. Obligate parasites are usually very specific as to the kind of host they attack, possibly because they have evolved in parallel with their host and require certain nutrients that are produced only by these plants or become available to the pathogen only in these hosts. Non-obligate parasites may attack many different plants and plant parts of varying age, possibly because they depend for their attack on nonspecific toxins or enzymes that affect substances or processes found widely spread among the plants. Some non-obligate parasites, however, will produce disease on only one or a few plant species. In any case, the number of plant species presently known to be susceptible to a single pathogen may be smaller than the actual number in nature since only a few species out of thousands have been studied for their

susceptibility to each pathogen. Furthermore, because of genetic changes, a pathogen may be able to attack hosts previously immune to it.

1.7 The Origin and Evolution of Parasitism

Parasitism is inherent to life. Parasites are found in every organism of all existing species on earth. Since the beginning of life parasitism was adopted by certain organisms to multiply. Actually parasitism must have occurred at an early stage of evolution. All living organisms have a uniform biochemical composition that points to a common origin in a common ancestor that lived a billion years ago. Life on earth was only possible as a consequence of parasitism in what was still a molecular world. In fact, life appeared on earth around 4.4-3.8 billion years ago as a consequence of molecular parasitism and the present day life forms still display relics of these ancient associations in their genomes. Later diversification of life forms and species radiation was also the consequence of these multiple associations. These ancient events represent the first steps towards a host-parasite way of life although still at the level of molecules. Molecular parasitism is clearly exemplified by transposable elements of the genome. Indeed, the DNA sequences called transposable elements are actually recognized as molecular parasites.

Transposable elements occur in both prokayotes and eukaryotes. Transposition is the insertion of an identical copy of the transposable element into a new genomic site of the host. These insertions can cause deletions, inversions, and chromosome fusions that result in considerable genome plasticity, thus contributing to biodiversity. They have a common origin with viruses and retroviruses and were identified first as 'control elements' by McClintock (1984).

The extra chromosomal transmissible genetic elements (viruses, plasmids and bacteriophages) are genome fragments that depend on the host cell to multiply and therefore they could not have preceded the cell itself. These elements could have been the origin of nuclear DNA as well as other cellular organelles. The extra chromosomal genetic elements and the transposable elements represent relics of primitive molecular parasites. They played a very important role in the evolution of life forms since their inclusion in the hosts' genomes' cell promoted genetic diversity. Certainly many of the alterations they induced were deleterious, but a number of them resulted in advantages for their hosts.

A failed episode of predation/parasitism resulted in the eukaryotic cell. Also, the prokaryotic cell exhibits clearly the parasitism that once was the causal

effect of their origin. Not only the nucleous but also the whole cell is a chimera, a polyheterogenic state derived from a long history of parasite associations. The association of microorganisms that resulted in mitochondria, chloroplasts and other organelles granted a significant increase in the complexity of the living organisms that resulted in the improvement of their capacity to occupy new ecological niches. Furthermore, the advent of the cell offered possible new niches for parasites.

1.8 Parasites and Infectious Diseases

Parasitic infections and parasitic disease are two strikingly distinct situations originated from a single process. A parasite is a necessary but not sufficient condition to launch a parasitic disease. Parasitism does not necessarily result in injury or benefit to the host. Parasitic disease is an eventual outcome of a given parasite in a given host from a given population in a certain environment during a particular life co-evolution period of both protagonists. In essence it is a unique result of association of parasite and host in a given environment. It matters not whether a molecular parasite or a multi-cellular parasite is involved.

A system is formed by the parasite, the host, and the environment where each one interacts and influences the other subsystem in such a way that any change in one subsystem affects the other two. Systems may exhibit order spontaneously. Submitted to natural selection and/or the inherent properties of a system, the parasite-host-environment system reacts to changes in each subsystem with different responses, according to the features of the stimulus. Such a stimulus can induce parasitic disease originated by any component of a subsystem. Parasitic disease is not an unbalanced occurrence in the host-parasite-environment relationship, but a natural phenomenon where some event has altered a component of the system and a specific reaction of a given individual host express signals and symptoms of a modified behavior of the whole system, i.e. disease.

The history of evolution and biodiversity is fundamentally a history of evolution of species interactions. An isolated individual is only an abstraction and cannot be conceived out of its environment. Parasites promoted the major factors that have influenced the organization and evolution of life. Zelmer (1998) proposed an evolutionary definition of parasitism. He concludes by placing all parasites within a shared evolutionary framework, with the host immune response as a constant and powerful selective factor. He criticizes the

view of transition of commensalism to parasitism as a definitive modification of the nature of a given symbiotic relationship.

1.9 Summary

Plants are producers of food in the world. They are living and continuously evolving with all the other living organisms. Organisms which directly or indirectly cause harm to them are called parasites. Plant diseases can be classified in as many comprehensive categories depending upon the requirement of the reader. Mostly, they are classified on the basis of the kind of pathogen causing the disease. The study of plant diseases is Plant Pathology and its history is as old as the history of mankind itself since man has always depended upon plant resources for its food. Due to this relation, any harm that comes to plants of economic importance in any form, is of great concern to man. Severe crop losses, forest destructions on account of plant diseases has led to the study advances in the field of plant pathology and also the study of course of evolution of parasitism in plants to achieve a better understanding of the pathogens in order to overcome and control them.

1.10 Glossary

- **Parasite**: an organism or virus living in or on another organism (host) from which it obtains its nutrient supply. A parasite is not necessarily a pathogen.
- **Parasitism**:a relation between organisms in which one lives as a parasite on another.
- **Pathogen**: a disease causing organism or agent.
- **Virulence**: the degree or measure of pathogenicity of a given pathogen; relative capacity to cause disease.
- **Translocation**: the long-distance transport of water, nutrients, chemicals, or food materials within a plant.
- **Necrosis**: the death of cells, often accompanied by black or brown darkening of the tissue.
- **Biotic**: pertaining to life and therefore living organisms. For example, plant diseases of a biotic origin are caused by living organisms such as insects, nematodes, etc.
- **Abiotic**: pertaining to physical and inorganic components. For example, diseases/disorders in plants can be caused by abiotic factors such as extremes of heat, light, moisture, lack of nutrients etc

- **Vector**: any living organism (e.g., insect, mite, bird, nematode, parasitic plant, human, etc.) that transmits a pathogen from an infected organism to an uninfected one.
- **Disease triangle**: a memory aid that diagrams the three important components necessary for disease: a susceptible plant, a virulent pathogen, and a favorable environment
- **Chlorosis**: an abnormally yellow color of plant tissues, resulting from partial failure to develop chlorophyll, caused by a nutrient deficiency or the activities of a pathogen.

1.11 Self-Learning Exercise

Section A : (Very Short Answer Type Questions)

1. The branch of science that deals with the study of plant diseases is_____.
2. The book _____ is the first Indian book, which gave information on plant diseases.
3. The Irish Potato Famine in 1845 was caused by _____.
4. The relationship between a parasite and its host is called _____.
5. _____live most of the time on dead organic matter but, may attack living plants and may become parasitic.
6. _____depicts the three important components necessary for disease: a susceptible plant, a virulent pathogen, and a favorable environment.

Section B : (Short Answer Type Questions)

1. What is a disease? How Plant diseases are classified on the basis of spread and severity of infection?
2. Differentiate between Localised and Systemic infection.
3. How is eukaryotic cell a failed example of parasitism evolution?

Section C : (Long Answer Type Questions)

1. Write an essay on the origin and evolution of parasitism.
2. How do plant diseases affect the human society?

3. How can plant diseases be classified on the basis of causal organisms?

Answer key of Section – A

1. Plant pathology
2. *Vraksha Ayurveda*
3. *Phytophthora infestans*
4. Parasitism
5. Facultative parasites
6. Disease Triangle

1.12 References

- Plant Pathology by G.N. Agrios
- Introduction to Plant Pathology by Richard N. Strange
- Principles of plant pathology by R.S. Singh

Unit - 2

Host Pathogen Interaction-I: Pathogen Attack

Structure of the Unit:

2.0 Objectives

After studying this unit, you will understand the various mechanisms by which the different plant pathogens initially interact with their host plants and set the initial course of disease development by going through different attacking modes in terms of-

- Adhesion methods of different pathogens
- Penetration methods using physical and chemical means
- Colonization of host tissues

2.1 Introduction

By going through this unit, you will understand the plant disease development in the initial stages. You will come to know about the infection process of a pathogen in a plant as to how after coming in contact with the suitable host and on getting favourable environment; the pathogens start to establish themselves.

Therefore for a pathogen to infect a plant, it must be able to make its way in to and through the plant, obtain nutrients from the plant, and neutralize the defense reactions of the plant. Pathogens accomplish these activities mostly through secretions of chemical substances that affect certain components or metabolic mechanisms of their hosts. Penetration and invasion, however, seem to be aided by, or in some cases entirely by, the result of the mechanical force exerted by certain pathogens on the cell walls of the plant.

2.2 Infection Process

Plants exist in a world which is full with microorganism. The microorganisms continue to grow in the same environment as the plants and trees throughout the growing season or for many years. The surfaces of these plants are constantly exposed to bacteria, fungi, nematodes, and possibly parasitic plants. Plant pathogens have accumulated many adaptations to enable themselves to adhere to plants, overcome the plant defense mechanisms, and colonize plant tissues for growth, survival, and reproduction. Once established inside the plant, they have at least temporarily escaped the intense competition from saprophytic organisms on plant surfaces and in the soil.

The "infection process" can be broadly divided into following three phases:

- Adhesion
- Penetration
- Colonisation

It encompasses the germination or multiplication of an infective propagule in or on a potential host till the establishment of a successful parasitic relationship between the pathogen and the host. The process of infection is influenced by properties of the pathogen, the host and the external environment. If any of the stages of the infection process is inhibited by any of these factors, the pathogen will not be able to cause disease in the host.

While some parasites colonise the outside of the plant (ectoparasites), pathogens may also enter the host plant by penetration, through a natural opening (like a stomatal pore) or via a wound. The symptoms of the diseases, produced by these pathogens, result from the disruption of respiration, photosynthesis, translocation of nutrients, transpiration, and other aspects of growth and development.

2.3 Adhesion

Before a pathogen can penetrate a host tissue, a spore must germinate and grow on the surface of the plant. Many fungi, on encountering their host or some other solid substrate, germinate or start producing germ tubes which may differentiate into infection structures. Adhesion is also crucial to the successful parasitism of plants by pathogens. In fungal-substratum adhesion that occurs on the plant host surface before penetration, adhesion serves multiple functions.

The various functions of a plant-pathogen adhesion can be as follows:

- Adhesion keeps propagules of pathgens from being displaced by not being blown or rinsed by water and/or wind from a potentially suitable environment.
- It is required for host penetration via mechanical pressure,
- It is required for thigmodifferentiation.
- It is required for thigmotropism.
- It facilitates interaction between pathogen and host.
- It increases the surface area of contact with its host.
- It also limits germination to potential host tissue (which is required for contact stimulated germination).

Many bacteria produce fimbriae and they play a role. In the case of motile pathogens, they must find the host and negotiate its surface before entering the host. Some pathogens develop specialised penetration structures, such as appressoria, while others utilise pre-existing openings in the plant's surface, such as wounds or stomatal pores. Plant viruses are often transported and introduced into the plant via vectors such as fungi or insects.

The initial contact between infective propagules of a parasite and a potential host plant is called inoculation. Pathogens use a variety of stimuli to identify a suitable entry point. Several fungi use topographical cues on the plant surface to guide them towards a likely stomatal site. Once the hypha reaches a stoma, volatile compounds escaping from the pore appear to provide a signal for the formation of a specialised penetration structure, the appressorium (Fig 2.1). Sugars, amino acids and minerals secreted by plants at the leaf surface can non-specifically trigger spore germination or provide nutrition for the pathogen. Some pathogenic spores will not germinate in the absence of these substances.

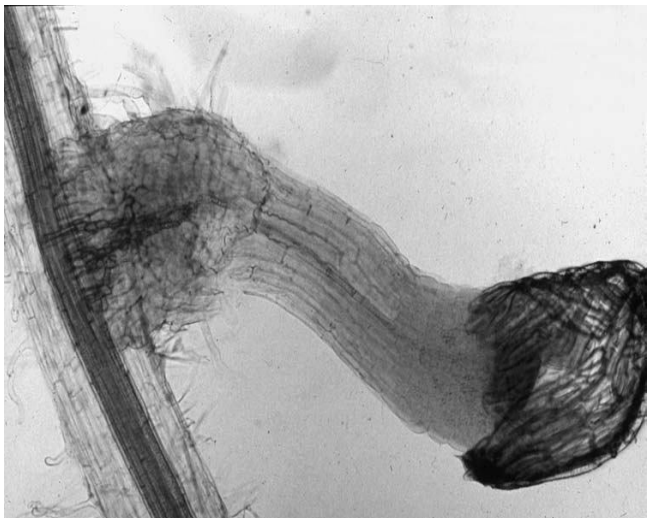


Fig. 2.1 : Germinating seed of *Striga hermonthica* giving rise to a appressorium which has attached to a host root

Pathogen development is influenced by temperature, moisture, light, aeration, nutrient availability and pH, whether contact is required can depend upon the environmental conditions. The conditions necessary for survival and successful infection differ between pathogens.

2.4 Penetration

Pathogens normally exploit every possible pathway to enter their host, although individual species of pathogen tend to have a preferred method. The host plays an equally important role in penetration of the pathogen by providing certain stimuli. The stimuli provided by the host for germination, growth and the differentiation of infection structures can be hydrophobicity, hardness, chemical components and topographical features of the host plant. Several chemical components of host plants have been implicated in the germination of propagules of plant pathogens and the differentiation of infection structures. In particular, the wax on the surface of aerial parts of the plant is a rich source of diverse compounds, which may play these roles. The topologies of plant surfaces provide signals to many fungal pathogens. For example, rust fungi usually enter their hosts through stomata, their topology triggering the development of infection structures. For rust fungi which enter via stomata, locating a stoma may be facilitated by responding to other topological signals. For example, germ tubes of *P. graminis f. sp. tritici* (Fig 2.2) orient themselves at right angles to leaf veins which, owing to the manner of their distribution, maximize the chance of the tube encountering a stoma.

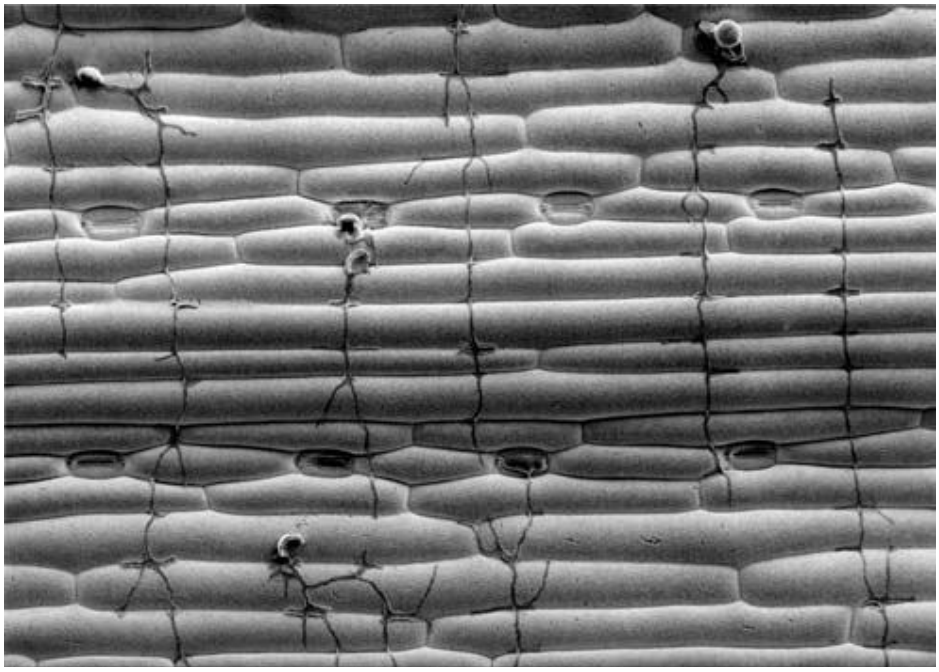


Fig. 2.2 : Germ tubes of *P. graminis f. sp. tritcii* orienting themselves at 90^0 to the grooves on the host surface corresponding to the epidermal cell junctions

Stimulants may also play an important role in the establishment of infection by aerial organisms. In particular, pollen and intact anthers are a rich source of nutrient and may enhance the virulence of facultative pathogens.

The pathogens utilize a wide range of tactics to penetrate the host. Fungal pathogens often use **direct penetration** of the plant surface to enter the host. This requires adhesion to the plant surface, followed by the application of **mechanical pressure** and then **enzymatic degradation** of the cuticle and cell wall, in order to overcome the physical barriers presented by the plant's surface. Many pathogens largely depend on the various chemicals such as toxins, growth regulators and various polysaccharides, which they produce, in order to fight the defenses of plants against them.

2.4.1 Direct Penetration

Most simple pathway for pathogen entry is via a **pre-existing opening** in the plant surface. This can be a natural opening or a wound. Pathogenic bacteria and nematodes often enter through stomatal pores when there is a film of moisture on the leaf surface. Fungi can also penetrate open stomata without the

formation of any specialised structures. Some fungi form a swollen appressorium over the stomatal aperture and a fine penetration hypha enters the air space inside the leaf, where it forms a sub-stomatal vesicle, from which infection hyphae emerge and form haustoria in surrounding cells. Also vulnerable to pathogen invasion are hydathodes, pores at the leaf margin that are continuous with the xylem. Under particularly humid conditions, droplets of xylem fluid (guttation droplets) can emerge at the surface of the leaf where they can be exposed to pathogenic bacteria, which then enter the plant when the droplet retreats back into the hydathode as the humidity decreases. Lenticels are raised pores that allow gas exchange across the bark of woody plants. They exclude most also use more unusual openings, such as nectaries, styles and ectodesmata. Entry through a wound does not require the formation of specialised structures, and many of the pathogens that utilise wounds to enter the plant are unable to penetrate the plant surface otherwise. Most plant viruses enter through wounds, such as those made by their insect vectors.

Fig. 2.3 : Pathogen Entry Through Natural Opening

2.4.2 Mechanical **Forces**

Exerted on Host Tissues by Pathogens

Although for many plant pathogens a capacity to breach the cell walls of their hosts is not required for entry since they rely on wounds, natural openings or vectors, many fungal pathogens achieve entry by mechanical force or enzyme activity or a combination of both. Viruses are usually introduced directly through the plant cells by insects therefore they do not exert mechanical forces. Many fungi are known to apply mechanical forces on the plant they are about to attack. When fungus lands on a plant surface, and contact is established, diameter of the tip of the hypha or radical in contact with the host increases and forms the flattened, bulb-like structure called the appressorium. This increases the area of adherence between the two organisms and securely fastens the pathogen to the plant. From the appressorium, a fine growing point, called the

penetration peg arises and advances into and through the cuticle and the cell wall.

Many fungi develop considerable pressure on a restricted area by producing melanized appressoria which adhere tightly to surfaces and within which massive turgor pressures are developed. The pressure needed for the hypha to penetrate the cell wall is achieved by first firmly attaching the appressorium to the plant surface with proteinaceous glue. The cell wall of the appressorium then becomes impregnated with melanin, making it watertight, and capable of containing the high turgor pressure that builds up within the appressorium. The point of the appresorium that is in contact with the cuticle is called the penetration pore, and the wall is thinnest at this point. The increasing turgor pressure causes the pore to herniate, forming a penetration peg, which applies huge pressure to the host cuticle and cell wall.

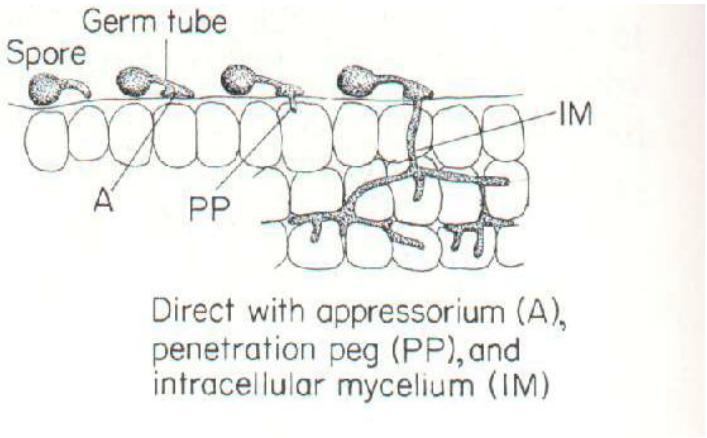


Fig. 2.4 : Steps of Pathogen Spore Germination on host Tissue

2.4.3 Chemical Forces

Weapons of Pathogens

Although some pathogens may use mechanical force to penetrate plant tissues, the activities of pathogens in plant are largely chemical in nature. Therefore, the effects caused by pathogens on plants are almost entirely the result of biochemical reactions taking place between substances secreted by the pathogen and those present in or produced by the plant. Viruses don't themselves directly produce chemicals but they induce host cells to produce the chemicals and that particular chemical may or may not be a chemical already made by the infected host cell. The main groups of substances secreted by pathogens in plants that seem to be involved in production of disease, either directly or indirectly, are enzymes, toxins, growth regulators and

polysaccharides (plugging substances). These substances vary greatly as to their importance in pathogenicity and their relative importance may be different from one disease to another. In general, plant pathogenic enzymes disintegrate the structural components of host cells, breakdown inert food substances in the cell, or affect components of its membranes and the protoplast directly, thereby interfering with its functioning systems. Toxins seem to act directly on protoplast components and interfere with the permeability of its membrane and with its functions. Growth regulators exert a hormonal effect on the cells and their increase or decrease their ability to divide and enlarge. Polysaccharides seem to play a role only in the vascular diseases, in which they interfere passively with the translocation of water in the plants.

(i) Enzymes

During the degradation of the cuticle and wall, a succession of genes are switched on and off in the pathogen, so that cutinase, followed by cellulase, then pectinase and protease are produced, attacking the cuticle, cell wall and middle lamella in the order that they are encountered.

Considerable evidence has accumulated that implicates degradative enzymes in pathogenesis or virulence. Early work was particularly concerned with pectic enzymes, which are likely to be important not only directly in ingress and destruction of structural materials, but also indirectly as a source of nutrient for the pathogen, since the depolymerization of pectic substances to monomers or oligomers of a low degree of polymerization would be readily assimilated. However, partial depolymerization may give rise to oligomers that function as elicitors of defense reactions. More recently, other enzymes such as lipases, cutinases and proteases have been investigated, in some instances with particular reference to the ability of an organism to penetrate its host. A further point for consideration is that some enzymes are able to kill cells.

(a) Cutinases : Cutin is the main component of the cuticle. The upper part of the cuticle is intermixed with waxes, whereas its lower part, in the region where it merges into the outer walls of epidermal cells, is admixed with pectin and cellulose. Cutinases break down cutin molecules and release monomers as well as oligomers of the component fatty acid derivatives from the insoluble cutin polymer e.g. *Fusarium* spp and *Botrytis cinerea*.

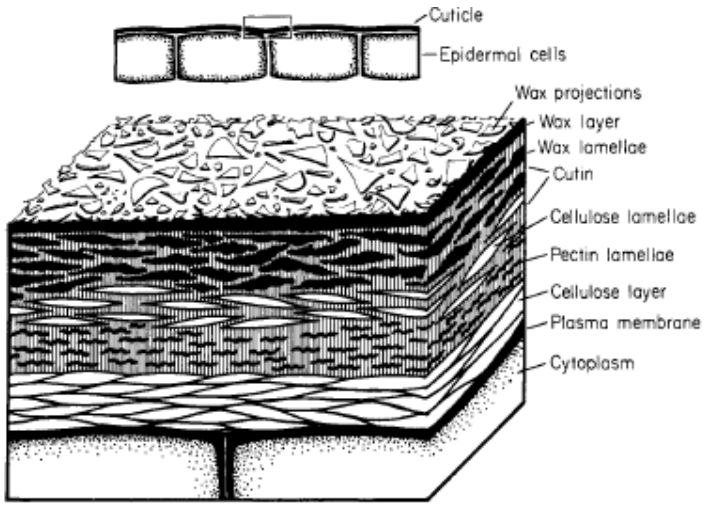
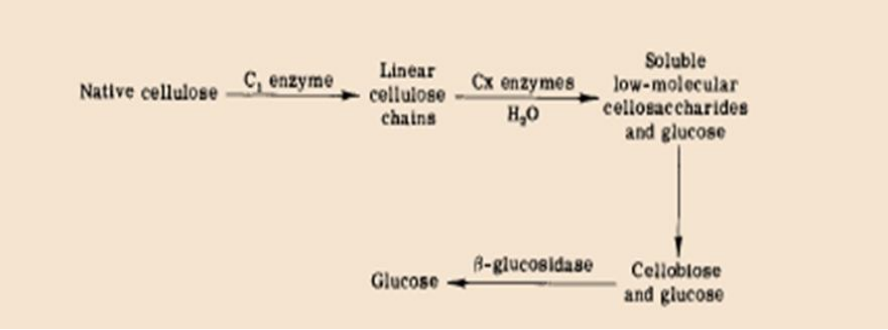


Fig. 2.5 : Schematic Representation of Structure and Composition of Cuticle of Leaf Epidermal Cells

(b) **Pectinases :** Pectin substances constitute the main components of the middle lamella i.e. the intercellular cement that holds in place the cells of plant tissues. Several enzymes degrade pectic substances and are known as pectinases or pectolytic enzymes. The first group of pectic enzymes is pectin methyl esterases, which remove small branches off the pectin chains. The second group of pectic enzymes is chain splitting pectinases called polygalacturonases. They split the pectic chain by adding a molecule of water and breaking the linkage between two galacturonan molecules. Pectin lyases which are the third group of pectic enzymes split the chain by removing a molecule of water from the linkage, thereby breaking it and releasing products with an unsaturated double bond. Examples of pathogens include *Ralstonia solanacearum, Didymella bryoniae*.

(c) **Cellulases :** Cellulose is also a polysaccharide, but it consists of chains of glucose (1-4) D-glucan molecules. The glucose chains are held to one another by a large number of hydrogen bonds. Glucose is produced by a series of enzymatic reactions carried out by several cellulases and other enzymes. One cellulase (C1) attacks native cellulose by cleaving cross-linkages between chains. A second cellulase (C2) also attacks native cellulose and breaks it into shorter chains. These are then attacked by a third group of cellulases (Cx) whch degrade them to the disaccharide cellobiose. Finally, cellobiose is degraded by the enzyme β- glucosidase

into glucose. Saprophytic fungi, mainly certain groups of basidiomycetes, and to a lesser degree, saprophytic bacteria cause the breakdown of most of the cellulose decomposed in nature. In living plant tissues, however, cellulolytic enzymes secreted by pathogens play a role in the softening and disintegration of cell wall material.



(ii) Toxins

Toxins are metabolites that are produced by invading microorganisms and act directly on living host protoplast, seriously damaging or killing the cells of the plant.

Such toxins –

- are extremely poisonous
- are very effective in low concentrations
- can potentially injure host cells
- can very seriously affect membrane permeability
- have the ability to deactivate or inhibit plant enzymes
- can also induce deficiency of essential growth factors.

Some toxins act as a general protoplasmic poison and affect many species of plant representing different families. Others are toxic to only a few plant species or varieties and are completely harmless to others. Many toxins exist in multiple forms that have different potency. Toxins may or may not be Host Specific.

(a) Non-host specific toxin or non host-selective toxins : Several toxic substances produced by phytopathogenic microorganisms have been shown to produce all or part of the disease syndrome not only on the host plant, but also on other species of plants that are not normally attacked by the pathogen in nature.

(1) Tabtoxin- is produced by the bacterium *Pseudomonas syringae pv tabaci* which causes the wildfire disease of tobacco, by strain

of *pv tabaci* occurring on other hosts such as bean and soybean and by other pathovars of *P. syringae* such as those occurring on oats, maize and coffee.

(2) **Phaseolotoxin**- is produced by the bacterium *Pseudomonas syringae pv phaseolicola*, the cause of halo blight of bean and some other legumes.

(3) **Tentoxin-** is produced by the fungus *Alternaria alternata* which causes spots and chlorosis in plants by many species.

(4) **Cercosporin-** is produced by the fungus *Cercospora* and by several other fungi. It causes damaging leaf spot and blight diseases of many crop plants such as *Cercospora* leaf spot of *Zinnia* and gray leaf spot of corn.

(b) **Host specific or host-selective toxins :** These are the substances produced by a pathogenic microorganism in such physiological concentrations which is toxic only to the hosts of that pathogen and shows little or no toxicity against non-susceptible plants.

(1) **Victorin or HV toxin –** is produced by the fungus *Cochliobolus victoriae*. This fungus infects the basal portions of susceptible oat plants and produces a toxin that is carried to the leaves, causes leaf blight and destroys the entire plant.

(2) **T-toxin-** is produced by race T of *Cochliobolus heterostrophus*, the cause of southern corn leaf blight. Race T is indistinguishable from all other *C. heterostrophus* races except for its ability to produce the T toxin.

(3) **HC-toxin-** is produced by Race 1 *of Cochliobolus carbonum* causing northern leaf spot and ear rot disease in maize.

(iii) Growth Regulators

Plant growth is regulated by a small number of groups of naturally occurring compounds that act as hormones and are generally called growth regulators. The most important growth regulators are auxins, gibberellins, and cytokinins, but other compounds, such as ethylene and growth inhibitors, play important regulatory roles in the life of the plant. Plant pathogens may produce more of the same growth regulators as those produced by the plant or more of the same inhibitors of the growth regulators as those produced by the plant. Pathogens often cause an imbalance in the hormonal system of the plant and bring about growth responses incompatible with the healthy development of the plant.

Pathogen growth regulators may cause imbalance in plant hormones by causing stunting of the plant, overgrowth leading to rosetting, excessive root branching, stem malformation, leaf epinasty, premature defoliation and may lead to bud growth suppression.

(a) **Auxins :** It occurs naturally in plants as indole-3-acetic acid (IAA). It is required for cell elongation and differentiation, and absorption of IAA to the cell membrane also affects the permeability of the membrane. Increased IAA levels occur in many plants infected by fungi, bacteria, viruses, nematodes and mollicutes, although some pathogens seem to lower the auxin level of the host e.g *Exobasidium azalea* causing flower gall, *Ustilago maydis* causative organism of corn smut.

(b) **Gibberellins :** These are normal constituents of green plants with a striking growth promoting effects. They speedup the elongation of dwarf varieties to normal sizes and promote flowering, stem and root elongation and growth of fruits. The foolish seedling diseases of rice, in which rice seedlings infected with the fungus *Gibberella fujikuroi* grow rapidly and become much taller than healthy plants is apparently the result, to a considerable extent at least, of the gibberellins secreted by the pathogen.

(c) **Cytokinins :** These are potent growth factors necessary for cell growth and differentiation, and also inhibit the breakdown of proteins and nucleic acids, thereby causing the inhibition of senescence, and have the capacity to direct the flow of amino acids and other nutrients through the point of high cytokinin concentration. Cytokinin activity increases in club root galls, in smut and rust – infected bean leaves. It is partly responsible for several bacterial galls of leafy gall diseae of sweet pea caused by bacterium *Rhodococus fasciens.*

(d) **Ethylene :** Produced naturally by plants and exerts a variety of effects on plants, including chlorosis, leaf abscission, epinasty, stimulation of adventitious roots and fruit ripening. In the fruit of banana infected with *Ralstonia solanacearum*, the ethylene content increases proportionately with the (premature) yellowing of the fruits, whereas no ethylene can be detected in the healthy fruits.

(iv) **Polysaccharides :** Fungi, bacteria, nematodes and possibly other pathogens constantly release varying amounts of mucilaginous substances that coat their bodies and provide the interface between the outer surface of the microorganism and its environment. The role of the

slimy polysaccharides in plant disease appears to be particularly important in wilt diseases caused by pathogens that invade the vascular system of the plant. Large polysaccharide molecules released by the pathogen in the xylem may be sufficient to cause a mechanical blockage of vascular bundles and thus initiate wilting.

2.5 Colonization

Once a pathogen has arrived in the vicinity of a potential host plant or, as may happen in the case of soil-borne pathogens, a plant root has arrived in the vicinity of a pathogen, subsequent events depend on the production and perception of signals by both partners. In soil, pathogens may be influenced by compounds exuded from the host root. Motile stages may be attracted or repelled and the germination of sessile propagules stimulated or inhibited. Air-borne pathogens generally rely upon large populations of propagules to ensure that at least some of them alight on a suitable host. At this point, adhesion is a necessity to prevent the propagule being washed off the plant and, for at least one fungal pathogen; adhesion has been established as a prerequisite for germination. Following adhesion, germination, which may be under the control of topological or chemical signals from the host, occurs and in some instances such signals lead to the differentiation of infection structures. These, too, require firm anchoring to the surface of the plant if any mechanical force is to be exerted.

A successful infection requires the establishment of a parasitic relationship between the pathogen and the host, once the host has gained entry to the plant. There are two broad categories of pathogens- biotrophs (those that establish an infection in living tissue) and necrotrophs (those that kill cells before colonising them, by secreting toxins that diffuse ahead of the advancing pathogen). These two kinds of pathogens are also sometimes known as 'sneaks' and 'thugs', because of the tactics they use to acquire nutrients from their hosts. . Biotrophs often feed through haustoria, which penetrate the host cell wall, almost certainly through the agency of degradative enzymes, and invaginate but do not penetrate the host plasma membrane.

Necrotrophs do not produce specialised penetration structures. Instead, they kill host cells by secreting toxins, then degrade the cell wall and middle lamella, allowing their hyphae to penetrate the plant cell walls and the cells themselves. The toxins produced by necrotrophs can be specific to the host or non-specific. Non-specific toxins are involved in a broad range of plant-fungus or plant-

bacterial interactions, and will therefore not usually determine the host range of the pathogen producing them. Necrotrophs often enter the plant through wounds and cause immediate and severe symptoms. For necrotrophs the role of degradative enzymes seems clear. They are required not only for penetration and colonization of plant tissue but also to reduce the high molecular weight components of these tissues to products which they can metabolize. In the case of soft rotting organisms, this often results in the 'mushy' symptoms that give these diseases their name.

An intermediate category of parasite is the hemibiotrophs, which start off as biotrophs and eventually become necrotrophic, employing tactics from both classes of pathogen. . In hemibiotrophic infections, intercellular hyphae can form haustoria in living mesophyll cells, but as the lesion expands under favourable conditions, those heavily parasitised cells at the inner, older part of the colony collapse and die.

Pathogens that colonise the surface of plants, extracting nutrients through haustoria in epidermal or mesophyll cells are termed ectoparasites. The haustoria are the only structures that penetrate the host cells. Some parasites colonise the area between the cuticle and the outer wall of the epidermal cells, penetrating host epidermal and mesophyll cells with haustoria. These are called **sub-cuticular infections**. Pathogens can also form colonies deeper in the plant tissues. These are mesophyll and parenchyma infections, and can be necrotrophic, hemibiotrophic or biotrophic relationships. Colonization of the host by viruses is a special case. Viruses move from cell to cell through plasmodesmata but they may be replicated at sites that are some distance away. In the case of systemic infections long-distance movement of viruses occurs through the phloem or xylem and normally requires an intact capsid protein. Once in the conducting tissues of the plant, movement of the virus and unloading follows as that of solutes but the mechanisms remain unknown.

Viruses, mildews and rusts develop specialised biotrophic relationships with their hosts. Intercellular hyphae of downy mildew colonise host mesophyll cells and form haustoria. The mildew sporulates and the infected cells eventually die, although necrosis is delayed and contained, compared to that caused by necrotrophic pathogens. Rust fungi can also delay senescence in infected cells while they sporulate. Vascular infections usually cause wilting and discoloration as a result of the physical blockage of infected xylem vessels. True vascular wilt pathogens colonize the vascular tissue exclusively, although other pathogens can cause the same symptoms if they infect the vascular system

as well as other tissues. There are a few pathogens that manage to achieve systemic infection of their host. For example, many viruses can spread to most parts of the plant, although not necessarily all tissues. Some downy mildews can also systemically infect their host by invading the vascular tissue and growing throughout the host, causing deformation, rather than necrosis. Finally, there are some pathogens that complete their entire life cycle within the cells of their host, and may spread from cell to cell during cytokinesis. These are endobiotic infections.

2.6 Summary

Since many pathogens have to breach the barriers of plant waxes, cutin and suberin that cover plants as well as plant cell walls before establishing a parasitic relation with their hosts. Some soil-borne pathogens locate their hosts through chemical signals and these are also important in subsequent events such as the germination of propagules, chemotropism of germ tubes and the differentiation of infection structures, the last of these also being influenced by physical features of the host. Adhesion is often required for successful penetration, particularly where this is achieved by the exertion of mechanical force. However, enzymes that degrade the surface layers of plants, such as waxes, cutin and suberin are also critical for entry by many pathogens. Once past these surface layers the pathogen usually has to breach the cell wall and for this a range of pectolytic enzymes, cellulases and xylanases as well as enzymes involved in the degradation of lignin are required. In some instances, other enzymes are inferred to have important roles to play in pathogenicity or virulence such as proteases and membranlytic enzymes. The products of degradative enzymes acting on host tissues are sources of nutrition for necrotrophic pathogens but the subtler biotrophic pathogens feed through specialized structures called haustoria. Viruses and viroids lack motility and therefore face a particular problem in colonizing their hosts after entry. Long distance spread is by passive movement in the xylem or phloem.

2.7 Glossary

- **Adhesion**: the act or state of adhering; state of being adhered or united.
- **Appressorium**: an enlarged fungal filament that adheres to the surface of the host, prior to penetration.
- **Penetration**: initial invasion of a host by a pathogen.

- **Penetration Peg**: a structure found in some plant parasitic fungi. The penetration peg is a specialised, narrow, hyphal strand located on the underside of an appressorium that penetrates the epidermal cell wall.
- **Propagules**: any part of an organism capable of independent growth (e.g., a spore, a mycelial fragment, etc.).
- **Hydathode**: a specialized epidermal leaf structure with one or more openings through which water is discharged from the leaf interior to its surface.
- **Hemibiotroph**: a parasite that initially forms an association with living cells of the host, much like a biotroph, and then in the later stages of infection it becomes necrotrophic, actively killing host cells.
- **Necrotroph**: an organism (parasite) that causes the death of host tissues as it grows through them, obtaining its energy from the dead cells.
- **Biotroph**: an organism that can live and reproduce only on another living organism. A biotroph is completely dependent on the host organism as a source of nutrients, i.e. it is an obligate parasite.
- **Ectodesmata**: Ectodesmata are pits in the outer wall of epidermal cells, which connect to valleys in the cuticle surface, exposing the plasma membrane. Plant metabolites leak from the ectodesmata and accumulate in the valleys on the leaf surface, providing nutrients for pathogens and saprophytes.
- **Ectoparasite**: a parasite that lives and feeds from the exterior of its host's cells or tissues.
- **Haustorium**: a specialized branch of a fungal hypha formed inside a living cell of the host plant in order to obtain nutrients.
- **Thigmotropism**: oriented growth of an organism in response to mechanical contact, as a plant tendril coiling around a string support.
- **Topology:** study of those properties of geometric forms that remain invarian t under certain transformations, as bending or stretching.
- **Herniate**:to protrude abnormally from an enclosed cavity or from the body so as to constitute a hernia.

2.8 Self-Learning Exercise

Section A : (Very Short Answer Type Questions)

1. The obligate parasitic fungi absorb their nourishment from the host cells through the _____.
2. Most simple pathway for pathogen entry is via ____in the plant surface.
3. Any part of an organism capable of independent growth is termed as _____.
4. _____is an enlarged fungal filament that adheres to the surface of the host, prior to penetration.
5. An organism that can live and reproduce only on another living organism is a _____.

Section B : (Short Answer Type Questions)

1. Write characteristics of various toxins produced by the pathogens.
2. What are the various ways in which pathogens can directly penetrate into the host tissues?
3. Write the importance of plant-pathogen adhesion mechanism.
4. How does the topology of a plant surface affect the infection by a pathogen?
5. Name a few toxins produced by the pathogens

Section C : (Long Answer Type Questions)

1. Give a detailed account of phenomenon of infection process with special reference to penetration.
2. Describe the role of various enzymes in the infection process.
3. Write differences between necrotrophs, biotrophs and hemi-biotrophs.

Answer key of Section – A

1. Haustoria
2. Pre existing opening
3. Propagule
4. Appressorium
5. Biotroph

2.9 References

- Plant Pathology by G.N. Agrios
- Plant disease by R.S. Singh
- Principles of plant pathology by R.S. Singh
- Introduction to Plant Pathology by Richard N. Strange

Unit - 3

Host Pathogen Interaction-II: Defense Mechanism

Structure of the Unit:

3.0 Objectives

After studying this unit, you will understand the various mechanisms by which the plants act to defend themselves against attack from the vast array of viruses, bacteria, fungi, parasitic plants, nematodes and insects in their environment. Plants are equipped both with pre-formed, constitutive chemical and mechanical barriers as well as with inducible defense systems. In this unit, you will get a comprehensive knowledge of plant defense mechanisms in terms of:

- Pre existing or Pre-Infective Defense Mechanisms
- Post Infective or Induced Defense Mechanisms
- Plant Defense Elicitors in response to Pathogen Attack
- Signal transduction pathway in Plants' Defense System

3. 1 Introduction

Adjustment is probably, one of the most important quality of a natural system that ensures its efficient working and survival, be it the host plant or pathogen. On planet earth, the green plants (autotrophs) constitute the only biological system capable of converting solar energy (electro-magnetic radiations) into chemical energy. These are always surrounded by a whole world of microbes and insects which are dependent directly or indirectly on the producers. These organisms, in a way, exploit these natural resources even if it is harming the plants. Plants as a biological system resist this exploitation, at all levels and by all means. The co evolution, forced by co-existence with pathogen, has led to the development of defense mechanism in plants. Thus, resistance against any 'harmful act' has become a natural and universal response of plant system. The resistance against parasites/pathogen is the heritable trait of plants by virtue of which they resist attack by parasites/pathogens or their activities. The defense mechanism(s) has ensured the survival of plants in spite of living amongst some of the most potentially devastating pathogens.

3.2 Pathogenesis and Host Response

Analysis of most of the host parasite relationships reveals that on the pattern of pathogenesis, the plants on their part, do exhibit defense mechanisms (structural and chemical) as soon as challenged by the pathogen. The moment pathogen propagules come in contact with host surface, the plants due to their inherent characters guard themselves using several naturally occuring physical and chemical barriers (preexisting) resisting penetration, and if at all the penetration occurs, the host reacts by different means resulting in formation of physical and chemical barriers(Fig 3.1). Thus the plant defense mechanisms can be studied according to the sequence of the events that lead to a disease attack on them.

A. Pre Existing or Pre Infective Defense mechanism

a. Physical or Structural Barriers

b. Biochemical Barriers

B. Post Infective or Induced Defense mechanism

a. Physical or Structural Barriers

b. Biochemical Barriers
c. Signal Induced Responses

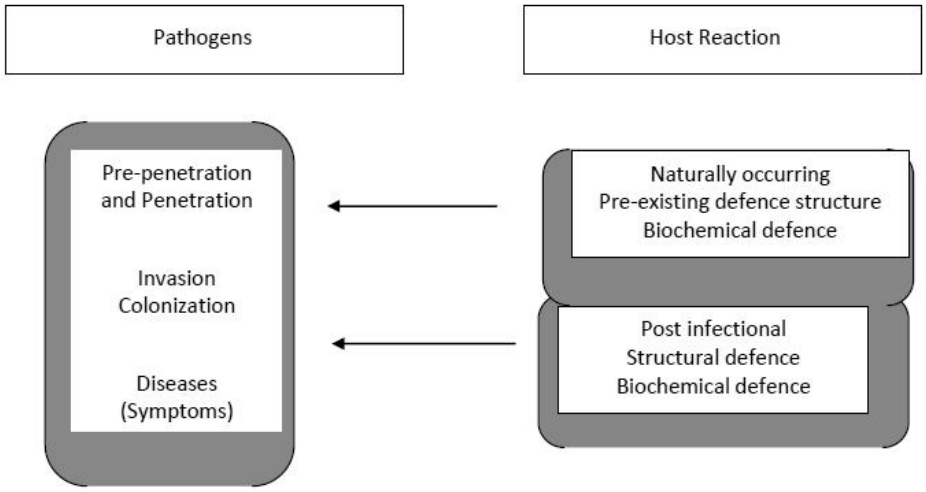


Fig. 3.1 : Defense Mechanisms: Pre-infective or Post infective

3.3 Pre existing or Pre-Infective Defense Mechanism

3.3.1 Physical or Structural Barriers

The first line of defense in plants is present in its epidermal surface. Several characters of the plants surface function as barriers to penetration which pathogen must breach to enter the host. The Pathogens enter the plant host by penetrating the epidermis along with cuticle and cuticular wax and number of natural openings existing before the onset of the pathogenesis can obstruct penetration. If the pathogen succeeds in penetration; it encounters pre-existing internal structural barriers. The external and internal structural barriers existing before pathogen attack are also called Pre-existing defense structures or passive/static or anti-infection structures.

- **Wax and cuticle :** The cuticle covers the epidermal cells of plants and consists of pectin layer, a cutinized layer and a wax layer. Cutin is composed of fatty acids. Waxes are mixture of long chain aliphatic compounds which prevent the retention of water on plant surface essential for spore germination. A negative charge usually develops on leaf surfaces due to fatty acids. This condition repels air-borne spore / propogules. Only few pathogens are known to dissolve cutin enzymatically. Examples:

Monilinia fructicola penetrates cuticle of cherry leaves but not of *Gingko biloba* leaves; the latter contains abundant cutin than the former. *F. solani f sp. pisi* induces the enzyme cutinase production by specific antibodies and inhibitors.

- **Epidermal layer :** Epidermis is the first layer of living host cells that comes in contact with the attacking microbes. The toughness of epidermis is due to the polymers of cellulose, hemicelluloses, lignin, mineral substances, polymerized organic compounds, suberin, etc. Potato tubers resistant to *Pythium debaryanum* contain higher fibre. Silicon accumulation in epidermal walls provides resistance against fungal attack. Suberization of epidermis confers protection in citrus plants against *Xanthomonas axonopodis pv. Citri*. A functional defense mechanism has been observed in some varieties where stomata open late in the day only when moisture on leaf surface has dried and the infective propagules have become non-functional.
- **Hydathodes** are natural openings on the edges of leaves that serve to excrete excess water from the interior. They are easy entry points of bacterial pathogens. Similar to hydathodes, are the nectarthodes in inflorescence of many plants. They secrete sugary nectar and this serves as a barrier to those organisms that cannot tolerate this condition and thus, cannot enter through nectaries. Leaf hairs on leaves and on nectaries also resist entry of pathogens. High hairlines of leaves and pods in chickpea are resistant character against *Ascpchyta rabei*. Groundnut varieties showing resistance to *Cercospora* leaf spots have thick epidermis-cum cuticle and compact palisade layer, few and smaller stomata and high frequency of trichomes on the abaxial surface of leaf.
- **Lenticels** are opening in outer corky walls of plants involved in gaseous exchange. They are weak in defense unless the cork cells within them are suberized. After suberization and periderm formation, lenticels are more resistant to invasion by pathogens.

3.3.2 Biochemical Barriers

Plants secrete different chemicals which directly interfere with activities of the pathogen and reduce the effect of pathogenesis, thereby preventing or reducing infection. These chemicals and the biochemical conditions that develop may act either directly through toxins or lytic enzymes on the invader, or indirectly through stimulating antagonistic plant surface microflora. Such protective

compounds against pathogens pre-existing in plants are called **Constitutive Antibiotics** and those, which are formed in response to wounds are called Wound **Antibiotics**.

- **Release of anti-microbial compounds :** Plants, in the normal phases of growth and development, release certain gases as well as organic substances from the epidermal surfaces of leaves and roots, commonly known as leaf and root exudates. These exudates are a mixture of many substances containing sugars, amino acids, organic acids, enzymes, glycosides, etc. These materials have profound effect on the nature of surrounding environment, particularly the micro flora and fauna of phyllosphere and rhizosphere. Although these substances are ideal nutrients for microbes and help in the germination and growth of several saprophytes and parasites, there are a large number of inhibitory substances also present in these exudates. These inhibitory substances may directly affect the microorganisms or they may also encourage certain groups to dominate the environment and function as antagonists of the pathogen.
- **Inhibitors present in the plant cells :** In many host-parasite interactions, pre-existing toxic substances in the host cells form the basis of resistance. In resistant variety, these substances are formed in abundance while in susceptible variety; they may be less or completely absent. Several phenolic compounds, tannins and some fatty acid-like compounds such as di-enes which already pre-exist in high concentrations in host cells have been implicated for the resistance of young tissues against parasitic fungi such as *Botrytis*. Many such compounds are potent inhibitors of many hydrolytic enzymes. Several other kinds of pre-formed compounds such as saponins (glycosylated steroidal or triterpenoid compound), tomatine in tomato and avenacin in oats, have antifungal membranolytic activity. The fungal pathogens which lack enzymes (saponinases) that breakdown the saponins are prevented from infecting the host. Several pre-formed plant proteins have been reported to act as inhibitors of pathogen proteinases or hydrolytic enzymes. Similarly, lectins (proteins that bind to certain sugars) cause lysis and growth inhibition of many fungi. Plants surface cells also contain variable amounts of hydrolytic enzymes such as glucanases, chitinases, etc, which may cause breakdown of pathogen cell wall components.
- **Absence of Recognition factors in plants :** The first step in infection process is the cell-to-cell communication between host and pathogens. Plants of many species or varieties may not be infected by pathogen if their

surface cells lack specific recognition factors. If the pathogen does not recognize the plant as one of its hosts, it may not adhere to the host surface or it may not produce infection substances such as enzymes, or structures (appressoria, haustoria). These recognition molecules can be a variety of oligosaccharides, polysaccharides or glycoproteins.

- **Absence of Host receptor and sites for toxins :** In many host- parasite interactions, the pathogen produces host specific toxins, which are responsible for symptoms and disease development. The molecules of toxin are supposed to attach to specific sensitive sites or receptors in the cell. Only the plants that have such sensitive sites become diseased.
- **Essential nutrients and growth factors :** The fact that many facultative saprophytes and most of the obligate parasites are host specific and sometimes are so specialized that they can grow and reproduce only on certain varieties of only those plant species, suggests that for these pathogens the essential nutrients and growth factors are available only in these hosts. Absence of these nutrients and stimulus automatically make the other varieties and species unsuitable hosts for such pathogens.

3. 4 Post Infective or Induced Defense Mechanism

Plants have to face the wide variety of pathogens (enemies) throughout their life span standing at one place. Thus nature and the process of evolution have built in them a strategically designed pre-existing (structural and biochemical) defense mechanism. The real value of this system has not been critically examined. It appears that these pre-existing defense mechanisms help plants in warding-off most of microbes as non pathogens. But it does not seem to be sufficient. Disease occurs when pre-exisiting defense mechanism are not enough to check the entry of pathogen and a pathogen avoids timely-eliciting active defense system in plant tissue or habits active defense response by secreting metabolic toxins.

The induced/active defense mechanism in plants may operate at different levels-

- Induced Physical or Structural Defense
- Induced Biochemical Defense

The activation or induction of defense mechanism may be both specific and non-specific type. Several structural changes are known to be induced by a range of biotic or abiotic elicitors. These dynamic defense mechanisms prevent

further colonization or spread of pathogen. Active defense in plants involves cellular defenses that rely upon preformed surveillance systems which are encoded by resistance genes. The receptor-proteins are strategically located in cell membrane to detect the pathogen or factor translocated by pathogens. The ability of plant to mount an active defense response is again under genomic control.

3.4.1 Induced Physical or Structural or Histological Barriers

Even after the establishment of infection in plant cells, the host defense system tries to create barriers for further colonization of tissues. This may be accomplished at various levels.

- **Lignification :** Lignified cell wall provide effective barrier to hyphal penetration(Fig 3.2 a). They also act as impermeable barrier for free movement of nutrient causing starvation of pathogen as in Potato infection by *Phytophtora infestans* or Cucumber infection by *Cladosporium cucumerium.*
- **Suberization :** In several plants, as soon as the infection occurs in the cells, the infected cells become surrounded by suberized cells (Fig 3.2 c). This isolates them from healthy tissue. Corky layer formation is a part of natural healing system of plants as in common scab of potato and rot of sweet potato.
- **Formation of Abscission layers :** Abcission layers are naturally formed in plants as a device for dropping –off older leaves and mature fruits. Many plants use this device as a defense mechanism also by dropping-off infected or invaded plant tissue or parts, along with pathogen (Fig 3.2 b). Shot holes in leaves of fruit trees are a common feature.
- **Tyloses Formation :** The tyloses are normally formed into the older xylem vessels of plants by protrusion of walls of xylem parenchyma cell through the way of pits. The size and number of tyloses physically block the vessel. The tyloses are inductively formed much ahead of infection, thus blocking the spread of pathogen (Fig 3.2 d). It suggests biochemical elicitors and movement of tyloses inducing factors (TIF) up the stem as in Sweet potato infection by *Fusarium oxysporum f. sp. Batatas.*
- **Gum deposition :** The gums and vascular gels quickly accumulate and fill the intercellular spaces or within the cells which surround the infection thread and infective haustoria (Fig 3.2 e), resulting in the death or starvation of infective propagules.

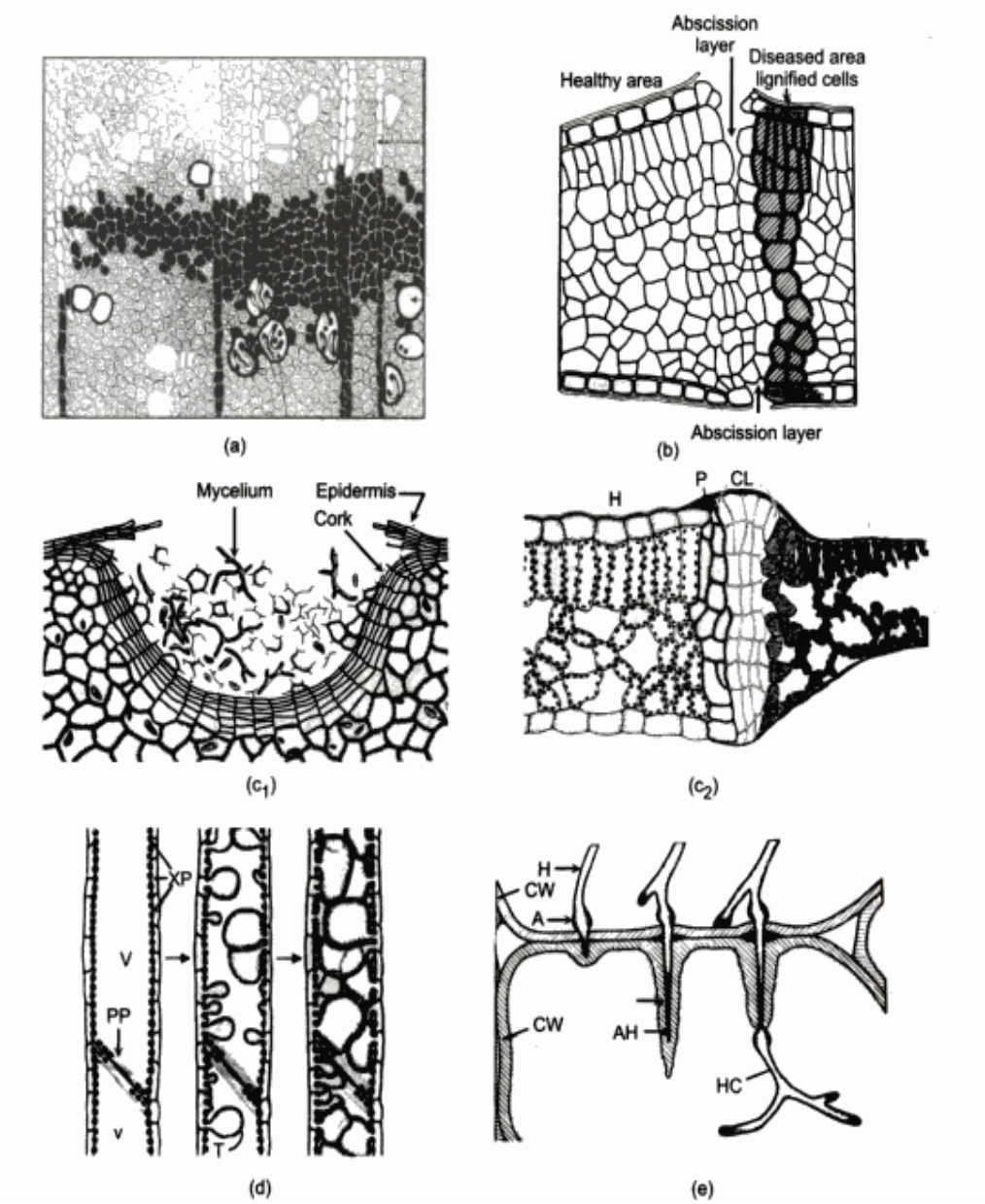


Fig. 3.2 : Mechanism of host resistance:

(a) Lignification (b) Abscission layer formation (c) C1 & C2 Cork layer formation (d) Tyloses formation (e) Sheathing of infection threads

3.4.2 Induced Biochemical Changes

The induced biochemical changes in host plants are the last line of host defense. This response may vary from a plant or a specific plant tissue; from susceptible to resistant variety as per their genetic potential. The role of biochemical factor in host defense is based on the following four attributes:

1. The substance is associated with protection against disease at the site where protection occurs.

2. The substance can be isolated from the host showing protection against the disease.
3. Introduction of isolated substance to the appropriate susceptible host confers protection.
4. The nature of protection so induced resembles that of the natural agents of a resistant plant.

- **Toxic substances production :** Rapid production, suitable modifications and/or accumulation of chemicals toxic to pathogen up to effective concentrations is an important component of overall active defense strategy of plants. Slow production or accumulation or low levels of similar chemicals have reported in susceptible host plants also.
- **Role of Phenolic compounds :** The phenolic compounds, viz., chlorogenic acid, caffeic acid and oxidation products of floretin, hydroquinone, hydroxyquionones and phytoalexins are main toxic chemicals produced to inhibit pathogen or its activities. Some of these are phe-formed toxic chemicals while others may be *de novo* synthesized or modified to more toxic forms. The enzymes involved in chemical pathways are already present in host cell (pre-existing).
- **Role of Phytoalexins :** Most common response of plants to stress, whether biotic (pathogens/insects) or abiotic (wounding), is the production and accumulation of substrates that can inhibit the growth and activities of the biotic factors or may help in healing process. Muller and Borger proposed the concept of phytoalexins in their study on hypersensitive reaction of potato to avirulent *P.infestans* strains. Phytoalexins are antibiotics produced in plant- pathogen interactions or as a result of reponse to injury or other physiological stimulation.
- **Role of new protein synthesized :** Post-infection changes in host cells involve production and modification of large number of proteins (structural and enzymatic), which have important role in defense mechanism. The enzymes are required for various synthetic pathways (normal or modified) for production of resistance related substances. In addition, phenol-oxidizing enzymes have vital role. The influence of these changes may be confined to infection site or nearby cells. Increased synthesis and activity of Phenyl Ammonia Lyase (PAL) has been reported in several bacterial and *vital* pathogens in resistance reactions. PAL plays a key role in synthesis of phenols, phytoalexins and lignin. The effectiveness of resistance depends on

speed and amount of synthesized products and their hurried movement to neighbouring healthy tissues to create timely defensive barriers.

- **Inactivation of enzymes and toxins :** The role played by chemical weapons (toxin and enzymes) of pathogens during pathogenesis is well established. The necrotrophs and hemibiotrophs employ more of these substances by causing more damage in tissues as compared to specialized obligate parasites. The defense strategy of resistant plants works through activity of phenols, tannins and protein as enzymes inhibitors. The phenolics are not anti-fungal but make pathogen ineffective by neutralizing their enzymes. In immature grape fruits catechol-tannin is known to inhibit enzymes produced by *Botrytis cinerea*. Toxins are known to be involved in pathogenesis. The resistance to toxins, in host, will be resistance to pathogens. This can be achieved by detoxification or lack of receptor sites for these toxins.
- **Role of altered biosynthetic pathway :** The post infection metabolism of host tissue is changed (stress physiology) to cope up with the advancing activities of pathogen. New enzymes (proteins) are produced in an effort to synthesize defense related substances. Most of these compounds are formed through Shikimic acid pathway and modified acetate pathway. Respiration in diseased tissue is invariably increased; a part of glycolysis is replaced by pentose pathway, which yields four carbon compounds. It is possible that in early stages of infection the gene regulation of host cell is influenced and some specific genes are triggered on in order to make new substances required for active defense.
- **Active defense to pathogens :** Induction of host resistance, structural or biochemical seems to be universal in plants. Active defense responses have been reported against all classes of pathogens (fungi, bacteria, viruses and nematodes). Active defense response may lead to incompatible host-pathogen interaction.

Thus, on entry of the pathogen, a temporary increase in cellular metabolic activities occurs in the host. Due to stress caused by increased metabolic activity cells die rapidly showing hypersensitive reaction. Rapid death of cells is correlated with increased degree of resistance in most diseased systems. When the infected tissues are reaching the necrotic stages, metabolism of neighboring tissues is also increased and phenolics and other compounds are accumulated. In this process, the synthesized compounds move from healthy to diseased tissues. The reactions expressed by hypersensitivity form common

phenols, phytoalexins and other abnormal substances. The oxidized products of phenolics may detoxify the toxins or inactivate other weapons of the pathogen. When spread of the pathogen is checked, the neighboring healthy tissues with accelerated metabolic activities try to isolate the damaged parts by forming new tissues and eliminate the disease/pathogen.

Host defense, pre-existing or induced, is a multi-component strategy where several factors work together to fashion the final outcome.

3.5 Elicitors

Elicitors, in biology, are compounds that, when introduced into a living organism, signal the activation or synthesis of another compound. An example of such a molecule is jasmonic acid, which stimulates the biosynthesis of delta-viniferin in grapevine cell cultures. Another example is chitosan which is used in agriculture as a natural biocontrol agent. The recognition of a potential elicitor results in several early responses including rapid ion fluxes, activation of kinase cascades and the generation of active oxygen species (AOS). These early events are followed by other defense responses including induction of hypersensitive response (HR), a localized form of programmed cell death (PCD) limiting pathogen spread, further reinforcement of the cell walls, and production of antimicrobial compounds such as defense proteins and phytoalexins. Many of the plant defense responses are mediated by an interacting set of signal molecules including jasmonic acid (JA), ethylene (ET) and salicylic acid (SA). Originally the term elicitor was used for molecules capable of inducing the production of phytoalexins, but it is now commonly used for compounds stimulating any type of plant defense. Eventually, the induction of defense responses may lead to enhanced resistance.

Elicitors differ from hormones (compounds produced in one part of an organism to cause a change in another part of that organism) as elicitors do not have to be produced within the organism in which they are eliciting a response and these are usually not naturally occurring in the organism. This broader definition of elicitors includes both substances of pathogen origin (exogenous elicitors) and compounds released from plants by the action of the pathogen (endogenous elicitors). Elicitors do not have any common chemical structure, but belong to a wide range of different classes of compounds including oligosaccharides, peptides, proteins and lipids. These act as signal compounds at low concentrations, providing information for the plant to trigger defense, distinguishing elicitors from toxins, which may act only at higher

concentrations and/or affect the plant detrimentally without active plant metabolism.

Most elicitors seem to fall into two broad categories. Many of them are constitutively present in the pathogen cell wall as structural components, e.g. glucan and chitin fragments as well as bacterial flagellin and lipopolysaccharides (LPS). Another class of elicitor plays a role as virulence determinants, e.g. harpins, products of *avr* genes, while the function of some elicitors remains elusive. In certain plant–pathogen interactions the main elicitors for the plant response might be produced due to the activity of plant cell wall-degrading enzymes. These enzymes that are often essential virulence factors and provide the attacking pathogen with nutrients, also release pectic fragments (oligogalacturonides, OGAs) that can act as endogenous elicitors. This vast array of elicitor compounds precludes the presence of a common elicitor motif and suggests that plants have the ability to recognize a number of structurally distinct molecules as signals for pathogen defenses. Early signal transduction pathway studies with elicitors revealed striking similarities between plants and animals, in molecules which use to perceive and transmit signals associated with invaders. These observations highlight the conservation of types of elicitors produced during defense-related signaling system in the different living kingdoms throughout evolution that have led to the innate immunity system.

Various general elicitors described in plant defense reactions-

1. Oligogalacturonides- Pectic fragments from plant cell wall
2. Chitosan- Chitin fragments from fungus cell wall
3. β-Heptaglucosan- Component of the mycelia cell walls of certain oomycetes
4. Lipopolysaccharides -Gram-negative bacteria
5. Elicitins (cryptogein)- Proteins from oomycetes
6. Avr2, Avr4, Avr5, Avr9 -Products of the corresponding *avr* genes
7. Pep-13-Oligopeptide of 13 amino acids
8. Flg22 22–amino acid N-terminal fragment of bacterial flagellin
9. BcPG1- Endopolygalacturonase from *Botrytis cinerea* Grapevine
10. AvrPto- *Pseudomonas syringae pv. tomato*

3.6 Signal Transduction Pathway

Plants continuously defend themselves against attack from fungi, bacteria, viruses, invertebrates, etc. They do not have immune system but rather possess

preformed and inducible defense resistance. Plant disease resistance often depends on ability of recognition signals activation to initiate response.

Plant pathogen attacks are perceived through pathogen derived compounds or plant-derived molecules that elicit defense reactions. Despite the large variety of elicitors, general schemes for cellular elicitor signaling leading to plant resistance are known. The early signaling events that happen after elicitor perception, including changes in the activities of plasma membrane proteins, reversible protein phosphorylations, production of nitric oxide and active oxygen species and variations in free calcium concentrations in cytosol and nucleus. These events occur within the first minutes to a few hours after elicitor perception. One specific elicitor transduction pathway can use a combination or a partial combination of such events which can differ in intensity depending on the stimulus. The links between the signaling events allow amplification of the signal transduction and ensure specificity to get appropriate plant defense reactions. Disease spreads only in susceptible plants, as they are unable to recognize the pathogen or respond too slowly. In such a case, Plant pathogen interaction is generally **Compatible** (Fig 3.3) as the pathogen produces characteristics symptoms.

When a putative pathogen is able to get over the host barriers and if the plant recognizes the invader, a rapid induction of defense responses by the resistant plant can prevent the pathogen from developing which makes this interaction **Incompatible** (Fig 3.4) as the plant resist symptoms development and pathogen reproduction.

Biologically, signal transduction refers to any process by which a cell converts one kind of signal or stimulus into another. Signal transduction at the cellular level refers to the movement of signals.

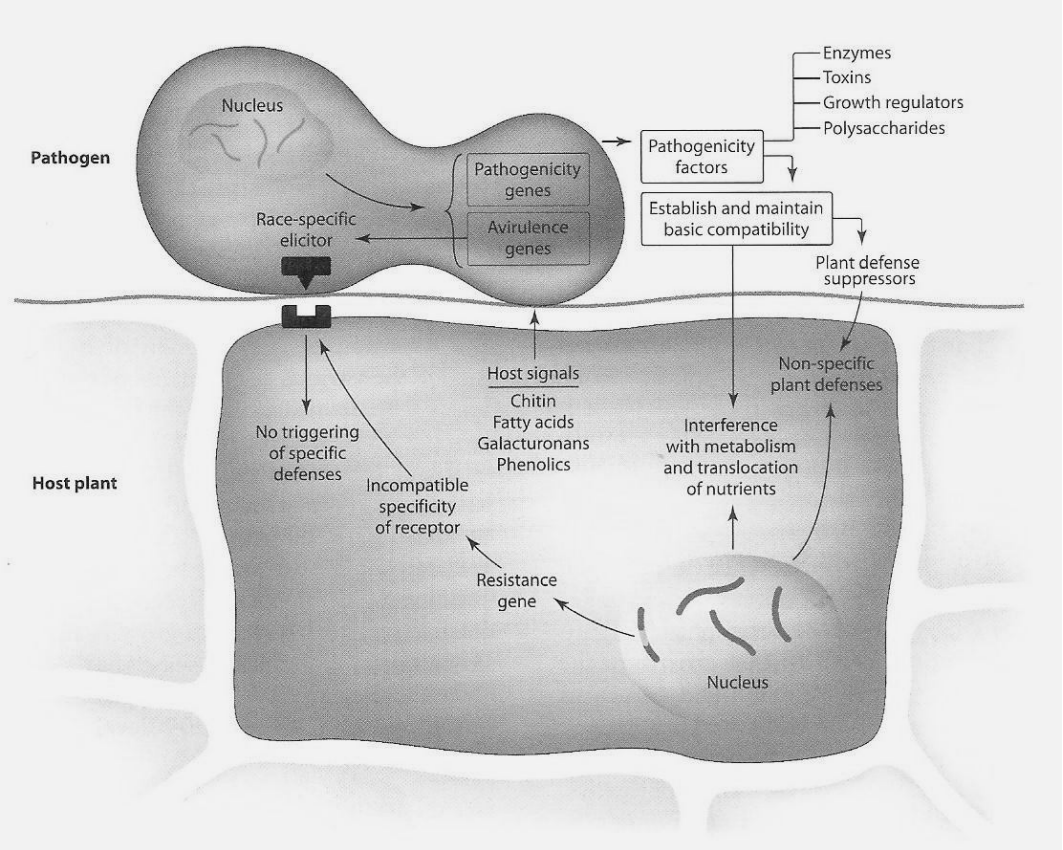


Fig. 3.3 : Compatible Reaction

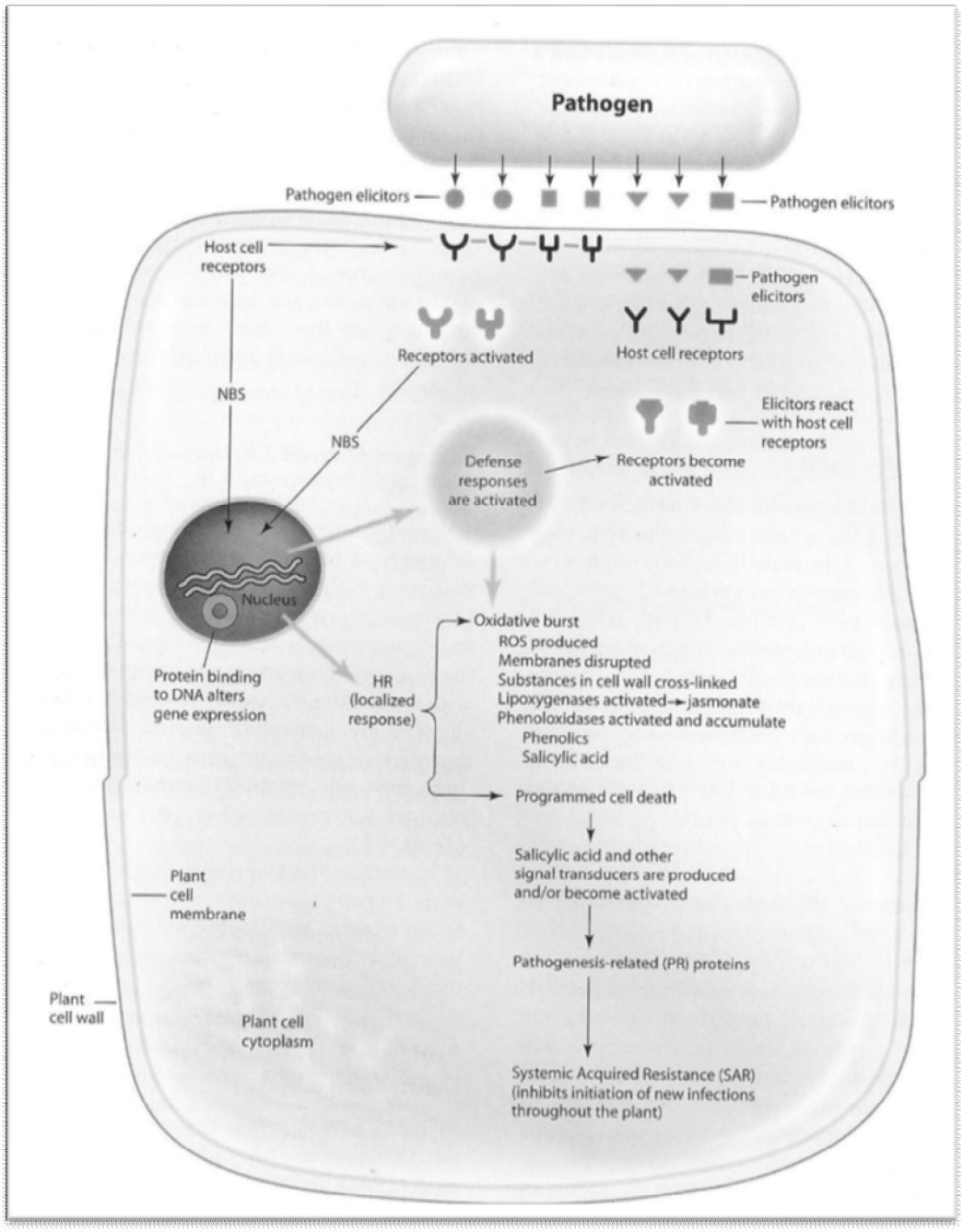


Fig. 3.4 : Incompatible Reaction

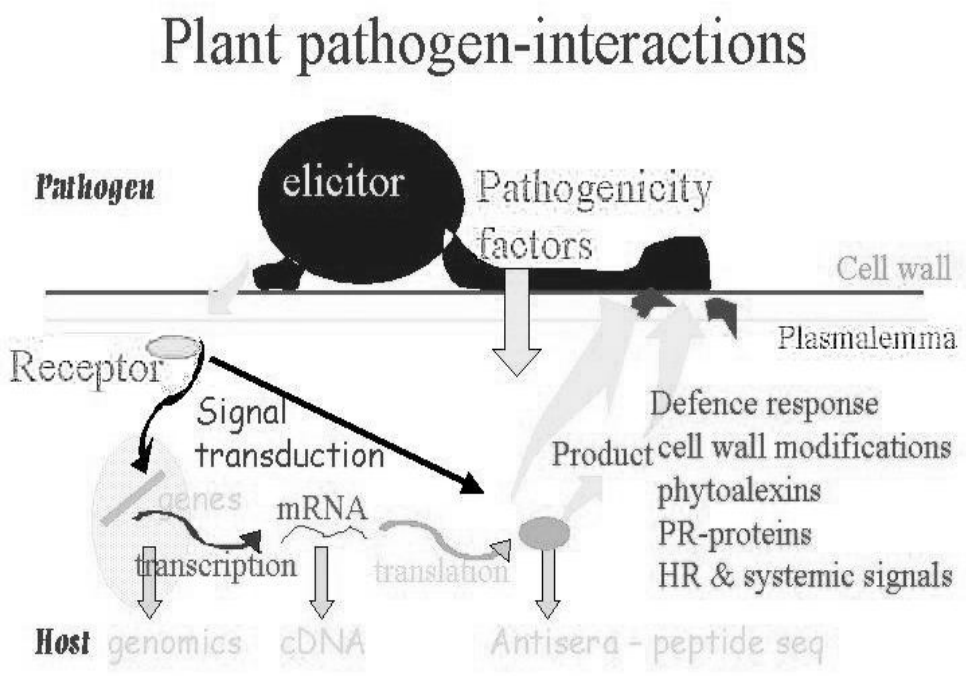


Fig. 3.5 : Generalised View of Signal Transduction

Steps of Signal Transduction during Host Pathogen Interactions:

- Recognition
- Signal perception
- Signal transduction
- Signal response
- Termination of signalling events

3.6.1 Recognition

It is the first step in interaction and a pre requisite to initiate the compatible or incompatible reaction. It depends upon that if the initial recognition signal received by pathogen favors its growth and development, the reaction is compatible and the outcome is a disease. If signal suppresses pathogen growth and activity, the reaction is incompatible and the disease will be aborted. Recognition depends on generation of elicitors by the pathogen. Recognition of pathogen triggers a large range of inducible defense mechanisms which lead to resistance in plants. Various mechanisms induced at site of infection like synthesis of antimicrobial compounds called phytoalexins, alteration in synthesis of cell wall structural proteins and response is generated which is involved in defense signal transduction.

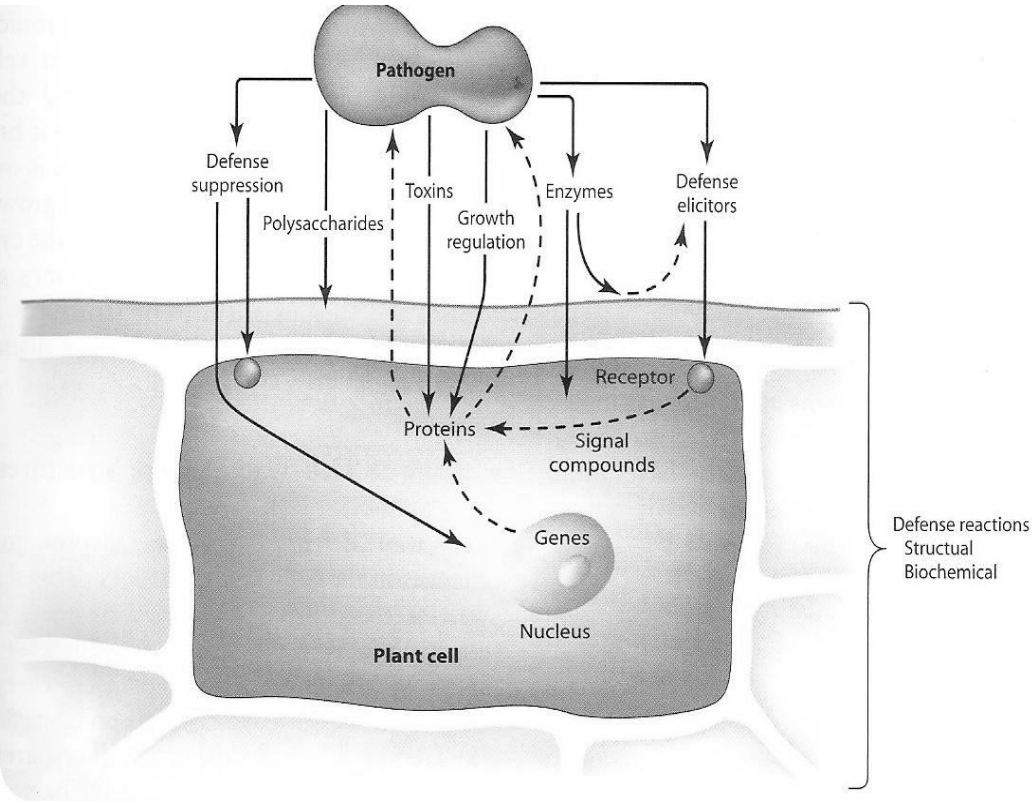


Fig. 3.6 : Host Pathogen Recognition

3.6.2 Signal Perception

It is a surface level phenomena in which elicitor from the pathogen are recognized by host receptor. It can be explained by Flor gene-for-gene hypothesis and consist of a receptor-ligand interaction.

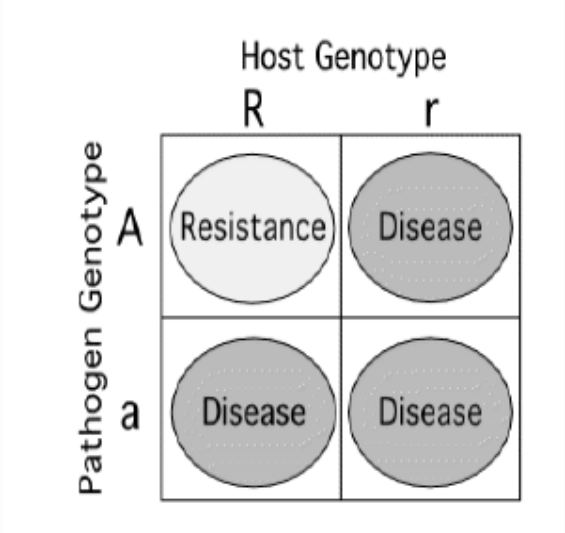


Fig. 3.7 : Flor Gene-for- Gene Hypothesis

In many plants, resistance to diseases is known to be genetically controlled by plant resistance (R) genes and pathogen avirulent (*avr*) genes (Flor 1947). It also has been postulated that R gene products act as receptors of Avr proteins, either directly or indirectly (elicitor-receptor model). This molecular interaction leads to the initiation of a signaling cascade responsible for defense response activation. However, for most matching Avr-/R-protein pairs, Avr protein binding has not been yet demonstrated. Biochemical studies have resulted in the characterization of some elicitor binding proteins that may be part of the recognition complex. Transmembrane receptor-like protein kinases (RLKs) constitute one of the most likely categories of receptors involved in pathogen perception. Some of these serine/threonine kinases have been identified as resistance or R genes, others as induced by pathogens or elicitors.

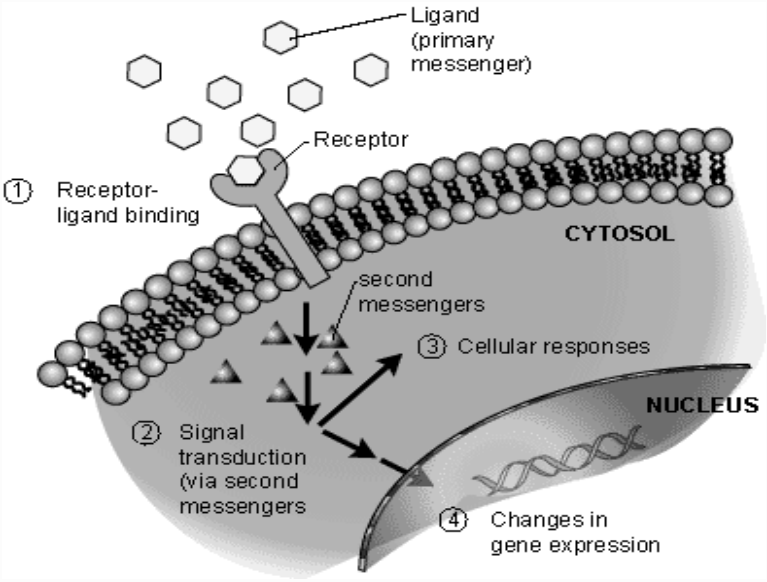


Fig. 3.8 : Receptor Ligand Interaction

However, triggering resistance is not always due to specific Avr products but instead, proceeds due to the action of general elicitors. As discussed in previous paragraphs, elicitors include compounds belonging to different chemical families of proteins, glycoproteins, glycans, lipids and synthetic molecules. They are constituents of the pathogen or secreted by it, or they are released from the plant or pathogen cell walls by hydrolytic enzymes from the pathogen or the plant. Early events, such as protein phosphorylation or activation of plasma membrane proteins, mobilize or generate directly or indirectly diverse signaling molecules (such as free calcium, nitric oxide [NO], active oxygen species [AOS]) which regulate many processes, interconnecting branch

pathways that amplify and specify the physiological response through transcriptional and metabolic changes.

3.6.3 Signal Transduction

After perception next step is signal transduction. Perception of environmental signals, mediated by specific receptors likely initiates internal signal transduction pathways. Signal Transduction Pathways refers to a series of sequential events, such as protein phosphorylations, consequent upon binding of ligand by a transmembrane receptor, that transfers a signal through a series of intermediate molecules until final regulatory molecules, such as transcription factors, are modified in response to the signal. There are two major pathways by which signal can be transduced:-

a. Via G-Protein: G-proteins are composed of three subunits: α, β, γ, where specificity is mainly determined by α. The α-subunit consists of two domains: GTPase domain and α-helical domain. Activation results in conformational changes around certain regions in GTPase domain.

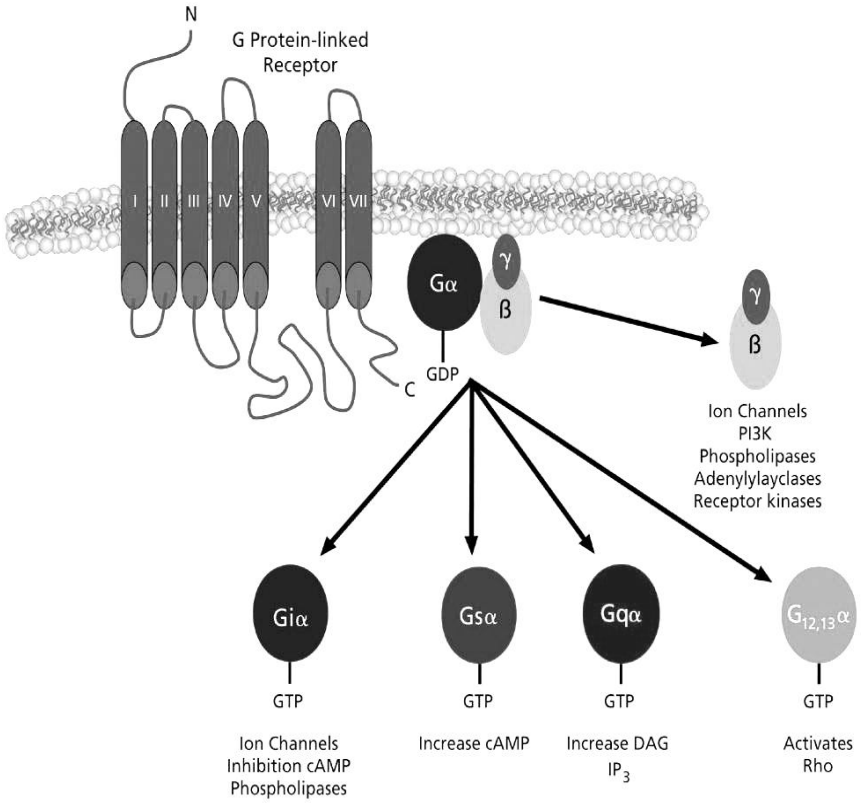


Fig. 3.9:G Protein Coupled Receptor Signal Transduction pathways

b. **Via Mitogen-activated Protein Kinase (MAPK) Cascades:** MAPK cascades fulfill essential functions in transduction of extracellular signals to cellular and nuclear responses. MAPK is activated by dual phosphorylation of a threonine catalyzed by MAPK kinase (MAPKK) MAPKKs activated by serine/threonine phosphorylation by a MAPKK kinase (MAPKKK). All MAPK pathways operate through sequential phosphorylation events to phosphorylate transcription factors and regulate gene expression.

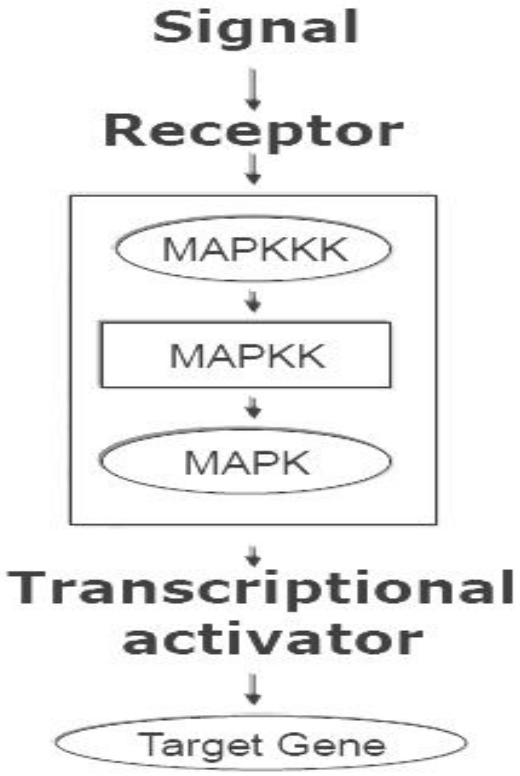


Fig. 3.10 : MAPK mediated Signal Transduction Pathways

3.6.4 Signal Response (Through Secondary Messengers)

The interaction between plant and pathogen are specific, complex and dynamic. Signals for activation of various defenses initiate in response to recognition. The outcome of interaction is dependent on initial sensing of the organism via exchange of molecular signal through signaling cascades and modified gene expression. Recognition is the first step by which eventually defense signal response is generated using a wide Secondary Messenger System. Many secondary molecules are involved in signal transduction process. Signaling outcome leads to massive changes in gene expression.

Secondary messenger system exists in plants to transmit the primary elicitation signal of pathogen and/or host. These are

- Active Oxygen Species
- Calcium Ion Influx
- Protein Phosphorylation
- cAMP
- Salicylic Acid
- Methyl Jasmonate and Jasmonic Acid
- Ethylene
- Nitric Oxide

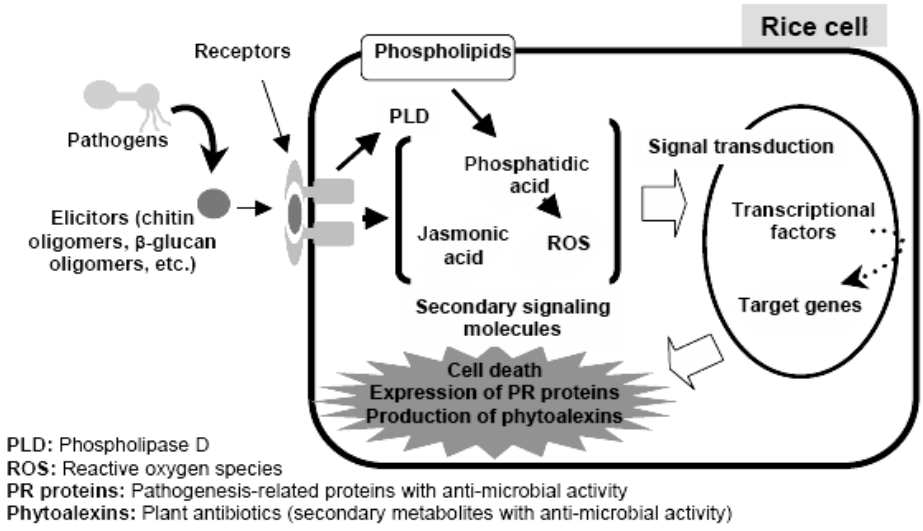


Fig. 3.11 : Secondary Messenger Activation against Pathogen Elicited Response

- **Active Oxygen Species:** Several Active Oxygen Species (AOS) involved in signal transduction are H_2O_2, Superoxide anion (O^{-2}), Singlet oxygen and Hydroxyl radical (OH). Active oxygen species may directly attack the pathogenic organisms, and they may indirectly deter subsequent invasion by the pathogenic organisms by causing a rapid cross-linking of phenolic components of the cell wall. Of these H_2O_2 is the most important secondary messenger.

- **Calcium Ion Influx:** Calcium (Ca^{2+}) is a very important second messenger. Regulatory functions of Ca^{2+} ion are exerted mostly by the small cytoplasmic protein Calmodulin. On binding Ca^{2+}, calmodulin undergoes a major conformational change which allows it to bind to calmodulin-dependent enzymes like Calmodulin-activated protein phosphatase and the calmodulin-dependent protein kinase (CDPK). Small change in cytoplasmic Ca2+ concentrations 'switches on' the active form of calmodulin. Ion fluxes occur across plasma membrane which includes efflux of K+ ions and influx of Ca^{2+}.
- **Cyclic Adenosine Mono Phosphate (cAMP):** Transmission of the cAMP signal proceeds via the cAMP-dependent activity of protein kinase A (PKA) and subsequent phosphorylation of target proteins. The major activity of PKA in developing germ tubes is the mobilization of carbohydrates and lipids to the appressorium site and is, therefore, pivotal to the production of functional appressoria. In some fungi, cAMP signaling is required for the initiation of appressorium development; high intracellular cAMP concentrations are required during differentiation of conidia and emergence of the appressorium germtube.
- **Salicylic Acid:** Salicylic acid (SA) is a phenolic compound commonly present in plant kingdom. It is one of the most important signal molecules which act locally in intracellular signal transduction. SA suppresses the H_2O_2 degrading activity of catalase. Targeting of R-gene mediated resistance leads to the expression of salicylic acid (SA). SA was shown to move from the infected leaf into the other parts of the plant. SA expression results in Hypersensitive Response HR (Hypersensitive Response includes the formation of necrotic lesions around the infection sites due to profound burst of reactive oxygen species and Systemic Acquired Resistance (SAR). SAR is an inducible plant defense state, the activation of which depends mostly on the accumulation of SA. Salicylic acid (SA) is a very essential signal molecule for the onset of SAR.
- **Methyl Jasmonic and Jasmonic Acid:** These are naturally occuring compounds in plants. JAs affect a variety of physiological processes and mediate plant responses to stresses by pathogen. In very low

concentrations, they induce different enzymes involved in plant defense such as Phenylalanine Ammonia Lyase (PAL) and lipoxygenase.

- **Ethylene:** Ethylene is a volatile plant hormone which is synthesized from amino acid methionine. Ethylene is produced upon wounding or infection by pathogen as well as by treatment by elicitors of defense responses. The increased production of ethylene is one of the earliest chemically detectable events in pathogen-infected plants or treated plants with elicitors.
- **Nitric Oxide:** Nitric oxide (NO) signaling involves cGMP- dependent pathways. Biosynthesis of NO is catalyzed by nitric oxide synthase (NOS) enzyme. AOS are also known to work with nitric oxide (NO) in defense responses.

3.6.5 Signal Termination

The signal should terminate when it is induced and responded to stop. The terminating events are in correspondence with the deactivation of pathogen induced signals or host induced protective mechanism.

3.7 Summary

Pathogens are agents of disease. These infectious microorganisms, such as fungi, bacteria, and nematodes, live off of the plant and damage its tissues. Plants have developed a variety of strategies to discourage or kill attackers. The first line of defense in plants is an intact and impenetrable barrier composed of bark and a waxy cuticle. Both protect plants against pathogens. A plant's exterior protection can be compromised by mechanical damage, which may provide an entry point for pathogens. If the first line of defense is breached, the plant must resort to a different set of defense mechanisms, such as toxins and enzymes. Additionally, plants have a variety of inducible defenses in the presence of pathogens. In addition to secondary metabolites, plants produce antimicrobial chemicals, antimicrobial proteins, and antimicrobial enzymes that are able to fight the pathogens. Plants can close stomata to prevent the pathogen from entering the plant.

Some defense reactions occur within minutes, while others may take several hours. Recognition of potential pathogens is central to plants' ability to defend themselves against harmful microbes. Microbial elicitors constitute a bewildering array of compounds including different oligosaccharides, lipids, peptides and proteins. Plants are able to recognize pathogen-derived molecules;

elicitors that trigger a number of induced defenses in plants through various signaling cascades.

3.8 Glossary

- **Constitutive**: constantly present, whether there is demand or not.
- **Gene Expression**: conversion of the information encoded in a gene via transcription and translation, resulting in the production of a protein and the appearance of the phenotype determined by that gene.
- **Hypersensitive Response (HR)**: the rapid and localized cell death at the site of infection in resistant interactions between plants and pathogens, the state of being extremely or excessively sensitive. It often refers to an extreme reaction by a plant to an invading pathogen in which the plant tissue around infected sites dies in order to prevent further spread of the infection.
- **Lignification**: the deposition of lignin around the tip of a fungal hypha penetrating a host plant cell. Lignification is an active defense mechanism and its function is to inhibit the growth of the hypha into other cells.
- **Obligate Parasite**: an organism that is only capable of living as a parasite in association with its host plant. The term is synonymous with biotroph.
- **Phytoalexin**: a substance produced in higher plants in response to a number of stimuli (chemical, physical or biological) that inhibits the development of a microorganism.
- **Tyloses**: a balloon-like outgrowth from a parenchyma cell that expands through a pit in a xylem vessel wall and into the lumen of the vessel, either blocking it completely or partially.
- **Elicitor**: a molecule produced by the host which induces a response by the pathogen. Conversely, an elicitor can be produced by a pathogen, eliciting a response in the host.
- **Gene-for-gene hypothesis**: the hypothesis that corresponding genes for resistance and virulence exist in the host and pathogen, respectively.
- **Systemic Acquired Resistance (SAR)**: the reduced disease symptoms on a portion of a plant distant from the area where a hypersensitive response occurred or other stimulus was applied; a rapid and coordinated defense response against a variety of pathogens as a signal travels throughout the plant.

- **Apposition**:growth of a cell wall by the deposition of new particles in layers on the wall.
- **Avirulence (*avr*) gene**: a gene in a pathogen that causes the pathogen to elicit an incompatible (defense) response in a resistant host plant, and may enhance pathogen virulence in a susceptible host plant. The outcome of the interaction of an avirulence gene product with its corresponding plant resistance (R) gene product is usually a hypersensitive reaction.

3.9 Self-Learning Exercise

Section A : (Very Short Answer Type Questions)

1. The protective compounds against pathogens pre-existing in plants are called _____ in nature.
2. Oligogalacturonides and Elicitins are examples of _____.
3. _____is a pre requisite to initiate the compatible or incompatible reaction.
4. _____ is a very essential signal molecule for the initiation of SAR.
5. _____ exists in plants to transmit the primary elicitation signal of pathogen and/or host.
6. Inositol Triphosphate (IP3), Diacylglycerol (DAG) and Ion channels are generally activated by _____.

Section B: (Short Answer Type Questions)

1. Define Pathogenesis.
2. Write short note on Flor Hypothesis.
3. What is the difference between a compatible and an incompatible reaction?
4. How is epidermis in plants important in plant defense?
5. Elucidate a few biochemical measures adopted by plants to overcome the pathogens.

Section C : (Long Answer Type Questions)

1. List some of the secondary messengers with their role in plant defense.
2. What features of plants are induced in relation to disease resistance?
3. Discuss the signal transduction pathway in terms of pathogen attack.

Answer key of Section – A

1. Constitutive
2. Elicitors
3. Recognition
4. Salicylic Acid
5. Secondary Messengers
6. G- Protein

3.10 References

- Plant pathology by G.N. Agrios
- Principles of plant pathology by R.S. Singh
- Introduction to Plant Pathology by Richard N. Strange

Unit - 4

Host Pathogen Interaction-III: Pathogenesis and Disease Development

Structure of the Unit:

4.0 Objectives

After studying this unit, you will understand the disease symptoms in plants and various changes that alter the host physiology in response to the pathogen attack as well as you will also come to know about factors that affect the disease development in terms of-

- Different terms of symptomology of disease
- Alterations in host normal metabolism

- Factors affecting the disease growth

4.1 Introduction

Unhealthy plants have special distress signals that show when pathogens have attacked. Plants will respond to the stress of infection with a wide range of symptoms, from leaf spots and damaged fruit to wilting and even death. Apart from symptoms on the plant, its internal mechanisms are also potentially affected leading to either development of the disease or its abortion. The pathogen, the host and the environment interact, usually in numerous ways in this respect. It is the balance of these interactions that determines whether or not disease develops to destructive levels in a particular situation.

4. 2 Symptom Production

Diseases generally involve a progression of symptoms that can vary significantly. The progression of symptoms is one of the most important characteristics associated with problems caused by biotic agents. Diseases can result in primary and secondary symptoms. For example, decayed roots on a tree may be a primary symptom while the toppling over of the tree by wind is a secondary symptom. At later stages of a disease, secondary invaders may also obscure the original disease symptoms so that symptoms observed at the later stages of the disease are not typical of the symptoms developed in response to the original pathogen. The vast majority of plant pathogens are fungi, however, plant diseases are also caused by insects, bacteria, nematodes, viruses and phytoplasmas. Disease-like symptoms can also be caused by abiotic factors, such as temperature, light, chemical agents and water or nutrient deficiencies.

Symptoms of plant disease can be broadly categorized into following major classes:

- Death and Destruction of Host Tissue or Necrosis
- Abnormal Growth and Differentiation
- Wilting
- Metaleptic symptoms
- Proleptic Symptoms
- Discolouration of Host Tissue.

Some parasites, called necrotrophs, secrete enzymes that kill host tissue, extract nutrients from the cells and then live in the dead tissue. The necrotic lesions caused by pathogens can be localised or extensive.

Local **necrotic** lesions appear as discrete necrotic areas, while extensive, or spreading lesions spread until the whole organ or plant is killed. **Abnormal growth and differentiation** results from deviation from the complex balance of interrelated reactions that take place in plants. Parasites can alter the hormonal balance in plants causing an abnormal increase in the size or number of cells, resulting in abnormal growth and differentiation, for example, the formation of galls. **Wilting** occurs when water loss is greater than water intake. It results from either: interference with water and nutrient absorption at the roots, interference with water conduction within the plant (i.e. infection of the vascular tissue), or loss of control of transpiration. **Metaplastic symptoms** are those which form when tissues change from one form to another. **Proleptic symptoms** result from the development of tissues earlier than usual. **Discolouration** of tissue is most commonly by chlorosis or mosaics of leaves, both of which can have a number of causes. Anything that interferes with the production of chlorophyll causes leaves to turn yellow, or chlorotic. Mosaic is a symptom of many virus infections and is characterised by alternating light and dark green areas on the leaves.

4.2.1 Death and Destruction of Host Tissue or Necrosis

Necrosis is caused due to necrosis or death of plant cells. The affected plant tissue usually turns brown to black in color. Necrotic symptoms could appear in any part of the plant such as in storage organs, in green tissues, or in woody tissues.

1. **Necrosis in Storage organs :** Death of cells in storage organs terminates in decomposition or decay referred to as a rot.
    a. **Soft rots or wet rots: Soft rots or wet rots** are those where the pathogen breaks down the host cell walls, resulting in the exudation of juices from the infected tissue. The organ becomes mushy or pulpy and a foul smell often develops due to colonization by secondary invaders. Many fungi and bacteria cause soft rots on several fruits and vegetables. Species of the fungus *Rhizopus* and bacterium *Erwinia* are two such commonly found pathogens causing soft rots.
    b. **Dry Rot:** In a **dry rot**, the storage organ becomes hard and dry
    c. **Mummifications**: In some diseases, there is rapid loss of water and the infected organs become shriveled, wrinkled, and leathery. Dry rots showing such symptoms are referred to as **mummifications**.
2. **Necrosis in Green tissues :** Necroses on green tissue are termed differently based on the nature of symptoms and the type of green tissue.

- **Damping off**: **damping off** refers to the sudden wilting and topping over of seedlings as a result of extensive necrosis of tender tissue of the roots and stem near the soil line, due to the attack of soil-borne pathogens such as fungus *Pythium*. This fungus is known to cause damping off in an assortment of seedlings such as those of brinjal, chilli, mung beans, tobacco and tomato.
- **Spot**: A **spot** refers to a well-defined area of gray or brown necrotic tissue. Spots are very common on leaves and fruits and are probably the most familiar necrotic symptom.
- **Shot Hole**: Sometimes the necrotic tissue within a leaf spot may crack and fall off from the surrounding green tissue leaving an empty space. Such a symptom is known as a **shot hole**.
- **Flecks** or **Specks:** Minute or very small spots are sometimes referred to as **flecks** or **specks**.
- **Blotch**: When dark mycelia of a fungal pathogen appear on the surface of necrotic spot, blotting the leaves, shoots, and stems as large and irregular spots, the symptom is referred to as a **blotch**.
- **Streaks** and **Stripes**: Both **streaks** and **stripes** occur in grasses and are elongated areas having dead cells. Streaks occur along the stem and veins, while stripes are in the laminar tissues between veins.
- **Net necrosis**: **Net necrosis** is a symptom resulting from an irregular pattern of anastomoses between streaks or stripes.
- **Blights: Blight**s are characterized by the rapid death of entire leaves including the veins or parts of the leaves. Blights also could occur on flowers and stems.
- **Scorches: Scorches** resemble blights, but there, necrosis occurs in irregular patterns between veins and along leaf margins.
- **Firing: Firing** is sudden drying, collapse and death of entire leaves. Firing occurs in response to the activity of root rot and vascular wilt pathogens.
- **Scald: Scald** is the term used to describe the blanching of epidermal and adjacent tissues of fruits and occasionally of leaves.
- **Blast:** The sudden death of unopened buds or inflorescence is referred to as **blast**.

- **Shelling:** Extensive necrosis of fruits that resemble in premature dropping is called **shelling**.

3. **Necrosis in Woody tissues :** Necrosis of woody tissue often brings about various types of die-back symptoms.
   - **Dieback**: **Dieback** is the extensive necrosis of a shoot from its tip downwards.
   - **Canker**: Restricted necrosis of the bark and cortical tissue of stems and roots is termed as a **canker**. In cankers, necrotic tissue in the sunken lesions is sharply limited, usually by a callus from adjacent healthy tissue.
   - **Gummosis**: When woody tissues are diseased, they may exude different kinds of substances. When the exudate is gummy, the symptom is called **gummosis**.
   - **Resinosis**: It is called **resinosis** when the exudate is resinous.
   - **Bleeding**: If the exudate is neither gummy nor resinous, it is described as **bleeding.**
4. **Abnormalities in Growth and Differentiation :** Many disease symptoms are associated with growth changes in diseased plants. These could be caused by either reduced growth due to **Hypoplasia** and **Atrophy** or excessive growth due to **Hyperplasia** and **Hypertrophy**.

Reduced Growth

- **Hypoplasia** : **Hypoplasia** is the failure of plants or plant organs to develop fully due to a decreased production of the number of cells. Hypoplasia results in plants or plant parts of sub-normal size.
- **Atrophy**: **Atrophy** is the reduction in the size of plant cells produced. This also results in stunted plants or plant parts.
- **Dwarfing: Dwarfing** is the failure of a plant or a plant part to attain its full size.
- **Rosetting: Rosetting** is a condition where the internode of a plant does not elongate, and hence, the leaves appear close together in a cluster.

Excessive Growth

- **Hyperplasia: Hyperplasia** is the enlargement of a plant tissue due to excessive increase in the number of plant cells produced. Hyperplasia results in overdevelopment in size of plants or plant organs.

- **Hypertrophy: Hypertrophy** is excessive growth due to the enlargement of individual cells. This condition also results in the overdevelopment in size of plants or plant organs.

Hyperplasia and hypertrophy could result in the enlargement of leaves and fruits, and the enlargement of stems and roots.

Enlargement of leaves and fruits *:* Several symptoms expressing enlargement of leaves and fruits are commonly observed among diseased plants.

- **Curling: Curling** which is the bending of the shoot or the rolling of the leaf, is a result of over-growth on one side of an organ. Often viral diseases cause such leaf distortions due to irregular growth of the lamina.
- **Shoe-string effect**: Extreme reduction of the leaf lamina brings about the symptom known as the **Shoe-string effect**.
- **Savoying:** The puckering or crinkling of leaves due to different growth rates in adjacent tissue is known as **savoying**.
- **Scab**: Overgrowth of epidermal and underlying tissues of leaves, stems, fruits and tubers may result scab formation. **Scab** consists of raised, rough, and discrete lesions. These are often sunken and cracked, giving a typical scabby appearance.
- **Blister**: Localized swellings or enlargement of epidermal cells due to excessive accumulation of water is termed **intermuscence** and the diagnostic symptom is the appearance of a **blister**.

Enlargement of stems and roots : Symptoms causing enlargement of stems and roots are termed differently based on their nature.

- **Sarcody:** Excessive accumulation of food material in stems, above a constricted area produces a swelling termed **sarcody**.
- **Tumefaction (Galls, Clubs and Knots**): Localized swellings that involve entire organs are termed **tumefaction**. Commonly exhibited tumefactions are **galls**, **clubs**, and **knots**.
- **Fasciculation**: Excessive development of adventitious organs results in **fasciculation**, which is the clustering of organs around a focal point. Such examples include witch's broom and hairy root.
- **Witch's broom: Witch's broom** is a broom-like mass proliferation due to the dense clustering of branches of woody plants.
- **Hairy root: Hairy root** results due to excessive development of roots.

- **Fasciation: Fasciation** is the broadening or flattening of cylindrical organs such as stems.
- **Proliferation:** The continued development of any organ after it has reached a stage beyond which it normally does not grow is known as **proliferation**.
- **Callus:** The outgrowth of tissue in response to wounding is known as a **callus**. Callus formation is found to form around most cankers.

4.2.2 Wilting

Wilting is due to loss of turgor in plant tissue resulting in the dropping of plant parts. They are common symptom in diseases where the pathogen or the toxic metabolites it produces affects the vascular tissue of the host plant. Interference in water transport brought about by the infection of these vascular pathogens leads to wilting. Unlike wilting due to low soil moisture, wilting due to the activity of these pathogens cannot be overcome by watering the plants. Infected plants eventually die.

4.2.3 Metaplastic symptoms

Metaplastic symptoms are those which form when tissues change from one form to another.

- **Phyllody**: Such symptoms include **phyllody**, the development of floral organs into leaf-like structures.
- **Juvenillody: Juvenillody**, the development of juvenile seedlings on mature plants.
- **Rusting: Rusting**, a superficial browning of surfaces of leaves, fruits and tubers due to suberization.

4.2.4 Proleptic symptoms

Proleptic symptoms result from the development of tissues earlier than usual.

- **Prolepsis**: **Prolepsis**, when there is premature development of a shoot from a bud in plants.
- **Proleptic Abscission: Proleptic Abscission**, the premature formation of **abscission** layers.
- **Restoration: Restoration,** the unexpected development of organs that are normally rudimentary.

4.2.5 Discolouration of Host Tissue

Changes in the color of plant tissue are a common symptom of plant disease.

- **Yellowing**: Often these color changes are brought about by the **yellowing** of normal green tissue due to the destruction of chlorophyll or a failure to form chlorophyll. Such repression of leaf color may be complete or partial.
- **Albication**: When color repression is complete, it is known as **albication**.
- **Chlorosis**: More common Symptom, partial repression is referred to as **chlorosis**.
- **Mosaic**: Patches of green tissue alternating with chlorotic areas are described as a **mosaic**. Mosaic is a symptom caused by many viruses. Based on the intensity and the pattern of discoloration, mosaics are termed differently.
- **Mottling**: Irregular patches of distinct light and dark areas are known as **mottling**.
- **Streaking: Streaking** is still other distinct type of discoloratios. Streaking is the formation of elongated chlorotic lines.
- **Ring Spots**: Ring spots are circular masses of chlorosis with a green center.
- **Vein clearing** and **vein banding**: **Vein clearing** and **vein banding** are yet other common color changes on leaves.
- **Virescence**: Chlorophyll may also develop in tissues normally devoid of it. Thus usually white or colored tissue becomes green in color. This is called as **virescence**.
- **Anthocyanescence: Anthocyanescence** is due to the overdevelopment of anthocyanin and result in the development of a purplish coloration. Color changes can also take place in flowers. Such an example is the color break virus-affected tulips.

4.3 Physiological Changes due to infection

During phytopathogen interactions, the host develops a variety of defense reactions. Plant pathogens include fungi, bacteria, oomycetes, and viruses. Pathogens affect plants in different ways as they have devised different strategies to invade a plant, as well as to feed on and reproduce in the plant. While necrotrophs have little effect on plant physiology, since they kill host cells before colonising them, biotrophic pathogens become incorporated into and subtly modify various aspects of host physiology, such as respiration, photosynthesis, translocation, transpiration and growth and development. The respiration rate of plants invariably increases following infection by fungi,

bacteria or viruses. The higher rate of glucose catabolism causes a measurable increase in the temperature of infected leaves. An early step in the plant's response to infection is an oxidative burst, which is manifested as a rapid increase in oxygen consumption, and the release of reactive oxygen species, such as hydrogen peroxide (H_2O_2) and the superoxide anion (O_2^-). The oxidative burst is involved in a range of disease resistance and wound repair mechanisms. In resistant plants, the increase in respiration and glucose catabolism is used to produce defense-related metabolites via the pentose phosphate pathway. In susceptible plants, the extra energy produced is used by the growing pathogen.

Phytopathogen interactions not only trigger the different defense responses accordingly as discussed in the previous unit but also lead to changes in secondary metabolism based on the induction of defense programmes as well as to changes in primary metabolism which affect growth and development of the plant. Phytopathological studies also take into consideration the physiological status of the infected tissues in relation to photosynthesis; assimilate partitioning, and source–sink regulation in relation with the infection development.

4.3.1 Changes in Primary Metabolism

Changes in plant metabolism have been discussed in details in previous unit. Cell wall strengthening by callosic and papillae formation, cell wall apposition, lignin deposition as well as hydrolytic PR proteins have been reported as first line of defense mechanism developed by plants. Components of these cell wall make ups are of primary metabolite origin. Upon contact with pathogens or with non-pathogenic micro-organisms or elicitors, Ion Fluxes, Phosphorylation/Dephosphorylation of proteins, and the production of signalling molecules such as Salicylic Acid, Jasmonic Acid, Ethylene, and Active Oxygen Species are activated. This leads to the regulation of gene expression and the induction of defense responses, for example, cell wall strengthening and the accumulation of phytoalexins and pathogenesis related (PR) proteins. Salicylic acid (SA), Jasmonic acid, Ethylene and numerous other secondary messengers are produced in order to act as important signal molecules in successful resistance development in plants.

- **Effects on Photosynthesis :** Pathogens also affect photosynthesis, both directly and indirectly. Pathogens that cause defoliation rob the plant of photosynthetic tissue, while necrotrophs decrease the photosynthetic rate by damaging chloroplasts and killing cells. Biotrophs affect photosynthesis in varying degrees, depending on the severity of the infection. A biotrophic

infection site becomes a strong metabolic sink, changing the pattern of nutrient translocation within the plant, and causing net influx of nutrients into infected leaves to satisfy the demands of the pathogen. A decrease in photosynthesis has been reported in incompatible interactions. The plants seem to switch off photosynthesis and other assimilatory metabolism to initiate respiration and other processes required for defense. But studies also show that decrease in photosynthetic activity is not necessarily preceded by the repression of photosynthetic genes.

- **Sugar Accumulation:** The down-regulation of photosynthesis and the simultaneous increased demand for assimilates very often leads to a transition of source tissue into sink tissue during plant–pathogen interactions. One indication for the induction of a sink status in infected leaves is the increase of cell wall invertase activity. Cell wall invertases are extracellular enzymes which cleave sucrose in the apoplast into glucose and fructose. The resulting hexoses are transported by hexose transporters into the cell. The cleavage of extracellular sucrose will also result in the decreased export of assimilates from the tissue. Enhanced expression and activity of cell wall invertases has been reported in several plant–pathogen interactions. Similarly, reduced sucrose export from infected source leaves has been observed.

  Pathogen attack first initiates a series of rapid changes resulting in a decline in photosynthesis and an increase in respiration, photorespiration, and invertase enzyme activity. The mechanisms and pathways which mediate these rapid changes are largely unknown. The electrophilic oxylipin 12-oxo-phytodienoic acid is a compound which has been shown to accumulate after pathogen infection and to result in a decrease in photosynthesis very shortly after application, suggesting that it might be involved in the decrease in photosynthesis upon pathogen challenge. Hexoses released by the action of increased invertase activity act as signalling molecules and repress photosynthetic genes. This down-regulation of photosynthetic genes, in turn, again decreases the net photosynthesis rate. The depletion, diversion and retention of photosynthetic products by the pathogen stunts plant growth, and further reduced the plant's photosynthetic efficiency.

- **Carbohydrate Metabolism**

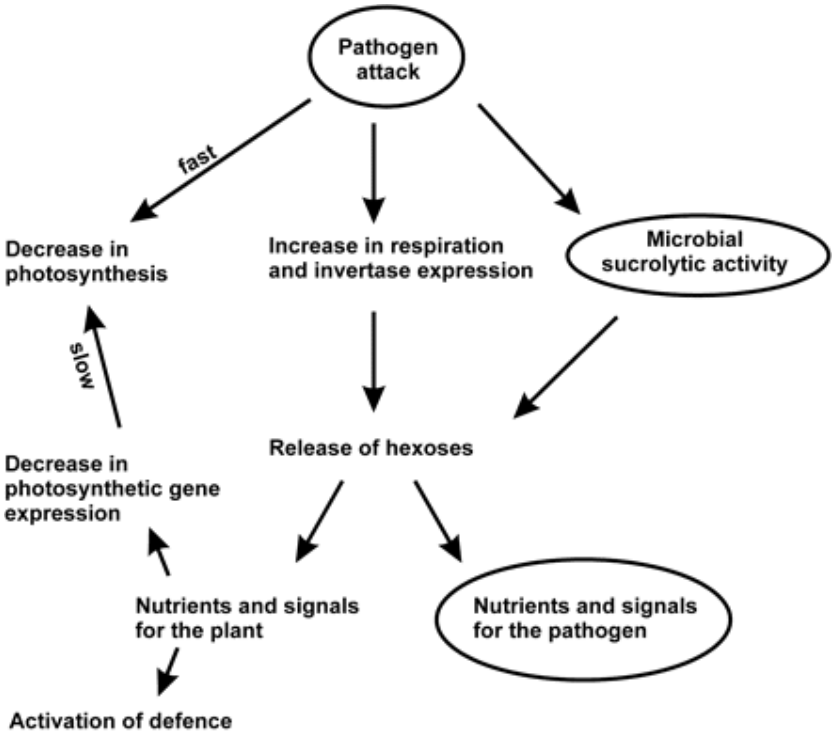


Fig. 4.1 : **Model of changes in carbohydrate metabolism in response to infection with biotrophic pathogens. Components showing the microbe activity are encircled**.

There are certain factors which contribute to the mutual relation between carbohydrate metabolism and development of disease/resistance. First, the carbohydrate status affects the defense as well as general metabolism of the plant. Second, sugars are not only nutrients and signals for the plant partner but also for the microbial partner. Therefore, changes in assimilate levels may influence the spreading of the pathogen and might regulate gene expression of the pathogen. Third, certain pathogens also possess extracellular sucrolytic enzymes such as invertases, fructoexohydrolases, and levansucrases which when expressed, the pathogen would be able to alter the hexose and sucrose levels in the apoplast of the host tissue. And this may directly or indirectly interfere with the plant metabolism at any level.

4.3.2 Changes in Secondary Metabolism

In natural systems, plants face a plethora of antagonists and thus posses a myriad of defense and have evolved multiple defense mechanisms by which they are able to cope with various kinds of biotic and abiotic stress. Generally,

it is difficult to assign a change in the physiology of metabolism of a plant to a specific stress factor as normally a complex variety of various stress factors affects the plant simultaneously. The role for secondary metabolites in the plant's interaction with its environment is widely recognized. The primary metabolites deriving from photosynthesis are channeled into different metabolite pathways for the synthesis, storage and modification (hydroxylation, glycosylation, acetylation, etc) of myriads of compounds and for use to cope abiotic and biotic stresses. Plants produce a high diversity of natural products or secondary metabolites with a prominent function in the protection against predators and microbial pathogens on the basis of their toxic nature and also important for the communication of the plants with other organisms, and are insignificant for growth and developmental processes. There are three major groups of secondary metabolites *viz* terpene, phenolics and N and S containing compounds. Gibberellins, a group of plant hormones are diterpenes, which play various detrimental roles in numerous plant defense processes. Psoralin, a basic linear furacoumarin is known for its use in the treatment of fungal defense. Lignin is a highly branched polymer of phenyl-propanoid groups, formed from three different alcohols *viz.,* coniferyl, coumaryl and sinapyl which oxidized to free radicals (ROS) by a ubiquitous plant enzyme-peroxidase, reacts simultaneously and randomly to form lignin. Lignifications block the growth of pathogens and are a frequent response to infection or wounding. Sulphur containing secondary metabolites includes Phytoalexins, Thionins, Defensins and Allinin which have been linked directly or indirectly with the defense of plants against microbial pathogens. Phytoalexins are synthesized in response to bacterial or fungal infection or other forms of stress that help in limiting the spread of the invading pathogens by accumulating around the site of infection, appears to be a common mechanism of resistance to pathogenic microbes in a wide range of plants. Many of these changes are linked to a rapid apoptotic response, resulting in the death of one or a few invaded plant cells, known as the hypersensitive response (HR). Defensins and Thionins are S-rich non-storage plant proteins which are synthesized and accumulated after microbial attack and such related situations as they all have antifungal and/or anti-bacterial activity.

High concentrations of secondary metabolites might result in a more resistant plant. Their production is thought to be costly and reduces plant growth and reproduction. Therefore, defense metabolites can be divided in to constitutive substances, also called **prohibitins** or **phytoanticipins** and induced metabolites

formed in response to an infection involving *de novo* enzyme synthesis, known as **phytoalexins**. Phytoanticipins are high energy and carbon consuming and are recognized as the first line of chemical defense that potential pathogens have to overcome. In contrast, phytoalexin production may take two or three days, as by definition, first the enzyme system needs to be synthesized.

4.3.3 Other Physiological Changes due to Infection

Pathogens affect water relations in the plants they infect. Biotrophs have little effect on transpiration rate until sporulation ruptures the cuticle, at which point the plant wilts rapidly. Pathogens that infect the roots directly affect the plant's ability to absorb water by killing the root system, thus producing secondary symptoms such as wilting and defoliation. Pathogens of the vascular system similarly affect water movement by blocking xylem vessels. **Growth and development** in general are affected by pathogen infection, as a result of the changes in source-sink patterns in the plant. Many pathogens disturb the hormone balance in plants by either releasing plant hormones themselves, or by triggering an increase or a decrease in synthesis or degradation of hormones in the plant. This can cause a variety of symptoms, such as the formation of adventitious roots, gall development, and epinasty (the down-turning of petioles).

4.4 Factors Affecting Disease Development

The amount of disease that develops in a plant community is dependent on properties of the host, the pathogen and the environment. The environment can affect both the susceptibility of the host (e.g. by creating stress in the plant) and the activity of the pathogen (e.g. providing moisture for spore germination). The pathogen and the host can affect each other's performance. The plant can also change its environment, by creating a microclimate around it.

Factors that affect disease development

Pathogen	Host	Environment
• Presence of pathogen • Pathogenicity • Adaptability • Dispersal efficiency • Survival efficiency • Reproductive fitness	• Susceptibility • Growth stage & form • Population density & structure • General health	• Temperature • Rainfall / Dew • Leaf wetness period • Soil properties • Wind • Fire history • Air pollution • Herbicide damage

4.4.1 Pathogen Factors

The presence or absence of a pathogen is the main factor that determines whether disease will occur or not. Introduction of a pathogen to an area from where it was never present earlier can cause major outbreaks of disease in such plant communities. The amount of disease that develops is often determined by the pathogenicity of the pathogen. The term pathogenicity relates to both the virulence (infection ability) and the aggressiveness (the vigour of the infection) of the pathogen. Pathogenicity is dependent on the pathogen's multiplicative ability, dispersal efficiency and survival fitness.

The **adaptability** of the pathogen is also crucial in determining its ability to infect resistant hosts or to survive changed environmental conditions. Adaptability is determined by the pathogen's genetic flexibility and reproductive efficiency. The spread of a disease and the development into epidemics is reliant on the pathogen's ability to **disperse** rapidly over long distances. The spores of cereal rusts, for instance, can be blown over vast distances in a few days, while soil-borne pathogens have little scope for extensive spread. And for a pathogen wnich is carried in seed has to depend upon external dispersing agencies. For a pathogen to cause disease in successive seasons, it must be able to **survive** the intervening time. Some pathogens form spores or sclerotia that can survive in the soil for years, while others colonise alternative plant species until the season of their primary host comes around again. Beyond the mere presence of a pathogen, the **number of infective propagules** available to infect plants is a crucial factor in determining

the amount of disease that develops. Generally, as the number of propagules increases, the level of disease increases, levelling off when the amount of disease reaches very high levels and there are few uninfected plants available. The survival of propagules, and therefore the number of propagules available to cause disease, is however heavily influenced by environmental factors.

4.4.2 Host Factors

The development of disease in a plant community depends on the presence of suitable hosts that are **susceptible** to that particular pathogen. If the majority of the population is susceptible to the pathotypes of a pathogen in the vicinity, an epidemic can occur. The best way of controlling disease is by planting species or cultivars that are not susceptible to pathogens of that area. The occurrence of disease can also be influenced by the host plant's **growth stage and form**. Some diseases are common in seedlings, while others are typical of mature plants. The growth stage of the population can also affect the microclimate around the plants; for example, the humidity and sunlight levels under the canopy. The **population structure and density** will also affect the development of disease in a plant community. The density of the main host species and the proportion of other plants that are not hosts within the community will determine the rate and extent of epidemic development. Crop plants tend to be densely planted, with no other species in amongst them, making them more susceptible to rapid spread of disease. Extensive, dense plantations can result in tremendous epidemics, particularly if a new pathogen is introduced to the area. In addition, the general **health** of the host plant before infection is important in determining the success of a disease. Necrotrophs do well on poorly growing plants, while biotrophs thrive on a healthy host plant.

4.4.3 Environmental Factors

The presence of a pathogen against a particular plant will generally not cause serious disease unless the environmental conditions are favourable. This includes the aerial environment and the soil (*edaphic*) environment. Human attempts of controlling disease usually involve manipulating the environment of the community in certain way. For example, breeding wheat cultivars which tolerate dry conditions allows farmers to plant the crop in areas that are not favourable for pathogens causing powdery mildew and leaf rust. Properties of the **aerial environment** that influence disease development include moisture levels, temperature and pollution.

Moisture is particularly important to pathogenic bacteria and fungi. Rain drop splashes play an important role in the dispersal of some fungi and nearly all

bacteria and a period of leaf wetness is necessary for the germination of most airborne spores. By using water for dispersal, propagules are dispersed at a time when they are likely to be able to germinate as well. Because the process of germination and infection takes time, the duration of leaf wetness also influences the success of the infection. The duration necessary for infection varies with temperature. Usually, a longer period of leaf wetness is needed to establish an infection in cooler temperatures, as germination and infection are generally accelerated in warmer conditions.

Temperature also affects the incubation, or latent period (the time between infection and the appearance of disease symptoms), the generation time (the time between infection and sporulation), and the infectious period (the time during which the pathogen keeps producing propagules). The disease cycle speeds up at higher temperatures, resulting in faster development of epidemics. The period of leaf wetness, combined with temperature information can be used to predict outbreaks of some diseases (infection periods) and be used to timely take up preventative treatments, such as spraying. A recently recognised aspect of aerial environment that can influence disease in plants is air **pollution**. A high concentration of pollutants can affect disease development and, in extreme cases, damage the plants directly by causing acid rain.

The **edaphic (soil) environment** affects soil-borne diseases, largely by determining the amount of **moisture** available to pathogens for germination, survival and motility. Germination and infection success also rely on the temperature of the soil. The **fertility** and **organic matter content** of the soil can affect the development of disease. Plant defenses are weakened by nutrient deficiency, although some pathogens, such as rusts and powdery mildews, thrive on well-nourished plants. Other diseases thrive in soils that are specifically low in organic matter.

The environment can affect both the susceptibility of the host and the activity of the pathogen. The pathogen can affect the host and the host can influence the pathogen in numerous ways. Similarly, the host can influence the environment such as by influencing the microclimate. Development of disease requires the interaction of a virulent pathogen and a susceptible plant host in an environment that favours the development of disease.

4.5 Summary

There are a number of the changes induced by different pathogens which reveal the complexity and divergence of the symptoms produced by them. The

symptoms that are produced are the result of the interference in the plant metabolic machinery either by the plant itself, in a defensive response to the disease, or induced by the pathogen. Certain metabolic signals such as hexoses are responsible for regulation of carbohydrate and defense metabolism. The amount of disease that develops in a plant community is determined by the factors related to the host, the pathogen and the environment. An understanding of these factors and their interactions for a particular disease in a particular locality allows prediction of disease outbreaks and intervention to reduce the amount of disease.

4.6 Glossary

- **Pathogenesis**: the sequence of processes in disease development that describes a pathogen's association with its host. The sequence begins with initial contact between the pathogen and host and ends when the pathogen is no longer associated with that host (i.e. when the host/pathogen dies or the pathogen moves to another host).
- **Sign**: an indication of disease from direct observation of a pathogen or its parts.
- **Symptom**: an indication of disease by reaction of the host e.g. canker, leaf spot, wilt.
- **Susceptible**: prone to develop disease when infected by a particular pathogen.
- **Phytoanticipin:** these are constitutive phytoalexins i.e. phytoanticipins are synthesised by the plant at a constant rate and therefore always present in the tissues of the plant, whereas phytoalexins are only produced in response to a stimulus such as a pathogenic invasion. The distinction between phytoalexins and phytoanticipins is not always clear as some compounds may be phytoalexins in one species and phytoanticipins in another species. In general, the distinction between the two compounds depends on when they are produced (either before or after infection) and the extent to which the compound is antimicrobial (phytoalexins are antimicrobial).
- **Alkaloid**: a nitrogen-containing ring compound produced by plants that causes physiological effects in animals.
- **Latent period**: the time between infection and the production of new inoculum; the time after a vector has acquired a pathogen and before it can be transmitted.

- **Sclerotia**: a vegetative resting body of a fungus composed of a compact mass of hyphae with or without host tissue, usually with a darkened rind.

4.7 Self-Learning Exercise

Section A : (Very Short Answer Type Questions)

1. The development of floral organs into leaf-like structures is called _____.
2. The enlargement of a plant tissue due to excessive increase in the number of plant cells is _____ whereas excessive growth due to the enlargement of individual cells is _____.
3. Death and destruction of host tissue is referred to as _____.
4. Cell wall_____ are extracellular enzymes which cleave sucrose into glucose and fructose.
5. _____is particularly important to pathogenic bacteria and fungi for spore germinations.
6. _____ are those which form when tissues change from one form to another.

Section B : (Short Answer Type Questions)

1. Why necrotrophs thrive well on poorly growing plants?
2. Write the importance of edaphic (soil) factors in disease development.
3. Discuss some secondary metabolites with their functions in plant defense.
4. How is photosynthesis affected after pathogen attack?
5. Enumerate the factors affecting disease development.
6. Which is more severe- Necrosis or Wilting? and why?
7. Write the difference between phytoalexins and phytoanticipins.

Section C : (Long Answer Type Questions)

1. Write an essay on the plant disease symptoms.
2. How does pathogen attack affect the metabolism in plant?
3. How does environment influence the development of a disease?

Answer key of Section A

1. phyllody
2. hyperplasia, hypertrophy
3. necrosis
4. invertases
5. Moisture
6. Metaplastic Symptoms

4.8 References

- Plant pathology by G.N. Agrios
- Principles of plant pathology by R.S. Singh
- Introduction to Plant Pathology by Richard N. Strange

Unit -5

Epiphytotics & Plant Disease Forecasting

Structure of the Unit:

5.0 Objectives

After going through this unit you will be able to understand about:

- Epiphytotic
- Factors responsible for Epiphytotic
- Types of Epidemics
- Plant Disease Forecasting
- Methods to study Plant Disease Forecasting
- Types of Disease warning System available

5.1 Introduction

When a pathogen spreads to and affects many individuals within a population over a relatively large area and within a relatively short time, the phenomenon

is called an epidemic. An epidemic has been defined as any increase of disease in a population. The study of epidemics and of factors that influence them is called epidemiology.

Forecasting of plant disease means prediction of the occurrence of plant disease in a specified area ahead of time, so that suitable control measures can be undertaken in advance to avoid losses. The forecasting of plant diseases requires the complete knowledge of the influence of weather conditions on the appearance and severity of disease, and of epidemiology of plant diseases. The knowledge of weather conditions and epidemiology in relation to plant diseases may be utilized in the prediction of plant diseases

5.2 Epiphytotic

Epidemiology or Epiphytology is the study of the outbreak of Disease, its course, intensity, cause and effects and the various factor governing it. Several internal and external factors of particular host plants play an important role in the development of epidemics involving those hosts.

Based on the occurrence and geographical distribution the epiphytotic are classified as follows:

Endemic or Endophytotic : When a disease is more or less occurring constantly every year in a moderate to severe form in a country or locality then it is called as endemic disease. eg: wart disease of potato (*Synchytrium endobioticum*) is endemic in Darjeeling, Citrus Canker (*Xanthomonas axonopodis pv citri*) in Asia.

Epidemic or Epiphytotic : It is a sudden outbreak of a disease periodically over a widespread area in a devastatingly severe form causing severe losses or complete destruction. This is constantly present in a locality but it assumes severe form only on occasions. This is because of the occurrence of favourable environment responsible for the rapid development of disease. Eg: wheat stem rust (*Puccinia graminis tritici*) and powdery mildew (*Erysiphe graminis* var *tritici)*, late blight of potato (*Phytophthora infestans*). Certain disease are endemic in one area and become epidemic in another area. Eg: citrus canker is endemic in Asia but epidemic in the introduced place Florida (U.S.A.)

Pandemic**:** When an epidemic disease spreads over continents or subcontinents and involves mass mortality it is considered as pandemic. The outbreak of black stem rust of wheat in India during 1947 is best example for a pandemic disease.

Sporadic: Disease which occurs at regular intervals over limited areas or locations are called sporadic. They occur relatively in few distances,eg Fusarium wilt of cotton*(Fusarium oxysporum var vasiinfectum*).

5.1.1 Factors Responsible for Disease epiphytotic

An epidemic may cause widespread and mass destruction of crop in a short time or may persist for long periods depending upon the three following factors responsible for the disease:

1. Host 2. Pathogen 3. Environment 4. Effect of human cultural practices

1. Host factors that affect the development of epidemics

Several internal and external factors of particular host plants play an important role in the development of epidemics involving those hosts.

a. **Levels of Genetic Resistance or Susceptibility of the Host :** The host plants carrying vertical resistance do not allow a pathogen to become established in them and thus no epidemic can develop. Host plants carrying horizontal resistance will probably become infected, but the rate at which the disease and the epidemic will develop depends on the level of resistance and the environmental conditions. Susceptible host plants lacking genes for resistance against the pathogen provide the ideal substrate for establishment and development of new infections. Therefore, in the presence of a virulent pathogen and favorable environment, susceptible hosts favour the development of disease epidemics.

b. **Degree of Genetic Uniformity of host plant :** When genetically uniform host plants, particularly with regard to the genes associated with disease resistance, are grown over large areas, a greater likelihood exists that a new pathogen race will appear that can attack their genome and result in an epidemic. This phenomenon has been observed repeatedly, for example, in the C*ochliobolus* (*Helminthosporium*) blight on Victoria oats and in southern corn leaf blight. For similar reasons of genetic uniformity, the highest of epidemic development generally occur in vegetatively propagated crops, intermediate rates in self-pollinated crops, and the lowest rates in cross-pollinated crops.

c. **Type of crop :** In disease of annual crops, such as corn, vegetables, rice, and cotton, and in foliar, blossom, or fruit disease of tree and vines, epidemic generally develop much more rapidly (usually in a few weeks) than they do in disease of branches and stems of perennial woody crops

such as fruit and forest trees, for example, Tristeza in citrus, pear decline, Dutch elm disease, and chestnut blight, take years to develop.

d. **Age of Host Plants :** Plants change in their susceptibility to disease with age. In some plant-pathogen combinations, for example, *Pythium* damping-off and root rots, downy mildews, peach leaf curl, systemic smuts, rusts, bacterial blights, and viral infections, the hosts (or their parts) are susceptible only during the adult period (adult resistance). With several disease, such as rusts and viral infections plant parts are actually quite resistant to infection while still very young, become more susceptible later in their growth and then become resistant again before they are fully expanded.

2. Pathogen Factors that Affect Development of Epidemics

a. **Levels of virulence:** Virulent pathogens capable of rapidly infecting the host ensure faster production of larger amounts of inoculums and thereby disease than pathogens of lesser virulence.

b. **Quantity of inoculum near hosts :** The greater the number of pathogen propagules (bacteria, fungal spores and sclerotia, nematode eggs, virus-infected plants, etc.) within or near fields of host plants, the more inoculum reaches the hosts and at an earlier time, thereby greatly increasing the change of an epidemic.

c. **Type of Reproduction of the pathogen :** All pathogens produce many offspring, but some of them, such as most fungi, bacteria, and viruses, produce a great number of offspring then others. A few fungi, all nematodes, and parasitic plants produce relatively small numbers of offspring. Even more important is the fact that some pathogens (most fungi, bacteria, and viruses) have short reproduction cycle and therefore can produce many generations in a single growing season. These are the polycyclic pathogens that usually cause rusts, mildews, and leaf spots, and they are responsible for most of the sudden, catastrophic plant-disease epidemics in the world. Some soil fungi, such as *Fusarium* and *Verticillium*, and most nematodes usually have one to a few (up to four) reproductive cycles per growing season. For these latter pathogens, the number of offspring and, especially, the conditions of dispersal limit their potential to cause sudden and widespread epidemics in a single season. Several pathogens, such as the smuts and several short-cycle rusts, require an entire year to complete a life cycle (monocyclic

pathogens) and can therefore cause only one series of infections per year.

d. **Ecology of the Pathogen :** Some pathogens, such as most fungi and all parasitic higher plants, produce their inoculums (spores and seeds, respectively) on the surface of the aerial parts of the host. From there, spores and seeds can be dispersed with ease over a range of distances and can cause widespread epidemics. Other pathogens, such as vascular fungi and bacteria, mollicutes, viruses, and protozoa, reproduce inside the plant. In this case, spread of the pathogen is rare or impossible without the help of vectors. Therefore, such pathogens can cause epidemics only when vectors are plentiful and active. Still other pathogen, such as soil borne fungi, bacteria, and nematodes, produce their inoculum on infected plant parts in the soil, within which the inoculum disperses slowly and presents little danger for sudden or widespread epidemics.

e. **Mode of Spread of the pathogen:** The spores of many plant pathogenic fungi, such as those causing rusts, mildews and leaf spots, are released into the air and can be dispersed by air breezes or strong winds over distances varying from a few centimeters to several kilometers. These kinds of fungi are responsible for the most frequent and most widespread epidemics. In terms of their ability to cause sudden and widespread epidemics, the next most important group of pathogens includes those whose inoculum is carried by airborne vectors. Many of the viruses are transmitted by aphids, whiteflies, and some other insects. Mollicutes and fastidious bacteria are transmitted by leafhoppers, planthoppers, or psyllids. Some fungi (such as the cause of Dutch elm disease), bacteria (such as the cause of bacterial wilt of cucurbits), and even nematodes (such as the cause of pine wilt disease) are disseminated primarily by beetles. Pathogens that are transmitted by wind-blown rain (primarily fungi causing diseases like anthracnoses and apple scab, and most bacteria) are almost annually responsible for severe but somewhat localized epidemics within a field, a country, or a valley. Pathogens carried with the seed or other vegetative propagative organs (such as tubers or bulbs) are often placed in the midst of susceptible plants, but their ability to cause epidemics depends on the effectiveness of their subsequent transmission to new plants.

3. Environmental factors that affect development of Epidemic

The most important environmental factors that affect the development of plant disease epidemics are moisture, temperature, and the activities of humans in terms of cultural practices and control measures.

a) Moisture: Abundant prolonged, or repeated high moisture, whether in the form of rain, dew, or high humidity, is the dominant factor in the development of most epidemics of diseases caused by fungi (blights, downy mildews, leaf spots, rusts, and anthracnose),bacteria (leaf spots, blights, soft rots), and nematodes. Moisture not only promotes new succulent and susceptible growth in the host but more importantly it increases sporulation of fungi and multiplication of bacteria. Moisture facilitates spore release by many fungi and the oozing of bacteria to the host surface, and it enables spores to germinate and zoospores, bacteria, and nematodes to move. The presence of high levels of moisture allows all these events to take place constantly and repeatedly and leads to epidemics.

b) Temperature: Epidemics are sometimes favoured by temperatures higher or lower than the optimum for the plant because they reduce the plant's level of horizontal resistance. At certain levels, temperatures may even reduce or eliminate the vertical resistance of host plants. Plants growing at such temperatures become "stressed" and predisposed to disease, provided the pathogen remains vigorous.

Low temperature reduces the amount of inoculum of fungi, bacteria, and nematodes that survives cold winters. High temperature reduces the inoculum of viruses and mollicutes that survives hot summer temperatures. In addition, low temperatures reduce the number of vectors that survives the winter. Low temperature occurring during the growing season can reduce the activity of vectors.

The most common effect of temperature on the epidemics, however, is its effect on the pathogen during the different stages of pathogens, that is, spore germination or egg hatching, host penetration, pathogen growth or reproduction, invasion of the host, and sporulation when temperature stays within a favourable range for each of these stages, a polycyclic pathogen can complete its infection cycle within a very short time (usually in a few days). As a result, polycyclic pathogens produce many infection cycles within the growing season. Since, with each infection cycle, the amount of inoculum is multiplied many fold (100 times or more), and since some of the new inoculum is likely to spread to new plants, more infection cycle

result in more plants becoming infected by more and more pathogens, thus leading to the development of a severe epidemic.

4. Effect of Human Cultural Practices and Control Measures

a. **Site Selection and Preparation :** Low-lying and poorly drained and aerated fields, especially if near other infected fields, tend to favour the appearance and development of epidemics.

b. **Selection of Propagative Material :** The use of seed, nursery stock, and other propagative material that carries various pathogens increases the amount of initial inoculum within the crop and greatly favours the development of epidemics. The use of pathogen-free or treated propagative material can greatly reduce the change of epidemics.

c. **Cultural practices :** Continuous monoculture, large acreages planting to the same variety of crop, high level of nitrogen fertilization, no-till culture, dense plantings, overhead irrigation, injury by herbicide application, and poor sanitation all increase the possibility and severity of epidemics.

d. **Disease control measures:** Chemical sprays, cultural practices (such as sanitation and rotation), biological controls (such as using resistant varieties), and other control measures reduce or eliminate the possibility of an epidemic. Sometime however, certain controls, for example, the use of a certain chemical or planting of a certain variety, may lead to selection of virulent strains of the pathogen that either are resistant to the chemical or can attack the resistance of the variety and thus lead to epidemics.

e. **Introduction of New Pathogens:** The ease and frequency of worldwide travel have also increased the movement of seeds, tubers, nursery stock, and other agricultural goods. These events increase the possibility of introducing pathogens into areas where the hosts have not had a chance to evolve resistance to these pathogens. Such pathogens frequently lead to severe epidemics. Examples are chestnut blight, Dutch elm disease, and citrus canker caused by the bacterium *Xanthomonas campestris pv. citri*.

5.3 Measurement of Plant Yield Loss

Disease severity is usually expressed as the percentage or proportion of plant area or fruit volume destroyed by a pathogen.

Yield loss almost always results in economic loss from disease. Economic loss occurs whenever economic returns from the crop decrease either because of reduced yield or because of the cost of agricultural activities undertaken to reduce damage to the crop, or both.

The level of disease, that is, the amount of plant damage, at which control costs just equal incremental crop returns is called the economic threshold of the disease. The economic threshold of a crop-pathogen system varies with the tolerance level (damage threshold) of the crop, which depends on the growth stage of the crop when attacked, crop management practices, environment, shift in pathogen virulence and new control practices.

5.4 Patterns of Epidemics

The patterns of an epidemic in terms of the numbers of lesions, the amount of diseased tissue or the numbers of diseased plants is given by a curve that shows the progress of the epidemic over time, called the disease-progress curve. The point of origin and shape of a disease-progress curve reveal information about the time of appearance and amount of inoculum, changes in host susceptibility during the growing period, recurrent weather events, and the effectiveness of cultural and control measures. Disease-progress curves, because they are affected by weather, variety, etc., vary somewhat with location and time but they are generally characteristic for some groups of diseases.

The pattern of an epidemic, in terms of changes in the number of lesions, the amount of diseased tissue, and the number of diseased plants as it spreads over distance, is given by a curve that is called the dispersal or disease-gradient curve. Because the amount of disease is generally greater near the source of inoculum and decreases with increasing distance from the source, most disease-gradient curves are quite similar, at least in the early stage of the epidemic.

From the data collected at various time intervals and used to plot the disease-progress curve of a disease, one can obtain the epidemic rate of the disease, which is the rate of growth of the epidemic. The epidemic rate, generally designated r, is the amount of increase of disease per unit of time (per day, week, or year) in the plant population under consideration. The patterns of epidemic rates are given by curves called rate curves, and these curves are different for various groups of disease.

5.5 Modeling of Plant Disease Epidemics

An epidemic is a dynamic process, it begins on one or a few plants and then, depending on the kind, magnitude and duration of environmental factors that influence the host and pathogen, increases in severity and spreads over a larger geographic area until it finally dies down. Epidemics come to a stop when all host plants are either killed by the pathogen, become resistant to the pathogen as they age, or are harvested. In many cases epidemic slow down or come to a stop when the weather turns dry or unseasonably cold. For phenomena, observations, measurements, mathematical formulas, and computer are used extensively to study the development and to predict the size, path, and time of attack in any given location.

In an effort to improve our ability to understand and predict the development of an epidemic, plant pathologists since the late 1960s have been developing models of potential epidemic of the most common and serious disease. The construction of a model takes into account all of the components and as many the subcomponents of a specific plant disease for which there is information for quantitative treatment, that is, for treatment by mathematical formulas.

With the help of computer various programmes are written by plant pathologists that allow simulation of epidemics of several plant diseases. One of the first computer simulation programs, called EPIDEM, was written in 1969 and resulted from modeling each stage of life cycle of a pathogen as a function of the environment. It was designed to simulate early blight epidemic of tomato and potato caused by the fungus *Alternaria solani.* Subsequently, computer simulators were written for *Cercospora* blight of celery (CERCOS), for *Mycosphaerella* blight of Chrysanthemums (MYCOS), for southern corn leaf blight caused by *Cochliobolus* (*Helminthosporium*) *maydis* (EPICORN), and for apple scab caused by *Venturia inaequalis* (EPIVEN). A more general and more flexible plant disease simulator, called EPIDEMIC, was written primarily for stripe rust of wheat but could be easily modified for other host-pathogen systems. Computer simulation programs are now available for numerous plant diseases.

In a computer simulation of an epidemic, the computer is given data, describing the various subcomponents of the epidemic and control practices at specific points in time (such as at weekly intervals). The computer then provides continuous information regarding not only the spread and severity of the disease over time, but also the final crop and economic losses likely to be

caused by the disease under the conditions of the epidemic as given to the computer.

Computer simulation of epidemic is extremely useful as an educational exercise for student of plant pathology and also for farmers so that they can better understand and appreciate the effect of each epidemic subcomponent on the final size of their crop loss. Computer simulations of epidemics are, however, even more useful in actual disease situations. There, they serve as tools that can evaluate the importance of the size of each epidemic subcomponent, at a particular point in time of the epidemic, by projecting its effect on the final crop loss. By highlighting the subcomponents of an epidemic that are most important at a particular time, the simulation serves to direct attention to management measures that are effective against these particular epidemic subcomponents. In subsequent evaluations of the epidemic, the computer evaluates not only the current status of the disease but also the effectiveness of the applied management measures in controlling the epidemic.

5.6 Plant Disease Forecasting

Disease forecasts are predictions of probable outbreaks or increase in intensity of disease. It involves well organized team work and expenditure of time, energy and money. The forecasting services are utilized for giving information to the farmers of a particular area, that weather conditions are favorable for the appearance of disease in epidemic form and control measures should be adopted in due course of time, to check the disease incidence.

It is used as an aid to the timely application of chemicals. Among the first spray warning services to be established for growers, were the grapevine downy mildew forecasting schemes in France, Germany and Italy in the 1920s. Disease forecasting methods are available for the following plant diseases.

S. No.	Plant Disease	Countries
1.	Grapevine downy mildew	Australia, France, Germany, Greece, Italy, Romania, Spain, USSR, Yugoslavia
2.	Cucurbit downy mildew	U.S.A.
3.	Potato late blight	Australia, Brazil, Finland, France, Germany, Greece, Japan, the Netherland, Norway, Peru, U.K., U.S.S.R.
4.	Tobacco blue mould	Canada, U.S.A.

5.	Apple and pear scab	Australia, Canada, Netherland, New Zealand, U.S.A.
6.	Sugarbeet root rot	U.S.A.
7.	Wheat brown (leaf) rust	U.S.A.
8.	Corn bacterial wilt	U.S.A.
9.	Sugarbeet curly top	U.S.A.

Being able to forecast plant disease epidemic is intellectually stimulating and also an indication of the success of modeling or computer simulation of particular diseases. However, it is extremely useful to farmers in the practical management of crop disease. Disease forecasting allows the prediction of probable outbreaks or increases in intensity of disease and, therefore, allows us to determine whether, when, and where a particular management practice should be applied. In managing the diseases of their crops, growers must always weigh the risks, costs, and benefits of each of numerous decisions. For example, they must decide whether or not to plant a certain crop in a particular field.

To develop a plant disease forecast, one must take into account several characteristics of the particular pathogen, host and environment. In general, for most monocyclic (such as root rot of peas and Stewart's wilt of corn) and for a few polycyclic diseases that may have a large amount of initial inoculum (such as apple scab), disease development may be predicted by assessing the amount of initial inoculum. For polycyclic diseases (such as late blight of potato) that have a small amount of initial inoculums but many infection cycles, disease development can best be predicted by assessing the rate of occurrence of the infection cycles. For diseases in which both the amount of initial inoculum and the number of disease cycles are large for example beet yellows both factors must be assessed for accurate prediction of disease epidemics

5.6.1 **Information needed for disease forecasting**

Sometime the forecasting of plant diseases may be known as applied epidemiology. Hence, knowledge of epidemiology (development of disease under the influence of factors associated with the host, pathogen) is necessary for accurate forecasting. The factors of epidemic and its components should be known in advance before forecasting is done.

The information required for forecasting are:

1. Host factors

a. Prevalence of susceptible varieties in the given locality

b. Response of host at different stages of the growth to the activity of pathogen e.g. some diseases are found during seedling stages while others attack grown up plants.
c. Density and distribution of the host in a given locality. Dense populations of susceptible variety invite quick spread of an epidemic. Growing susceptible varieties in scattered locations and that too in a limited area are less prone to epiphytotic.

2.**Pathogen factors**

It is often difficult or impossible in the absence of the host to detect small population of most pathogens. Inoculum propagules of soil borne pathogens, such as fungi and nematodes, are estimated after extraction or trapping from soil. Airborne fungal spores and insect vectors are estimated by trapping them in various devices. Following parameters should be taken care of while forecasting a disease.

a. Amount of primary (initial) inoculum in the air, soil or planting material
b. Dispersal of inoculums
c. Spore germination
d. Infection
e. Incubation period
f. Sporulation on the infected host
g. Re-dispersal / Dissemination of spores
h. Perennating stage
i. Inoculum potential and density in the seed, soil and air

3. Environmental factors

Monitoring weather factors during a plant disease epidemic presents enormous difficulties. They are as follows:

a. Temperature
b. Humidity
c. Light intensity
d. Wind velocity

4. Requirements or conditions for disease forecasting

There are five main requirements which must be satisfied before a useful and successful disease forecast is made.

1. The disease must cause economically significant damage in terms of yield or quality. Damage assessment is essential to develop strategy for controlling a disease. e.g., annual estimation of yield loss caused by barley powdery mildew (*Erysiphe polygoni)* in England and wales had ranged

from 6 to 13%, Potato late blight can cause a yield loss of 28% if the disease reaches the 75% stage by mid-August. Diseases like apple scab and potato common scab reduces the quality of the produce lower the value of the harvested crop and cause considerable financial loss to the growers.

2. Control measures must be available at an economically acceptable cost.
3. The disease must vary each season in the timing of the first infections and its subsequent rate of progress. If it does not, there is no need for forecasting.
4. The criteria or model used in making a prediction must be based on sound investigational work carried out in the laboratory and in the field and tested over a number of years to establish its accuracy and applicability in all the location where its use is envisaged.
5. Growers must have sufficient man power and equipments to apply control measures when disease warning is given. Long-term warnings or prediction are more useful than short-term warning or predictions.

5.6.2 Methods of Disease Forecasting

On the basis of disease-weather relationship the forecasting services have been introduced in several countries like Great Britain, Germany, U.S.A, Canada and Russia for late blight of potato and France, Germany and Italy for downy mildew of vine. Disease forecasting requires field observations on the pathogen characters, collection of weather data, variety of the crop and certain investigations and their correlations. Usually the following methods are employed in disease forecasting.

1. Forecasting based on primary inoculums

Presence of primary inoculum, its density and viability are determined in the air, soil or planting material. Occurrence of the viable spores or propagules in the air can be assessed by using different air trapping devices (spore traps). In the case of soil-borne diseases the primary inoculums in the soil can be determined by monoculture method.

Presence of loose smut of wheat, ergot of pearl millet and viral diseases of potato can be detected in the seed lots at random by different seed testing methods. Seed testing methods can be used to determine potential disease incidence and enable decision to be made on the need for chemical seed treatment. The extent of many virus diseases is dependent on the severity of the preceding winter which affects the size of vector population in the growing season. E.g. sugarbeet yellows virus.

2. Forecasting based on weather conditions

Weather conditions *viz.,* temperature, relative humidity, rainfall, light, wind velocity etc., during the crop season and during the winter crop season are measured. Weather conditions above the crop and at the soil surface are also recorded.

Monitoring weather factors during a plant disease epidemic presents enormous difficulties. The difficulties arise from the need for continuous monitoring of several different factors (temperature, relative humidity, leaf wetness, rain, wind and cloudiness) at various locations in the crop canopy or on plant surfaces in one or more fields. In the past, measurements were made with mechanical instruments that measured these environmental variables roughly or infrequently and recorded the data inconveniently as ink traces on chart paper. Since the 1970, however, several types of electronic sensors have been developed that produce electrical outputs easily recorded by computerized data loggers. Such computerized sensors have greatly improved studies of weather in relation to disease and have facilitated the acceptance and use of predictive systems for disease control on the farm.

Several types of traditional and battery-operated electrical instruments are used to measure various weather factors. Temperature measurements are made with various types of thermometers, hygrothermographs thermocouples, and especially with thermistors (the latter are semiconductors whose electrical resistance changes considerably with temperature). Relative humidity measurement are made with a hygrothermograph (which depends on the contraction and expansion of human hair in relation to relative humidity changes), with a ventilated psychrometer (consisting of a wet- and dry-bulb thermometer or a wet and dry thermistor), or with an electrode-bonding sulfonated polystyrene plate (whose resistance change logarithmically with relative humidity). Leaf wetness is monitored with string- types sensors that constrict when moistened or slacken when dry and either leave an ink trace in the process or close or break an electrical circuit. Several types of electrical wetness sensors are available that can be either clipped onto leaves or placed among the leaves; they detect and measure the duration of rain or dew because either of the latter helps close the circuit between two pairs of electrodes. Rain, wind, and cloudiness (irradiance) are still measured by the traditional instrument (rain funnels and tipping-bucket gauges for rain, cups and thermal anemometers for wind speed, vanes for wind direction, and pyranometers for

irradiance). Several of these instruments, however, have become adapted electronic monitoring.

In modern weather-monitoring systems, the weather sensors are connected to data-logging devices. The data may be read on a digital display, or they are transmitted to a cassette tape recorder or a printer. From the cassette the data may be transferred to a microcomputer. There they may be viewed, processed in several computer languages, organized into separate matrices for each weather variable, plotted and analyzed. Depending on particular disease model used, accurate weather information provides the most useful basis to predict sporulation and infection and therefore provides the best warning to time disease management practices, such as the application of fungicides.

3. Forecasting based on correlative information

Weather data of several years are collected and correlated with the intensity of the diseases. The data are compared and then the forecasting of the disease is done. Forecasting criteria developed from comparisons of disease observation with standard meteorological data have been provided for diseases like *Septoria* leaf blotch of wheat, fire blight of apple and barley powdery mildew.

4. Use of computer for disease forecasting

In some advanced countries forecasting of disease is made by use of computers. This system gives the result quickly. One such computer based programs in the USA is known as 'Blitecast' for potato late blight. In a computer simulation of an epidemic, the computer is given data describing the various subcomponents of the epidemic and control practices at specific points in time (like weekly intervals). The computer than provide continuous information regarding not only the spread and severity of the disease over time, but also the final crop and economic losses likely to be caused by the disease under the conditions of the epidemic as given to the computer.

Computer simulation of epidemics is extremely useful for students of plant pathology and also for farmers so that they can better understand and appreciate the effect of each epidemic subcomponent on the final size of their crop loss.

Computer simulation of epidemic serves as tools that can evaluate the importance of the size of each epidemic subcomponent at a particular point in time of the epidemic, by projecting its effect on the final crop loss.

Examples of well developed forecasting systems are given below.

a) **Early and late leaf spots of groundnut :** A technique has been developed for forecasting early and late leaf spots of groundnut in the U.S.A. When

the groundnut foliage remains wet for a period greater than or equal to 10 h and the minimum temperature is 21^{o} C or higher for two consecutive days or nights, the disease development is forecasted.

A computer programme has been developed in the USA. This is accurate and is widely used in the USA. The data on hours for day with relative humidity (RH) of 95% and above and minimum temperature (T) during the RH observations for the period, for the previous 5 days are fed to the computer. Calculations are rounded to whole numbers. The T/RH index for each of the five days is calculated e.g., when hours of the RH 95% equal 10 and the minimum temperature during the period equals 21.1^{O} C the T/RH index is 2.0. The T/RH indices for days 4 and 5 are summed. If the total index exceeds 4 disease is forecasted. If the index is 3 or less no disease is forecasted.

b) **Late blight of potato :** In the USA a forecasting programme has been developed for late blight of potato (*Phytophthora infestans).* The initial appearance of late blight is forecasted 7 to 14 days after the occurrence of 10 consecutive blight favourable days. A day is considered to be blight favourable when the 5 day average temperature is 25.5^{o} C and the total rainfall for the last 10 days is more than 3.0 cm. A computerized version (Blitecast) has also been developed in the U.S.A. for forecasting potato late blight. Blitecast is written in Fortran IV. When a farmer desires blight cast (blitecast) he telephones the blight cast operator and reports the most recently recorded environmental data. The operator calls for the blight cast programmes in the computer *viz*., typewriter terminal and feed the new data into the computer. Within a fraction of second the computer analyses the data and series of a forecast and spray recommendations to the operator who relays in to the farmer.

The entire operation can be completed during standard three minutes telephone call. The system makes one of the four recommendations viz., no spray. Late blight warning, 7 days spray schedule or 5 days spray schedule. The last 5 days spray schedule is issued only during severe blight weather. In West Germany, 'Phytoprog' is the programme used. It is based on measurements of temperature, relative humidity and rainfall. Phytoprog provides a negative prognose (an indication of when the usual routine spray application should be dispensed with).

a. **Rice blast :** In India, forecasting rice blast (*Pyricularia ozyzae*) is done by correlative information method. It is predicted on the basis of minimum

night temperature 20 to 26^{o} C in association with high relative humidity of 90% or above. Computer based forecasting system has also been developed for rice blast in India.

b. **Wheat stem rust :** Forecasting wheat stem rust epidemic is done by analyzing the rain samples which give precise data for inoculum present in the air. Moreover several wind trajectors are also prepared to survey the air-borne primary inoculums and its deposition. It has been observed that primary inoculum comes from south India, to the plains of central and north India.

c. **Brown stripe downy mildew of corn** : The forecasting of brown stripe downy mildew of corn (*Sclerophthora raysise* var. *zeae*) which is restricted to India is done on the basis of average rainfall 100 to 200 cm or more accompanied by low temperature (25^{o} C or less). Sproe trapping techniques of acquisition of biological data for consecutive forecasting models are important. Spore traps have been widely used in to complete disease with weather conditions. Spore trapping is useful for understanding epidemiology of a disease and behaviour of the pathogens. This helps in developing models on dispersal of pathogens or on epidemiology of the disease and to formulate methods of management. Methodology of spore trapping depends on the following objectives of the worker.

   1. Biology of the pathogen
   2. For infection forecasting
   3. Spore dispersal gradients
   4. Management of the disease

In epidemics of air-borne plant diseases the number of spores of the pathogen landing on the plant which depends on the number of spores in the atmosphere above the crop is an important factor for the quantitative sampling of the atmosphere (number of spores per unit volume of air). For trapping and estimating these studies different types of traps are used. The following spore traps are usually employed in trapping of fungal spores.

Cylindrical rods or microscopic glass slides: it helps to gather data on the spore arrival in a locality. In this, the surface of microscopic slide is smeared with grease and made sticky. In the method, quantitative estimation is not possible as number of spores collected is very low.

1. **Hirst's volumetric spore trap (Hirst 1952)** : In this instrument, air is sucked into at a controlled rate and impinged on to a glass slide moved by a clockwork mechanism past the orifice. It gives continuous count of spores

in 24 hours. The number of spores per unit volume of air at any given time can thus be calculated.

2. **Rotorod sampler or rotorod spore trap (Sutton and Jones 1976)** : It comprises of a 'U' shaped rod attached at its mid point to the shaft of a small battery operated electric motor. In this equipment the surface of the rod is covered with a Vaseline strip of transparent cellophanes to catch spores which can be taken of and mounted on a glass slide. From the area of the strip and the speed of rotation, the volume of air sample can be calculated.
3. **Anderson cascade spore sampler** : It is a device where Petri plates with nutrient agar are used to collect the spores.
4. **Bourdillon slit sampler** : Air is sucked in a chamber by vacuum pump which strikes the rotating Petri dish containing agar medium. The agar medium retains the spores sucked in the air. Concentration of viable spores is calculated after counting germinated spores in the medium.
5. **Burkard's 7 day volumetric spore trap :** This device records spores in the air drawn by a pump on 7 days basis on a cellophane strip wrapped on a drum rotating inside a chamber.
6. **Jet spore trap** : In the above sampling methods, the viability of the spores cannot be determined. To overcome this, living plants have been used as spore trap. A jet spore trap in which spores are impacted in an air jet into a column of still air, through which they fall, to settle on leaf segments exposed at the base of the chamber. In this trap, suitable cultivars of host plants can be employed to determine number of viable spores.

5.7 Disease Warning System

In many states and countries different types of warning system are functional for one or more plant diseases. The purpose of these systems is to warn farmers of the impending onset of an infection period or to inform that the infection period has already occurred so that they can take appropriate control measures to stop recent infections from developing or prevent further infections from occurring.

The warning system starts with a grower, an extension agent, or a private consultant surveying certain fields on a regular basis or when the weather conditions are likely to favour maturation of the primary inoculums or appearance of the particular disease. When mature inoculums or traces of disease are found the country extension office is notified which in turn notify

the state extension plant pathologist who collates all reports about the disease from around the state and by electronic mail, telephone, fax or in writing notifies all concerned country agents(Pest alert). They in turn notify the farmers in their country. For diseases of potential regional or national epidemic consequences the state extension plant pathologist notifies the federal plant disease survey office of the U.S. Department of Agriculture which in turn notifies all of adjacent state extension plant pathologists that may be affected by that plant disease.

Since 1970s computerized warning systems are in use for certain diseases in some states.Some of them such as BLITECAST use centrally located computers that process weather data either collected on the farm by individual growers and electronically transmitted or phoned when certain weather conditions prevail or at certain intervals. The computer then process the data , determine that weather an infection period is likely to occur or cannot occur, and makes a recommendation to the grower as to weather to spray or not and what material to apply.

After 1980s small special purpose computers have been developed that have field sensors and can be mounted on a post in a farmers field. Such units (like Apple Scab predictor) monitor and collect data in the field on temperature, relative humidity, duration of leaf wetness, rainfall amount ,analyse the data automatically ,make predictions of disease occurrence, intensity and make recommendation for disease control measures.

5.8 Development of Expert System in Plant Pathology

Expert systems are computer programmes that try to equal or surpass the logic and ability of an expert professional in solving the problem. The dependability of an expert system is proportional to the knowledge of the experts who produced it. Expert system can use data in any format and can suggest a solution to the problem. These expert systems are frequently used for diagnostic purposes for identifying the cause of a disease by the symptoms and related observations. By incorporating infection model of the important diseases of a crop into the knowledge base of the computers the expert system can advice growers of disease potentials on the basis of the actual occurrence of infection periods and provides pesticide recommendations and its amount and timing of application.

The most important part of creating an expert system is the quality of the experts providing the knowledge that is inputed in the system which is later on represented in the form that can be converted into computer code. Once a

prototype expert system is generated it is first tested for logic and accuracy. It is also reviewed and if necessary revised by the other experts.

The availability of computer has allowed plant pathologists to write programs that allows simulation of epidemics of several plant diseases. One of the first computer simulation programs called EPIDEM was written in 1969 and resulted from modeling each stage of life cycle of a pathogen as a function of the environment. This program was designed to stimulate early blight epidemics of tomato and potato caused by *Alternaria solani*.

BLITECAST (1975) is a computerized forecasting system for potato late blight and considered to be the precursor to expert systems. A number of expert systems have been developed for the diagnosis or management of diseases of tomato (TOM), grape (GrapES), wheat (CONSELLOR), peach (CALEX) and wheat (Morecrop) etc.

The word Morecrop stands for 'Managerial Options for Reasonable Economic Control of rusts and other pathogens" which is designed to provide disease management options in different geographical regions and agronomic zones of the Pacific North West.

Expert systems are primarily used for high value horticultural crops that require frequent application of pesticides as part of their disease and pest management.

5.9 Summary

Plant disease epidemiology is the study of disease in plant populations. Plant disease epidemiology is often looked at from a multi-disciplinary approach, requiring biological, statistical, agronomic and ecological perspectives. Biology is necessary for understanding the pathogen and its life cycle. It is also necessary for understanding the physiology of the crop and how the pathogen is adversely affecting it. Agronomic practices often influence disease incidence for better or for worse. Ecological influences are numerous. Native species of plants may serve as reservoirs for pathogens that cause disease in crops. Statistical models are often applied in order to summarize and describe the complexity of plant disease epidemiology, so that disease processes can be more readily understood. The comparisons between patterns of disease progress for different diseases, cultivars, management strategies, or environmental settings can help in determining how plant diseases may best be managed. Policy can be influential in the occurrence of diseases, through actions such as restrictions on imports from sources where a disease occurs.

The elements of an epidemic are referred to as the "disease triangle" a susceptible host, pathogen, and conducive environment. Plant disease forecasting systems are based on assumptions about the pathogen's interactions with the host and environment, the disease triangle.

Good disease forecasting systems must be reliable, simple, cost-effective and applicable to many diseases. It involves well organized team work and expenditure of time, energy and money. An example of a multiple disease/pest forecasting system is the EPIdemiology, PREdiction, and PREvention (EPIPRE) system developed in the Netherlands for winter wheat that focused on multiple pathogens. Correct choice of a model is essential for a disease forecasting system to be useful. Plant disease forecasting models must be thoroughly tested and validated after being developed.

5.10 Glossary

- **Epiphytotics:** A widespread and destructive outbreak of a disease of plant.
- **Epidemic:** A widespread and severe outbreak of a disease.
- **Eradicant:** A chemical substance that destroy a pathogen at its source.
- **Exclusion:** Control of plant disease by excluding the pathogen or infected plant material from disease free areas.
- **Chemotherapy:** Control of plant disease with chemicals that are absorbed and are translocated early.
- **Cross Protection:** The phenomenon in which plant tissues infected with one strain of virus are protected from infection by other strains of the same virus.
- **Pandemic:** Very widely distributed, referring to plant disease.
- **Susceptibility:** The inability of plant to resist the effect of a pathogen.

5.11 Self -Learning Exercise

Section A : (Very Short Answer Type Questions)

1. Quarantine laws were first enacted in
   (a)USA (b) India (c) France (d) China
2. Abiotic disease are caused by
   (a) Saprophyte (b) Parasites (c) Viruses (d) Unfavourable environment
3. Sporadic disease are disease which occur
   (a) Regularly year after year (b) Only here and there at irregular intervals (c) Remain present continuously (d) none

4. Epiphytotic diseases

(a) Occur here and there at irregular intervals (b) spread widely but occur periodically (c) Regularly present in a certain region (d) None.

Section B : (Short Answer Type Questions)

1. Define the term epiphytotic.
2. What do you mean by epidemics?
3. What is sporadic disease?
4. Define plant disease forecasting.
5. Name computerized forecasting system for potato late blight.

Section C : (Long Answer Type Questions)

1. Discuss in detail about the factors responsible for causing disease.
2. Explain in detail about the various patterns of epidemics.
3. Write a detail note on the plant disease forecasting.
4. Discuss in detail about the basic information required for plant disease forecasting.
5. Discuss the methods used for plant disease forecasting.
6. Explain the role of computer in plant disease forecasting.

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Unit - 6

Plant Disease Management – I: Physical, Chemical, Biological, Cultural

Structure of the Unit:

6.0 Objectives

After going through this unit you will be able to understand about:

- plant disease management
- types of management practices
- limitations and advantages of different management practices

6.1 Introduction

The critical shortage of food supplies in several countries and rapid increase in population in recent years brought to the forefront the enhanced necessity to protect as much of the crop yields as possible by reducing the losses caused by infection and diseases.

To control the plant diseases, it becomes necessary to know about the causal organism of the disease, its life-history and the meteorological conditions which influence the host and parasite interaction and stimulate the increased chances to cause loss in production.

6.2 Types of Control Method

Control measures may be divided into two main groups-

(1) Prophylaxis and (2) Immunization or disease resistance

Prophylaxis includes the protection of the host from exposure to the pathogen, from infection or from the environmental factors favourable to disease development.

Immunization or disease resistance implies the improvement of resistance of the host to infection and to disease development. This method is generally used as a means of control by the development of strains of the host through hybridization and/or selection, which are more resistant to one or more pathogens.

The term Prophylaxis includes a wide range of control measures. Such variations of control measures are being considered under three sub-groups-

(1) Exclusion, (2) Eradication, (3) Direct protection

(i) Exclusion of the Parasite

The term exclusion of the parasite from the host means the measures are designed to keep away the pathogen from entering the area in which the host is growing or to reduce its population to such a minimum extent to which is harmless to the host. These measures include quarantine regulation; inspection and certification, etc.

Quarantines : Many plant diseases have been introduced in our country from time to time from some other part of the world. The possibility of the entry of the pathogen in a new area has been increased several fold with the extension of transport facilities.

In several agriculturally advanced countries the plant quarantine legislation is strictly followed to check the movement of diseased plant material or of fungi, bacteria or viruses which are responsible for causing plant diseases.

In India "Destructive Insects and Pests act" was passed by the legislature in 1914 and it has been amended from time to time.

Inspection and Certification : In several agriculturally advanced countries the certification of plant and its products are being done. The certificate must contain that the living plants were thoroughly examined on a date which must be not more than fourteen days prior to shipment and are healthy.

There is a provision for inspection of any consignment and for its treatment or destruction. Inspection and certification are compulsory useful measures to

prevent the introduction of diseases from a foreign country or from one part of the country to another.

(2) Eradication of The Parasite

Eradication of the pathogen can be done in several ways such as- crop rotation, removal of infected parts, elimination of alternate host, destruction of wild hosts and weeds, rogueing. For example the intensity of the black rust of wheat has been greatly reduced in USA by systematic removal of barberry bushes, the alternate host of the black rust. On the other hand hand, sugarcane smut is best controlled by roguing.

Crop Rotation : Most of the serious parasitic fungi cannot multiply in the absence of the host plant. They may survive in the soil for sometime, but if the proper host plants are absent their number may deteriorate and they may in due course even go out of existence. A parasite may persist in the soil on diseased plant debris after the crop has been harvested. Many of the pathogenic organisms survive until the host residue persists as a substrate for their saprophytic existence.

In general the crop rotation eliminates the source of inoculum by a 3 or 4 year duration of rotation with non host crops.

Removal of Infected Parts : The destruction of diseased parts of plants in the field removes the main foci of infection and thus breaks the chain. Citrus canker, caused by *Xanthomonas citri*, can be effectively controlled by the removal of affected plant parts. Destruction of affected tree tops and practice of general sanitation by burning all diseased parts is an effective protection against the breakout of the disease.

Elimination of Alternate Hosts

In some of the destructive diseases in which long-cycled, heteroecious rust fungi are concerned, the eradication of alternate host is a possible means of control. The barberry eradication campaign in the control of black rust of wheat in United States had been proved effective.

Destruction of Wild Hosts and Weeds : A large number of diseases, particularly viral diseases, are harboured in wild hosts and weeds. Removal of such wild hosts is a good method of control. The yellow vein mosaic of bhindi persists on a wild host (Hibiscus *tetraphylus*) in nature and the systematic removal of this wild host has proved successful in combating the disease. Similary, effective control has been obtained in sugarcane against mosaic, by

the systematic removal of maize from the neighbouring fields, as the latter acts as a collateral host for the perpetuation of the virus infection chain.

Roguing : This practice consists of destruction of affected plants from the fields at an early stage by removing foci of infection and preventing wide dissemination of the pathogen. This makes one of the routine methods of control of viral diseases in plants. The virus-infected plants should be removed and destroyed in early stage to check the spread of viral diseases. The control of yellow mosaic of bhindi has been obtained through these practices.

(3) Direct Proction

6.2.1 Physical Methods

The physical agents most commonly used in controlling plant diseases are temperature (high or low), dry air, unfavorable light wavelengths and different types of radiation. With some crops, cultivation in glass or plastic greenhouses provides physical barriers to pathogens and protects the crop from some diseases. Similarly, plastic or net covering of row crops may protect the crop from infection by preventing pathogens of vectors from reaching the plants.

1. Soil Sterilization by Heat

Soil can be sterilized in greenhouses and in seed beds and cold frames, by the heat carried in live or aerated steam or hot water. The soil is steam sterilized either in special containers (soil sterilization), into which steam is supplied under pressure, or on the greenhouse benches, in which steam is piped into and is allowed to diffuse through the soil. At about 50^0 C, nematodes, some oomycetes fungi and other water molds are killed, whereas most plant pathogenic fungi and bacteria, along with some worms, slugs and centipedes are usually killed at temperatures between 60 and 70^OC. About 82^oC, most weeds, the rest of the plant pathogenic bacteria, most plant viruses in plant debris and most of the insects are killed. Heat-tolerant seeds of weed and some plant viruses like tobacco mosaic virus (TMV) are killed at or near the boiling point (between 95 and 100^o C). The soil sterilization is generally completed when the temperature in the coldest part of the soil has remained for at least 30 minutes at 82^o C or above, the temperature at which almost all plant pathogens in the soil are killed. Heat sterilization of soil can also be achieved by producing the heat electrically rather than supplied by steam or hot water.

It should be taken care that excessively high or prolonged high temperatures should be avoided during soil sterilization because such conditions destroy all normal saprophytic micro flora in the soil and also result in release of toxic levels of some (e.g., manganese) salt and accumulation of toxic levels of

ammonia (by killing the nitrifying bacteria before they kill the more heat-resistant ammonifying bacteria), which may damage or kill plants planted afterward.

2. Hot-Water Treatment of Propagative Organs

Hot-water treatment of certain seeds, bulbs, and nursery stock is used to kill any pathogens with which they are infected or which may be present inside seed coats, bulb scales, etc., or which may be present in external surfaces or wounds. In some diseases, seed treatment with hot water was the only means of control for many years like in the loose smut of cereals, in which the fungus overwinters as mycelium inside the seed where the chemicals cannot be reached. Similarly, treatment of bulbs and nursery stock with hot water frees them, such as nematodes like *Ditylenchus dipsaci* in bulbs of various ornamentals and *Radolpbolus similis* in citrus rootstocks.

The effectiveness of the method is based on the fact that dormant plant organs can withstand higher temperatures than those their respective pathogens can survive for a given time. The temperature of the hot water used and the duration of the treatment vary with the different host-pathogen combinations. Thus, in the loose smut of wheat the seed is kept in hot water at 52^{o} C for 11 minutes, whereas bulbs treated for *Ditylenchus dipsaci* are kept at 43^{o}C for 3 hours.

3. Hot-Air Treatment of Storage Organs

Treatment of storage organs with warm air (curing) removes the excess moisture from their surfaces and hastens the healing of wound, thus preventing their infection by certain weak pathogens. For example, keeping sweet potatoes at 28 to 32^{o}C for 2 weeks helps the wounds to heal and prevents infection by *Rhizopus* and by soft-rotting bacteria. Also, hot-air curing of harvested ears of corn, tobacco leaves, etc. removes most moisture from them and protects them from attack by fungal and bacterial saprophytes. Similarly, dry heat treatment of barley seed at 72^{o} C for 7 to 10 days eliminates the leaf streak and black chaff-causing bacterium *Xanthomonas campestris pv. translucens* from the seed with negligible reduction of seed germination.

4. Control by Eliminating Certain Light Wavelengths

Alternaria and *Botrytis* are examples of plant pathogenic fungi that sporulate only when they receive light in the ultraviolet range (below 360nm). It has been possible to control diseases on greenhouse vegetables caused by several species of these fungi by covering or constructing the greenhouse with a special UV-absorbing vinyl film that blocks transmission of light wavelength below 390 nm.

5. Drying Stored Grains and Fruit

All grains, legumes and nuts carry with them a variety and number of fungi and bacteria that can cause decay of these organs in the presence of sufficient moisture. Such decay, however, can be avoided if seeds and nuts are harvested when properly mature and then are allowed to dry in the air or are treated with heated air until the moisture content are reduced sufficiently (to about 12 percent moisture) before storage. Afterwords they are stored under condition of ventilation which do not allow buildup of moisture to a high levels (above 12 percent) that would allow storage fungi to become activated. Fleshy fruits, such as peaches and strawberries, should be harvested later in the day, after the dew is gone as to ensure that the fruit does not carry surface moisture during storage and may result in decay of the fruit by fungi and bacteria.

Many fruits also can be stored dry for a long time and can be kept free of disease if they are dried sufficiently before storage and if moisture is kept below a certain level during storage. For example, grapes, dates, and figs can be dried in the sun or through warm air treatment to produce raising, prunes, and dried dates and figs, respectively, that are generally unaffected by fungi and bacteria as long as they are kept dry. Even slices of fleshy fruits such as apples, peaches, and apricots can be protected from infection and decay by fungi and bacteria if they are sufficiently dried by exposure to the sun or to warm air currents.

6. Disease Control by Refrigeration

Refrigeration is probably the most widely used and effective method of controlling postharvest diseases of fleshy plant products. Although low temperatures at or slightly above the freezing point do not kill any of the pathogens that may be on or in the plant tissues but they do inhibit or greatly retard the growth and activities of all such pathogens and thereby reduce the spread of existing infections and the initiation of new ones. Most perishable fruits and vegetables should be refrigerated as soon as possible after harvest, transported in refrigerated vehicles and kept refrigerated until they are used by the consumer. Regular refrigeration of succulent fruits and vegetable should be done as soon as possible. Regular refrigeration of these succulent fruits and vegetables is sometimes preceded by a quick hydrocooling or air cooling of these products, aimed at removing the excess heat carried in them from the field to store as quickly as possible to prevent the development of any new or latent infections. The magnitude of infection from growers to consumers is immense.

7. Disease Control by Radiation

A different types of electromagnetic radiation such as ultraviolet light, X rays and gamma rays, as particulate(as alpha particle and beta particles) have been studied for their ability to control postharvest diseases of fruits and vegetables by killing the pathogens present on them. Some satisfactory results were obtained in experimental studies using gamma rays to control postharvest infections of peaches and strawberries but the major disadvantage with this treatment is the dosage of radiation required to kill the pathogen may also injure the plant tissues on which the pathogens exist. Also the method of treatment of foodstuffs, although found safe and properly licensed by the USDA is vigorously opposed by certain segments of the population. So far, no plant diseases are commercially controlled by radiation.

6.2.2 Chemical Methods

Chemical based pesticides are generally used to protect plant surfaces from infection or to eradicate a pathogen which has already infected a plant. A few chemical treatments however, are aimed at eradicating or greatly reducing the inoculum before it comes in contact with the plant. They include soil treatment disinfection of warehouses, sanitation of handling, equipment and control of insect vectors of pathogens.

1. Soil treatment with chemicals

Soil to be planted with vegetables, strawberries, ornamentals, trees, or other high value crops, such as tobacco, is frequently treated with chemical to control primarily the nematodes but occasionally also of soil born fungi such as *Fusarium* and *Verticillium,* weeds and bacteria. Certain fungicides are applied to the soil as dusts, liquid drenches or granules to control damping of seedling, blights, crown and root rots and other diseases in fields where irrigation is possible.The fungicides is sometimes applied with the irrigation water particularly in sprinkler irrigation. The fungicides used for soil treatment includes metalaxyl, diazoben, pentachloronitrobenzene (PCNB), captan and chloroneb. The last two are used primarily as seed treatments. Most soil treatments are aimed at controlling nematodes and the materials used are volatile gases or produce volatile gases that penetrate the soil throughout.Some nematicides however are not volatile but dissolve in soil water and distribute through the soil.

2. Fumigation

The most promising method of controlling nematodes and certain other soilborne pathogens and pests in the fields has been through the use of chemicals usually called fumigation. Some of them like Chloropicrin,

Methylbromide, Dazomet and Metam sodium, either volatilize as they are applied to the soil or decompose into gases in the soil .These materials are general purpose preplant fumigant and are effective against a wide range of soil microorganisms including nematodes, many fungi, insect, certain bacteria and weeds. Contact nematicides like Fensulfothion, Carbofuran, Ethoprop and Aldicarb are of low volatility are effective against nematodes and insects, and can be applied before and after planting of many crops which are tolerant to these chemicals.

Nematicides used as soil fumigants are available as liquids under pressure, emulsifiable liquid concentrates and granules. These materials are applied to the soil either by spreading the chemical evenly over the entire field (broadcast) or by applying it only to the rows to be sowed with the crop (row treatment). In both cases the fumigation is applied through delivery tubes attached at the back of tractor-mounted chisel-tooth injection shanks or disks spaced at variable widths and usually reaching 6 inches below the soil surface. The nematicides is sealed in the soil instantly by a smoothing and firming drag or can be mixed into the soil with disk harrows or rototillers. Highly volatile nematicides are immediately covered with polyethylene sheeting and are left in place for at least 48 hours. When small areas are to be fumigated the most convenient method is through injection of the chemical with a hand applicator under trap that has been placed over the area. The edges of the trap are covered with soil prior to injection of the chemicals. Application may also be made by placement of small amounts of granules in holes or furrows six inches deep and 6-12 inches apart, which should be immediately covered with soil. In all cases of preplans soil fumigation with phytotoxic nematicides several days to 2 weeks must elapse from the time of treatment seeding or planting in the field to avoid plant injury. The effective of the fumigants however, is based on diffusion in a gaseous state through the pores.

The distance the vapors move is influenced by the size and continuity of soil pores by the soil temperature (the best range in between 10^0 and 20^0C) by of soil moisture (best at about 80 percent of field capacity) by the type of soil (more material is required for soil rich in colloidal or organic matter) and by the properties of the chemical itself. The nematicides with low volatility, such as carbofuran, do not diffuse through the soil to any great extent and must be mixed with the soil mechanically or by irrigation water or rainfall.

In practice chemical nematode control in the field is generally obtained by preplant soil fumigation with one of the nematicides applied only before

planting. These chemicals are nonspecific and control all types of nematodes. Chloropicrin, methyl bromide and metamsodiam are expensive broad-spectrum nematicides.

3. Direct protection by chemicals

One of the most commonly known means of controlling plant diseases in the field, greenhouse and somethimes in storages is by the use of chemical compounds that are toxic to the pathogens. These chemicals either inhibit germination, growth and multiplication of the pathogen or are outright lethal to the pathogen. Depending on the kind of pathogens they affect, the chemicals are called fungicides, bactericides, nematicides, viricides and herbicides. Some chemicals are broad-spectrum pesticides that are toxic to almost all kinds of pathogens. About 60 percent of all the chemicals (mostly fungicides) used to control plant diseases is applied to fruit and about 25 percent to vegetables.

Most of the chemicals are used to control diseases of the foliage and of other above ground parts of plants. Others are used to disinfest and/or protect from infection seeds, tubers and bulbs, some are used to disinfest the soil, others to disinfest warehouse, to treat wounds, to protect stored fruit and vegetables from infection.

Methods of Application of Chemicals : Chemicals used to control plant diseases are applied directly to plant or to the soil with the help of various types of equipment.

Foliage Sprays and Dusts : Chemicals applied as sprays or dusts on the foliage of plants are usually aimed to control fungal diseases and to a lesser extent control of bacterial diseases. Most fungicides and bactericides are protectants and must be present on the surface of the plant in advance of the pathogen in order to prevent infections. Their presence usually does not allow fungus spores to germinate, or the chemicals may kills spores to germinate, or may kill spores on germination. Contact of bacteria with bactericides may inhibit their multiplication or causes their death.

Some newer fungicides have a direct effect on pathogens which already invaded the leaves, fruit and stem and in this case they act as eradicants (they kill the fungus inside the host or may suppress the sporulation of the fungus without killing it). Some fungicides have a partial systemic action because they can be absorbed by parts of the leaf tissues and translocated internally into the the leaf area. Some bactericides such streptomycin, tetracyclines and some other antibiotics are also systemics when applied by injection.

Some newer systemic fungicides, like metalaxyl and the sterol inhibitors triadimefon and fenarimol are so effective in post infection applications that they can be used as rescue treatment of crops.

Fungicides and bactericides applied as sprays are generally more efficient in creating a protective residue layer on the plant surfaces when applied as dusts. Neither dusts nor sprays stick well when applied during a rain. Some other compounds like lime may be added to the active chemical in order to reduce its phytotoxicity and make it safer to the plant. The compounds with low surface tension called surfactants are often added to fungicides so that they spread better and thereby increasing the contact area between fungicide and the sprayed surface. Some compounds with good sticking ability are added to increase the adherence of the fungicide to the plant surface.

In agricultural field with sprinkler irrigation available, some control of foliar disease by systematic fungicides can be obtained by applying protectant or systemic fungicides to the foliage system.

Many fungicides and bactericides are protection in their action and they may be at the plant surface before the pathogen arrives or at least before it has time of germination entry and establishment itself in the host, because most of the pathogenic spores require a film of water on the leaf surface or at least atmospheric humidity near saturation before they can germinate. For these reasons young, exponding leaves, twigs and fruits may have to be sprayed by chemicals more often than mature tissues. The interval between sprays on mature tissue may vary from 7 to 14 day or longer depending on the particular disease, the frequency and duration of rains, the persistence or residual life of the fungicide, and the season of the year.

Types of Chemicals : A large number of chemicals have been developed to date for crop protection as fumigant, soil treatment, sprays, dust paints and pastes.

A. Inorganic Chemicals

- Copper Compounds: Bordeaux mixture (after the name of Bordeaux region in France) was used against the downy mildew of grape is the product of reaction of Copper sulphate and calcium hydroxide. It was the first Aungicide developed and most widely used copper fungicide in the world. It controls many fungal and bacterial leaf spot, blight, downy mildew and cankers.

- Inorganic Sulphur Compounds: The element sulphur is probably the oldest fungicide known. It is used as a dust, wettable powder, paste or liquid to control powdery mildew, certain rust, leaf blight and fruit rots.
- Carbonate Compounds: Sodium bicarbonate and bicarbonate salts of ammonium, potassium and lithium plus 1% superfine oil have shown the inhibitory and fungicidal property towards powdery mildew fungi of roses.
- Phosphate and Phosphonate Compounds: Spraying cucumber or grape plants with solutions of either monopotassium phosphate or dipotassium phosphate has given satisfactory results towards powdery mildew disease.
- Film forming Compounds: The compounds like antitranspirant polymers, mineral oils when applied on plant surfaces before inoculations with the pathogen significantly reduce the intensity of infection.These film forming polymers are permeable to gases , non phytotoxic and resist for at least one week and biodegradable.

B. Organic Chemicals:

1. Contact Protective Fungicides:

- **Organic Sulphur Compounds:** The dithiocarbamates are one of the most important versatile and widely used groups of modern fungicides. They include thiram, ferbam, nabam, maneb and zineb. All of these compounds are toxic to fungi mainly because they are metabolized to the isothiocyanate radical(-N=C=S)which inactivates the sulfhydryl groups(-SH) in amino acids and enzymes within pathogen cells and inhibits their production in host cells.

Thiram is a compound containing two molecules of dithiocarbamic acid and mostly used for seed and bulb treatment of vegetables and flowers.

Ferbam is a three molecule containing dithiocarbamic acid reacted with one atom of ironand used to control foliage diseases of fruit trees and ornamentals.

Maneb is a broad spectrum fungicide containsing manganese and used to controlfoliage and fruit disease of many vegetables.

Zineb is a safe multipurpose foliar and soil fungicide to control leaf spots, blights and fruit rots of vegetables, flowers and fruit trees.

Aromatic Compounds: many compounds have aromatic benzene ring which are toxic to microorganisms and have been developed into fungicide commercially. Most of them inhibit the production of compounds that have $–NH_2$ and –SH group.

Pentachloronitrobenzene (PCNB) and Terraclor is a long lasting soil fungicide and used to control soil borne disease of vegetables e.g *Botrytis, Sclerotinia and Rhizopus.*

Chlorothalonil (brand name Bravo) is a excellent broad spectrum fungicide against many leafspots, blights, downy mildews, rust, scab and fruit rots.

Biphenyl is volatile and applied by impregnating shipping materials with it and this compound volatilizes in storage and protects the stored fruits.

Heterocyclic Compounds: It includes some of the best fungicides like Captan, Iprodione and Vinclozolin.

Captan is used to control leaf spot, blights and fruit rots on fruit crops and vegetables. It is also used as seed protectant.

Iprodione (Rovral) is a broad spectrum foliage contact fungicide.It inhibits spore germination and mycelial growth and shows mostly preventative and early curative activity.

Vinclozolin (Ornalin or Vorlan) is a contact and protective fungicide.and used as spray on strawberries, lettuce and ornamentals.

2. Systemic Fungicides of Organic Compounds:

Systemic fungicides are absorbed through the foliage or roots and are translocated within the plant through the xylem. These fungi generally move upward in the transpiration stream and may accumulate at the leaf margin. Almost all systemic fungicides are site specific inhibiting only one or perhaps a few specific steps in the metabolism of the fungi they control.

- Acylalanines: the most common is Metalaxyl which is effective against the oomycetes *Pythium, Phytopthora* and downy mildews. It is sold as Ridomil for use in soil in conjuction with a companion broad spectrum fungicide on leaves.
- Benzimidazoles: it includes important systemic fungicides like Benomyl, Carbendazim, Thiabendazole and Thiophanate.
- Benomyl sold as Benlate is a safe broad spectrum fungicide effective against large number of pathogens causing powdery mildew of all crops, scab of apples.
- Thiabendazole (Mertect) is a broad spectrum fungicide effective against many imperfecti fungi. It is commonly used as a post harvest treatment for the control of storage rots of citrus, apples and bananas.

- Thiophnate (trade name Topsin) is effective against several root and foliage fungi affecting turf grasses and vegetables.
- Oxathiins: it was the first systemic fungicide discovered. They are selectively concentrated in cells of these fungi and inhibit succinate dehydrogenase activity an enzyme important in mitochondrial respiration.
- Organophosphate Fungicides: they primarily include fosetyl-Al sold as Aliette. It is very effective against foliar, root and stem diseases. This compound has been reported to stimulate defense action and synthesis of phytoalexins against oomycetes.
- The other compounds like Kitazin, Edifenphos are found effective against rice blast.
- Pyrimidines:They include diamethirimol(Milcurb), ethrimol(Milstem) which are effective against powdery mildews of various crop plants
- Triazoles: It includes several excellent systemic fungicides like triadimefon(Bayleton), triadimenol(Baytan), Butrizol(Indar) and Difenoconazole(Score).They are applied as foliar sprays, seed and soil treatments and show long protective and curative activity against a broad spectrum of foliar, root and seedling diseases caused by many ascomycetes , imperfect fungi and basidiomycetes.

Some other excellent systemic fungicides of different chemical composition are Chloroneb used as seed treatment for beans, soybeans and cotton.

Ethazol (Truban) is a seed soil and turf fungicide effective against damping off and root and stem rots.

Imazalil (Fungaflor) is effective against many ascomycetes and imperfect fungi causing powdery mildews.

Some other chemically diverse compounds which are excellent protectant fungicides are Dodine (Cyprex) used against apple scab and gives long lasting protection and also a good eradicant.

Fentin hydroxide (Super Tin) is a broad spectrum fungicide with suppressant or antifeeding properties on many insects.

Zinc is sometimes used as Zinc naphthenate for disinfection and preservation of wood.

3. Antibiotics

These are the substances produced by one microorganism and toxic to another microorganism. Most of these antibiotics are of branching bacteria origin

(*Streptomyces*) or some fungi (*Penicillium*) which are mostly toxic to bacteria, mollicutes and certain fungi.

Generally only few antibiotics are available for plant disease control for example Streptomycin, Tetracyclines and cycloheximide.

Streptomycin obtained from actinomycetes *Streptomyces griseus.* It acts by binding to bacterial ribosomes and prevent protein synthesis. It is used against broad range bacterial plant pathogens and several oomycetous fungi.

Tetracyclines are active against many bacteria and mollicutes.

In Japan blasticidin is used against rice blast fungus *Magnaporthe grisea.*

- Plant oils and Petroleum Oils: The oils obtained from seeds of several plants such as sunflower, olive, corn and soybean gave excellent control on powdery mildew of apple.
- **Nematicides:** They are broad spectrum volatile soil fumigants that are active against not only nematodes but also insect, fungi, weed seeds. The four main groups are halogenated hydrocarbons, organophosphates, isothiocyanates and carbamates.

Halogenated Hydrocarbons: Methyl Bromide is better useful against nematodes and weed seeds and also for above ground control of dry wood termites. It affects organism because it is soluble in the lipid and disrupts the function of membranes and nervous systems.

Organophosphates: It includes the insecticides phorate (Thimet) and Disulfoton (Disyston), Fenamiphos (Nemacur). They are available as water soluble liquid and granules.They are effective against only the nematodes and not on soil fungi. They inhibit the nerve-transmitter enzyme cholinesterase and result in paralysis and ultimate death of affected nematodes.

Isothiocyeanates: It include metam sodium (Vapam), Vorlex and Dazomt (Mylone). These are applied as injection, incorporation and irrigation in soil at least 2 weeks prior to planting. All of them are effective against nematodes, soil insects, weeds and soil fungi.

Carbamates: It includes Aldicarb (Temik), Carbofuran (Furadan) and Carbosulfan(Advantage). They are active against nematodes, soil insects and some foliage insects. They inhibit the enzyme cholinesterase and causes paralysis and death of affected nematodes and insects.

Mechanism of Action of chemicals : The complete mechanism by which the chemicals control the plant diseases is yet unknown but such chemicals act by inhibiting the ability of the pathogen to synthesize certain cellwall substances,

by acting as solvents , by damaging the cell membrane of pathogen, by forming complexes and inactivating enzymes and causing general precipitation of proteins of the pathogens.

Resistance of Pathogens : The pathogens can become resistant to chemicals and antibiotics when continuously exposed and widespread use of these chemicals is done. Several plant pathogens have also developed strains that are resistant to certain fungicides. The protectant fungicides such as Thiram, Maneb, or Captan when used no resistant strain of pathogens were observed, presumably because these fungicides affect several vital process of the pathogen and too many gene changes. Resistance to same fungicides, all of which contained a benzene ring began to appear in the 1960 when *Penicillium* strains resistant to diphenyl, *Tilletia* strains resistant to hexachlorobenzene and *Rhizoctonia* strains resistant to PCNB were found to occur naturally.

In some cases, strains resistant to the fungicides appeared and become widespread after only two years of use of chemicals. Today several important fungal pathogens, for example *Alternaria, Botrytis, Colletotrichum, Fusarisum, Verticillium, Aspergillus, Penicillium, Phytophthora, Phythium and Ustilago,* are known to have produced strains resistant to one or more of the systemic fungicides. This is because systemic fungicides are specific in their action and affect only one or perhaps two steps in a genetically controlled event in the fungus, as a result resistant population can arise quickly either by a single mutation or by selection of resistant individual in a population.

The most common mechanisms by which pathogens develop resistance to various fungicides and bactericides is by (1) decreased permeability of the pathogen cell membranes to the chemical, (2) detoxification of the chemical through modification of its structure or through binding it to cell constituents (3) decreased conversion to the real toxic compound, (4) decreased affinity at the reactive site in the cell (5) bypassing a blocked reaction through a shift in metabolism and (6) compensation for the effect of inhibition by producing more of the inhibited product(e.g an enzyme).

The good systemic or nonsystemic fungicides that become ineffective because of the appearance of new resistant strains can continuues to be used, and the resistant strains can still be controlled to a practical level through changes in the methods of deployment of the fungicide. This can be achieved by using mixture of specific systemic fungicides, by alternating sprays with systemic and protectant fungicides, or by spraying during half the season with systemic and the other half with protectant fungicides. In each these schedules, the systemic

or specific action chemical carries most of the weight in controlling the disease, while the protectant or nonspecific chemical reduces the possibility or survival of any strains of the pathogen that may develop resistance to the systemic or specific action chemical.

Although most chemical used to control plant diseases are much less toxic than most insecticides, especially the nematicides are extremely toxic. Also, some have adverse genetic effects, causing morphologic and physiological abnormalities in test animals. For this reason, a number of restrictions are imposed in the licensing, registration, and use of each chemical.

In the united states, both the food and drug administration (FDA)and the Environmental Protection Agency (EPA) keep a close watch on the registration production and use of pesticides. It is estimated that only 1 out of 10,000 new compounds synthesized by the pesticide industry turns out to be a successful pesticide.

Once a chemical is approved for a certain crop, two important restrictions on the use of the chemical must then be observed (1) the number of days that must elapse before harvest of a crop after use of a particular chemical on the crop and (2) the amount of the chemical that can be used per application must not exceed a certain amount. If either of these restrictions in not observed it is likely that, at harvesting of crop especially vegetables and fruits, carries on it a greater amount than is allowed for the particular chemical, and the crop then must be destroyed.

As a result of all mandated and voluntary precautions regarding the use of pesticides, the food stuffs reaching the U.S. markets is either free of all pesticides or carry minimal residues within the legal limits less than 1 percent of samples tested has residues over the allowed tolerances.

Disinfestation of warehouse : Stored products can be protected from becoming infected by pathogen left over in the warehouse from previous years by first cleaning thoroughly the storage rooms and by removing and burning the debris. The wall and floor are washed with bleach, a copper sulfate solution (1 pound in 5 gallon water) or some other sanitizing agent. Warehouses that can be closed airtight and in which the relative humidity can be kept at nearly 100 percent while the temperature is betwwen 25^0 and 30^0c can be effectively fumigated with chloropricin (tear gas) used at 1 pound per each 1000 cubic feet. In all case the fumigants should be allowed to act for at least 24 hours before the warehouse doors are opened for aeration.

6.2.3 Biological Methods

Biological control of pathogens is "the total or partial destruction of pathogen population by other organisms", occurs routinely in nature.According to Garrett (1965) "any condition under which, or practice where by, survival or activity of a pathogen is reduced through the agency of any other living organism except man itself, with the result that there is reduction in incidence of disease caused by the pathogen". For example, there are several disease in which the pathogen cannot develop in certain areas either because the soil (called suppressive soil), contains microorganism antagonistic to the pathogen or because the plant that is attacked by a pathogen has also been naturally inoculated with antagonistic microorganisms before or after the pathogen attack. Sometimes the antagonistic microorganisms may consist of avirulent strains of the same pathogen that destroy or inhibit the development of the pathogen, as happens in hypovirulence and cross protection. In some cases, even higher plants reduce the amount of inoculum either by trapping available pathogens (trap plants) or by releasing into the soil substances toxic to the pathogen. Agriculturalists have increased their efforts to take advantage of such natural biological antagonisms and to develop strategies by which biological control can be used effectively against several plant diseases. Biological antagonisms are expected to become an important part of the control measures used against many more diseases.

1. Suppressive Soils

Several soilborne pathogens, such as *Fusarium oxysporum* (the cause of vascular wilts), *Gaeumannomyces graminis* (the cause of take-all of wheat), *Phytophthora cinnamomi* (the cause of root rots of many fruit and forest trees), *Pythium* spp. (a cause of damping-off), and *Heterodera avenae* (the oat cyst nematode), develop well and cause severe diseases in some soils, known as conducive soils, whereas they develop and cause much milder diseases in other soils, known as suppressive soils. The mechanisms by which soils are suppressive to different pathogens may involve biotic and/or abiotic factors and may vary with the pathogen. They operate primarily by the presence of one or several microorganisms antagonistic to the pathogen in such soils. Such antagonists occur through competition for food, through direct parasitizing of the pathogen, production of antibiotics, production of lytic enzymes that do not allow the pathogen to reach high enough populations to cause severe disease.

A number of antagonistic microorganisms have been found to increase in suppressive soils; most common pathogen and disease suppression has been shown to be caused by fungi for example *Trichoderma, Penicillium* and

Sporidesmium, or by bacteria of the genera *Pseudomonas, Bacillus* and *Streptomyces*. The suppressive soil added to conducive soil can reduce the amount of disease by introducing microorganism antagonistic to the pathogen. For example, soil amended with soil containing a strain of a *Streptomyces scabies* which causes potato scab, results in potato tubers significantly free from pototo scab.

In several diseases continuous cultivation (monoculture) of the same crop in a conducive soil, after some years of severe disease, eventually leads to reduction in disease through increased population of microorganisms antagonistic to the pathogen. For example, continuous cultivation of wheat or cucumber leads to reduction of take all of wheat and *Rhizoctonia* damping-off of cucumber.Similarly continuous cropping of watermelon variety crimson sweet allows the buildup of antagonistic species of *Fusarium* related to that causing Fusarium wilt of watermelon with the result that Fusarium wilt is reduced rather than increased. The suppressiveness is due to antagonistic microflora can be shown by pasteurization of the soil at 60^0C for 30 minutes which completely eliminates the suppressiveness.

2. Antagonistic Microorganisms

- **Soilborne Pathogens :** The mycelium and resting spores or sclerotia of several phytopathogenic soil fungi such as *Pythium, Phytophthora, Rhizoctonia and Sclerotinia* are invaded and parasitized (mycoparasitism) or lysed (mycolysis) by several fungi which as a rule are not pathogenic to plants. Several non plant pathogenic fungi, including some oomycetes, chytridiomycetes and hyphomycetes and some pseudomonad and actinomycetous bacteria infect the resting spores of several plant pathogenic fungi. Among the most common mycoparasitic fungi are *Trichoderma harzianum* which has been shown to parasitize mycelia of *Rhizoctonia* and *Sclerotium,* to inhibit the growth, of many other fungi, for example, *Pythium, Phytophthora* and *Fusarium* and to reduce the diseases caused by most of these pathogens.

Other common mycoparasitic fungi are *Laetisaria arvalis* a mycoparasite and antagonist of *Rhizoctonia* and *Pythium. Talaromyces flavus* which parasitizes *Verticillium* and control Vetricilllium wilt of eggplant. Some *Pythium* species parasitize species of *Phytopthora*.

Along with fungi, in addition some bacteria of the genus *Bacillus, Enterobacter* and *Pseudomonas* have been shown to parasitize /inhibit the pathogenic fungi *Sclerotium ceptivorum*, *Phytophthora sp* and *Pythium* sp.

The mycophagous nematode *Aphelenchus avenae* parasitizes *Rhizoctonia* and *Fusarium.*

The dagger nematode *Xiphenema* and the cyst nematode *Heterodera* are parasitized by the nematophagous fungi *Catenaria auxiliaries.*

Pasteuria penetrans, a bacterium parasitize the nematode *Meloidogyne javanica.*

- **Aerial Pathogens:** Many other fungi have been shown to antagonize and inhibit numerous fungal pathogens of aerial plant parts. For example, *Chaetomium* sp. and *Athelia bombacina* suppress *Venturia inaequalis* ascospore and conidia production in fallen and growing leaves respectively. *Tuberculina maxima* parasitizes the white pine blister rust fungus *Cronartium ribicola* and *Verticillium lecanii* parasitize serevel rusts.*Nectria inventa* and *Gonatobotrys simplex* parasitizes two pathogenic species of *Alternaria*.

Mechanisms of action : The mechanism by which antagonistic microorganisms affect pathogen populations are not always clear but they are generally attributed to one of four reasons (1)direct parasitism or lysis and death of the pathogen(2) competition with the pathogen for food(3) direct toxic effects on the pathogen by antibiotic substance released by the antagonist(4)indirect toxic effect on the pathogen by volatile substances(ethylene).

Many of the antagonistic microorganisms mentioned above are naturally present in crop soils and exert a certain degree of biological control over one or many plant pathogens regardless of human activities. Human however have been attempting to increase the effectiveness of antagonists either by introducing new and larger population of antagonists in field where they are lacking and/or by adding soil amendments that serves as nutrients or otherwise stimulate growth of the antagonistic microorganisms and increase their inhibitory activity against the pathogen. Unfortunately, both approaches are effective in the laboratory and in their greenhouse neither has been particularly successful in the field. New microorganisms when added to the soil of a field cannot compete with the existing microflora and cannot maintain themselves for very long also. Thus their potential for eventual disease control is quiet limited.

3. Control through Trap Plants

If a few rows of rye, corn, or other tall plants are planted around a field of beans, peppers, or squash many of the incoming aphids carrying viruses who preferably attack the beans, peppers, and squash will first stop and feed on the peripheral taller rows of rye or corn. Because most of the aphid borne viruses are non persistent in the aphid many of the aphids lose the bean, pepper or squash infecting viruses by the time they move onto these crops. In this way trap crops reduce the amount of inoculum that reaches a target crop.

Trap plant are also used against nematodes in a different way .Some plants that are not actually susceptible to certain sedentary plant-parasitic nematodes produce exudates that stimulate eggs of these nematodes to hatch. The juveniles enter this plant but are unable to develop into adults and eventually they die. Such plants are also called **trap crops**. By using trap crop in a crop rotation program, grower can reduce the nematode population in the soil for example *Crotalaria* plant trap the juveniles of the root-knot nematodes two or with water or through soil (the other).

4.Control of insect vector : When the pathogen is introduced or disseminated by an insect vector control of insect vector is as important as and sometime easier than the control of the pathogen itself. Application of insecticides for the control of insect carrier of fungus spore and bacteria has been fairly successful and is a recommended procedure in the control of several such insect-carried pathogens.

In the case of viruses, mollicutes, and fastidious bacteria, however, of which insects are the most important disseminating agents, insect control has been helpful in controlling the spread of their diseases only when it has been carried out in the area and on the plants on which the insects overwinter or feed before they enter the crop. Controlling such diseases by killing the insect vectors with insecticides after they have arrived at the crop has seldom proved adequate, even with good insect control; enough insects survive for sufficiently long period to spread the pathogen. Nevertheless, appreciable reduction in losses from certain such disease has been obtained by controlling their insect vectors.

Success in reducing virus transmission by insects has been achieved by interfering with the ability of the aphid vector to acquire and to transmit the virus rather than by killing the insects. The interference is provided by spraying the plants several times each season with a fine-grade mineral oil. Such oil seems to have little effect on the probing and feeding behavior of the aphids and is not particularly toxic to the aphids, but it interferes with the transmission

of nonpersistent, semipersistent, and even some persistent aphid-borne viruses. Control of aphid-borne viruses by oil sprays has been successful with some viruses (eg; cucumber mosaic virus on cucumber and pepper, and potato virus Y on Pepper)

6.2.4 Cultural Practices

Several methods have been developed which bring about artificial manipulation of rhizosphere and thereby reducing the incidence of disease. Some of them are as follows:

- **Raising of Beds :** The foot rot of ginger caused by *Pythium myriotylum* occurs in the low bed areas which are subjected to flooding and are ill-drained and which favour the aquatic? This disease can be controlled by an improved cultural practice, by planting the crop on raised beds. The raised bed system has removed those conditions which favoured the pathogen.
- **Change in Planting Season:** The leaf of sugarcane (*Puccinia sacchari*) and blast of ragi have been controlled effectively by this method. It is a case of disease escape. The crops are grown during September-October instead of in june-july and thus escape the pathogens. Heavy rust infection may be early sowing of wheat in the plains of north India.
- **Obtaining seed From Disease Free Localities: T**he foot rot of ginger caused by *Pythium myriotylum* is prevalent in South India, which can be controlled by importing seed-rhizomes from disease free arid zones of Northen India, where the disease is practically absent because of the dry climate soils and moderate rainfall.
- **Proper Manuring:** Application of a balanced dose of nitrogen helps in the reduction of blast (*Pyricularia oryzae*) and leaf spot of rice. Nitrogenous fertilizers increase the rust incidence of wheat while potassic and phosphatic manure, reduce the intensity of rust.
- **Mixed Cropping:** The practice of mixed cropping has helped effectively the spread of infectious diseases. The root rot of cotton caused by *Rhizoctonia bataticola* and the wilt of pigon pea caused by *Fusarium udum* in Uttar Pradesh have been successfully overcome by such practice.
- **Soil hygiene:** Field sanitation is an important factor in the healthy and vigorous growth of plants. Uncleaned field constitute a danger to the well being of plant as remnants of previous crops in the field harbor disease organisms. Crops grown under such conditions become exposed to attack of

disease and get seriously damaged. Rotten crops of cotton and sugarcane generally get easily infected by soil borne diseases. To get rid of diseases that perennate in the crop residue in the soil, the surest method is to collect all parts of diseased plants after harvest and burn them.

- The incidence of a parasitic organism in the soil can be reduced by keeping the land fallow and planting non susceptible crops.
- **Amendment of soil Conditions:** When the crop plants are not grown under ideal conditions they are more susceptible to infection and amendment of such factors would control the disease. Soil improvement with a view to controlling disease includes measure devesed to increase host residence and adjustment of soil temperature, soil moisture, soil texture and soil reaction.
- The activity of the organisms which are sensitive to soil pH can be controlled by the adjustment of the reaction of the soil.
- **Soil Sterilization and Partial Sterilization:** The soil may be sterilized completely or only partially whereby not all organisms are destroyed, but only certain group. To sterilize a soil, it is placed in glass or clay containers and heated under pressure, 15-20 pounds, for 2-3 hours, by using flowing steam for 1-2 hours, on six consecutive days, complete sterilization can also be obtained.

The partial sterilization of soil is to destroy certain injurious insects on pathogenic fungi but not kills the whole soil population.

Hot weather cultivation and burning trash seems to be a beneficial agricultural practice. Soil sterilization with chemical, cannot be recommended for large scale application in the fields.

6.3 Summary

Seven groups of pathogenic agents are recognized on economically useful plants. They comprise of bacteria, fungi, viruses, nematodes, insects (excluding vectors), parasitic flowering plants and a heterogeneous group which includes mineral deficiencies and excesses and unfavourable environmental conditions.

Different control methods, *Physical, Chemical Biological and Cultural* methods or combinations of such methods (integrated control) are available to control the plant disease. The efficiency of the different control methods depends on the pest density at the time of application. Biological agents for example will have maximum effect when the pest is plentiful but insecticides and environmental measures are not influenced by density. The

impact of any one control measure is governed by the net increase in production of the crops. Any planning strategy must take all the characteristics *viz.* biological and abiotic parameters into account and well-organized and properly-timed attack on the total pathogenic population.

6.4 Glossary

- **Abiotic:** Absence of life, a disease not caused by living organisms
- **Allelopathy:** Ability of one species to inhibit or prevent the growth of another species through the production of toxic substance(s)
- **Alternate host:** One of two different plants that a parasitic fungus (e.g. rust) must infect to complete its life cycle
- **Antibiotic:** A chemical compound produced by one microorganism that inhibits or kills other living organisms
- **Biocide:** A compound toxic to all forms of life
- **Biocontrol:** use by humans of one species of organism to eliminate or control another species of organism through the use of competition, parasitism or antagonism between the organisms
- **Crop rotation:** The successive planting of different crop species in an area; used to improve soil fertility and reduce disease and pest problems.
- **Cultural practices:** How plants are grown, including application of nutrients, irrigation practices, types of cultivation, etc.; also a method used for disease management
- **Exclusion:** Control of disease by excluding the pathogen or infected plant material from crop production areas (e.g. by quarantines)
- **Fungicide:** Chemical used to control fungal diseases. Despite the name, most fungicides only slow down or prevent the spread of disease ; only a few actually kill the fungus.
- **Soil Drench:** Application of a solution or suspension of a chemical to the soil, especially pesticides to control soilborne pathogens.
- **Yield:** The desired product resulting from growth or cultivation of a plant.

6.5 Self -Learning Exercise

Section A : (Very Short Answer Type Questions)

1. Antibiotic used in disease control are

   (a) Streptomycin (b) Penicillin (c) Griesofulvin (d) All

2. Dithane and Zineb are

   (a) Pesticides (b) Minerals (c) Fungicides (d) Vitamins

3. Some chemicals do not kill the fungicide but inhibit the growth of mycelium are

   (a) Fungitoxic (b) Fungistatic (c) Fungicidal (d) Fungistatis

4. Bordeaux mixture is

   (a) Mixture of Calcium sulphate and slaked lime (b)Mixture of Calcium carbonate and nitric acid (d) Mixture of copper and iron (d) All of the above

5. Mixed cropping helps

   (a) In effectively checking the spread of infectious diseases (b) To increase the nitrogen content in the soil (c) To decrease the nitrogen content in the soil (d) None of the above

6. The pathogen can be eradicated by

   (a) Crop rotation (b) Eradication of alternate and collateral host (c) Biological control (d)All of the above

7. Dazomet is

   (a) An oil (b) Nematicide (c) A wettable powder (c) All of the above

8. The advantage of oil in disease control is

   (a) Superior disease control (b) Effectiveness at very low dosage (c) Excellent spreading and sticking properties on leaf surfaces (d)All of the above

Section B : (Short Answer Type Questions)

1. Name the type of prophylactic measures used for the control of plant disease.
2. Define quarantine.
3. What do you mean by crop rotation?
4. What is Rogueing?
5. Define the term soil hygiene.
6. What are antagonistic plants?
7. Define trap crops.

Section C : (Long Answer Type Questions)

1. Discuss in detail about the preventive measures used to control plant diseases.
2. Explain in detail about the physical methods of pest control in plants.
3. Write a detail note on the chemical control measures used to control plant pathogens.
4. Discuss in detail about the biological method of control in plants.
5. Discuss cultural control measures used in plant protection.

6.6 References

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Unit -7

Plant Disease Management-II: Biotechnology and Breeding

Structure of the Unit:

7.0 Objectives

After going through this unit you will be able to understand:

- the role of biotechnology in Plant Disease management
- insect Resistance and its induction through biotechnology
- virus resistance can be induced in plants.
- use of conventional plant breeding methods can be used for Plant Disease Management

7.1 Introdution

Transfer of genes between plant species has played an important role in crop improvement for many decades. Plant improvement whether as a result of natural selection or the efforts of plant breeder has always relied upon evolving, evaluating and selecting the right combination of alleles. Useful traits such as resistance to diseases, insects and pests have been transferred to crop varieties from noncultivated plants. Since 1970, rapid progress is being made in developing tools for the manipulation of genetic information in plants by recombinant DNA methods. The overall process of genetic transformation involves introduction, integration and expression of foreign gene(s) in the

recipient host plant. Plants that carry additional, stably integrated and expressed foreign genes transferred (transgenes) from other genetic sources are referred to as transgenic plants. The development of transgenic plants is the result of integrated application of DNA technology, gene transfer methods and tissue culture techniques

The capacity to introduce and express diverse foreign genes in plants was first described in tobacco by *Agrobacterium* mediated and vectorless approach. The list of plant species that can be transformed by vector-mediated *(Agrobacterium)* and vectorless methods has been growing continuously and at present transformation capability has been extended to more than 120 species in at least 35 families.

7.2 Biotechnology in Pest Management

The application of genetic engineering to crop agriculture has been targeted towards the generation of transgenic plants having resistance to viruses, insects, herbicides or post harvest deterioration.

Resistance to Biotic Stresses

Genetic transformation has led to the possibility of transforming crops for enhanced resistance to insects and pathogens and it is rapidly moving towards commercialization. These advances form the basis of a chemical-free and economically viable approach for pest and disease control.

Resistance to biotic stresses has been discussed under the following headings.

1. Insect resistance
2. Virus resistance
3. Disease resistance

7.2.1 Insect Resistance

Progress in engineering insect resistance in transgenic plants has been achieved through the use of insect control protein genes of *Bacillus thuringiensis.* Insect resistance was first reported in tobacco (Vaeck *et al,* 1987) and tomato (Fischhoff*, et al.,* 1987). Today insect-resistant transgenes, whether of plant, bacterial or other origin can be introduced in plants to increase the level of insect resistance. Approximately 40 different genes conferring insect resistance have been incorporated into crops. Genes conferring insect resistance to plants have been obtained from microorganisms like *Bt* gene from *Bacillus thuringiensis; ipt* (isopentyl transferase) gene from *Agrobacterium tumefaciens,* cholesterol oxidase gene from a *Streptomyces* and *Pht* gene from *Photorhabdus luminescens.* Resistance genes from higher plants can be

grouped into two categories: (i) proteinase and amylase inhibitors and (ii) lectins: snowdrop lectin (GNA, pea lectin, jacalin, rice lectin, etc.). Resistance genes of animal origin are serine proteinase inhibitors from mammals and tobacco hornworm *(Manduca sexta).*

Bacillus thuringiensis (Bt) is an entomocidal bacterium that produces an insect control protein. Bt genes code for the Bt toxin,differing in their spectrum of insecticidal activity.Most of this toxin is active against Lepidopteran larvae but some are specific for dipteran and coleopteran insects.The toxicity of this toxin lies in a large protein which accumulates as crystal protein (delta-endotoxin) inside the bacteria during sporulation and convert into active form upon infection by susceptible insect by disrupting the ion transport across the brush borders/ membranes of susceptible insect.

Bt toxin is not harmful to beneficial insects, mammals and humans. This gene segregates as a single dominant gene.

Bt strains contain a great diversity of delta endotoxin encoding genes. The cloning and sequencing of first insecticidal protein encoding genes was published in 1981.Today more than 100 crystal protein gene sequences have been published, each with a specific activity spectrum.

Both the full length and truncated forms of *Bt* delta - endotoxins have been introduced in plants, conferring demonstrable resistance to tobacco pests *(M. sexta),* tomato pests *(Heliothis virescens* and *Helicoverpa zea)* and potato pests *(Phthorimeae operculella).* The first plants produced were capable of synthesizing the entire protoxin, but expression of the gene was weak and the resulting small quantity of delta - endotoxin gave little or no insect resistance. Further development ultimately led to optimization of the *cry* gene expression in plants. The first generation insecticidal plants have been introduced to the market for commercial purposes.

Other microorganism-derive resistance genes: Cholesterol oxidase (CO) protein present in the *Streptomyces* culture filtrate showed acute toxicity to boll weevil larvae. This gene has been engineered into tobacco. Isopentenyl transferase *(ipt)* gene from *Agrobacterium tumefaciens* codes for a key enzyme in the cytokinin biosynthetic pathway. Expression of *ipt* in tobacco and tomato by a wound inducible promoter has resulted in a decrease in leaf consumption by the tobacco hornworm *(M. sexta)* and reduced survival of the peach potato aphid *(Myzus persicae).*

A. **Resistance Genes from higher Plants**: A number of efforts are directed towards discovery of non-Bt toxin genes having insecticidal activity. A

number of non-Bt insecticidal proteins interfere with the nutritional needs of the insect. Currently there are two major groups of plant-derived genes that are used to confer insect resistance on crop plants by retarding insect growth and development.

Proteinase inhibitors: Since 1938 it was known that plants contain peptides acting as protease inactivating proteins (PIPs). The different proteinases are serine, cysteine, aspartic and metallo proteinases. They catalyze the release of amino acids from dietary protein, thereby providing the nutrients crucial for normal growth and development of insects. The proteinase inhibitors deprive the insect of nutrients by interfering with digestive enzymes of the insect. Two such proteinase inhibitor genes are as follows:

(i) **Cowpea trypsin inhibitor gene (CpTI)**: *CpT I,* found in cowpea *(Vigna unguiculata),* is the most active inhibitor identified to date. This inhibitor gene produces antimetabolite substances that provide protection against the major storage pest Bruchid beetel *(Callosobruchus maculatus).* This gene is also harmful to various Lepidopteran insects *(Heliothis virescens),* Spodopteran insects *(Manduca sexta)* Coleopteran insects *(Callosobruchus, Anthonomus grandis),* Orthopteran insects *(Locusta migratoria)* but not harmful to mammals.

(ii) **α -Amylase inhibitor**: Genes for three a-amylase inhibitors have been expressed in tobacco but the main emphasis has been on transferring the gene of α -amylase inhibitor (aAl-Pv) isolated from adzuki bean *(Phaseolus vulgaris).* It works against *Zabrotes subfasciatus* and *Callosobruchus chinensis.* This α -amylase inhibitor protein blocks the larval feeding in the midgut. The larvae secrete a gut enzyme called α -amylase that digests the starch. By adding a protein that inhibits insect gut α -amylase, the weevil can be starved and it dies.

Lectins constitute another large family of proteins that can be used as insect toxins for genetic engineering of insect resistance. These are plant glycoproteins. A latest lectin identified is from snowdrop *(Galanthus nivalis),* known as GNA shows activity against aphids. The gene for this protein has been successfully used in genetic engineering studies and expressed in different species including potato, oilseed rape and tomato. The laboratory tests with modified

potato showed that GNA did not increase the mortality but considerably reduced fecundity. An important feature of this protein is that it also acts against piercing and sucking insects. One disadvantage is that the protein works well only when it is ingested in large quantities, i.e. when insects are exposed to microgram levels in diet incorporation bioassays. A number of lectin encoding genes (wheat- germ agglutinin, jacalin and rice lectin) have been expressed in transgenic plant.

B. **Resistance genes from animals:** Resistance genes involved are primarily serine proteinase inhibitors from mammals and the tobacco homworm *(Manduca sexta)*. Based on *in vitro* screening of inhibition of proteolysis midgut extracts of a range of lepidopteran larvae, bovine pancreatic trypsin inhibitor (BPTI), α-antitrypsin (α AT) and spleen inhibitor (SI) have been identified as promising insect resistance proteins and have been transferred to a range of plants.

7.2.2 Virus Resistance

The development of molecular strategies for the control of virus diseases has been especially successful because of the relatively small genomic size of plant viruses. There are a number of different strategies for using molecular technology to integrate or create new resistance factors in plant virus systems. The approach is to identify those viral genes or gene products, which when present at an improper time or in the wrong amount, will interfere with the normal functions of the infection process and prevent disease development.

Coat-protein-mediated cross protection: The concept of cross protection is the ability of one virus to prevent or inhibit the effect of a second challenge virus. If susceptible strain of a crop is inoculated with a mild strain of a virus, then the susceptible strain develops resistance against more virulent strain. Powell-Abel *et al.* (1986) first demonstrated that transgenic tobacco expressing tobacco mosaic virus (TMV) coat protein showed resistance similar to that occurring in viral-mediated cross protection. A number of coat protein genes from different virus groups have been found to provide resistance when expressed in transgenic plants. Coat protein mediated resistance in many systems is correlated with the inhibition of virus replication at the initial point of infection. This resistance takes the form of reduced numbers of infection sites on inoculated leaves, showing that an initial step in the virus life cycle has been disrupted. TMV cross protection may result from the coat protein of the protecting virus preventing uncoating of the challenge virus RNA. Most of the

system in which coat-protein-mediated resistance has been used in several crops such as tobacco, tomato, potato, alfalfa, melons, sugarbeet, squash, rice, maize etc.

An example of important negative strain virus is tomato spotted Wilt virus (TSWV). Here the genomic RNA is tightly associated with nucleocapsid (N) protein. This protein helps in wrapping of viral RNA and also in regulation of transcription to replication switch during the infection cycle.

Nonstructural protein-mediated resistance*:* A number of *v*iruses encode nonstructural proteins that are necessary for replication. Several of these nonstructural "replicase" proteins have been found to provide a high degree of resistance to virus infection when expressed in transgenic plants. Golemboski *et al*. (1990) first demonstrated this phenomenon by expressing the 54 kDa open reading frame (ORF) of TMV in transgenic tobacco. Transgenic tobacco has been developed against pea early browning virus (PEBV) and potato virus X (PVX). Pinto *et al.* (1999) developed rice yellow mosaic virus resistant transgenic plants of rice by expressing the replicase gene. Blocking of viral movement through the expression of defective viral movement protein to impede viral infection has been tested in transgenic tobacco and other crops.

Antisense- and sense-mediated resistance: Another pathogen derived strategy that has been investigated for the control of plant viruses is the transgene expression of antisense and recently sense segments of viral RNAs. The principle of this strategy is to bind up viral RNA with complementary RNA sequences expressed by the plant. Inappropriate RNA-RNA base pairing would potentially prevent accessibility of the viral RNA for replication or gene expression. This antisense and sense constructs could be used to block initial steps important in the establishment of viral infection. Antisense protection has been demonstrated in tobacco expressing complementary RNA to the coat protein.

Satellite RNA protection: Satellite RNAs are class of small (approximately 300 nucleotides), single stranded RNA molecules that are dependent upon a helper virus for replication and virion packaging to cause infection at some other host. Satellite RNA depends on virus for its replication and transmission, even though it is unrelated to viral genome. These satellite RNA species have been associated with several viruses. A number of satellite RNAs have been shown to modulate the replication and symptoms of their helper viruses. Changes in symptom development range from severe necrosis to almost complete symptom attenuation, depending on the associated satellite RNA.

Satellite RNAs that attenuate symptoms can be potentially used to reduce the disease severity of the helper virus. It is used in developing transgenic crops to confer resistance. The deliberate inoculation of a mild strain of CMV (cucumber mosaic virus) with a symptom attenuating satellite RNA successfully protected tobacco, pepper, tomato and cucumber plants from a virulent strain of CMV and reduced yield losses. Tien and Gusui (1991) reported that 121 transgenic tomato plants expressing an attenuating CMV satellite RNA gave 50% increase in yield over control plants when injected with a severe strain of CMV. This strategy is limited to those virus systems in which attenuating satellite RNAs are found.

Pathogen targeted resistance: Anti-viral proteins: A class of polypeptides variously called antiviral or ribosome-inactivating proteins (RIPs) has been identified in a number of plant species of which the best-known source is pokeweed *(Phytolacca americana).* Three distinct pokeweed antiviral proteins (PAPs) have been identified. The ribosome-inhibiting function is due to their ability to modify ribosomal RNA and thereby to interfere with polypeptide translation. Lodge *et al.* (1993) generated transgenic plants of tobacco and potato with PAP isolated from *Phytolacca* that were resistant to PVX, PVY, and CMV. Transgenic tobacco expressing pokeweed antiviral protein was found to be resistant to TMV.

Ribozyme-mediated resistance: Ribozymes are essentially RNA-based RNA restriction enzymes capable of catalytically cleaving RNA molecules at specific sites. The ability to direct ribozyme cleavage provides a potentially useful strategy to control plant virus diseases, especially since the majority of agriculturally important plant viruses have RNA genomes. Thus, transgene expression of ribozymes designed to cleave viral RNAs could be used to disrupt viral replication and disease development.

Several different types of ribozymes with different sequences and structures have been identified. Edington and Nelson (1992) have tested the ability of a ribozyme to confer resistance to virus infection *in vivo* in a protoplast system. Results demonstrated that a ribozyme directed against the replicase ORF of TMV was effective at reducing viral accumulation in protoplasts by as much as 90% in the first 24hours post infection.

7.2.3 Disease Resistance

A large number of plant defense response genes encoding anti-microbial proteins have now been cloned. Most of these are transcriptionally activated in response to infection or exposure to microbial elicitor macromolecules. The

products of defense response genes may include (i) hydrolytic enzymes, e.g. chitinase, 1-3 β-D glucanase and other pathogenesis related (PR) proteins, (ii) ribosome inactivating proteins (RIPs), (iii) antifungal proteins (AFPs), (iv) biosynthetic enzymes for the production of anti-microbial phytoalexins, (v) wall-bound phenolics, osmotins, thionins, lectins etc. and (vi) hydrogen peroxide.

Pathogenesis-Related protein: These are low molecular weight proteins, which accumulate to significant levels in infected plant tissues. The important types of PR proteins are tobacco PR-1, PR-2 (β 1-3 glucanase), PR-3 (chitinases), PR-4 (hevein like) PR-5 (thaumatin like and osmotin) etc. The ability of hydrolytic enzymes to break chitin and glucan in the cell walls of fungal pathogen has been exploited to develop crop resistance to pathogens. Various chitinase genes of plants have been isolated and characterized. The first report was in tobacco where a bacterial chitinase gene obtained from soil bacteria *(Serratia marcescens)* was stably integrated and expressed in tobacco leaves. A basic chitinase gene of bean under the control of strong constitutive promoter of CaMV 35S has been constitutively expressed to high levels in transgenic plants of tobacco and *Brassica napus.* This expression resulted in significant protection of the plants from post-emergent damping off caused by the pathogen *Rhizoctonia solani.* In the case of *Brassica napus* the protection was delay rather than complete inhibition.

Antimicrobial Protien: Plants and other organism may contain antimicrobial protein that may directly are not associated with induced defense response but their presence exhibit resistance towards pathogens. These are ribosome inactivating proteins (RIPs), cysteine rich proteins (lecithins, defencins, thionins, lysozyme etc)

Barley α- thionin gene when transferred to tobacco showed resistance against *Pseudomonas syringae* pv *tabaci* and P. *syringae* pv *syringae*. Antibacterial proteins of nonplant origin include lytic peptides, lysozymes, and iron-sequestering glycoproteins. Lytic peptides are small proteins with an amphipathicα-helical structure, whose effect is to form pores in bacterial membranes (e.g. cecropin, attacin). Cecropins have been expressed in transgenic potato and tobacco (Mourgues *et al.,* 1998) and attacins in apple plants. A cecropin gene cassette when transferred to tobacco and rice showed resistance against P *syringae* and bacterial pathogens.

BacteriophageT4 lysozyme gene transferred to potato showed resistance to *Erwinia carotovora* subsp. *Atroseptia.* The introduction of a human lysozyme gene is an effective approach to protect crops against both fungal and bacterial diseases.

Active oxygen species (AOS), including H_20_2, also has a defense mechanism. Transgenic potato expressing a H_20_2-generating fungal gene for glucose oxidase was found to have high levels of H_20_2 and enhanced levels of resistance both to fungal and bacterial pathogens.

A different approach is offered by the fungal endo α 1,4-D polygalacturonases, which were partly responsible for dissolution of the plant cell wall and are presumably essential for efficient colonization. All dicotyledons investigated so far possess special inhibitors of these endopolygalacturonases (polygalacturonases inhibiting proteins, PGIPs), which show no activity against the endogenous pectinases of the plant.

The molecular targets of several fungal or bacterial toxins from plant pathogens are now known. Toxin-inactivating enzymes have been successfully used to engineer resistance. The bacterial haloblight pathogen of bean, *Pseudomonas phaseolicola,* produces a tripeptide toxin, phaseolotoxin, which causes the chlorotic halos. Phaseolotoxin inhibits the enzyme ornithine transcarbamylase (OC). Bacteria have been selected which contain a phaseolotoxin-insensitive OC and the gene encoding this enzyme has been cloned and transferred to tobacco, where its expression has been shown to prevent the symptoms caused by application of the toxin. The systemic xylem-invading pathogen *Xanthomonas albilineans* produces a family of low-molecular-weight toxins (albicidins) that selectively block prokaryote DNA replication and causes the characteristic chlorotic symptoms by blocking chloroplast development.

Phytoalexins: These are low molecular weight antimicrobially active secondary metabolites synthesized by the plant in response to an infection, which contribute to the resistance of plants to diseases.During infection these stored phytoalexins (in special cell or organelles in inactive form) are mobilized, while genes for biosynthetic pathways are induced and synthesis of more phytoalexins begins. Resveratrol is one of the most commone phytoalexin synthesized in some species.

Various attempts to isolate disease resistance and avirulence genes have gained momentum in the past few years primarily because of the development of map based cloning and gene tagging strategies. HM1 gene from maize, which confers resistance to *Cochliobolus carbonum,* has been cloned by transposon

tagging. This gene encodes NADPH dependent HC-toxin reductase that inactivates the fungal HC toxin. Resistant genes like *Arabidopsis* Rps2 and *RPM1,*Pto, Cf9, Cf2, Cf4 from tomato; tobacco N gene have been cloned. A number of avirulence genes have also been cloned *like* A vr 9 and Avr4 of *Cladosposium fulvum,* NIP1 *of Rhynchosporium secalis* etc.

Introduction of resistance (R) gene from a plant variety resistant to a certain pathogen into susceptible varieties is one the strategies which is developed in tomato plants with *Pto* resistance gene that confer resistance against *Pseudomonas syringae* pv *tomato*. Tobacco plants transgenic for *Pto* were resistant to *Pseudomonas syringae* pv *tabaci* expressing avr Pto. Rice Xa21 gene confers resistance to over 30 distinct strains of the bacterium *Xanthomonas oryzae* pv *oryzae* that causes leaf blight i*n* rice.

7.3 Breeding for Disease Resistant Varieties

The value of resistance in controlling plant disease was recognized in the early 1900s. The advances in the science of genetics and the advantages of planting a resistant instead of a susceptible variety made the breeding of resistant varieties possible and desirable. Scientific breeding for disease resistance originated with Sir R Biffen, who identified a single recessive gene for resistance to wheat yellow rust. Nearly every crop was then bred to include disease resistance (R) genes, many by introgression from compatible wild relatives.

The breeding of resistant varieties as one part of broader plant breeding programmes is more popular and more intensive today. Its usefulness and importance are paramount in the production of food and fiber. Todays cultivated crop plants are the result of selection and breeding of plant lines that evolved naturally in one or many geographic areas over millions of years. The evolution of plants from their ancient ancestors to present-day crop plants has occurred slowly and has produced countless genetically diverse forms of these plants. Many such plants still exist as wild types at the point of origin or in the areas of natural spread of the plant.

Since beginning of agriculture, some of the wild plants in each locality have been selected and cultivated and thus produced numerous cultivated lines or varieties. The most productive of these varieties were perpetuated in each locality from year after year and those that survived the local climate and the pathogens continued to be cultivated. Nature and pathogens eliminated the weak and susceptible ones while the farmers selected the best yielders among the survivors. Surviving varieties had different sets of major and minor genes

for resistance. In this fashion, selection of crop plants continued wherever they were grown, with people in each locality independently selecting varieties adapted to the local environment and resistant to local pathogens. Thus, numerous varieties of each crop plant were cultivated throughout the world and by their own genetic diversity, contributed to make the crop locally adapted but in overall genetically nonuniform and thereby safe from any sudden outbreak of a single pathogen over a large area.

Each plant species is affected by approximately 100 different kinds of fungi, bacteria, mollicutes, viruses and nematodes. In general a single plant is attacked by hundreds thousands of a single kind or different kind of pathogen. Although such plants may suffer damage to a lesser or greater extent, many survive all these attacks and manage to grow well to produce appreciable yields. When new plant breeds or cultivars are bred, they must be maintained and propagated. Some plants are propagated by asexual means while others are propagated by seeds. Seed propagated cultivars require specific control over seed source and production procedures to maintain the integrity of the plant breeds results. Isolation is necessary to prevent cross contamination with related plants or the mixing of seeds after harvesting. Isolation is normally accomplished by planting distance but in certain crops, plants are enclosed in greenhouses or cages. Breeding for disease resistance began when plants were first domesticated. Breeding efforts is a continuous procedure because pathogen populations are always under pressure of selection for increased virulence, new pathogens appear, evolving cultivation practices and changing climate can reduce resistance and/or strengthen pathogens and plant breeding for other traits can disrupt prior resistance. A plant line with acceptable resistance against one pathogen may lack resistance against others.

Plant Breeding programme for inducing resistance in plants typically includes:

1. Identification of plants that may be less desirable in other ways, but which carry a useful disease resistance trait, including wild strains that often express enhanced resistance.
2. Crossing of a desirable but disease-susceptible variety to another variety that is a source of resistance.
3. Growth of breeding candidates in a disease-conducive setting, possibly including pathogen inoculation. Attention must be paid to the specific pathogen isolates, to address variability within a single pathogen species.
4. Selection of disease-resistant individuals that retain other desirable traits such as yield, quality and including other disease resistance traits.

Resistance is termed durable and long lasting if it continues to be effective over multiple years of widespread use as pathogen populations evolve. Crops such as potato, apple, banana and sugarcane are often propagated by vegetative reproduction to preserve highly desirable plant varieties, because for these species, outcrossing seriously disrupts the preferred traits. Vegetatively propagated crops may be among the best targets for resistance improvement by the biotechnology method of plant transformation to manage genes that affect disease resistance.

7.4 Summary

As agricultural production increases to meet the demands of a growing world population, so has the biotechnology research to control plant disease. Diseases can be caused by a variety of complex plant pathogens including fungi, bacteria, viruses and nematodes, and their management requires the use of techniques in transgenic technology, biochemistry and genetics. The development of rDNA technology, gene transfer and tissue culture techniques have enabled to provide new solutions to age old problems.

The first generation application of genetic engineering to crop agriculture has been targeted to generate transgenic plants expressing foreign genes that confer resistance to viruses, insects, and diseases etc.

Agrobacterium mediated approach for transfer of foreign gene has been growing continuously.

Bacillus thuringiensis is an entomocidal bacterium that produces an insect control protein, the *Bt* toxin which are active against lepidopteran larvae.

There are a number of different strategies for using molecular technology to integrate or create new resistance towards virus in various plants ex; coat protein mediated cross protection.

A number of plant denfense responsive genes encoding anti microbial proteins have been cloned whose products may include hydrolytic enzymes, ribosome inactivating proteins, antifungal proteins etc.

Scientific breeding for disease resistance originated with Sir Biffen, who identified a single recessive gene for resistance to wheat yellow rust. Nearly every crop was then bred to include disease resistance (R) genes, many by introgression from compatible wild relatives.

The breeding of resistant varieties as one part of broader plant breeding programs is more popular and more intensive today. Its usefulness and importance are paramount in the production of food and fiber. Today's

cultivated crop plants are the result of selection and breeding of plant lines that evolved naturally in one or many geographic areas over millions of years.

Breeding efforts is a continuous procedure because pathogen populations are always under pressure of selection for increased virulence, new pathogens appear, evolving cultivation practices and changing climate can reduce resistance and/or strengthen pathogens, and plant breeding for other traits can disrupt prior resistance.

7.5 Glossary

- **Clone**: A genetic replica of an organism created without sexual reproduction.
- ***Bt* crops**: Crops that are genetically engineered to carry a gene from the soil bacterium *Bacillus thuringiensis* (*Bt*). The bacterium produces proteins that are toxic to some pests but non-toxic to humans and other mammals
- **Genetic modification**: The production of heritable improvements in plants or animals for specific uses, via either genetic engineering or other more traditional methods.
- **Insect-resistant crops**: Plants with the ability to withstand deter or repel insects and thereby prevent them from feeding on the plant.
- **Recombinant DNA (rDNA)**: A molecule of DNA formed by joining different DNA segments using recombinant DNA technology.
- **Transgene:** A gene from one organism inserted into another organism by recombinant DNA techniques.
- **Transgenic organism:** An organism resulting from the insertion of genetic material from another organism using recombinant DNA techniques.

7.6 Self -Learning Exercise

Section A : (Very Short Answer Type Questions)

1. Generally which carrier is used for production of transgenic plants
   (a)Agrobacterium plasmid (b) Bacillus plasmid (c) Viral DNA (d) All
2. Which of the following have insect resistant gene/
   (a)Bacillus thuringiensis (b) *Bacillus subtilis*
   (c) *Bacillus anthracis* (d) *Pseudomonas sps*
3. In which plants, plant breeding is useful

(a) Genetically incompatible plants (b) sexually incompatible plants (c) Both of the above (d) In genetically related plants.

4. Which bacterium is called as natural genetic engineer?
   (a) *Pseudomonas aeuriginosa* (b) *Agrobacterium tumifaciens*
   (c) *Pseudomonas tumifaciens* (d) All
5. Cry I Protien is insecticidal to
   (a) Coleoptera (b) Lepidoptera (c) Orthoptera (d) Diptera

Section B : (Short Answer Type Questions)

1. Define the term Plant disease management.
2. What do you mean by genetic transformation?
3. What is *Bt* toxin?
4. Define lectins.
5. What is Phytoalexin?

Section C : (Long Answer Type Questions)

1. Discuss the role of biotechnology in introducing Insect resistance.
2. Discuss in detail about the factors responsible for introducing virus resistance in plants.
3. Explain the role of biotechnology in introducing disease resistance in plant disease management.
4. Write a detail note on role of plant breeding in inducing disease resistance in plants.
5. Discuss the merits and demerits of plant biotechnology in the production of plants against biotic stress.

7.7 References

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Unit - 8

Molecular Plant Pathology

Structure of the Unit:

8.0 Objectives

After going through this unit you will be able to understand:

- the role of genes in causing disease
- the mechanism of causing variability in both plants and pathogen
- the different stages of variation in pathogens
- the types of plant resistance to pathogens
- the genetics of virulence in pathogens and resistance in host plants

8.1 Introduction

Genetic information of all organism is encoded in DNA in a linear form, in the order of the four bases (A, adenine; C, cytosine; G, guanine; and T, thymine). Each triplet of adjacent bases, codes for a particular amino acid. A gene is a stretch of a DNA molecule, usually of about 100 to 500 or more adjacent triplets, that codes for one protein molecule or in a few cases one RNA molecule.

In eukaryotes, the coding region of a gene is often interrupted by noncoding stretches of DNA called introns. When a gene is active one of its DNA strands is used as a template and is transcribed into a RNA strand. Some genes code only for a RNA and that RNA is either a transfer RNA (tRNA) or a ribosomal RNA (rRNA). Most genes encode proteins, however, and the transcription product is a messenger RNA (mRNA). The mRNA then becomes attached to ribosomes who with the help of tRNAs, translate the base sequence of the mRNA strand into a specific sequence of amino acids that folds into a specific shape and forms a particular protein. Different genes code for different proteins. Some of the proteins are part of the structure of cell membranes, but most act as enzymes. It is the proteins that give cells and organisms their characteristic properties such as shape, size and color, determine what kinds of chemical substances are produced by the cell and regulate all activities of cells and organisms.

Not all genes in a cell are expressed at all times the additional stretches of DNA called promoters, enhancers, silencers or terminators regulates the functioning of on, off of a particular gene and act as signals for genes to be expressed or stop being expressed. They also act as signals for production of RNAs and proteins that themselves act as promoters, enhancers of gene expression or as repressors, terminators of gene expression.

The host pathogen interaction genes in the one organism are triggered to be expressed by a substance produced by the other organism. For example, genes for cellwall-degrading enzymes in the pathogen are apparently induced by the presence of monomers or oligomers of host cell wall macromolecules that are substrates for these enzymes. Similarly the genes for defense reactions in the host, like production of phytoalexins, is triggered to produce by expression of certain signal compounds activated by inducer molecules (elicitors) produced by the pathogen.

8.2 Genes and Disease

A number of plants, such as tomato, apple or wheat, become diseased as a result of infection by a pathogen. The pathogen is often specific for that particular host plant. For example the fungus *Fusarium oxysporum* sp. *lycopersici* causing tomato wilt attacks only tomato and has absolutely no effect on apple.

What makes possible the development of disease in a host is the presence in the pathogen of one or more genes for pathogenicity, specificity and for virulence against the particular host.

The gene or genes for virulence in a pathogen are usually specific for one or a few related kinds of plants that are hosts to the pathogen. Also, the genes and gene combination who makes a plant susceptible (host)to a particular pathogen are present only in that one kind of plant and possibly a few related kinds of plants. All plants also have preformed and induced defenses that provide resistance against most pathogens. A pathogen that is virulent on one kind of plant and is not able to attack other kind of plants is known as non host resistance.

Few pathogens are able to attack many kinds(sometimes hundreds) of host plants,such pathogens tend to be necrotrophs and can attack so many hosts apparently because they either have many diverse genes for virulence, or because their genes of virulence somehow have much less plant specificity than those of the commonly more specialized pathogens. Each species of plant, however, seems to be susceptible to a fairly small number of different pathogens, usually less than a hundred for most plants.

Why all the plants are not attacked by their pathogens and why are those that are attacked not usually killed by the pathogens? The answer is complex, but basically it happens because plants, through evolution or through systematic breeding, have acquired, in addition to the genes that make them susceptible to a pathogen, one or usually numerous genes for resistance that protect the plants from infection or from severe disease. When a new gene for resistance to a pathogen appears or is introduced into a plant, the plant becomes resistant to all or most of the previously existing individuals of the pathogen. Such pathogens contain one and usually more than one gene for virulence, but if they do not contain the additional new gene for virulence that is required to overcome the effect of the new resistance gene in the plant, they cannot infect the plant and the plant remains resistant. Thus, even one new gene for resistance to a pathogen can protect plants that have the gene from becoming infected by all of most pre existing races of the pathogen at least for several months and possibly for several years.

8.2.1 Mechanism of Variability

In host plants and in pathogens, such as most fungi and higher plants and nematodes, which usually reproduce by means of a sexual process, variation in the progeny is introduced primarily through segregation and recombination of genes during the meiotic division of the zygote. Bacteria and even virus

exhibits variations as a result of sexual recombination. In many fungi, heteroploidy and certain parasexual processes lead to variation.

Two mechanisms of variability *viz.* mutation and recombination occur in both plants and pathogens.

(a) Mutation: Amutation is an abrupt change in the genetic material of an organism, which is then transmitted in a hereditary fashion to the progeny. Mutations represent changes in the sequence of bases in the DNA, either through substitution of one base for another or throughaddition or deletion of one or many base pairs. Additional changes may be brought about by amplification of particular segments of DNA to multiple copies, by insertion or excision of a transposable element, that is, a movable DNA segment, into a coding or regulatory sequence of a gene and by inversion of a DNA segement.

Mutations occur spontaneously in nature in all living organisms. Mutations in single-celled organismsms (bacteria, fungi with a haploid mycelium and in viruses) may be expressed immediately after their occurrence. Most mutations are usually recessive and in diploid or dikaryotic organisms, mutations can remain unexpressed until they are brought together in a homozygous condition.

The mutations for virulence probably occur rarely than mutations for any other inherited characteristics but because of large number of progeny produced by pathogens, it is probable that large numbers of mutants differing in virulence from their parent are produced in nature every year.Considering that only a few genetically homogeneous varieties of each crop plant are planted continuously over enormous land areas for a number of years, the threat of new, more virulent, mutants appearing and attacking a previously resistant variety is a real challenge for the farmers. Additionaly once a new factor for virulence appears in a mutant this factor will take part in the sexual or parasexual processes of the pathogen and may produce recombinants possessing virulence quite different in degree or nature from that existing in the parental strains.

Three different types of adaptations are brought about by changes in genetic material of the cytoplasm have been shown in pathogens. Pathogens may acquire the ability to tolerate previously toxic substances to utilize new substances for growth and to change their virulence toward host plants. Several

characteristics of plants are also inherited through extranuclear DNA, including resistance or susceptibility to infection by certain pathogens.

B) **Recombination:** Recombination occurs primarily during the sexual reproduction of plants, fungi and nematodes whenever two haploid (N) nuclei, containing slightly different genetic material, unite to form a diploid (2N) nucleus, called a zygote. The zygote sooner or later divides meiotically and produces new haploid cells (gametes, spores, mycelium). Recombination of genetic factors occurs during the meiotic division of the zygote as a result of genetic crossovers in which parts of chromatids (and the genes they carry) of one chromosome of a pair are exchanged with parts of chromatids of the other chromosome of the pair. Recombination of the genes of two parental nuclei takes place in the zygote and the eventual haploid nuclei or gametes resulting after meiosis are different both from the gametes that produced the zygote and from one another.

8.2.2 Stages of variation in Pathogens

The entire population of a particular organism on the earth for example a fungal pathogen, has certain morphological and other phenotypic characteristics in common and makes up the species of pathogen, such as *Puccinia graminis,* the cause of stem rust of cereals. Some individuals of this species, however, attack only wheat or only barley, or oats and these individuals make up groups that are called varieties or special forms *(formae specialis)* such as P. *graminis* f. sp. *tritici* or *P. graminis tritici, P. graminis hordei* and *P. graminis avenae* . Even within each special form some individuals attack some of the varieties of the host plant but not the others, some attack another set of host plant varieties, and so on, with each group of such individuals making up a race. Example, there are more than 200 races of *Puccinia graminis tritici (race* 1, race 15, race 59, and so on). Occasionally, one of the offspring of a race can suddenly attack a new variety or can cause severe symptoms on a variety that it could barely infect before. This individual is called a variant. The identical individuals produced asexually by the variant make up a biotype. Each race consists of one or several biotypes (race 15A etc).

The appearance of new pathogen biotypes may be very dramatic when the change involves the host range of the pathogen. If the variant has lost the ability to infect a plant variety that is widely cultivated, this pathogen simply loses its ability to procure a livelihood for itself and will die without even making its existence known to us. If, on the other hand, the change in the variant pathogen enables it to infect a plant variety cultivated because of its resistance to the

parental race or strain, the variant individual, being the only one that can survive on this plant variety, grows and multiplies on the new variety without any competition and soon produces large populations that spread and destroy the heretofore resistant variety. This is the way the resistance of a plant variety is said to be "broken down," brought by the change in the pathogen, not the host plant.

8.2.3 Types of Plant Resistance

Plants are resistant to certain pathogens either because they belong to taxonomic groups that are outside the host range of these pathogens (nonhost resistance) or because they possess genes for resistance (R genes) directed against the avirulence genes of the pathogen (true, cultivar-specific, or gene-for-gene resistance), or because the plants escape or tolerate infection by these pathogens (apparent resistance)

Each kind of plant is a host to a small and different set of pathogens that make up a small proportion of the total number of known plant pathogens. While in other sense, each kind of plant is a nonhost to the vast majority of known plant pathogens. Nonhosts are completely resistant to pathogens of other plants, usually even under the most favorable conditions for disease development (nonhost resistance). The same species of plants, however, that are nonhosts to most pathogens are susceptible, to a lesser or greater extent, to their own pathogens.

The variation in susceptibility to the pathogen among plant varieties is due to different kinds and different number of genes for resistance that may be present in each variety. The effects of individual resistance genes vary from large to minute, depending on the importance of the functions they control. A variety that is very susceptible to a pathogen isolate obviously has no effective genes for resistance against that isolate. The same variety, however, may (or may not) be susceptible to another pathogen isolate obtained from infected plants of another variety.It is likely that the variety also contains several genes for resistance, each specific against a particular pathogen isolate.

True Resistance: Disease resistance that is genetically controlled by the presence of one, a few, or many genes for resistance in the plant is known as true resistance. In true resistance, the host and the pathogen are more or less incompatible with one another, either because of lack of chemical recognition between the host and the pathogen or because the host plant can defend itself against the pathogen by the various defense mechanisms either already present

or activated in response to infection by the pathogen. There are two kinds of true resistance: horizontal and vertical.

i). **Horizontal Resistance:** All plants have certain, but not always the same, level of possibly unspecific resistance that is effective against each of their pathogens. Such resistance is called nonspecific, general, quantitative, adult-plant, field or durable resistance and most commonly as horizontal resistance.This resistance is controlled by many genes (dozens or hundreds) therefore also called as Polygenic or multigene resistance.

Each of these genes alone is rather ineffective against the pathogen and may play a minor role in the total horizontal resistance (minor gene resistance).

These many numbers of genes involved in horizontal resistance exert their influence by controlling the numerous steps of the physiological processes in the plants that provide the material and structures which makes the defense mechanism of the plant.

The horizontal resistance does not protect the plants from becoming infected but slows down the development of individual infection loci on a plant and thereby slows down the spread of the disease and development of epidemics in the field.

This type of resistance is quantitatively inherited.

ii). **Vertical Resistance:** Many plant varieties are quite resistant to some races of a pathogen while they are susceptible to other races of the same pathogen. In other words, depending on the race of the pathogen used to infect a variety, the variety may appear strongly resistant to one pathogen race and susceptible to another race under a variety of environmental conditions. Such resistance differentiates clearly between races of a pathogen since it is effective against specific races of the pathogen and ineffective against others. Such resistance is called strong, major, specific, qualitative or differential resistance, but it is more commonly referred to as vertical resistance.

Vertical resistance is always controlled by one or few genes (monogenic or oligogenic resistance). These genes are referred as R genes, control a major step in the recognition of the pathogen by the host plant and therefore play a major role in the expression of resistance. In the presence of vertical resistance the host and pathogen appear

incompatible. The host may respond with a hypersensitive reaction, may appear immune or may slow pathogen reproduction.

Vertical resistance inhibits the development of epidemics by limiting the initial inoculum or by limiting reproduction after infection.

Complete resistance may be provided by a single resistance gene. It is desirable to combine, or pyramid more than one resistance gene (R1R2, R1R3, R1R2R3) in the same plant which then is resistant to all the pathogen races to which each of the genes provides resistance. For example wheat has 20 to40 genes for resistance against the leaf rust fungus *Puccinia recondite*. Each gene for resistance such as R2 makes the plant resistant to all the races of the pathogen that contain the corresponding gene for avirulence. This pathogen race and its avirulence gene(A2) are detected because the pathogen attacks plants that lack the particular gene for resistance (R2).

There are, however, several plant diseases in which resistance is controlled by genetic material contained in the cytoplasm of the cell. Such resistance is sometimes referred to as cytoplasmic resistance. The two best known cases of cytoplasmic resistance occur in corn in which resistance to two leaf blights, the southern corn leaf blight caused by *Bipolaris (Helminthosporium) maydis* and the yellow leaf blight caused by *Phyllosticta maydis,* is conferred by the lack of a gene in the mitochondria of normal cytoplasm of various types of corn that encodes a receptor for the host-specific toxin produced by each pathogen. The presence of such a gene in the mitochondria of Texas male-sterile cytoplasm makes all corn lines with Texas male-sterile cytoplasm susceptible to these pathogens.

In any area and almost every year, limited or widespread plant disease epidemics occur on various crop plants. Under certain conditions or circumstances however some very susceptible plants or varieties of these crops may remain free from infection or symptoms and thus appear resistant. The apparent resistance to disease of plants known to be susceptible is generally a result of disease escape or tolerance to disease.

Disease escape occurs whenever genetically susceptible plants do not become infected because the three factors necessary for disease (susceptible host, virulent pathogen and favorable environment) do not coincide and interact at the proper time or for sufficient duration. Plants

may escape disease from soil bornepathogens because their seeds germinate faster, or their seedlings harden earlier than others and before the temperature becomes favorable for the pathogen to attack them. Some plants escape disease because they are susceptible to a pathogen only at a particular growth stage (young leaves, stems, or fruits; at blossoming or fruiting; at maturity and early senescence), and therefore, if the pathogen is absent or inactive at that particular time, such plants avoid becoming infected.

In many cases, plants escape disease because they are interspersed with other types of plants that are insusceptible to the pathogen, and the amount of inoculum that reaches them is much less than if they were in monocultural plantations; because their surface hairs and wax repel water and pathogens suspended in it; because their growth habit is too erect or otherwise unfavorable for pathogen attachment and germination; or because their natural openings, such as stomata, are at a higher level than the rest of the leaf surface or open too late in the day, by which time the leaves are dry and the germ tubes of spores, such as of *Puccinia graminis,* have desiccated.

A number of environmental factors play crucial roles in plant disease escape in almost every location. For example temperature determines the geographical distribution of most pathogens and plants growing outside the range of that temperature escape disease from such pathogens. Most commonly, however, plant disease escape increases in temperature ranges that favor plant growth much more than they do the growth of the pathogen.Temperature outside certain ranges inhibit sporulation of fungi as well as spore germination and infection, and increases the chances for disease escape. The low temperature also reduces the mobility of many insect vectors or pathogens allowing more plants to escape disease.

Lack of moisture due to low rainfall is another most common cause of disease escape in plants. The plants grown in dry areas are generally free of apple scab, late blight, downy mildew because all these disease requires a film of water on the plant.

The wind blowing from wrong direction at right time may increase disease escape by carrying spores and vectors away from the crop plant

and also by drying up plant surface quickly before the pathogen has time to germinate and infect.

The lateness, rapid growth, resistance to bruising, unattractiveness to vectors and tolerance to low temperatures are often bred into crop varieties to help them escape specific diseases.

Escape from disease is a manageable quality and farmers through many cultural practices like using disease free,vigorous seed, choosing proper soil, planting date, depth of sowing, utilizing proper crop rotation, sanitation, interplanting and multilines attending to insect and vector control etc.

Tolerance to Disease

Tolerance to disease is the ability of plants to produce a good crop even when they are infected with a pathogen. Tolerance results from specific, heritable characteristics of the host plant that allow the pathogen to develop and multiply in the host while the host, either by lacking receptor sites for or by inactivating or compensating for the irritant excretions of the pathogen, manages to produce a good crop. Tolerant plants are generally susceptible to the pathogen, but they are not killed by it and generally show little damage.

8. 3 Genetics of Virulence in Pathogens and Resistance in Host Plants

Infectious plant diseases are the result of the interaction of at least two organisms, the host plant and the pathogen. The properties of each of these two organisms are governed by their genetic material, the DNA, which is organized in numerous segments making up the genes.

It is known that the host reaction i.e the degree of susceptibility or resistance of host to various pathogens is an inherited characteristic. This knowledge has been used quite effectively in breeding and distributing varieties resistant to pathogens causing particular diseases. The pathogens consist of a multitude of races, each different from others in its ability to attack certain varieties of a plant species but not other varieties. When a variety is inoculated with two appropriately chosen races of a pathogen, the variety is susceptible to one race but resistant to the other. Conversely, when the same race of a pathogen is inoculated on two appropriately chosen varieties of a host plant, one variety is susceptible while the other is resistant to the same pathogen. This clearly indicates that, in the first case, one race possesses a genetic characteristic that

enables it to attack the plant, while the other race does not and in the second case, that the one variety possesses a genetic characteristic that enables it to defend itself against the pathogen, so that it remains resistant, while the other variety does not. When several varieties are inoculated separately with one of several races of the pathogen, it is again noted that one pathogen race can infect a certain group of varieties; another race can infect another group of varieties, including some that can and some that cannot be infected by the previous races and so on.

The studies on the inheritance of resistance versus susceptibility in plants prove that single genes control resistance and their absence allows susceptibility. Studies of the inheritance of avirulence versus virulence in pathogens prove that single genes control avirulence and their absence allows virulence. Studies of their interactions prove that R genes in the plant are specific for *avr* genes in the pathogen. Thus, varieties possessing certain genes for resistance react differently against the various pathogen races and their genes for avirulence. The progeny of these varieties react to the same pathogens in exactly the same manner as did the parent plants, indicating that the property of resistance or susceptibility against a pathogen is genetically controlled (inherited). Similarly, the progeny of each pathogen causes on each variety the same effect that was caused by the parent pathogens, indicating that the property of virulence or avirulence of the pathogen on a particular variety is also genetically controlled (inherited).

Under favorable environmental conditions, the infection (susceptibility) or noninfection (resistance) in each host–pathogen combination is predetermined by the genetic material of the host and of the pathogen. The number of genes determining resistance or susceptibility varies from plant to plant, as the number of genes determining virulence or avirulence varies from pathogen to pathogen.

The gene for gene concept: The coexistence of host plants and their pathogens side by side in nature indicates that the two have been evolving together. Changes in the virulence of the pathogens appear to be continually balanced by changes in the resistance of the host and vice versa. In this a dynamic equilibrium of resistance and virulence is maintained and both host and pathogen survive over considerable periods of time. The stepwise evolution of virulence and resistance is explained by the gene for-gene concept, according to which for each gene that confers virulence to the pathogen there is a corresponding gene in the host that confers resistance to the host and vice versa.

The gene-for-gene concept was first proved in the case of flax and flax rust, but it has now been shown to operate in many other rusts and smuts, powdery mildews, apple scab, late blight of potato and other diseases caused by fungi.Generally in the host the genes for resistance are dominant (R) while genes for susceptibility that lack resistance are recessive (r). In the pathogen the genes for avirulence (inability to infect) is usually dominant (A) while genes for virulence are recessive (a). Each gene in the host can be identified only by its counterpart gene in the pathogen and vice versa. Of the four possible gene combinations only the AR interaction is incompatible (resistant) i.e the host has a certain gene for resistance (R) that recognizes the corresponding specific gene for avirulence (A) of the pathogen. In the Ar combination, infection results because the host lacks genes for resistance (r) and so the pathogen can attack it with its other genes for virulence. In aR, infection occur because although the host has a gene for resistance, the pathogen lacks the gene for avirulence that is recognized specifically by this particular gene for resistance and therefore no defense mechanisms (resistance) are activated. Finally, in the ar interaction, infection results because the plant has no resistance (r) and the pathogen, being a pathogen and therefore virulent (a), attacks it.

The researches suggest that genes for resistance appear and accumulate first in hosts through evolution and that they coexist with nonspecific genes for pathogenicity which evolve in pathogens. Genes for pathogenecity exist in pathogens against all host plants that lack specific resistance. When a specific gene for resistance appears in or is bred into the host, the gene enables the host to recognize the product of a particular gene for virulence in the pathogen. That pathogen gene is then thought of as the"avirulence" gene *(avrA)* of the pathogen that corresponds to the plant resistance gene R. The change in the function of the pathogen gene is because subsequent recognition of the *avrA* gene product (the elicitor molecule) by the receptor coded by the R gene triggers the hy-persensitive response reaction in the plant that keeps the plant resistant. Whenever a new gene for virulence that attacks the existing gene for resistance appears, often by mutation of an existing avirulence gene which then avoids gene-for-gene recognition, the resistance of the host breaks down. Plant breeders then introduce another gene in the plant for resistance, which recognizes the new gene for virulence of the pathogen and extends the resistance of the host beyond the range of the new gene for virulence in the pathogen. This produces a variety which is resistant to all races that have an avirulence gene corresponding to the specific gene for resistance until another

gene for virulence appears in the pathogen. When a variety has two or more genes for resistance (R_1, R_2, ...) against a particular pathogen, it means that each corresponds to one or two or more genes of former virulence (and now avirulence) in the pathogen (a_1, a_2, ...), each of which, once recognized by one of the genes for resistance in the host, subsequently functions as an avirulence gene. The gene combinations and disease reaction, types of hosts and pathogens with two for resistance or virulence are present in corresponding loci.

Susceptible (r_1r_2) plants lacking genes for resistance are attacked by all races of the pathogen, regardless of the virulence (aa) or avirulence genes (A,A_2) carried by the pathogen. Second, pathogen races or individuals designated a_1a_2, that is, which lack genes for avirulence (A_1A_2) for eachgene for resistance of the host (R_1R_2), can infect all plants that have any combination of these genes (R_1 **R_2,** R r_2, r_1R_2) since the a_1a_2 pathogen produces no elicitor molecules capable of triggering the host defense response. When a pathogen has one of the two genes for virulence (a_1 or a_2), that is, it lacks one of the two genes for avirulence (A_1 or A_2), then it can infect plants that have the corresponding gene for resistance (R_1 or **R_2,** respectively) but not plants that have a gene for resistance corresponding to a gene for avirulence in the pathogen (e.g., pathogen with genes A_1a_2 infects plant with $r_1R_2$but not those with R_1r_2, because R_1 can recognize the *avr* gene A_1 and triggers defenses against it).

Plant breeders apply the gene-for-gene concept every time they incorporate a new resistance gene into a desirable variety that becomes susceptible to a new strain of the pathogen. With the diseases of some crops, new resistance genes must be found and introduced into old varieties at relatively frequent intervals, whereas in others a single gene confers resistance to the varieties for many years.

Nature of Resistance: A microorganism is pathogenic because it has the genetic ability to infect another organism and to cause disease. A plant is immune to a pathogen because it is not attacked by the pathogen even under the most favorable conditions or it may show various degrees of resistance ranging from near immunity to complete susceptibility. Resistance in host may be conditioned by a number of internal and external factors that operate to reduce the chance and degree of infection. The first step in any infection is recognition of the host by the pathogen and perhaps the opposite, some type of recognition of the pathogen by the host. Therefore, the absence of a recognition factor(s) in the host could help it avoid infection by a particular pathogen. In general any heritable characteristic of the plant that contributes to localization and isolation

of the pathogen at the points of entry, to reduce harmful effects of toxic substances produced by the pathogen, or to inhibit reproduction and further spread of the pathogen contributes to the resistance of the plant to disease. As a result in most plant diseases the pathogen is usually localized after varying degrees of invasion and colonization of host tissues. There are only few diseases in which the pathogen is allowed to spread unchecked throughout the plant and to kill the entire plant. Furthermore, any heritable characteristic that enables a particular variety to complete its development and maturation under conditions that do not favor the development of the pathogen also contributes to resistance.The contribution of the genes conditioning resistance in the host seems to contain primarily of providing the genetic potential in the plant for development of one or more of the morphological or physiological characters that contribute to disease resistance.

The mechanisms by which genes control the physiological processes that lead to disease resistance or susceptibility are not yet clear, but they are, presumably, no different from the mechanisms controlling any other physiological process in living organisms.

It is found that for the production of an inducible enzyme or a fungitoxic substance needed for defense, a stimulant (elicitor), either secreted by the pathogen or caused by the activities of a pathogen, reacts with a receptor molecule of a host cell. This then transmits signals to other host cell molecules, activating plant defenses. On the other hand, if a pathogen mutant appears which does not secrete the particular elicitor that activates the defense reaction the pathogen infects the host without opposition and so causes disease. In the latter case, the resistance of the host is said to have broken down.

Resistance through hypersensitive Response: The hypersensitive response (HR) is a localized self-induced cell death at the site of infection of a host plant in response to infection by a race or strain of a pathogen that cannot develop extensively in this particular resistant plant cultivar. Thus, the plant species as a whole may be a host to the pathogen species, but individual cultivars (varieties) of the plant may be hosts (susceptible) or nonhosts (resistant) to a particular race or strain of the pathogen. Resistance through the hypersensitive response has been shown to be the result of gene-for-gene systems in which an avirulence *(avr)* gene in the pathogen corresponds to a resistance (R) gene in the host plant. Such gene-for-gene systems provide resistance through the hypersensitive response caused by obligate and facultative intracellular pathogens such as viruses, mollicutes, bacteria, fungi, and nematodes.

Irrespective of the pathogen type it is believed that resistance through the hypersensitive response is the result of recognition by the plant of specific signal molecules, the elicitors, produced by the avirulence genes of the pathogen and recognized by R gene-coded specific receptor molecules in the plant. Such recognition causes the activation of a cascade of host genes which then leads to the hypersensitive response, inhibition of pathogen growth, and thereby resistance.

Avirulence or *avr* genes were first identified by H. H. Flor in the 1950s, but since then numerous bacterial and fungal *avr* genes have been identified. The *avr* genes make a pathogen unable to induce disease (avirulent) on a specific variety of the host plant. In this way, *avr* genes determine the host range of the pathogen at the species and at the race—variety level.

In some cases two independent resistance (R) genes rnay correspond to a single *avr* gene, so there is apparently a gene-for-gene interaction. Some *avr* genes, when transferred artificially to other pathovars, are active in the new pathovars, making the recipient pathogen unable to infect their previously susceptible hosts and cause the hypersensitive response in these plants. In some host—pathogen systems, avr genes determine not only which cultivars of a host species the pathogen can attack but also which plant species it can attack. For example, an *avr* gene *(avrBsT) in* the tomato-infecting group of strains of the bacterium *Xanthomonas campestris pv. vesicatoria,* the pathogen of bacterial spot in tomato and pepper, enables the bacterium to induce the hypersensitive response on all cultivars of pepper. Loss of *avrBsT* from such tomato-infecting strains allows these strains to cause disease on normally resistant pepper cultivars.

Chemically A*vr* proteins are generally hydrophilic therefore water soluble, lacking stretches of hydrophobic amino acids that would enable them to be anchored in cell membranes. Avr proteins also lack stretches of amino acids known as "signal sequences" that would allow the proteins to be secreted into the external medium by the general secretory pathway. Therefore avr gene proteins are produced and either localized in the pathogen cytoplasm are secreted through membrane pores formed by proteins coded for hypersensitive response and pathogenicity *(hip)* genes, known as Hrp proteins (harpins). If harpins are secreted externally, the Avr proteins may act directly as elicitors. If they are localized in the pathogen cytoplasm, the *avr* gene proteins may act enzymatically to produce an elicitor molecule that is transported freely through the bacterial envelope. In either of the cases, the elicitor

reacts directly or indirectly with the product of the corresponding plant resistance Rgene.

Structurally *avr* genes are quite different; some of them have common structural characteristics that allow grouping of *avr* genes into distinct families. The best known example of *avr* gene group is *Xanthomonas* avr gene family, called *pth* (for pathogenicity) genes by some. Members of this gene family are found in different species and pathovars of the bacterium *Xanthomonas.* They encode proteins that, in their central part, have from 13 to 23 copies of a nearly identical 34-amino acid repeat unit.

Till now the functions of only a few *avr* genes have been determined. These include the *avrD* gene from the bacterium *Pseudomonas syringae pv. glycinea* and the *avrCf9* and *avrCf4* genes from the tomato leaf mold fungus *Cladosporium fulvum. The avr* gene products of these pathogens are precursors of elicitor peptides that are exported and specifically elicit the hypersensitive response in host cultivars carrying the corresponding R genes for resistance.

Plants transformed with mutant weakly eliciting or nonelicitor coat proteins expressed weaker or no hypersensitive response. In some viral infections the viral coat protein, which is produced within the cell, appears to function as a specific elicitor that activates the hypersensitive response in plant cultivars that carry the corresponding Rgene for that virus.

Most of the *avr* genes tested so far play no role in pathogenicity or virulence of the pathogen since, even when *avr* genes are inactivated by mutation, susceptible hosts continue to be susceptible. Some *avr* genesfor example, the *avrBs2* gene from the bacterium *Xanthomonas campestris* pv. *vesicatoria,* encode proteins that are also necessary for pathogenicity, as shown by the fact that this *avr* gene is present in all strains of this pathovar, whereas mutants lacking the *avr* gene lose pathogenicity on all susceptible hosts but they do not gain virulence on any previously resistant hosts. On the other hand, several avr genes, such as the *pthA* gene from *X. citri* and *avrb6* from X. *campestris* pv. *malvacearum,* both of them members of the *Xanthomonas avr/pth* gene family, encode proteins that act as pathogenicity or virulence factors.

The *hrp* genes are found only in gram-negative bacteria. It is an additional bacterial gene that seems to be essential for some bacteria to be able to cause visible disease on a host plant, to induce a hypersensitive response on certain plants that are normally not infected by the bacteria and to enable bacteria to multiply and reach high numbers in a susceptible host. Most bacterial species

have two distinct clusters of *hrp* genes. The larger *hrp* gene cluster consists of six to nine transcription units, with each transcription unit coding for several (1 to 12) proteins. Transcription of *hrp* genes is controlled by the presence of certain nutrients, by other bacterial regulatory genes, and by lp far unknown signal molecules of plant origin.*Several hrp* gene-coded proteins, called harpins, seem to be localized in the bacterial cell membrane. There they might be involved in forming a secretory apparatus involved in the outward translocation of bacterial Avr or *Pth* proteins that could interact with components of host plant cells. Some *hrp* genes also code for an ATPase enzyme that may play a role in energizing the secretory apparatus.

Along with *avr* and or *hrp* genes in plant infecting pathogen they are likely to possess one to several additional classes of genes that are essential either for the pathogen to cause disease (pathogenicity factors) or for the pathogen to express increased virulence (virulence factors) on only one or a few related hosts.Pathogenicity factors encoded by "pathogenicitygenes" *(pat)* and "disease-specific genes" *(dsp)* are those involved in steps crucial for the establishment of disease. Such genes include those essential for attachment of the Pathogen to the plant surface, germination and formation of infection structures on the plant surface, penetration of the host, and colonization of the host tissue. Genes involved in the synthesis and modification of the lipopolysaccharide cell wall of gram-negative bacteria may help condition the host range of the bacteria.

Some plant cell wall degrading enzymes (e.g., cutinases), some toxins (e.g., victorin and HC-toxin), hormones (e.g., indoleacetic acid and cytokinin), polysaccharides, proteinases, siderophores, melanin, etc., are produced by pathogens in pathogen-plant interactions in which they are essential for the pathogen to infect and cause disease on its host. In those cases, such factors function as pathogenicity factors. In other plant -pathogen systems the same compounds are helpful but not essential for disease induction and development. In these cases, these compounds are considered as virulence factors.

There is an unlimited number of virulence factors known, produced by pathogens. In addition to many cell-wall-degrading enzymes, toxins, hormones, and polysaccharides, almost all molecules /structures are amylases, lipases, signaling molecules such as homoserine lactone exopolysaccharides, flagella, etc. These compounds/ structures may be present on the pathogen surface or translocated to the extra-cellular environment of the pathogen and in a variety of ways could influence growth of the pathogen in the plant.

R gene: In spite of many and different kinds of plant pathogens that come in contact with a plant, in most of the cases the plants remain resistant to disease because they are not hosts to the vast majority of pathogens (nonhost resistance).When a plant is a host (susceptible) to a certain pathogen, some varieties of the plant may be susceptible, or more susceptible, to the pathogen while others may be resistant, or more resistant, to the pathogen, depending on the kind and number of resistance genes present in the plant, the prevailing environmental conditions and other factors. Even when a plant becomes attacked and diseased by a pathogen, however, a number of defense response (resistance) genes are activated. As a result, in most cases, the plant manages to limit the spread of the pathogen into a smaller or larger spot, lesion, canker, etc., through defense compounds and structures that block the further expansion of the pathogen. In a number of cases, however, plant varieties are resistant to certain pathogen races because they possess specific resistance (R) genes that enable the plant to remain resistant to pathogens carrying the corresponding avirulence (avr) genes.

In 1992, the first R gene, the maize *Hm1* gene, was located, isolated, and sequenced, and its function was described at the molecular level. The *Hml* R gene makes certain varieties of corn plants resistant to race 1 of the fungus *Cochliobolus carbonum* that causes a leaf spot disease on susceptible corn varieties.

After isolation of the *Hm1* gene, more than a dozen plant R genes were isolated from plants, sequenced, and transferred and expressed in other, susceptible, plants. The first such gene was the PTO gene of tomato because it confers resistance in tomato to the bacterial speck-causing strains of *Pseudomonas syringae* pv. *tomato* that carry the avirulence gene *avr-Pto.*

Some of the other R genes isolated from plants include the tomato Cf2, *Cf4,* and Cf9 genes, which confer resistance to the leaf mold-causing fungus *Cladosporium fulvum* races 2, 4, and 9 that carry the avirulence genes *avr2,* avr4, and *avr9,* respectively; the tobacco N' gene, which confers resistance to tobacco mosaic virus (TMV); the flax L^6 gene, which confers resistance to the rust fungus *Melampsora lini race* 6 carrying the *avr6* gene; the rice *Xa21* gene, which confers resistance to many races of the leaf-spotting bacterium *Xanthomonas oryzae;* and several *Arabidopsis* R genes.

The various plant R genes, regardless of the type of pathogen (bacterial, fungal, or viral) to which they confer resistance, have many structural similarities.

a. Most of the R genes exist as clustered gene families.

b. Depending on structure and function, R genes can be subdivided into five classes:

i R genes, like *Hml,* that encode a detoxifying enzyme.

ii. R genes, like PTO, that encode a serine-threonine protein kinase that plays a role in signal transduction.

iii. R genes, like *Xa21* of rice, that encode a protein rich in leucine repeats, function as receptors of kinase like proteins and transmit the signal to phosphokinases for further amplification.

iv. R genes, like the tobacco N' gene and, possibly, the flax L^6 gene and the various *Arabidopsis* genes, that encode proteins which are most likely cytoplasmic with (L^6) or without (N') attachment to the cell membrane. These cytoplasmic proteins, in addition to leucine-rich repeats, also have a site that binds to nucleotides; such proteins may serve as receptors that activate the translocation of a transcription factor from the cytoplasm to the nucleus where it activates transcription of the genes related to hypersensitive response.

v. R genes, like the tomato Cf 9 gene, that encode proteins which consist primarily of leucine-rich repeats and are located outside the cell membrane but are attached to the membrane with a transmembrane anchor. Such R gene-coded proteins may serve as receptors for the extracellular or intracellular elicitor molecules produced as the result of expression of the corresponding *avr* gene. For example, in the case of *avr9,* the elicitor molecule is a peptide consisting of 22 amino acids, and it binds to the receptor product of the Cf9 R gene.

The mechanisms by which R genes bring about disease resistance to a plant against a specific pathogen are not yet understood. It is believed that the elicitor molecule produced by an *avr* gene of the pathogen is recognized by a specific plant receptor encoded by an R gene. Following recognition of the elicitor by the receptor molecule, one or more kinase enzymes may become activated they further amplify the signal by phosphorylating and thereby energizing, other kinases and other enzymes. This leads to a cascade of biochemical reactions which is still unclear, result in the hypersensitive response (HR) and, thereby, localized host resistance at the point of attack by the pathogen.

During the evolutionary race for the survival of the plant from the pathogen a resistance gene R1 was evolved. To resist infection such an individual plant and its progeny (variety 1) were selected for survival and so the plant and and the R1 gene survived and multiplied.This evolutionary process described is supported by the fact that most of the R genes studied so far seem to be present in tandem arrays of multiple (up to 10 or more) related R genes. They exhibit different specificities but behave as though they are alleles of a single gene that cannot be separated during recombination, or exist as a clustered gene family. The various R genes isolated so far appear to have a portion (about 20 percent) of their nucleotide sequences identical, whereas a larger portion(about 50 percent) of the nucleotide sequences are similar. Such relationships among R genes may indicate an important mechanism by which plants, by reshuffling preexisting coding information, can more quickly respond to attack by a new pathogen by reformulating existing R genes into new R genes that then produce new specific receptors. Besides, the change of a pathogen from avirulence to virulence is caused by the loss of an avirulence gene through a loss of function mutation on that gene, an event much more likely to happen than the positive production of a new receptor on a plant by a newly formed R gene.

Other Plant Genes for Resistance: It is possible that the nonhost plant carries numerous R gene-coded receptors, one or more of which quickly recognize and fend off the pathogen, or, probably, some entirely different mechanisms are responsible for nonhost resistance.

The most common types of resistance genes in plants in natural populations, and quite often in cultivated crops are numerous "minor" genes for resistance. These may affect superficial or internal, structural or biochemical defenses, preexisting or induced on or after infection. Such minor genes are probably quite numerous in all plants. They are triggered into action by signal compounds produced by the pathogen or by the infected cells and in most cases, through their actions, produce defenses that manage to halt the advance of the pathogen and colonization of the host to a small lesion on whatever plant organ is attacked. Such minor defense genes do not always appear to effectively defend plants from pathogens, primarily because the pathogens can overcome their hosts by forming a number of small lesions they cause on the plants. Nevertheless, in most cases, these genes manage to halt the pathogen to a small lesion in each individual infection.

In general, plants defend themselves against pathogens by a combination of weapons from two arsenals: (1) structural characteristics that act as physical

barriers and inhibit the pathogen from gaining entrance and spreading through the plant and (2) biochemical reactions that take place in the cells and tissues of the plant and produce substances which either are toxic to the pathogen or create conditions that inhibit growth of the pathogen in the plant.

8.4 Summary

Different plants become diseased as a result of infection by the pathogen which is generally different for each kind of host plant. The gene/genes for virulence in a pathogen are usually specific for one or few related kind of plants that are host to the pathogen. Few pathogens are able to attack many kinds of host plants. Mutation and recombination are the two possible mechanisms to induce variation in both i.e host and the pathogen. These possible mechanisms induce various stages of variation in pathogens resulting in the evolution of different species, varieties, biotypes, races and pathovars. The variation in susceptibility to the pathogen among plant varieties is due to different kinds and numbers of gene for resistance. The true resistance refers to the resistance in plant because of the presence of few or many genes for resistance. The true resistances are of two types the horizontal and the vertical. Horizontal resistance is controlled by many genes while vertical resistance is controlled by one or few genes.

Disease escape in plants generally occur because the three factors necessary for disease i.e susceptible host, virulent pathogen and favourable environment do not coincide and interact at the proper time or for sufficient duration.

Tolerance to disease is the ability of plants to produce a good crop even when they are infected with a pathogen. The stepwise evolution of virulence and resistance is explained by the genefor-gene concept, according to which for each gene that confers virulence to the pathogen there is a corresponding gene in the host that confers resistance to the host, and. vice versa.

A microorganism is pathogenic because it has the genetic ability to infect another organism and to cause disease. A plant is immune to a pathogen because it is not attacked by the pathogen even under the most favorable conditions or it may show various degrees of resistance ranging from near immunity to complete susceptibility.

The hypersensitive response (HR) is a localized self-induced cell death at the site of infection of a host plant in response to infection by a race or strain of a pathogen that cannot develop extensively in this particular resistant plant cultivar.

Avirulence or *avr*genes were first identified by H. H. Flor in the 1950s.The *avr* genes makea pathogen unable to induce disease (avirulent) on a specific variety of the host plant. In this way, *avr* genes determine the host range of the pathogen at the species and at the race—variety level.

Along with *avr* and or *hrp* genes in plant infecting pathogen they are likely to possess one to several additional classes of genes that are essential either for the pathogen to cause disease (pathogenicity factors) or for the pathogen to express increased virulence (virulence factors) on only one or a few related hosts.Pathogenicity factors encoded by "pathogenicity genes" *(pat)* and "disease-specific genes" *(dsp)* are those involved in steps crucial for the establishment of disease.

8.5 Glossary

- **Biotype:** A subgroup within a species or race usually characterized by the common possessionof a single or few new characters.
- **Codon:** The coding unit consisting of three adjacent nucleotides which codes for specific amino acid.
- **Elicitors:** Molecule produced by the pathogen that induce a response by the host.
- **Horizontal Resistance:** Partial resistance equally effective against all races of a pathogen.
- **Pathovar:** In bacteria a subspecies or group of strains that can infect only plants within a certain genus or species.
- **Resistance:** The ability of an organism to exclude or overcome completely or in some degree, the effect of a pathogen or other damaging factor.
- Vertical Resistance: Complete resistance to some races of a pathogen but not to others.

8.6 Self -Learning Exercise

Section A : (Very Short Answer Type Questions)

1. Tranfer of gene in between different species is known as
   (a) Vertical gene flow (b) Horizontal gene flow
   (b) lateral gene flow (d) None
2. Intervening the noncoding sequences are called
   (a) Exons (b) Introns

(c) Codons (d) None

3. Which can serve as elicitors?

(a) Chitosan (b) Cell wall pectins

(c) Some glycoprotiens (d) All of these

4. In hypersensitive response the avr gene of the pathogen is responsible for generating

(a) Toxin (b) Receptor

(c) Elicitor (d) Repressor

Section B: (Short Answer Type Questions)

1. Define the term gene.
2. What do you mean by variability?
3. What is pathovar?
4. Define virulence.
5. Name the genes responsible for inducing resistance in plants towards pathogens.
6. Define disease escape.

Section C : (Long Answer Type Questions)

1. Discuss in detail about the mechanisms responsible for inducing variability in host and the pathogen.
2. Explain in detail about the role of genetics in inducing virulence in pathogen and resistance in host plants.
3. Write a detail note on the types of plant resistance found in plants.
4. Discuss in detail about the nature of resistance to disease.
5. Discuss the types of genes found in pathogen for inducing pathogenesis in host plant.

8.7 References

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Unit - 9

Fungal Diseases - I:

Classification and General

Structure of the Unit:

9.0 Objectives

After going through this unit you will be able to understand:

- the classification of plant pathogenic fungi
- general symptoms of fungal diseases.
- identification and differentiation of various fungi by distinguishing symptoms produced by different fungi in plants.
- explain various methods of disease cycles.
- explain disease control measures.

9.1 Introduction

Plant diseases are important as they cause damage to plants and plant products. The kind and amount of losses caused by plant diseases vary with plant, the pathogen, locality, environment, control method etc. Before the discovery of microorganisms as the causative agent (1850s) the classification of diseases was made by many authors on the effect they bring about in plants rather than

on the cause. However, after 1850, the diseases were classified on the basis of their cause rather than their effect. Some of these are made based on host, e.g. diseases of cereals, potato etc. or, based on symptoms like root rot, powdery mildews etc. The most satisfactory way to classify diseases is on the basis of their cause. In this unit various aspects of fungal diseases are discussed.

9.2 Classification of Fungi

The fungi have been divided into four classes-Phyco-, Asco-, Basidio- and Deuteromycetes. This has been accepted and followed by a number of workers (Gwynne-Vaughan and Barnes, 1937; Wolf and Wolf, 1947; Bessey, 1950; Gaumann, 1952; Alexopoulos, 1952; Smith, 1955). In these classifications, the conceptions regarding the last three classes have remained fairly constant, but the first class-Phycomycetes-has been lately undergoing revolutionary changes. Initially Phycomycetes was divided into two sub-classes; Oomycetes and Zygomycetes, on the basis of their reproductive structures. Later on it was suggested that this was not a natural group. In 1958, Sparrow divided the older class Phycomycetes into six groups and gave class ranks to all of them.

At one time slime molds were kept in the animal kingdom and called them Mycetozoa (DeBary, 1887; Bessey, 1950), but today most of the botanists consider them to be related to fungi. However, the myxomycetes differ so markedly from other fungi (multinucleate, free living, naked plasmodium, zoospores with two heterokont, whiplash type of flagella, asexual reproduction by sporangia and non-motile spores) that they should be placed in a separate division-the Myxomycophyta.

Further, the name-endings of fungal groups have been revised as per recommendations of the committee on International Rules of Botanical Nomenclature. According to it the names of the division of fungi should end in mycota, subdivision in mycotina, class in mycetes, subclass in mycetidae, order in ales, family in aceae and genus and species have no standard endings.

The broad classification adopted by Alexopoulos (1962) is given below:

Subdivision: 2 subdivisions

1. Myxomycotina (Myxomycophyta, Mycetozoa)

   Class: Myxomycetes

2. Eumycotina (Eumycophyta or true fungi)

The subdivision, Eumycotina, is divided into nine classes as follows:

1. Chytridiomycetes.
2. Hyphochytridiomycetes.
3. Plasmodiophoromycetes.
4. Oomycetes.
5. Trichomycetes.
6. Zygomycetes.
7. Ascomycetes.
8. Basidiomycetes.
9. Deuteromycetes.

Class 1. Chytridiomycetes

The one characteristic feature is the production of motile cells (zoospores or planogametes), each with a single posterior whiplash flagellum (Sparrow, 1958/1959). The other characteristics which may also be present in other fungi are: (1) coenocytic structure of the thallus which may be a multinucleate globose or oval structure, an elongated simple hypha or a well developed mycelium and (2) the conversion of the resting spore or zygote into a resting sporangium (diploid coenocytic thallus).

Key to the Orders of the Class- Chytridiomycetes

A. True mycelium lacking, rhizomycelium may be present- Chytridiales.

AA. True mycelium present.

B. Sexual reproduction by fusion of planogametes; thick walled resistant sporangia are formed - Blastocladiales.

BB. Sexual reproduction by fusion of a male planogamete with a female aplanlaogamete. Resistant sporangia are not formed- Monoblepharidales.

The order Chytridiales is divided into the following families:

a. Olpidiaceae: Holocarpic, parasitic on algae, mosses and flowering plants.

b. Synchytriaceae: Parasitic, holocarpic in which the sporangia are inoperculate which are enveloped in a common membrane.

c. Phylactidiaceae: Eucarpic, monocentric forms.

d. Megachytridiaceae: Eucarpic, polycentric forms with operculate sporangia.

e. Physodermataceae: Eucarpic, polycentric forms with characteristic turbinate cells.

The orders, BIastocladiales and Monoblepharidales, do not have genera of plant pathological importance.

Class 2. Hyphochytridiomycetes

These are fresh water or marine chytrid-like fungi whose motile cells are anteriorly uniflagellate, flagellum of the tinsel type. They are parasitic on algae and fungi; or saprobic on plants and insect debris in water in which they live. The class is divided into three families. These are:

a. Anisolpidiaceae- Holocarpic forms.

b. Rhizidiomycetaceae-Eucarpic, monocentric forms.

c. Hyphochytriaceae-Eucarpic, polycentric forms.

Class 3. Oomycetes

Fungi which reproduce asexually by means of biflagellate zoospores, each bearing one tinsel type flagellum directed forward and one whiplash type flagellum directed backward. Sexual reproduction is typically oogamous.

Key to the Orders of the Class- Oomycetes

A. Zoospores always formed within the sporangium, diplanetic, monoplanetic or rarely aplanetic.

B. Holocarpic or eucarpic hyphae when present without constriction- Saprolegniales. BB. Eucarpic, hyphae constricted - Leptomitales.

AA. Zoospores formed within the sporangium, or if not, then usually within an eyanescent vesicle arising from the sporangium, monoplanetic.

C. Holocarpic- Lagenidiales.

CC. Eucarpic-Peronosporales.

Class 4. Plasmodiophoromycetes

These are obligate endoparasites which usually cause hypertrophy in the host cells. The somatic phase is a plasmodium. Swarm cells and zoospores bear two unequal anterior flagella, both of the whiplash type. Examples of pathological importance: Plasmodiophora and Spongospora.

Class 5. Zygomycetes

Sexual reproduction is characterized by the production of a sexual resting spore called zygospore. Zygomycetes are classified into three orders: Mucorales, Entomophthorales and Zoopagales.

Key to the Orders of the Class- Zygomycetes

A. Chiefly saprobic, some weekly parasitic on plants, asexual reproduction by sporangia contajning one to many aplanospores, sometimes by conidia- Mucorales.

AA. Rarely parasitic on plants, sometimes saprobic; asexual reproduction by modified sporangia functioning as conidia, or by true conidia.

B. Modified sporangia functioning as conidia which are forcibly dischargedEntomophthorales.

BB. Conidia not forcibly discharged-Zoopagales.

The order Mucorales is divided into a number of families. These are:

a. Mucoraceae-Sporangia large, many spored with well-developed columella; the sporangia are all alike.

b. Thamnidiaceae -In this family two types of reproductive structures are formed -columellate sporangia, and smaller non-columellate sporangia termed sporangiola, often borne in whorls at the tips of the branches.

c. Choaneophoraceae- In this family, as in the Thamnidiaceae, both sporangia and sporangiola occur. The sporangia are usually columellate and often hang downwards. Spores are striat-e epispores and bristle-like appendages.

d. Cunninghamellaceae- Primary sporangia are totally lacking. sporangiola are borne on terminal or lateral vesicles and are finally reduced to conidia.

e. Piptocephalidaceae- Members are usually parasitic on other mucorales. Sporangia cylindrical with a linear row of spores.

f. Pilobolaceae- coprophilous.

g. Kickxellaceae- characterized by the formation of conidia on special structures called sporocladia.

h. Mortierellaceae- The sporangia are big and multispored but there is no

columella.

i. Endogoniaceae- characterized by special structures Sclerotial bodies called sporocarps. Sporangia and zygospores are produced within these sclerotial bodies.

The order Entomophthorales, are chiefly parasitic on insects. A characteristic feature of this order is that the mycelium breaks into hyphal bodies from which conidiophores arise. Conidia are violently discharged. Sexual reproduction occurs when two hyphae enlargements acting as gametangia copulate. Waterhouse (1973) recognized a single family, Entomophthoraceae, with six genera. Entomophthora and Basidiobolus are the two important genera.

The order zoopagales are specially adopted for parasitizing small animals, such as amoebae, rhizopods and nematodes. They reproduce sexually by the formation of zygospores.

Class 6. Trichomycetes

According to Martin (1961), the Trichomycetes are fungi with simple or branched filamentous thallus attached by a basal cell to the digestive tract or the external cuticle of the living arthropods.

Class 7. Ascomycetes

The mycelium is usually septate, frequently produces conidia, rarely ±, unicellular and reproduces by budding. Ascospores are produced within asci. Sexual fusion is by gametangia or through somatic hyphae.

Ascomycetes are classified into three subclasses on the basis of their ascocarps, method of ascus formation and structure of their asci. Three subclasses: Hemiascomycetidae, Euascomycetidae and Loculoascomycetidae

Key to the Subclasses of the Class Ascomycetes

A. Asci arising naked; no ascogenous hyphae or ascocarps produced-Hemiascomycetidae. AA. Asci produced in ascocarps, mostly from ascogenous hyphae.

B. Asci typically unitunicate, in an apothecium if bitunicate; ascocarps of various types - Euascomycetidae.

BB. Asci bitunicate, ascocarp an ascostroma-Loculoascomycetidae.

Key to the Orders of the Subclass 1. Hemiascomycetidae

A. Asci arising directly from zygotes, each derived from the copulation of two cells, or pathogenetically from single cells- Endomycetales.

AA. Asci arising from binucleate ascogenous cell which develop like chlamydospores,

from binucleate cells-Taphrinales.

The order Endomycetales is divided into the following families:

A. Asci multispored - *Ascoideaceae*.

AA. Asci mostly 1-8 spored.

B. Mycelium abundant.

C. Plasmogamy by copulation of uninucleate gametangia- *Endomycetaceae*.

CC. Plasmogamy by copulation of non-motile gametes released from gametangia-*Spermophthoraceae*.

BB. Mycelium scanty or lacking-*Saccharomycetaceae*.

The order Taphrinales is divided into the following families:

A. Ascogenous cells formed singly in the intercellular spaces, ascus multispored-*Protomycetaceae.*

AA. Ascogenous cells formed in a layer giving a hymenium of asci, at first eight spored but commonly becoming multi spored by the budding of the original eight spores*Taphrinaceae*.

Key to the Orders of the Subclass 2. Euascomycetidae

A. Saprobes or parasites of plants or animals, mycelium abundant, well developed.

B. Asci scattered within the ascocarp or united in spore balls within a spore-cyst,

evanescent. Series- Plectomycetes. .

C. Ascocarps sessile.

D. Ascocarps without ostioles-Eurotiales.

Family-*Ascosphaeriaceae*, *Gymnosphaeriaceae, Eurotiaceae*

DD. Ascocarps ostiolate-Microascales.

Family-*Microascaceae, Ophiostomataceae, Clavicipitaceae*

CC. Ascocarps stalked-Onygenales.

Family-*Onygenaceae, Trichocomaceae*

BB. Asci in a basal or peripheral layer, forming a hymenium, or in basal tufts; typically persistent, sometimes evanescent.

E. Ascocarps closed; mostly ostiolate. Series-Pyrenomycetes.

F. Ascocarp with perithecial wall of its own.

G. Mycelium largely superficial.

H. Mycelium white; asci in cleistothecia-Erysiphales.

Single family-*Erysiphaceae*

HH. Mycelium dark; ascocarps ostiolate-Meliolales.

Single family-*Meliolaceae*

GG. Mycelium within the substratum

I. Asci evanescent-Chaetomiales.

single family-*Chaetomiaceae*

II. Asci persistent.

J. Ascospores thread-like-Clavicipitales.

JJ. Ascospores not thread-like, sometimes needle shaped.

K. Ascocarps and stromata, if present, dark, membranous or carbonous.

L. Ascal bases not gelatinizing, mature ascus remains attached to the

inner perithecial wall-Sphaeriales.

Family-*Sordariaceae, Phyllachoraceae*

Diatrypaceae, Xylariaceae

LL. Ascal bases gelatinizing, mature asci loose

in theperithecial cavity and exuded through the ostiole-Diaporthales

Family-Diaporthaceae, Gnomoniaceae

KK.Ascocarps and stromata, if present, brightly

coloured, soft, fleshy or waxy-Hypocreales.

Family-*Hypocreaceae, Hypomycetaceae,*

Melanosporaceae

FF. Asci in ascostromata.

M.Ascocarp ostiolate, ostiole funnel-shaped-Coryneliales.

MM. Ascocarp opening irregularly or by an ostiole, but ostiole not funnel shaped-Coronophorales

EE. Ascocarp an open apothecium or a modified form thereof.

Series- Discomycetes.

N. Ascocarps above the ground.

O. Asci inoperculate.

P.Ascospores thread like-Ostropales.

Family-Stictidaceae

PP. Ascospores not thread-like, sometim

Shaped-Helotiales

Many families; *Phacidiaceae, Sclerotiniaceae*, *Geoglossaceae* and *Cyttariaceae* are important

OO. Asci operculate or suboperculate-Pezizales.

Family: *Sarcoscyphaceae, Pezizaceae, Helvellaceae*

NN. Ascocarp below the ground-Tuberales.

Includes several families

AA. Specialized exoparasites of insects or arachnids; thallus usually limited, sometimes consisting of only a few cells. Series Loboulbeniomycetes

1. Order : . Loboulbeniales, include several families.

Important family: *Loboulbeniaceae*

Simple Key to the Orders of the Subclass 3. Loculoascomycetidae

A. Locules uniascal; usually scattered at various levels in the ascostroma-Myriangiales.

Three families: *Elsinoaceae Myriangiaceae,Piedriaceae*

AA. Locules generally polyascal; arranged in a basal layer.

B. Stroma flattened, dimidiate (two halves very unequal or one half lacking altogether) - Microthyriales.

C. Stroma boat-shaped, opening by a longitudinal slit-Hysteriales.

CC. Stroma not boat-shaped.

D. Pseudoparaphysis present-Pleosporales.

Three families: *Venturiaceae, Pleosporaceae, Lophiostomataceae*

DD. Pseudoparaphysis lacking-Dothideales.

Four families: *Dothioraceae. Pseudosphaeriaceae, Capnodiaceae.*

Dothidiaceae.

Form Class 8. Deuteromycetes (the imperfect fungi).

Characteristic feature is absence of a perfect stage.

Key to the Form Orders of the class-Deuteromycetes

A. Reproduction by means of conidia, by oidia or by budding.

B. Reproduction by means of conidia borne in pycnidia-Sphaeropsidales.

Four form families: *Sphaeropsidaceae, Zythiaceae, Leptostromataceae and Excipulaceae*

BB. Conidia, when formed, not in pycnidia.

C. Reproduction by means of conidia borne in acervuli-Melanconiales.

Single family-*Melanconiaceae*

CC. Reproduction by means of conidia borne, otherwise by oidia or by budding

- Moniliales.

Five families: *Cryptococcaceae, Moniliaceae, Dematiaceae, Stilbellaceae, Tuberculariaceae*

AA. No reproductive structures known-Mycelia Sterilia. Rhizoctonia and Sclerotium are important genera

Class 9. Basidiomycetes

Dikaryotic phase is the predominant phase in the life cycle Basidiospores produced on the outside of a spore-producing body-the basidium. Excepting rusts the septum in most of the basidiomycetes is a dolipore septum.

Key to the Subclasses of the Class -Basidiomycetes

A. Basidium septate, deeply divided or consists of a teleutospore germinating into a promycelium; basidiospores usually capable of germinating by repetition; Subclass 1.- Heterobasidiomycetidae.

AA. Basidium not septate or deeply divided, basidiospores usually germinate by the germ tube; Subclass 2.- Homobasidiomycetidae.

Simple Key to the Orders of the Subclass Heterobasidiolnycetidae

A. Basidiocarp usually well-developed; mostly saprobic; some species parasitic on plants or scale insects-Tremellales.

Nine families: *Ceratobasidiaceae, Tulasnellaceae, Sirobasidiaceae, Dacrymycetaceae, Tremellaceae, Hyloriaceae, Phleogenaceae, Auriculariaceae and Septobasidiaceae*.

AA. Basidiocarp lacking or poorly developed; mostly parasitic on vascular plants.

B. Teleutospores present; plant parasites.

C. Basidiospores produced on sterigmata, forcibly discharged - Uredinales. CC. Basidiospores sessile; not forcibly discharged- Ustilaginales.

BB. Teleutospores lacking; resting spores may be present; saprobic,

Family-*Sporobolomycetaceae*.

Simple Key to the Families of the Order Uredinales

A. Teleutospore forming a septate promycelium upon germination.

B. Teleutospores free or variously united, but never in the form of layers or crust-*Pucciniaceae*.

BB. Teleutospores laterally united into layers, crusts or columns- *Melampsoraceae*.

AA. Teleutospores becoming septate during germination, without the formation of an external promycelium- *Coleosporiaceae.*

Simple Key to the Families of the Order Ustilaginales

A. Basidiocarps absent

B. Promycelium septate, basidiospores produced laterally from each cell of the promycelium - *Ustilaginaceae*.

BB. Promycelium non-septate, basidiospores in a terminal cluster- *Tilletiaceae*.

AA. Cup-shaped basidiocarp produced-*Graphiolaceae*.

Simple Key to the Orders of the Subclass Homobasidiomycetidae

A. Basidiocarp lacking, hymenium covering the surface of the parasitized plant tissuesExobasidiales. One family- *Exobasidiaceae*

AA. Basidiocarp present.

B. Hymenium present and exposed before the spores are mature. Series-Hymenomycetes

C. Hymenium borne in various ways, but if basidia line the pores or gills, texture of basidiocarp not soft and putrescent--Polyporales.

Family: *Thelephoraceae, Clavariaceae, Cantharellaceae, Hydnaceae,*

Meruliaceae, Polyporaceae

CC. Hymenium borne on lamellae (gills) or if lining the interior of pores then basidiocarp soft and putrescent-Agaricales.

Family: *Boletaceae, Paxillaceae, Russulaceae, Hygrophoraceae*,

Agaricaceae

BB. Hymenium present or absent; basidiocarps remaining closed at least until the spores have been released from the basidia. Series-Gasteromycetes

D. Gleba fleshy to castilaginous or if slimy then not exposed at maturity-Hymenogastrales.

DD. Gleba powdery, slimy or waxy; if slimy then exposed at maturity.

E. Glebal chambers usually not separated from peridium or from each other.

F. Gleba powdery

G. Hymenium present in early stages; spores mostly light coloured, small-Lycoperdales.

GG. Hymenium lacking or indistinct; spores mostly dark. large- Sclerodermatales.

FF. Gleba slimy and fetid; exposed on a receptacle-Phallales

EE. Glebal chambers forming waxy peridioles, or entire gleba separating as a unit from the, peridium- Nidulariales.

9.3 Symptomatology of Fungal Diseases

Symptomatology is the set of symptoms characteristic of plant infected by a pathogen. A particular pathogenic fungus causes a particular set of symptom on the host plant. Disease can be identified by specific symptoms caused by a particular fungus. Different types of symptoms caused by fungi are as follows:

(I) Damping Off and Seedling Blights : The term damping-off covers several soil borne diseases of plants to describe underground, soil line, or crown rots. Damping off and seedling blight occurs in germinating seeds and seedlings. The infection occurs from soil and affects the initial establishment of crop.

There are two types of damping off symptoms;

(a) Pre-emergence damping off: In this case sprouting seeds starts rotting before it breaks through the soil. Germinating seeds may be infected as soon as moisture penetrates the seed coat or a bit later as the radicle begins to extend, all of which rot immediately under the soil surface. Pre-emergence damping off is more common in cold wet soils when germination is slow. In field areas where such infections have taken place can be seen as bare spaces in uniform rows of seedlings due to low viability.

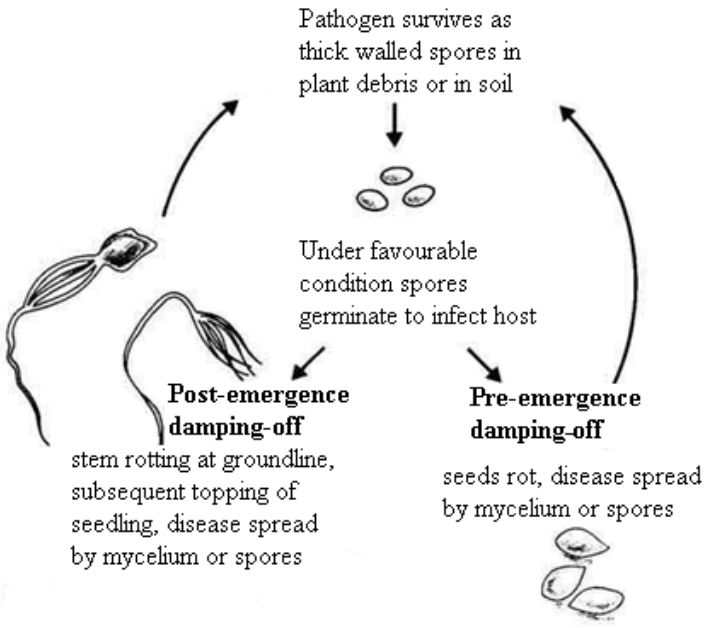

Fig. 9.1: Generalized disease cycle of Pre and post-emergence damping off symptoms

(b) Post-emergence damping off: This can also be termed as seedling blight. As soon as the seedlings emerge from soil, they rot or wilt. The cotyledons may break the soil surface only to whither and die or healthy looking seedlings may suddenly fall over.

At ground level, succulent stems have water-soaked, then necrotic and sunken zone and seedlings fall on the ground. In case of woody seedlings they wilt and remain upright, followed by root decay.

This type of damping off is common in green houses or outdoors in warm humid weather, especially in areas where seedlings are too crowded. Fungi that occur in the upper layers of soil are the most common causal agents of damping off of seedlings. Example: *Pythium debaryanum* and *Rhizoctonia solani.* Although other forms like *Fusarium, Botrytis* and *Phytophthora* also can cause this.

(ii) Root Rot and Foot Rot : Here there is a progressive rotting (disintegration or decay) of the root system as a result of which the basal portion of the stem (foot rot) also become rotted. Thus the aerial parts of plants are deprived of obtaining water and nutrients and, hence, exhibit various symptoms e.g. checking growth, the plant becomes stunted, leaves yellow and wilt, some of them drop and, eventually, the plant collapses and dies. The speed at which these symptoms appear on the plant is mainly dependent on the rate at which root and foot rotting occur and also on the ability of the plant to produce new roots from unaffected areas. This also depends on the ability of the plants to withstand water stress due to the loss of roots.

Rots are of two types (i) hard dry decay, and (ii) soft squashy ones. Example: black root rot of tobacco, peas, tomatoes caused by *Thielaviopsis basicola*; violet root rot of sugarbeet, clover, carrot etc *caused by Rhizoctonia crocorum* with its teleomorph *Helicobasidium purpureum*; root rot of plantation crops like tea, cocoa caused by *Armillaria* causing; rot of peas caused by *Aphanomyces* (Saprolegniales) etc.

(iii) Wilts : Wilt is loosing freshness of plants or to become flaccid when decayed root system of a plant cannot absorb enough water to replace that lost by transpiration. The first indication is that the lower petioles bend down so that an obtuse angle is produced between the stem and the petiole and this is called as epinasty (Wellman, 1941). Vein clearing

followed by yellowing also occur in the lower leaves. Progressively, these leaves become more chlorotic and eventually die. Similar symptoms develop on the upper leaves also leading to the death of the plant. The most common and important fungus that causes wilt is *Fusarium oxysporum.* f. *sp. lycopersici* on tomato and *Fusarium oxysporum* f. sp. *vasinfectum* on cotton.

Two species of *Verticillium* also cause wilt. One is mild and is known as facultative wilt and the other 'propagative wilt' which kills the crops.

Unlike the other wilts, *Fusarium* and *Verticillium* enter their hosts through roots, grow in cortical cells without doing much damage to the cortical tissue and then enter the vessels and establish, blocking them. This leads to epinasty yellowing, vascular browning and wilting. Metabolic changes that include production of ethylene, increased level of IAA and enzyme activity may account for some of these changes in the host plant. The symptoms of disease may be a combined form of blocking of vessels, production of toxins and other chemicals and increased enzyme activity.

(iv) Downy Mildews : Downy mildews develop as downy grey colour patches on the lower side of the leaves or stem bearing sporangiophores. The causal agent belongs to the Peronosporales of the Mastigomycotina.

All downy mildew forming fungi except *Phytophthora* belong to the family Peronosporaceae. The vegetative mycelia (nonseptate) develop in the intercellular spaces of the host tissue (leaf), absorb nutrients by intracellular haustoria which are simple and sac-like or forked. The sporangiophores produced in groups protrude through the stomata and bear sporangia at the tips of the ultimate branches. These groups of sporangiophores and sporangia have grey colour with the sporangia appearing like dewdrops which give the disease the name downy mildews.

Sporangia germinate and give rise to biflagellate zoospores which infect the host or the zoosporangia produce germ tube and initiate infection. These are obligate parasites, which invade the host tissue.

There are exceptions to the fact that downy mildews can be distinguished by means of sporangiophores.

One such infection is the downy mildews of the millet *Pennisetum typhoidum* caused by *Sclerospora graminicola,* which occurs widely in

India. The fungus which infects the inflorescence causes the glumes and stamens of the flower head to develop leaf-like structures so that instead of a close spike with grains developed on it, a loose green structure in formed. This is known as the green ear head. On the leaves it first produces sporangiophores and then causes long chlorotic streaks which split the leaves into threadlike structures. The latter stage has oospores of the fungus and this is known as leaf shred.

Another type of infection is the one caused by *Pseudoperonospora humuli,* the causal agent of downy mildew of hops, where infected root stocks give rise to swollen basal shoots.

(v) **Powdery Mildews :** Powdery mildews are also recognized by the fungal hyphae, conidia and conidiophores on the surface of the host like downy mildews. This gives an appearance to the host as if it is dusted with talcum powder. Powdery mildews also belong to a single family Erysiphaceae of the order Erysiphales of the Ascomycotina. Powdery mildews are obligate parasites having no saprophytic growth. Symptoms are dwarfing and stunting often with a slight reddening and curling of leaves even before the white mycelium becomes noticeable. Due to excessive respiration and withdrawal of plant nutrients the flower bud gets deformed as in powdery mildews of roses.

The vegetative phase consists of white mycelial tuft, which is formed of hyphae that bear upright conidiophores with colourless conidia in chains or singly. The masses of white mycelia, conidiophores and turgid dewdrop like conidia give a white powdery appearance to the host and, hence, the name powdery mildews.

When spores of powdery mildews germinate on the surface of the host, the germ tube does not penetrate the host tissue but produce a tangle of hyphae on its surface. The hyphal cells produce haustoria, which penetrate the host cell epidermis by means of slender tubes and once it enter it becomes round or pear spread. In *Erysipha graminis* these slender tubes branch several times thus increasing the absorbing surface.

(vi) **Rusts :** The name “rust” is derived from the orange-brown (uredo) spore masses, which many of these fungi produce on their hosts in pustules. Rust diseases are caused by members of the order Uredinales of the Basidiomycotina. Rusts are obligate parasites and attack Angiosperms, Gymnosperms and some ferns. The intercellular mycelium which

parasitisizes the host sends intracellular haustoria as in the downy mildews.

Many of the rusts are heteroceious, completing their life-cycle on two different kinds of plants. But some are monoecious (or autoceous) having all the spores form on a single host plant. Many of the rusts show physiological specialization. Within the same species, numerous strains and races occur.

Rusts with a complete life cycle has five different types of spores numbered from 0 to 4.

0. **Spermagonium :** These are also known as pycnia, which resemble the pycnidia of Sphaeropsidales. They are usually found on the upper side of the leaf. The single celled pycnospores or spermatia are discharged in sweet nectar-like liquid. These are produced from the monokaryotic hyphae and function in fertilization.

1. **Aeciospores (aecidiospores) :** These are produced on the lower surface of the same leaf. They are one-celled binucleate and formed in chain in a cup-like sorus, the aecidium having a peridium (wall which opens at or beyond the surface of the host). These aeciospores germinate; give rise to mycelium on which other spore types are produced but never aeciospores.

In the rust fungi there are five different morphological types of aecia. Each of these may be used as the basis for a 'form genus' in the absence of other definite structures:

(i) **Aecidium:** The aeciospores are produced in chain in a short cup-like structure bordered by a layer of cells called the peridium. This is the typical cluster cup found in many species of *Puccinia.*

(ii) **Peridermium:** The peridermium is typically long and well-developed than in aecium, otherwise it resembles aecidium. This is found in certain Gymnospermous rusts.

(iii) **Roestelia:** As in peridermium the peridium is long and well developed and generally more horn like. It tends to split longitudinally at maturity. This term is used for aecia produced by the genus *Gymnosporangium.*

(iv) **Caeoma:** Here there is no peridium and the pustule appears diffuse or indeterminate. This type is found in *Phragmidium and Melampsora.*

(v) **Uraecium:** Here also there is no peridium. The aeciospores are stalked and are like the uredospores known as urediod aecia.

2. **Uredium :** These are one-celled and binucleate spores also known as summer spores or red rust spores. This arises from the mycelium that is produced by aeciospores. Uredospores on germination give rise to further uredia. They are, therefore, known as repeating form. Uredospores are short lived and help in enlarging the distribution of the parasite on the host. Only in some species like *Puccinia vexans* thick-walled uredospores (amphispores) are formed which can survive adverse conditions.

3. **Telium :** These are also known as the winter spores or the black rust spores. Teliospores of majority of the rusts are thick walled and are resting or winter spores which also are binucleate. Over wintering is necessary in some rusts for germination of teliospores.

In majority of the rust, rust genera are grouped on the basis of the teliospores, e.g. *Uromyces-one* celled, stalked; *Puccinia,* two celled, stalked; *Phragmidium* several-celled, stalked; *Melampsora* one celled sessile; *Cronartium* one celled. Telia attached to form Telial column.

4. **Basidiospores :** When teliospores germinate they give rise to a basidium in which the two nuclei fuse, undergo meiosis and on this the basidiospores are formed. These basidiospores are uninucleate.

Rust spores which have all the spore forms 0-4, or those in which the spermagonia are missing are termed macrocyclic. Those with aecia and uredia or these plus spermagonia are termed demicyclic and those with telia only or telia plus spermagonia are termed microcyclic.

Some rusts have all the spore forms on one host and each spore form is capable of infecting the same host. Such a species is known as autecious e.g. *Trochodium.* Those which alternate between two unrelated hosts - and both are necessary for the completion of the life cycle - are heteroecious e.g. *Puccinia graminis tritici.*

(vii) **Smuts :** The disease is named so because of the sooty black masses of spores that they produce. They are caused by fungi belonging to the Ustilaginales of the Basidiomycetous fungi. Vegetative cells of the dikaryotic mycelium round up into thin walled chlamydospores. They are produced singly as in *Ustilago* and *Sphacelotheca* or may be produced in groups called spore balls as in *Georgefischeria.* These smut

spores may be smooth walled or sculptured in various ways. These spores are one celled and binucleate.

The smut spores on germination give rise to a germ tube of limited growth known as promycelium. This stage is common for all. The further stages of development vary in different groups of genera. The binucleate or the dikaryotic mycelium formed as a result of fusion between sporidia, mycelia or cells alone are capable of initiating infection.

Smut fungi are hetrothallic, parasitic with binucleate mycelium. Only few are obligate parasites compared to rusts. Mycelium is distributed in vegetative parts but sporulation occurs only in inflorescence.

(viii) Blight: Blight' is a common term used for diseases caused by a wide variety of organisms, including insects. In all these, leaf damage is sudden and serious. The most predominant symptom is death of the host tissue. Example: Late blight of potato and tomato caused by *Phytophthora infestans* belonging to order Peronosporales of class Oomycetes. After blossoming, large, dark green water-soaked spots appear on leaves in wet weather, first on the lower leaves. As the spot enlarges the center is shriveled and becomes dry dark brown to black. Under moist conditions at the edges of these lesions on the lower surfaces of the leaf a downy whitish growth appears. This consists of sympodially branched sporangiophores, which bear the sporangia at its tips.

Similar lesions are formed on the stem and petioles. There is a characteristic strong odor as tops are blighted.

Tubers are infected by the zoospores washed into the soil from leaves of stem. Small brown to purple discolourations of the skin appear on tubers on its upper side. They become depressed pits. Rotting occurs during storage mainly due to invasion of bacteria through these blighted spots. The fungus over-winters as oospores. But this also is a seedborne pathogen.

(ix) Anthracnose : The term anthracnose is derived from the Greek word *anthrax* meaning coal and *noses* meaning disease. The term literally means 'like coal'.

The term Anthracnose is used for two distinct types of diseases: (1) Characterized by typical necrotic spot or lesion of dead tissue, (2) the

other by the hyperplastic symptoms like a raised border around a depressed center.

The pathogen of anthracnose disease of grapes is *Sphaceloma ampelinum* an imperfect fungus belonging to the Melanconiales which is the asexual stage of an ascomycete *Elsinoe ampelina.* The chief symptom of it is a bird's eye-spot with a raised border.

This name was further extended to a disease of blackberries caused by a species of *Elsinoe* and then to the disease of *Phaseolus* bean caused by *Colletotrichum lindemuthianum.* The term anthracnose is used in an etiological sense for disease caused by *Sphaceloma, Colletotrichum* and *Gloesporium* conidial stages.

The term scab is used for raised hyperplastic symptoms and to lesions but the main objection is that both symptoms occur in the same disease and, hence, Anthracnose is generally used. The conidia are borne in acervuli, which are erumpent cushion-like masses of conidiophores. The conidia are hyaline one-celled and ovoid to oblong.

(x) Leaf Spots : In leaf spot diseases the area of necrosis is limited and the main symptom is only a spot of varying size and shape. These are very often with a brown, sometimes white center and a darker margin. They are caused by viruses, bacteria, fungi or even insects.

Leaf spot is caused by so many fungi but majority of the fungi that cause leaf spots belong to the Ascomycotina or Deuteromycotina.

Amongst imperfect fungi group the leaf spots are caused by members of the Moniliales.

Cercospora with 400 and above species is a form, which is mainly identified by their host. Several leaf spot diseases of economic importance are caused by species of *Cercospora.* Example: Two species of *Cercospora; Cercospora arachidicola* and *Cercospora personata* with their perfect stages as *Mycosphaerella arachidicola* and *Mycosphaerella brekelyii,* respectively cause the 'Tikka disease' of groundnut. Mycelium is both internal and external, inter and intra cellular. Both fungi enter the host through the stomata or by direct penetration.

Alternaria solani on potato and tomato causes leaf spots. This is known as early blight to distinguish it from the late blight of potato caused by

P. infestans. Another disease name target spot refers to *Alternaria* spot. These form concentric rings in the leaf lesions. The leaves of infected potato and tomato leaves drop prematurely and decrease the yield. *Alternaria brassicae* and *A. brassicola* cause dark leaf spots and are troublesome on cabbage and cauliflower.

Drechslera (*Helminthosporium)* causes several important leaf spots on Gramineae. The fungus initially penetrates the coleoptile and grows through it to the inner surface and infects the seedling then through the leaf base into the leaf. *H. gramineum* causes the leaf stripe of barley. Some workers reported that once it invades the seedling, the fungus becomes distributed in the tissue as in the loose smut or bunt of wheat. But N J G. Smith (1929) found that each leaf was infected by externally applied mycelium.

H. avenae causes the leaf stripe of oats. *H. oryzae* (perfect stage *Cochliobolus miyabeanus)* is an important species which causes a leaf spot of rice. On susceptible varieties, lesion development is so extensive that the leaves dry out before the plants mature. Lesions below the ear head result in broken heads or litter grains. Extensive mycelia and conidia occur on crop residues.

(xi) Leaf Curl: In leaves an increase in cells on either side of the midrib and a stimulation in growth of the palisade cells and to a lesser extent of cells of spongy parenchyma results in puckering and curling of the leaf. This is known as the leaf curl.

One of the spectacular diseases of this type occurs on peach and is caused by the fungus *Taphrina deformans* belonging to the order Taphrinales of Ascomycotina. The leaves show symptoms as soon as they emerge. Either a part of the leaf or the entire blade thickens and curls. The chlorophyll soon disappears and red or purple tints develop in the affected areas. Later, this appears to be covered by a greyish bloom due to the sporulation of the fungus. Leaves drop prematurely. This stimulates the dormant buds for the emergence of new leaves. These are not generally infected. Repeated losses of leaves in successive seasons reduce the vigour of the tree and its cropping capacity. Blossoms and young fruits are also attacked and they drop. Some of these infected fruits that mature will have prominent warty outgrowths with reddish tints even though they are not so well-developed as on leaves.

The asci of *Taphrina* are clearly seen on the leaf surface in sections. It forms an exposed layer on the host surface and hence, the old genus name *Exoascus.* Originally there are eight ascospores which bud and give rise to several blastospores in an ascus.

(xii) Leaf Galls or Blisters : This is caused by *Exobasidium* (these are hyperplastic abnormalities, which have a definite form). Other leaf galls are caused by rusts. The intercellular mycelium with branched haustoria enters the host cell. Basidia, which bear the 2-8 basidiospores, extend above the epidermal layers as the asci of *Taphrina.*

Infected region has grey or dark-brown cluster-like areas. When there is severe infection these blisters become confluent and the entire plant appears as if they are burnt as in the blister blight or fire blight often caused by *Exobasidium vexans.*

(xiii) Witches' Broom : Other species of *Taphrina* other than *T. deformans* of peach leaf curl causes witches' broom, e.g. *Taphrina cerasi* on cherry. The fungus stimulates shoot production by the host, which results in the production of a dense cluster of twigs that looks like a broom. These malformations are caused on a wide variety of hosts by insects or mites, viruses and parasitic plants. *Georgefischeria riveae* causes witches' broom on *Rivea hypocraterijormis.* Witches' broom of *Theobroma cocoa* is caused by *Marasmius perniciosus.*

(xiv) Club Root : Abnormal development of the root system results in club root disease. The most important one is the club root or the finger and toe disease of crucifers caused by *Plasmodiophora brassicae* belonging to the Plasmodiophorales of the Myxomycotina. The characteristic symptom is the club like swellings of infected roots. These swellings show marbled or mottled appearance and this distinguishes them from the crown gall of apples caused by *Agrobacterium.*

Symptoms due to interference in water and nutrients by the roots are exhibited in the shoot system. Growth is checked and there is yellowing and wilting of the foliage in the later stages of the disease.

(xv) Cankers : One of the important diseases is the canker of apple caused by *Nectria galligena.* Here the fungus enters only through wounds. If the wound is shallow the fungus is confined only to the cortex and there will not be any canker. The wound must penetrate to the wood for cankerous growth. A small amount of cambium will be killed. The

fungus enters the xylem via the medullary rays and grows up and down the vessels rhrough the pits. The host reacts to this invasion by the fungus. Its movement is blocked by gum formation and tyloses. Its lateral movement is checked by a wound callus.

(xvi) Scab : The common scab of potatoes is caused by *Streptomyces scabis*. Powdery or corky scab of potato is caused by *Spongospora subterranea.* In both, these pustules appear on the tubers.

The scab of apple is caused by *Venturia inaequalis.* On leaves lesions appear on the upper surfaces as areas lighter in colour. They do not have the normal lustre. As the lesions develop they become velvety, olive, brown to black and then finally lose their velvety appearance and appear as dry corky scab. Similar lesions appear on the fruit. Growth is reduced in the affected area and cracks appear around the lesion when the healthy parts enlarge.

9.4 Disease Identification

There are different ways to identify plant disease on the basis of symptoms, host, spore morphology etc.

On the basis of symptoms

Club root or the finger and toe disease of crucifers is caused by *Plasmodiophora brassicae* belonging to the Plasmodiophorales of the Myxomycotina. The characteristic symptom is the club like swellings of infected roots. These swellings show marbled or mottled appearance and this distinguishes them from the crown gall of apples caused by *Agrobacterium.*

Even different species of the same genera can be identified on the basis of symptoms. For example; *Cercospora personata* produce more circular necrotic lesions on both the surfaces, assume a dark brown colour and there are no halos around young spots. Leaf spots of *Cercospora arachidicola* are irregular, circular, often confluent and larger in size. A circular bright yellow halo which blends into the green of the leaf is there from the beginning. Halos are less distinct on the lower surface.

On the basis of morphology of spores

In smuts the morphology of smut spores or chlamydospores is the main features that determine the different species of smut fungi. In some, they are made up of all fertile spores as in *Georgefischeria* or it may consist of sterile elements which cover the viable spores e.g. *Urocystis.*

In smut disease, the variety and position of the sori also helps in identification of the disease. For example, the sori are covered in some only by host tissue as in *Ustilago* and *Tilletia.* In others a peridium of fungal tissue is also formed over the developing spores as in *Sphacelotheca, Sorosporium.*

The way smut spores germinate and further stages of development vary in different groups of genera. For example, in loose smut of oats and wheat caused by *Ustilago avenae, U. tritici,* the chlamydospores germinate to give rise to promycelium which becomes septate. Each of these cells by budding gives rises to small hyaline cells which are thin walled and uninucleate, known as the sporidia. The two sporidia of opposite strains fuses together to give rise a mycelium which is capable of infecting the host tissue.

In loose smut of barley (caused by *Ustilago nuda*) there are no sporidia produced. Mycelia develop from the promycelia and branches of this fuse to give rise to a dikaryotic mycelium. In wheat bunt caused by *Tilletia caries*, the promycelium is aseptate and 8 filiform septate sporidia develop terminally. Fusion occurs between adjacent cells of the sporidia, which then infect wheat.

On the basis of host

Some fungal species cause disease only in the presence of specific host only. This may be due to physiological specialization. For example; Black stem rust causing fungi *Puccinia graminis* have many physiological races which are similar in morphological structure but may be different in their capacity to produce disease in different host. Such physiological races are encountered in rusts, smuts, powdery and downy mildews.

Table 9.1: Physiological races of *Puccinia graminis*

Fungal Pathogen	Host
Puccinia graminis tritici	Wheat, barley and many wild grasses
P. graminis secalis	Rye, barley and many wild grasses
P. graminis avenae	Oats and wild grasses
P. graminis phlelpratensis	Timothy and other wild grasses
P. graminis agrostidis	Aragrostis spp.
P. graminis poae	*Poa pratensis* and other grasses

On the basis of fruit bodies

The Deuteromycotina, which includes a wide variety of organisms, three main groups can be identified on the basis of fruit bodies:

(1) conidiophores and conidia are borne in acervuli in Melanconiales.

(2) conidiophores and conidia arise within pycnidia in Sphaeropsidales.

(3) there is no special fruit bodies even though conidiophores are aggregated in some genera in Moniliales.

Fungi belonging to the first (Melanconiales) order produce anthracnose disease. Members of the Sphaeropsidales cause cankers and rots (fruits). The leaf spots are caused by members of the Moniliales.

9.5 Control measures

Scientific information gained on symptoms, etiology and mechanism of development of a particular disease is essential to devise control measures. Several factors like the kind of pathogen, the host, the host-pathogen interaction and many others are involved in devising control measures and this varies from one disease to the other. Generally, plants are treated as populations since the purpose is to save a population rather than individuals. Rarely, individual plants like certain trees or ornamentals are treated individually.

Control methods can be classified as follows: 1. Regulatory; 2. Cultural; 3. Biological; 4. Physical and 5. Chemical

1. Regulatory Methods

This method is mainly aimed at excluding a pathogen from a host or from certain geographical area. This is attained by keeping a pathogen away from the plant by preventing spread of pathogen through import of plants and seeds.

There are federal and state laws that regulate the conditions under which certain crops may be grown and distributed between different states as well as different countries. Such restrictions are implemented by means of quarantines, inspections of plants in the fields or warehouses or by eradication of certain host plants. Plants are sometimes grown on an international basis for seed production. This is done in areas which are pathogen free or areas from where pathogens are excluded by unfavorable environmental conditions, like low rainfall, low relative humidity or lack of vectors. This is known as avoidance or evasion.

(a) **Quarantines and inspections :** When a new pathogen is introduced into an area where it was absent previously, it may cause more catastrophic epidemic than that caused by an already existing pathogen in that area. The reason being the plants growing in that area had no opportunity to select resistance factors specific against that introduced pathogen. Similarly, there may be lack of organisms antagonistic to or compete with such pathogens. Thus the pathogen gets a large amount of susceptible tissue to grow and multiply unchecked. Example: Downy mildew of grapes is introduced in Europe.

However, plant quarantine is not all that fool-proof since pathogens can exist in the form of resistant spores etc. even after treatment on propagative organs or seeds. Precautions taken by plant quarantine stations include growing plants under observation for certain periods, before they are released, repeated serological tests of seed lots (ELISA), nucleic acid tests involving DNA probes and polymerase chain reaction (PCR), amplification of specific pathogen DNA sequences and inspection of imported nursery stock in the growers' premises etc.

(b) **Crop certification :** There are compulsory or voluntary inspection systems in various states. Growers who are interested in producing and selling disease-free plants submit them to these agencies voluntarily for inspection and indexing, obtain a certificate of disease-free plant material and then can sell the disease-free seeds or propagules.

(c) **Crop Isolation :** This is done for *Fusarium* wilt of banana (Panama disease) caused by *Fusarium oxysporum f. cubense* where cultivation is moved to a new previously uncultivated area.

Use of healthy seeds, either early or late planting, proper sites maintaining proper distance between rows of plants, planting in well-drained soil etc. are some of the methods to prevent disease development.

(d) **Use of pathogen-free material :** Disease-free propagatory materials like seeds, tubers, bulbs, etc. of a plant will help in getting disease free crops. Such disease-free propagules can be obtained by growing plants in (I) an area free of pathogen, (2) in an area not suitable for pathogen growth or (3) in an area not suitable for the vector of the pathogen.

Exclusion of pathogen from leaf surface can be done by spraying the leaves with dodecyl alcohol which will free the leaf surface of pathogen allow O_2 and

CO_2 to pass through but not water. It prevents diseases in plants like cucumber, tomato, beet, wheat and rice from powdery mildews.

(2) Cultural Methods

This includes control of disease through cultural manipulation of plants and is achieved by elimination of pathogen, increase resistance in plants, create unfavorable conditions for development of pathogen, obtain pathogen-free propagules through tissue culture, sanitation or crop rotation etc.

(a) **Eradication of infected host:** In spite of quarantine, if a pathogen is introduced into a particular area an epiphytotic (epidemic) follows. All hosts infected or suspected to harbour the pathogen should be eradicated and burned to prevent further spread. In some crops like potatoes, all types of pathogens overwinter in tubers that are left behind in the field. They produce infected plants in the spring allowing pathogen to come above ground, from where they are disseminated. Such volunteer plants should be eradicated. In warmer areas, volunteer plants grow during periods between plantings of crop e.g. tomato, such volunteer plants get infected by several pathogens since it is a crop-free season and serve as reservoirs of inoculum causing disease when plants are cultivated during season.

Some pathogens require two alternate hosts to complete their full life cycle e.g. *Puccinia graminis triticii* black stem rust of wheat requires wheat and barberry plant to complete its life cycle. In such cases, eradication of the wild or less economically important host will disrupt the life cycle of the pathogen.

(b) **Crop Rotation :** Soil-borne pathogens that infect either one or a few species of plants can sometimes be reduced if crops other than these are planted for 3 to 4 years in the soil. This is possible in case of soil invaders i.e. when pathogen survives only on host or as saprophyte on the residues of that particular host. However, when it is a soil inhabitant i.e. when it produces long lived spores that remain dormant for 5 to 6 years crop rotation is not successful. But in such cases crop rotation can reduce population of pathogens like *Verticillium* in soil. Field can be tilled and left without planting for an year or so. In areas of severe summer this reduces the population of pathogens.

(c) **Sanitation :** Ploughing under, infected plants will help in the inoculum on infected plant parts to go underground and rot. Reinoving infected

leaves of house or garden plants, pruning of infected branches, removing of infected flowers or fruits etc. prevent pathogen from growing into healthier plants or plant parts. These infected parts should be burned. Washing of hands before handling of certain plants like tomato, disinfecting of knives used in cutting propagative parts of plants, washing soil off farm equipment before taking it from one field to the other, cleaning the walls and floor of storage houses etc are helpful in reducing infection.

Seeds or propagative materials that are stored should be properly aerated to prevent germination of spores present on the seed surface. Plants in fields as well as green houses should be spaced properly, which prevents formation of high humid conditions on the plant surface, which will prevent invasion by pathogens like *Botrytis* and *Peronospora tabacina.* Soil drainage prevents pathogens like *Pythium.*

Choice of fertilizers and soil amendments should be such that they will create changes in soil pH, which is unfavourable for development of pathogen. Pathogens like *Fusarium, Sclerotinia sclerotiorum* etc. can be reduced by flooding followed by drying. Use of composted tree bark in nurseries reduces soil-borne pathogens.

(d) **Suppressive soils :** Some pathogenic fungi develop well and cause diseases in some soil known as conducive soils. The wilt fungus *Fusarium,* the cause of 'take all' of wheat *Gaeumannomyces graminis, Phytophthora cinnamomi* the causal agent of root rot of many fruit and forest trees, *Pythium,* the damping off fungus etc. causes severe disease in conducive soils. But in suppressive soils due to the antagonistic effect of microorganisms present in soil which produce antibiotics, or lytic enzymes the pathogen is reduced thus causing mild infection. Example: *Penicillium, Trichoderma, Sporidesmium* and other organisms like *Pseudomonas, Bacillus, Streptomyces* etc. will be helpful in reducing pathogens.

Reducing the amount of pathogen inoculum through antagonistic organisms will also result in mycolysis of pathogens e.g. the phytopathogenic organism *Rhizoctonia, Sclerotium* and several others are lysed by *Trichoderma harzianum.* Similarly, *Verticillium* is lysed by *Talaromycesjlavus* and *Botrytis* by *Pichia gulliermondii.*

Competition for food, direct toxic effect and indirect toxic effect by

volatile substances like ethylene released by metabolic activities of the antagonist also reduce pathogen populations in soil.

(e) **Cultural Practices:** Avoidance of pathogen *e.g.* Seed-born diseases like anthracnose of beans caused by *Colletotrichum lindemuthianum* plants can be grown in areas of low humidity.

(3) Physical Methods

The various physical methods include temperature (high or low) dry air, unfavourable wavelengths of light and various types of radiation.

(a) **Heat Treatment :** In green houses the soil used in seed beds are sterilized by live steam or hot water. At 50°C, Oomycetes and water moulds and at 60-72°C other phytopathogenic fungi and bacteria are irradicated. Weed seeds and TMV are heat tolerant and require 95-100°C. A temperature of 82°C for 30 minutes helps in destroying most of the pathogens in soil. However, temperature and time varies with different pathogens e.g. for loose smut of wheat 52°C for 11 minutes.

(b) **By elimination of certain light wavelengths :** Spores of *Alternaria, Botrytis, Stemphylum* etc. will sporulate only when they receive light in U.V. range (below 360 nm). This can be prevented by covering green houses with special U.V. absorbing vinyl film that blocks light below wavelength 390 nm.

Besides these, drying of stored grains and fruits till the moisture content is reduced to 12%, refrigeration of fleshy plant products, U.V., X-ray and gamma rays, particulate radiation like α and β particles etc. control post harvest diseases.

(4) Chemical Methods

Chemical fungicides are used either to protect the plant or to reduce a pathogen that has already infected a plant. Those that are used to prevent infection include soil treatment, (fumigation) disinfection of glass houses and warehouses, sanitation in handling equipment and control of insects by soil treatment. Before planting, the soil is treated with chemicals. These fungicides are applied to the soil as dusts, liquid in drenches or as granules. They are mainly meant to reduce damping off, seedling blight, crown and root rots etc.

Several hundreds of chemicals have been advanced to date for crop protection as fumigants, soil treatments, sprays, dusts, paints, pastes and systemics. Some important ones are the inorganic ones.

In irrigated soil especially in sprinkle irrigation fungicide is added to water. Metalaxyl, diazoben, pentachloronitrobenze (PCNB), captan and chloroneb are the fungicides that are used.

(a) Inorganic compounds

Phosphate and Phosphonate Compounds:

Powdery mildew diseases (PMD) can be controlled by spraying cucumber and grapes with solutions of either monopotassium phosphate (KH_2PO_4) or dipotassium phosphate (K_2HPO_4). Film-forming compounds like antitranspirant polymers, mineral oils, surfactants etc. applied to plant surface before they are infected prevent number of infections. Most of these polymers are non-toxic to plants, permeable to many gases, resist weathering for at least one week and are biodegradable.

Copper compounds:

Bordeaux mixture: This name was given after Bordeaux a region of France where it was developed and used against Downy mildew (DMD) of grape. This is still the most widely used fungicide throughout the world. It is the product of the reaction of $CuS0_4$ with CaOH (hydrated lime). It controls many fungal and bacterial leaf spots, blights, anthracnoses, mildews and cankers. However, it can cause burning of leaves or russeting of fruits like apples when applied in cool wet weather. Copper is the element toxic to fungus and sometimes to plants, whereas the role of lime is primarily that of a 'safener'.

Inorganic Sulfur compounds:

Element Sulfur is probably the oldest fungicide known. As dust, wettable powder, paste or liquid they are used to control Powdery mildews, certain rusts, leaf blights and fruit rots. It may cause injury to plants in hot (above 30°C) dry weather, especially to S-sensitive plants like tomatoes, melons and grape.

Carbonate compounds:

Sodium bicarbonate and bicarbonate salts of ammonium, Potassium, and Lithium plus 1% superfine oil was found to be effective against Powdery mildews of roses, to pathogen of cucumber, black spot fungus of roses, to southern blight fungus *Sclerotium rolfsii* to grey mould fungus *Botrytis cinerea.*

(b) Organic chemicals: 4 catagories : Organic Sulfur Compounds, Aromatic compounds, Organo $P0_4$ fungicides and antibiotics.

Organic Sulfur Compounds:

Dithiocarbamates are the versatile and modern widely used fungicides. Example: Derivatives of dithiocarbamic acid; thiram, ferbam, nabam, maneb, zineb and mancozeb. Dithiocarbamates are toxic to fungi mainly because they are metabolized to the isothiocyanate radical (-N = C = S*)*. This radical inactivates the sulydryl groups (-SH) in amino acids and in enzymes within pathogen cell and thereby inhibits the production and function of these compounds.

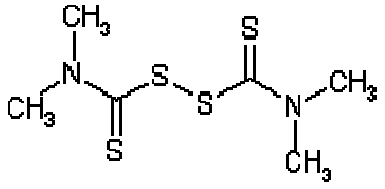

Fig. 9.2: Thiram

Thiram consists of two molecules of dithiocarbamic acid joined together. This is generally used for seed and bulb treatment for vegetables grasses and flowers. This is also used for the control of certain foliage diseases like rusts of lawns.

There is another group of dithiocarbamic acid derivatives having different molecular configurations the ethylenebisdithiocarbamate which include maneb and zineb. Maneb contains Manganese. Zineb is a multipurpose foliar and soil fungicide.

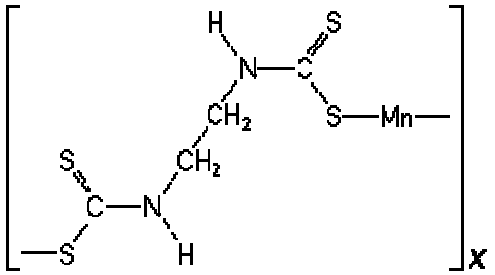

Fig. 9.3 : Maneb

2. Aromatic compounds:

Compounds with an aromatic (benzene) ring inhibit amino acids and Pentachlorobenzene enzymes with NH_2 and SH groups,e.g. Pentachloronitrobenzene (PCNB).

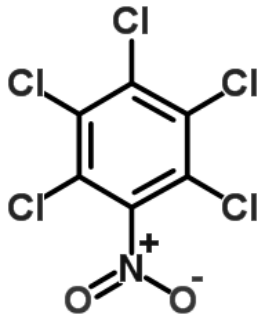

Fig. 9.4 : Pentachloronitrobenzene (PCNB)

Heterocyclic compounds: Some of the best fungicides like captan (Fig. 5) iprodione and vincozolin inhibit compounds having -NH_2 and -SH groups (amino acids and enzymes). Heterocyclic compounds are the best control of leaf spots, blights, fruit rots, seed protectants (e.g. pentachloronitrobenzene).

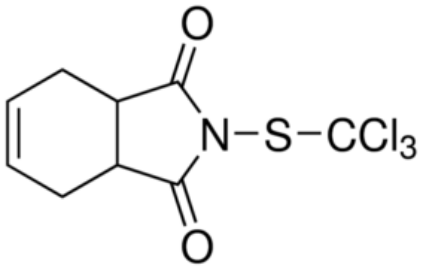

Fig. 9.5 : Captan

Systemic fungicides – Example: Metalaxyl. This is absorbed through leaf or root and circulated in the entire plant through xylem. Normally they move upward in transport stream and accumulate at leaf margins.

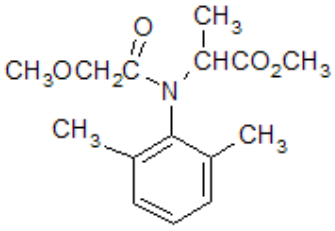

Fig. 9.6 : Metalaxyl

Acylalanines - Most important is metalaxyl used as a soil or seed treatment for Oomycetes *Pythium, Phytophthora* and several downy mildew diseases. It is water soluble and translocated easily in the plant.

Benzimidazoles: Benomyl, Carbendazim. Benomyl is the best systemic fungicide. Most benzimidazoles are converted to methyl benzimidazole on the

surface of plant. This interferes with nuclear division of sensitive fungi.

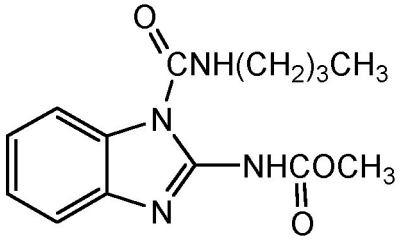

Fig. 9.7: Benomyl

3. Organo PO_4 fungicides

These include primarily fosetyl-Al sold as Aliette. It is effective against foliar, root and stem diseases by Oomycetes, *Phytophthora* and *Pythium* and downy mildews since it stimulates defense reaction and synthesis of phytoalexin against Oomycetes.

4. Antibiotics

Streptomycin, tetracycline and cycloheximide are some of the antibiotics used to control plant diseases.

5. Biological Control

Microorganisms like *Gliocladium virens* sold as Gliogard for control of seedling diseases of ornamental and bedding plants. *Trichoderma harzianum* is sold as F-stop for control of several soil borne phytopathogenic fungi. *Trichoderma harzianum / Trichoderma polysporum* sold as BINABT for control of wood decays.

Cultivation of resistant varieties is one of the most feasible methods. Use of biological organisms like hyperparasites, bacteriophages, mycophages and mycoparasites, parasites of nematodes (nematophagus fungi) and use of antagonistic plants are some of the methods used for control of plant diseases. Soil treatment is mainly used to control nematodes.

(vi) **Fumigation :** Fumigation of soil helps in controlling nematodes and certain soil borne fungi. Methyl bromide, Chloropicrin, dazomet and metam sodium are substances which are either volatised or decomposed in soil releasing volatile substances that kill fungal spores and nematodes.

(vii) **Disinfection of Warehouses :** Dried leaves and plant material of previous year should be removed and burned. The walls should be cleaned with bleach, copper sulphate solution (1 lb in 5 gallons of water)

and any other sterilising agent. It can also be fumigated with tear gas (Chloropicrin) for 24 hours.

(viii) Control of Insect and Vectors : Insecticides should be used to destroy insect vectors of fungal spores, bacteria or viruses that are phytopathogenic.

(ix) Resistance of pathogens to chemicals : *Tilletia* strain is resistant to hexachlorobenzene. *Rhizoctonia* strain is resistant to PCNB. *Venturia inaequalis* strain is resistant to do dine

9.6 Summary

Classification of Fungi

The fungi have been divided into four classes-Phyco-, Asco-, Basidio- and Deuteromycetes. This has been accepted and followed by a number of researchers. The broad classification adopted by Alexopoulos (1962) is discussed in this unit. As per this **2 subdivisions ;** Myxomycotina (Myxomycophyta, Mycetozoa) and Eumycotina (Eumycophyta or true fungi) are there. Myxomycotina has only one class Myxomycetes however subdivision, Eumycotina, is divided into nine classes: Chytridiomycetes, Hyphochytridiomycetes, Plasmodiophoromycetes, Oomycetes, Trichomycetes, Zygomycetes, Ascomycetes, Basidiomycetes, Deuteromycetes.

A particular pathogenic fungus causes a particular set of symptom on the host plant. Damping off and seedling blight occurs in germinating seeds and seedlings. The infection occurs from soil and affects the initial establishment of crop. In root rot or foot rot there is a progressive rotting (disintegration or decay) of the root system as a result of which the basal portion of the stem (foot rot) also become rotted. Wilt is loosing freshness of plants or to become flaccid when decayed root system of a plant cannot absorb enough water to replace that lost by transpiration. Downy mildews develop as downy gray colour patches on the lower side of the leaves or stem bearing sporangiophores whereas powdery mildews gives an appearance to the host as if it is dusted with talcum powder. Rust disease appears as orange –brown colour rusty patches on the host surface. Smut disease is disease is named so because of the sooty black masses of spores that they produce. In blight disease large, dark green water-soaked spots appear on leaves in wet weather, first on the lower leaves. As the spot enlarges the center is shriveled and becomes dry dark brown to black. Anthracnose is characterized by two distinct types of symptoms; one characterized by typical necrotic spot or lesion of dead tissue, the other by the hyperplastic symptoms

like a raised border around a depressed center. In leaf spot symptoms various forms of lesions are included. Besides these there are many other symptoms such as leaf curl, leaf gall or blisters, withches' broom, scab, cankers etc which are produced by specific fungi.

Disease can be identified on the basis of symptoms they produce on the host, their specific host or by spore morphology. Most of the fungi of Deuteromycotina are identified by the fruit body they produce.

Various control measures are classified as regulatory, cultural, biological, physical and chemical control. Regulatory method is mainly aimed at excluding a pathogen from a host or from certain geographical area. This includes quarantines and inspections, crop certification, isolation and use of pathogen free material. Cultural methods includes control of disease through cultural manipulation of plants and is achieved by elimination of pathogen, increase resistance in plants, create unfavorable conditions for development of pathogen, obtain pathogen-free propagules through tissue culture, sanitation, or crop rotation etc. The physical methods include temperature (high or low) dry air, unfavourable wavelengths of light and various types of radiation. Chemical methods include treatment of seed, soil or plant with the use of various chemical to control the disease. Use of biological organisms like hyperparasites, bacteriophages, mycophages and mycoparasites, parasites of nematodes (nematophagus fungi) and use of antagonistic plants are some of the methods used for biological control of plant diseases.

9.7 Glossary

- **Damping off :** destruction of seedling near the soil resulting in fall of seedling
- **Epiphytotic :** a widespread and destructive outbreak of a disease
- **Etiology :** study of living and non-living entities including environmental conditions
- **Haustorium :** a projection of hyphae into host cell which acts as a penetration or absorbing organ (pleural haustoria)
- **Heteroceous :** requiring two different kinds of hosts to complete its life cycle
- **Hyperplasia :** a plant outgrowth due to increased cell division
- **Hypertrpphy :** a plant outgrowth due to abnormal cell enlargment
- **Inoculum :** the pathogen, or its parts that cause disease

- **Lesions :** a localized area of discoloured, diseased tissue
- **Necrosis :** discolouration and death
- **Pathogen :** an entity that can cause disease
- **Primary infection :** the first infection of the plant by the **pathogen**
- **Pustules :** small blister like elavation of epidermis as spore emerge
- **Secondary infection :** infection caused by inoculum produced by primary infection

9.8 Self-Learning Exercise

Section A : (Very Short Answer Type Questions)

1. What is systemic disease?
2. Write note on epidemic diseases.
3. Give the name of causal agent of 'Tikka disease'.
4. Write the name of causal organism of club rot disease of crufers.
5. Write the name of host of *Puccinia graminis secalis.*

Section B : (Short Answer Type Questions)

1. Write note on epidemic diseases.
2. Differentiate between pre- emergence and post-emergence damping off.
3. Write a short note on identification of fungal pathogen on the basis of spores.
4. Write note on quarantine and inspection.
5. Write note on organic sulfur compounds.

Section C : (Long Answer Type Questions)

1. Explain the sympomatology of rusts.
2. Write detail note on cultural methods of disease control.
3. Write detail note on classification of diseses on the basis of symptoms.

9.9 References

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Unit-10

Fungal Diseases - II:

Plant Diseases Caused by Fungi

Structure of the Unit:

10.0 Objectives

- After going through this unit you will be able to understand:
- various plant diseases caused by fungi.
- various symptoms produced by fungi on their specific host plant.
- about systematic position and characteristic features of fungi causing a particular plant disease.
- the disease cycles of various fungal pathogens.
- about control measures of a particular disease.

10.1 Introduction

Most of the parasitic fungi cause diseases in plants. They are responsible of destroying crops worth billion of rupees. They may result even into a catastrophe if not checked and controlled. Disastrous Irish potato famine of 1945-49 caused by *Phytophthora infestans* was responsible for taking approximately one million lives. Similarly, tobacco yield was reduced by over 60% in North America and Middle East in 1962 because of the infection of *Perenospora tabacina*. In England over five million elm trees were destroyed during 1967-77 because of the infection of *Ceratocystis ulmi*. The common fungal diseases of plants are smuts, rusts, midews, blights, rots and wilts occuring on many plants of tremendous economic importance. In this unit important diseases caused by fungi are discussed.

10.2 Flag Smut of Wheat

The disease infects wheat and many grass species. The disease was first observed in Australia in 1868, since then it has been reported from all the wheat growing areas of the world including Japan, South East Asian countries, India, Pakistan, Middle East countries, Europe, South Africa and USA. The disease is prevalent in almost all important wheat growing areas of the world.

In India, Butler was the first person to report this disease from Layallpur in West Punjab (Butler, 1918). It is believed that the disease was introduced in India from Australia (Bedi, 1957). Bedi (1957) estimated that the incidence of disease in some parts of Punjab, Haryana and Himachal Pradesh is very high (upto 75%). In India the disease has now been observed in Punjab, Haryana, Madhya Pradesh, Himachal Pradesh and Rajasthan. During the late 1970s the disease caused 39-78% loss in Rajasthan (Bhatnager *et al*., 1978).

The disease in itself is not particularly damaging unless present at high levels but it can have serious consequences with regard to exporting grain or wheat products. The smut can persist in soil for long duration and under favourable conditions, it assumes serious proportions. Reduced yield due to complete loss of productivity of infected plants is the most significant effect of the disease.

Fig.10.1: Symptom of flag smut on wheat leaves

Symptoms

The disease attacks the stem, culms and leaves. Symptoms appear on all parts of the shoots but leaf and leaf sheaths are commonly affected. The infection is evident from the late seedling stage until maturity of the crop and as a rule every shoot of the plant is infected. Leaves are twisted laterally giving the appearance of hormone-type herbicide injury, begin to droop (flagging) and then wither. Soon these leaves are shed away and the whole plant is dead. Black powdery stripes appear on twisted and drooping leaves. Heading may be prevented thus culms remain sterile frequently bearing no grains. If grains are formed they are shrivelled. Affected plants are severely stunted. Excessive tillering is common and often the ears fail to emerge.

Usually systemic, the infection normally affects all tillers. Occasionally, only isolated tillers are affected. At or near heading, plants show long dark grey to black streaks, slightly swollen bands running parallel to the veins of the older leaves on the leaf blades and leaf sheaths. These streaks bear the smut sori which have a greasy appearance under epidermis in the mesophyll tissue. Sori commonly in leaves as narrow elongated blisters between the veins covered by the epidermis when young; later rupture to expose spore balls, splitting leaf to ribbons. Linear sori may also occur on the rachis, glumes and rhizomes of grasses.

The sori eventually erupt, giving the leaves a ragged appearance and exposing the black teliospores which are then dispersed, giving the plants the appearance of being covered in soot.

The disease can be detected in very early seedling stage of plant development by a characteristic twisting and bending of the coleoptile associated with subsequent formation of bleached spots on the coleoptile.

Pathogen

Urocystis agropyri (Preuss) A.A. Fisch. Waldh. 1867 (synonym U. *tritici*) belonging to subclass Ustilaginomycetes, order Urocystales is the causal agent of the disease flag smut. *Urocystis agropyri* causes flag smut on leaves of species in the family Poaceae. As a pathogen of grasses, it appears to have a wide host range and worldwide distribution. Some authorities, however, do not include the pathogen on wheat, identified as *U. tritici*, within *U. agropyri*. Rossman *et al*. (2006) place *U. agropyri* in the category of a "Threat to Major Crop Plants" and wheat and wheat straw imports are restricted in North America. The fungus has a biosynonym *Tuburcinia agropyri.*

The fungus forms small spore balls (diameter 18-52μ). Spore ball mass is powdery, dark brown to black. Spore balls are subspherical to oblong, 18-38 x 35-40 μm, composed of 1-5, usually 1-3, teliospores surrounded by a layer of smaller sterile peripheral cells. The teliospores are red-brown, smooth walled, globose to subglobose and 8-18μ in diameter. The peripheral sterile cells are hyaline to yellowish, sub-spherical to oblong, thin-walled, 3-12 μm diameter.

The teliospores germinate *in situ* to produce a short hypha (promycelium) with 3 to 4 basidiospores (sporidia) near the tip. Sporidia are cylindrical, aseptate or 1-2 septate and measure 12-15 x 3μ. The sporidia germinate while still attached to the promycelium and this forms the infection thread. The fungus strain(s) that affect wheat are specific to that crop; other strains of the fungus attack a number of grasses.

Fresh spores of the fungus are difficult to germinate. A preliminary drying favours germination.The temperature of 18-24^{Œ}C and pH 5.1-5.7 are reported favourable for spore germination.

Fig 10.2 : Spore balls encircled by sterile cells

Systematic position of pathogen

Division Eumycota
Subdivision Basidiomycotina
Class Teliomycetes
Subclass Ustilaginomycetes
Order Ustilaginales (Urocystales)
Family Urocystidaceae
Genus *Urocystis*
Species *agropyri*

Disease Cycle

The spore balls are windborne. The teliospores released from the leaves can either be blown onto grain of healthy plants, contaminating the grain or they can drop to the soil where they are very persistent. Teliospores survive in the soil or on stored seed for up to 4 years. They germinate in the soil at temperatures ranging from 40° to 86° F. Optimum germination occurs at 64° - 77° F.

When contaminated grain is sown or if healthy grain is sown into contaminated soil the teliospores germinate, producing a secondary spore type - the basidiospores. Seedling infection takes place soon after seed germination since the basidiospores fuse and the infectious hypha infects the coleoptile prior to emergence. The fungus, having penetrated the seedling then grows inside the plant, eventually producing the typical striping on the upper leaves late in the season, giving rise to a new generation of teliospores. The teliospores can survive in soil for several years so even where a break from cereals occurs, subsequent wheat crops may become infected.

Low soil moisture (10-15% of field capacity) and temperature between 50° - 68°F favours infection. The fungus over-winters as mycelium in seedlings, then systemically invades and sporulates within the upper portions of the plant.

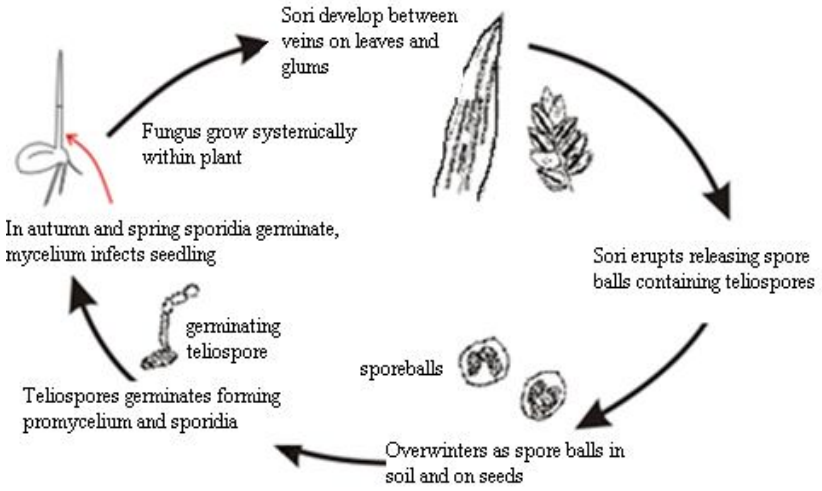

Fig 10.3: Disease Cycle of *Urocystis agropyri* causing flag smut of wheat

Control Measures

Chemical Control: Seed treatment with the systemic fungicide carboxin is highly effective. Treatment of the seed with Vitavax or Bavistin (2.5gm/kg seed) is also beneficial.

Cultural Control: Shallow planting is better than deep planting to reduce infection, probably because the seedling is in a susceptible stage for a shorter period of time. The disease is normally considered a problem on autumn sown wheat in countries with arid summers and mild winters. The disease is favoured by minimal cultivation practices which leave plant debris on the soil surface. Spring sown wheats are not affected by the disease. One to two year breaks from wheat can reduce inoculum levels and deep ploughing can help to remove to inoculum from the emerging seedlings.

10.3 Covered Smut of Barley

Covered Smut of Barley is widely distributed throughout the world. In India, it is found everywhere in areas of barley cultivation especially northern parts of India. The disease causes considerable damage to the susceptible varieties of the host. The fungus has been reported on other monocot species like *Agropyron* and *Elymus*.

Fig 10.4 : Smutted heads of barley

Symptoms

In Covered smut of barley, there are no symptoms of the disease before ear emergence. Detection of this disease is difficult, except in severe cases. Smutted heads appear stunted and the awns shrivel soon after flowering. Plant dwarfing and peduncle compaction have been observed in severe cases. The affected ear may emerge about the same time as the healthy ears but remain shorter and are usually retained within the sheath for a longer time before appearing, or may sometimes fail to emerge at all.

At ear emergence the ears seem to be normal except that the grains appear to be covered in a thin, whitish or grey membrane. Sori in spikelets, as a blackish brown coherent spore mass, at first covered by a membrane of host tissue origin (covered smut). If this is broken open it can be seen that the kernels in heads of infected plants are replaced by masses of dark brown smut spores held in place by the transparent membrane.

Persistent membrane enclosing the smut sorus until the plant is mature is the characteristic symptom of Barley Covered Smut. The membrane is relatively easily ruptured and as spores are released the symptoms become similar to those of loose smut. Frequently, masses of spores remain intact and appear in harvested grain.

Each smutted head contains many thousands of black spores, which contaminate the normal grain during harvest. The smut sori are mostly broken when the grains are threshed and then the spores get mixed with and stick to the healthy seeds. Smut sori sometimes develop as long strokes in leaf blades or in nodal tissue. Smutted heads are especially conspicuous at maturity. Infected plants are often shorter than healthy plants.

The pathogen is *Ustilago hordei (Pers.) Langerheim.* Many physiological races are known in the species (Shrivastava and Shrivastava 1974). The species of the pathogen are crop specific, mainly occurring on barley and oats. The fungus is obligate parasite, attacks cultivated barley.

Systematic position of pathogen

Division	Eumycota
Subdivision	Basidiomycotina
Class	Teliomycetes
Order	Ustilaginales
Family	Ustilaginaceae
Genus	*Ustilago*
Species	*hordei*

The smut spores or teliospores are globose to subglobose, 3.6-7.2 μ (more often 4.5 μ in diameter), light olive brown, paler on one side and smooth. The teliospores appear black in mass. The epispore is smooth. On germination, the spores give out a four celled promycelium which give rise to sporidia near the septa and the apex. Sporidia are uninucleate, ovate to oblong and may form fresh secondary sporidia by buding, especially in the presence of nutrition. Dikaryophase is attained by inter-sporidial anastomoses as well as hyphal anastomoses through which nucleus from one sporidia passes into the other. The dikaryotic sporidia germinate to produce infection thread which develops into the extensively branched mycelium in the host tissue.

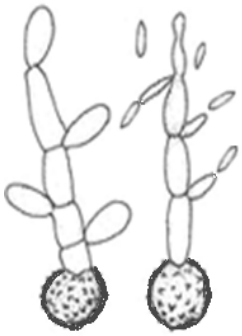

Fig. 10.5: (a) *Ustilago hordei* germination

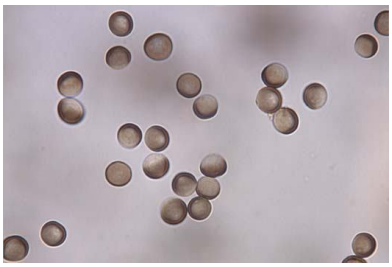

Fig. 10.5 (b): Teliospores of *U. hordei* of teliospore and budding of sporidia

Disease Cycle

Covered smut infection perennates through the externally seed borne smut spores which spread during harvest and other seed handling operations, when infected grains are mechanically broken and spread amongst healthy grain. Covered smut spores are carried on the outside of the seed and can remain viable for up to three years.

Teliospores can overwinter on barley seeds or in soil. Smutted heads are broken and crushed during threshing. They germinate at the same time as barley seeds, when adequate moisture is available. Teliospores germinate at 25-30 ° C in 16 hours, at 20 ° C in 24 hours, and at 5-10 ° C in 3 days. The teliospore germinates to form a four-celled basidium bearing four ovate to oblong basidiospores. When sporidia of opposite mating types are in close proximity, as they would be following teliospore germination, the released pheromones induce the formation of conjugation tubes, which fuse. Later the infectious dikaryotic mycelium is formed.

Primary infection occurs on the very young seedling by the sporidia. Infection occurs through the coleoptile in the first 8 days following seed germination. When infected seed begins to germinate, the spores also germinate on the seed coat of the germinating seed and penetrate the shoot of the seedling just before emergence. The mycelium advances through the host tissue and then grows in

between the plant cells just behind the growing point of the plant. As the plant grows, the mycelium maintains its position within the meristem until flower formation, when it penetrates the tissues of an ovary and forms the fungal mass in place of a seed. Spores are produced by the cells of the hyphae and these replace the kernel. Thus once the fungus enters the tissues of the head, black spore masses are produced in the head instead of normal grain. The teliospores are covered with a persistent membrane, being released at harvest.

Plants become resistant to covered smut infection after emergence and therefore, if an uninfected plant has emerged, it cannot become infected. Thus no infection occurs when the primary shoot of the host has grown out above the soil surface.

High soil moisture over a wide range of temperatures favors infection. Infection occurs at temperatures 14 to 25° C (20-24° C is optimal) in combination with a high extent of soil humidity.

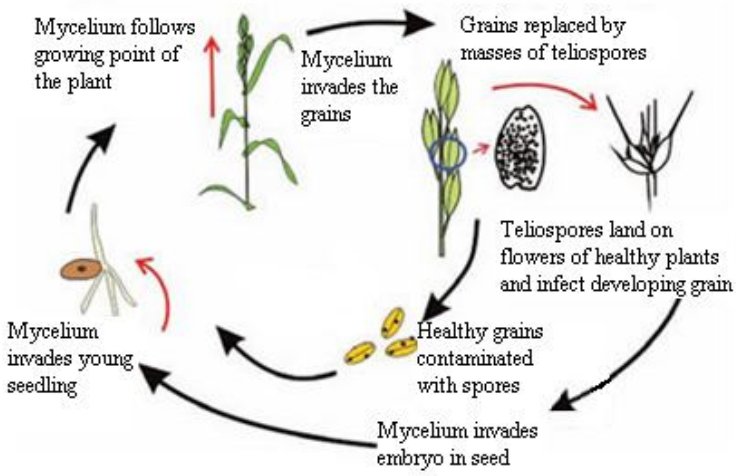

Fig. 10.5 (c): Disease cycle of covered smut of barley

Control Measures

Cultural control: Plants become resistant to covered smut infection after emergence and therefore, if an uninfected plant has emerged, it cannot become infected. Sowing disease free seed can help reduce the disease occurance.

Chemical control*:* Since the disease is externally seedborne effective and economic control can be achieved by seed treatment with protectant and systemic fungicides. Seed treatments can give excellent control. Agrosan GN (an organo mercurial) applied at the rate of 2.5g/Kg seed had been the most commonly recommended fungicide.

According to Grewal and Dharam Vir (1964) Cerasan and Agrosan 5W give complete control of the disease.

Sulphur dust (300 mesh) can also be used for seed treatment.

Treat the seed with a recommended systemic fungicides (Foliarflo-C, Vitaflo C, Vitavax 200FF, Proguard Plus, Proleaf Plus, Rancona C, Rancona Dimension).

Resistant Varieties

Varieties K 12, K18, K19, C50, C94 and C294 are moderately resistant whereas BHS 4, BM 23, LB 837 are highly resistant. In Punjab the variety C163 was found immune to the disease (Bedi and Singh, 1972).

10.4 Blast of Paddy

The disease "***Blast of Rice or Paddy"*** is also known as **"*rotten neck*"** of rice. This disease is worldwide in distribution occurring in at least 80 rice growing countries. The disease becomes more severe in humid areas, with plenty of rainfall. The disease is chiefly a foliage disease but attacks leaf sheath, rachis the joints of the culm and even the glumer also. Severe leaf infection occurs in the post transplant stage causing total distruction of the foliage. During neck infection stage half filled or totally chaffed earheads are formed. There is a tendency in the ears to break and fall off.

In India the damage to rice crop due to this disease is estimated as high as 75%. Many countries have repeated devastating epidemics due to considerable loss of rice crop.

Symptoms

It can affect all above ground parts of a rice plant: leaf, collar, node, neck, parts of panicle, and sometimes leaf sheath. Rice can have blast in all growth stages. However, leaf blast incidence tends to lessen as plants mature and develop adult plant resistance to the disease.

The symptom appears on leaves, leaf sheaths, rachis and even the glumes. Initial symptoms appear as white to gray-green lesions or spots, with dark green borders. Older lesions on the leaves are elliptical or spindle-shaped and whitish to grey centers with red to brownish or necrotic border. The most common and diagnostic symptom is diamond shaped lesions, wide in the center and pointed toward either end. On susceptible cultivars, older lesions often become light tan in color with necrotic borders. On resistant cultivars, lesions often remain small in size (1-2 mm) and brown to dark brown in color.

Fig. 10.6: Blast disease of rice caused by *Pyricularia oryzae;* infected leaves

On foliage, it first appears as 1-3 mm small, bluish flecks, which on younger leaves may enlarge considerably to several centimeters long. In large lesions, the central part becomes pale green or dull greenish green and the outer rim is of dark brown colour and water soaked in appearance. At later stage, the spots become grey or straw coloured in the centre. Similar spots appear on leaf sheaths. Lesions can enlarge and coalesce, growing together, to kill the entire leaves.

On mature inflorescence (rachis), brown to black spots are formed. Ear may also show similar spots. The most characteristic symptoms appear on culms. The neck (stem) becomes covered with grey fluffy mycelium and becomes shrivelled. Due to necrosis of tissues of neck the ear breaks down and collapses. This stage of the disease causes maximum damage.

Necks (portion of the stem that rises above the leaves and supports the seed head or panicle) are often infected at the node by the rice blast fungus and infection leads to a condition called rotten neck or neck blast. Infection of the necks can be very destructive, causing failure of the seeds to fill (a condition

called blanking) or causing the entire panicle to fall over as if rotted. Seeds are not produced when pedicels become infected, a condition called blanking.

Blast lesions can commonly be confused with Brown Spot lesions. Leaf blast lesions are usually elongated and pointed at each end, while brown spot lesions tend to be more rounded, brown in colour and have a yellow halo surrounding the lesion.

Pathogen

Blast is caused by the fungus *Magnaporthe oryzae*, formerly known as Magnaporthe grisea (T.T. Hebert) M.E. Barr). The asexual conidial stage of Magnaporthe oryzae is described by the name Pyricularia oryzae Cavara (formerly called P. grisea (Cooke) Sacc) an imperfect fungi. The imperfect stage is common in nature. The perfect stage of the fungus Magnaporthe grisea (T.T. Hebert) M.E. Barr. was reported from cultivated cereal and wild grasses in 1971. However the perithecial stge is rare in nature.

The fungus *Magnaporthe oryzae* is an ascomycete because it produces sexual spores (ascospores) in structures called asci, and is classified in the newly erected family Magnaporthaceae. The asci are foud within specialized structures called perithecia. The mycelium of *M. oryzae* is septate and the nuclei within the mycelium and spores of this fungus are haploid.

Mycelium of *Pyricularia grisea* is septate, mostly uninucleate branched hyphae. The spores, called conidia, are produced abundantly on lesions on specialized stalks, called conidiophores. From the mycelium single or fasciculate conidiophores arise, which are simple, rarely branched. They show sympodial growth and are septate, slender, denticulate and grayish in colour producing conidia at their apex (Fig. 16). Conidia are produced singly, one at a time in succession at the tips of conidiophores. The conidia are narrowly pyrifofm (pear shaped) to obclavate, 3-celled when mature with pointed or blunt apex, multinucleate. The conidium is two septate. The cells of the conidium are uninucleate. Each conidium produces several germ tubes on germination. Sclerotia may also be produced.

Under favorable conditions, the fungus sporulates in the center of the lesions on susceptible cultivars. Spores are produced on infected leaf, collar, panicle and seed, on conidiophores that extend beyond lesion surfaces; the conidiophores and spores en-masse may give the lesions a dusty grey appearance.

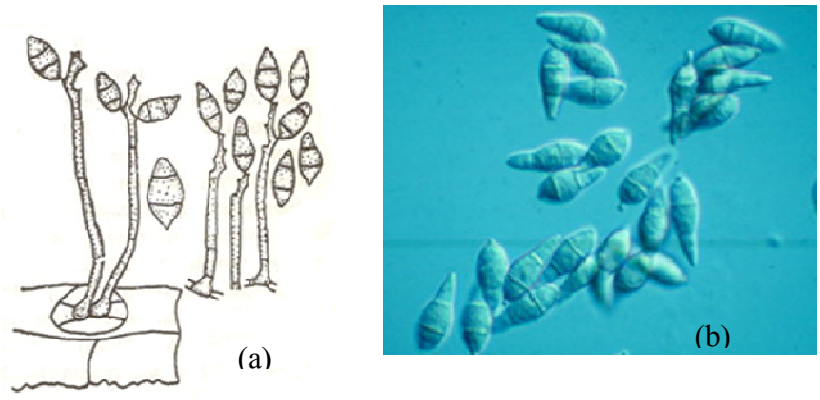

Fig. 10.7: (a) & (b) conidiophores and conidia of *Pyricularia oryzae*

The fungus is reported to produce a toxin, ***Pyricularin*** which is plant growth stimulant in low concentration but toxic to plants in higher concentration. It also produces wall-dissolving pectolytic enzymes which help in breaking down of the host cell walls. About thirty physiologic races of this fungus have been reported in India.

Systematic position of pathogen

	Anamorph	Telomorph
Division	Eumycota	Eumycota
Subdivision	Deuteromycotina	Ascomycotina
Class	Hyphomycetes	Discomycetes
Subclass	_	Sordariomycetidae
Order	Moniliales	Pezizales
Family	Moniliaceae	Magnaporthaceae
Genus	*Pyricularia*	*Magnaporthe*
Species	*grisea*	*grisea*

Disease Cycle

The fungus is reported to survive on a number of collateral hosts. In South and plains of North, the fungus rarely survives in soil due to high temperature and probably it survives on grass hosts and early-sown paddy crop. The conidia from them may be disseminated by wind to cause secondary spread. Rice plants

may be attacked at three different stages *viz.* seedling stage and rapid tillering stage (15-30 days after transplantation) during which leaves are attacked and the ear or neck- emergence stage, when neck infection occurs.

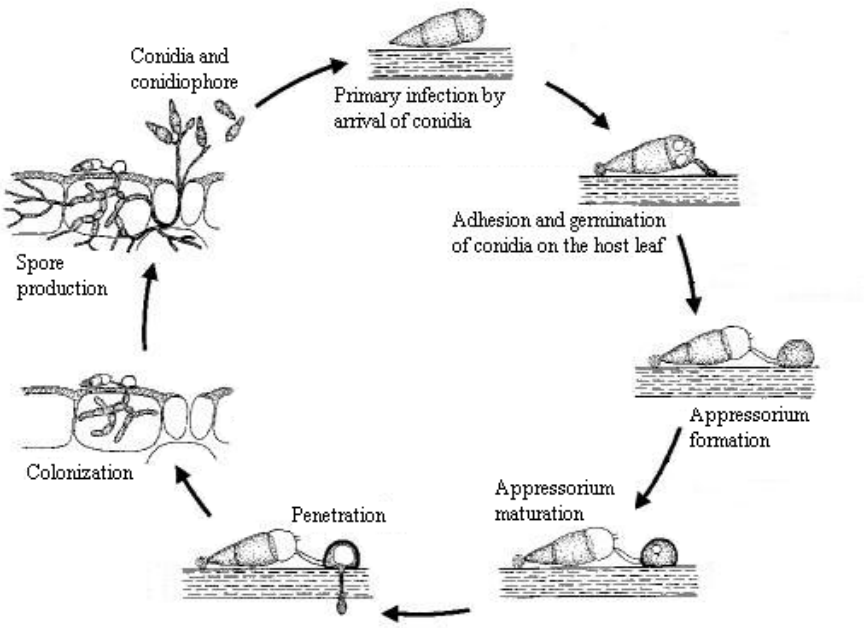

Fig. 10.8 : Disease **cycle of blast of paddy**

Addition of nitrogenous manures to soil predisposes the plants to infection. Varieties with high silicon content in leaves are normally resistant to disease. Environmental factors, particularly temperature and moisture, are important in relation to extent of damage. They affect the incidence as well as spread of disease in various ways. Conidia are not produced below 88% relative humidity and at least 90% saturation is essential for their production in large numbers.

The role of hight temperature and humidity in incidence of disease has been investigated by Subramanian (1967) and Suryanarayanan (1967). A night temperature of 20°C alternating with a day temperature of 30°C with 14 hours of light and 10 hours of darkness is most favourable for infection. Data on meteorological factors in relation to the disease have been utilised in forecasting the disease.

Control Measures

Cultural Control

(1) Field sanitation by Destruction of weed host can control the spread of the disease.

(2) Early planted crops usually show less disease than late planted crops.

Chemical Control

(1) Seed treatment: Seed treatment with organo- murcurials such as Agrosan GNis effective in controlling the externally seed borne disease. Mixture of aureofungin and copper sulphate can also be used as effective seed treatment.

(2) Foliar spray: A variety of chemicals are used. Coppesan, Blitox-50, Cupravit, Bordeaux mixture, Ceresan wet, Verdasan, Brestanol, Benlate controlled the disease effectively.

(3) Antibiotics: Most effective are Kitazin, Inazin, Blastin, Kasumin etc.

Resistant varieties

Varieties T-603 and T-141 (in Orissa), A-67, A-90, A-200, A-249 (in Maharashtra) have been found resistant. In Bihar varieties Alkulu, Kululu, GS-397, GS-480, ADT-20Suchi, Gennibera and Kamala was reported to be resistant.

10.5 Smut of Jowar

There are four types of smut of jowar *(Sorghum)* each having different symptoms and pathogen.

These are Grain smut (Kernel smut / Covered smut / Short smut), Loose smut, Head smut and Long smut.

Fig. 10.9: Smutted earheds of jowar showing

(a) Grain smut (b) Loose smut (c) Head smut (d) Long smut

1. Grain smut (Kernel smut / Covered smut / Short smut)

The first time the Covered Smut of Sorghum was registered in Russia was in 1890. Now the disease is shown on sorghum and Sudan grass everywhere in all territories of the former USSR. It is present in Africa, Asia, Australia and Central. North and South America and is coextensive with the cultivation of sorghum. In India, it is one of the most serious diseases of the crop in the states of Tamilnadu, Andhra Pradesh, Andhra Pradesh, Uttar Pradesh, Madhya Pradesh and Maharashtra. Up to 25% of the plants have been found to be affected in certain areas, and the value of the grain destroyed was computed at several millions sterling (Butler, 1918). The decrease in the yield of grains due to this disease comes to about 6 to 10 per cent.

Symptoms

The pathogen of the disease has a suppressive action on the growth and development of affected plants. This disease cannot be recognised until the earheads come out. The first signs of infection of sorghum with the covered smut are noticeable after ejection of inflorescences, when smut sori start to develop instead of the flower elements. More often all ovaries of the inflorescences are affected, but occasionally part of them remains unaffected and normal caryopses are formed. The affected ovaries turn into conical porcelain white sori (spore sacs) which contain black powder consisting of millions of chlamydospores of the fungus. Thus individual grains are replaced by smut sori. The majority of the grains in an ear are converted into sori. The sori are oval or cyclindrical, grey sac, measuring 5-15 mm in length and 3-5 mm in breadth and are covered with a tough creamy skin (peridium) which often persists unbroken up to threshing. The interior of the sorus is completely filled with the spore powder, except a slender, sometimes curved, central column of hard tissue, the columella which is hollowed into depressions at the surface. These depressions are filled with black spore mass. The columella is composed of the host tissues and consists of parenchyma traversed by fibro-vascular bundles. Sometimes the columella is branched at the tip.The peridium collapses during harvest releasing teliospores and polluting grain.

Diseased panicle does not show hypertrophy and glume shape and size remain unchanged.

Pathogen

The fungus *Sphacelotheca sorghi* (Link) Clint. is the causal organism of covered smut of sorghum. Synonyms of the fungus are *Sporisporium sorghi* Link, *Ustilago sorghi* Passerini, *Ustilago tulasnei* Kuhn, *Cintractia sorghi-vulgaris* Clinton, *Endothlaspis sorghi* Sorokin, *Sphacelotheca sorokiniana* Ciferri and *Tilletia sorghi* (Ehrenb. ex Link) Tul. & C. Tul.

The disease can only be recognized after the emergence of earheads. The diseased earheads form spore sacs in place of normal grains. The spores of the species are round to shortly oval, dark brown in mass but olive brown singly, smooth walled and 5-9μ (generally 6μ) in diameter. They are often united in loose balls which break up into individual spores when placed in water. Recent studies on grain smut caused by S. *sorghi* have indicated the presence of variations in the types of sori, varietal reaction and cultural characteristics of monosporic isolates obtained from different localities (Ranganathaiah and Govindu, 1970).

Germination of spores may take place immediately or after they have been kept dry for upto six and a half years. There are two types of germination in water. In one case a promycelium of 3 cells is formed and sporidia are budded off laterally and at the apex the apical sporidium appears as a fourth cell of the promycelium. These sporidia are spindle shaped 10-12.5x 2-3μ in size and do not budd off secondary sporidia. In other case, the promycelium directly develops into a branched or unbranched infection hypha.

Systematic position of pathogen

Division	Eumycota
Subdivision	Basidiomycotina
Class	Teliomycetes
Order	Ustilaginales
Family	Ustilaginaceae
Genus	*Sphacelotheca*
Species	*sorghi*

The fungi can grow and develop at temperatures from 10 to 32°C. The soil temperature 18-23°C and soil humidity 15-20% during the period of seed germination are optimal for the contamination of plants. The fungus belongs to the biological group heterotroph.

Tarr (1962) has tabulated the reactions of eight physiologic races on ten differential sorghum varieties. Hybridization has been recorded between S. *sorghi,* S. *reiliana* and S. *cruenta.*

Disease Cycle

It is an externally seed borne disease. The disease occurs on *kharif j*owar between September and November and on rabi jowar from December to February. The source of infection is the infected seeds. Seeds get contaminated at the time of the threshing of the grains.The threshing of diseased and healthy earheads together is the main source of infection. During threashing the sori are broken and the spores get lodged on the surface of healthy seeds where they remain dormant until the next season when they germinate with the germination of the seed.

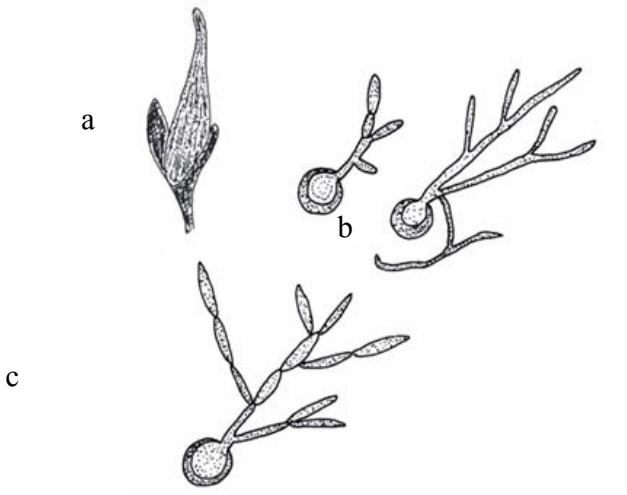

Fig. 10.10: *Sphacelotheca sorghi*

(a) smutted grain (b) germinating chlamydospores (c) budding of sporidia

Fig. 10.11: A sorus magnified and open sorus with central columella

Infection takes place between the commencement of the germination of the seed and the appearance of the seedling above the soil in the field. Infection

occurs best on slow germinating seeds checked by cold since the optimum temperature for spore germination is only 20°-30°C. Penetration is chiefly through the radicle or mesocotyl region. The pathogen of the disease in its development cycle forms teliospores / chlamydospores (wintering stage of the fungus) and sporidia. Teliospores are spherical or roundish, tinted, smooth, 4.8 to to 8.5 mm in diameter. At sprouting they form three or four cellular basidium with laterally and apically growing sporidia. Sporidia are oblong, with slightly rounded ends. Contamination of the plants occurs only during the sprouting stage.

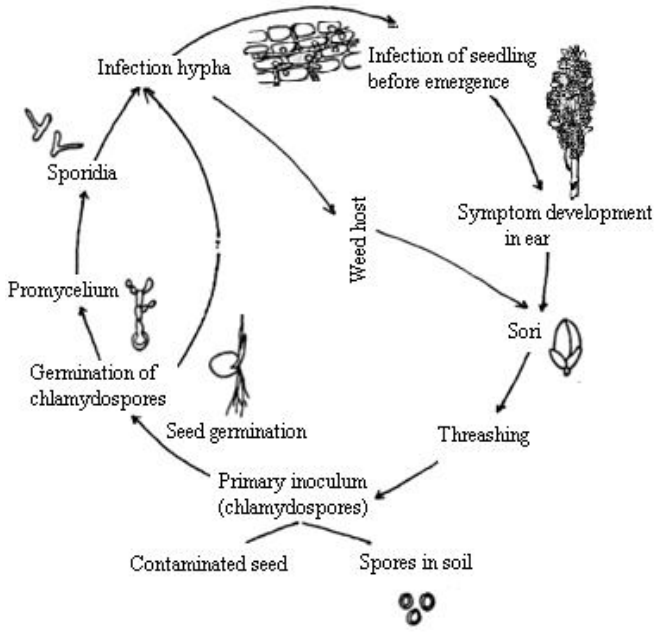

Fig. 10.12 : Disease cycle of Grain smut of sorghum

Environmental conditions influence the intensity of infection. Based on germination studies, Kulkarni (1922) concluded that temperature plays an important part in the infection and distribution of the disease. A temperature of 25°C or so is very congenial for the development of this smut. Medium to low soil moisture coupled with medium to low soil moisture are conducive to maximum infection.

The smut infects all the cultivated species of *Sorghum* and also S. *halepense* and S. *sudanense.*

Control Measures

Cultural control: Infection is mainly seed borne and occurs only at the seedling stage. Butler (1918) stated that there is field evidence to show that the spores

from a previous crop do not persist in the soil. Therefore, with the help of seed disinfection, the disease can be practically controlled.

Protective measures are cultivation of resistant varieties and hybrids, presowing seed treatment. Use of disease free seeds, crop rotation, collection of the smutted ear heads in cloth bags and burial in soil can reduce the occurrence of disease.

Chemical control: The seed treatment with sulphur (300 mesh fine) at the rate of 110 g per 27.2 kg of seed controls the disease effectively. Treatment of the seed with Captan or Thiram at 4 g/kg.

2. Loose smut of Sorghum

It is distributed in Africa, Asia, Europe and North, Central and South America. It is less common than the grain smut. In India, it occurs in the states of Andhra Pradesh, Maharashtra, Karnataka, Madhya Pradesh and Tamil Nadu. The gain as well as fodder yield may be reduced due to the effect of this smut not only on the grain but also on the plant growth.

Symptoms

The affected plants can be detected before the ears come out. They are shorter than the healthy plants by about a foot, with thinner stalks and marked tillering. The ears come out much earlier and are less compact than the healthy ones. The glumes are hypertrophied (2.5 mm in length) and the earhead gives a loose appearance than healthy. All the spikelets are infected and sori may be borne on glumes and pedicels, besides the essential organs of the spikelets. The floral bracts tend to elongate and proliferate.The sorus may include the transformed pistil and the stamens. The columella persists after the spores have been discharged. The size of the sorus varies with the variety of the host (Fig. 22)

The Pathogen

The pathogen responsible for loose smut is *Sphacelotheca cruenta* (Kohn) Potter (Synonym *Ustilago cruenta* J.G. Kühn). Its current name is *Sporisorium cruentum* (J.G. Kühn) Vánky. The spores are formed in the ovaries and floral bracts. The sorus is covered by a thin membrane which ruptures very early, exposing the spores even as the head emerges from the sheath. The covering of the sorus is made up of loosely joined rounded and grey fungal cells. In the centre of each sorus is a long conical unbranched columella of host tissue as in grain smut, but is longer and more curved. The spore mass is dark brown. The spores are globose to subglobose, light yellowish brown, wall 1 µ thick, finely

echinulate under oil immersion, 6-10 μ in diameter. They germinate to form a four celled promycelium with laterally borne sporidia. Sometimes, the promycelium may develop into branched or unbranched hyphae without forming sporidia. At lower temperature sporidia are formed while at higher temperature direct germination occur. Sporidial cultures may be obtained on Czapek's agar. This species has two sex groups and can hybridize with S. *sorghi* and S. *rdiana.* Hybridization has been recorded between S. *cruenta* as well as both *S. sorghi* and S. *reiliana.*

Systematic position of pathogen

Division	Eumycota
Subdivision	Basidiomycotina
Class	Teliomycetes
Order	Ustilaginales
Family	Ustilaginaceae
Genus	*Sphacelotheca*
Species	*cruenta*

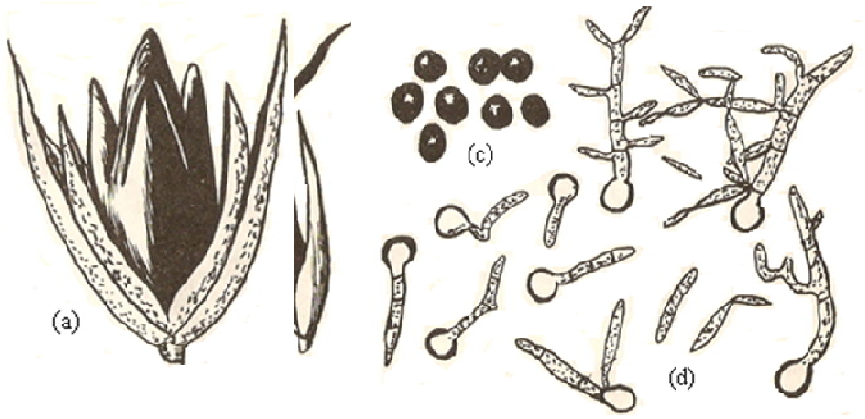

Fig. 10.13: (a) Sorus involving stamens (b) exposed columella after the spores have fallen (c & d) spores and their germination

Disease Cycle

As in grain smut S. *cruenta* is mainly externally seed borne. It is stated to be soil borne to some extent in dry soils. Spore remains viable for four years in the laboratory, but as they germinate readily in water soil transmission is unimportant. Germination of spores can occur at 8°-38°C but the optimum lies between 18°-32S°C. Spread within the crop by floral infection by air-borne spores has been observed. Low temperature and low soil moisture as well as

deep sowing favour infection. Infection occurs at the time of germination of seeds and before emergence of seedlings. Entry into the seedling takes place through the radical, mesocotyl and hypocotyls.

Control Measures:

Control measures are the same as those for grain smut of sorghum.

3. Head smut of Sorghum

It is found in Africa, Asia, Australia, Europe and North, Central and South America. The disease is not very common in India and is economically not important. In India, it is reported from Andhra Pradesh, Karnataka, Bombay, Madhya Pradesh, Uttar Pradesh, Punjab and Tamil Nadu.

Symptoms

The disease is not noticeable by a difference in either the size or the growth of the plant. The entire inflorescence is converted into a big sorus, about 10-13 cm in length and 4.6 cm in breadth. Thus the inflorescence is invariably totally destroyed in the infected plants. The sorus consists of the conductive elements of the suspect surrounded by the spore mass and covered by a whitish grey membrane of fungal tissue, which ruptures, before the head emerges from the boot leaf to expose a mass of brown smut spores. Spores are embedded in long, thin, dark colored filaments which are the vascular bundles of the infected head.

Pathogen

The pathogen responsible for heet smut of sorghum is *Sphacelotheca reiliana* (Kuhn) Clinton. Biosynonym *Sorosporium reilianum* (Kuhn) McAlpine and *Sporisorium reilianum* (Kuhn) Langdon and Fullerton.

The sorus is composed of loosely united spores and the conductive tissues of the suspect which is initially enclosed by a fragile fungal membrane. The reddish-brown to black chalmydospores are finely echinulate, irregular to spherical and 9·14 μ in diameter. The spore mass is powdery and is quickly dispersed to expose a tangled mass of vascular strands of the host or sometimes a single central columella. Germination of chalmydospores results in development of a promycelium and sporidia.

Systematic position of pathogen

Division	Eumycota
Subdivision	Basidiomycotina
Class	Teliomycetes
Order	Ustilaginales
Family	Ustilaginaceae
Genus	*Sphacelotheca*
Species	*reiliana*

Disease Cycle

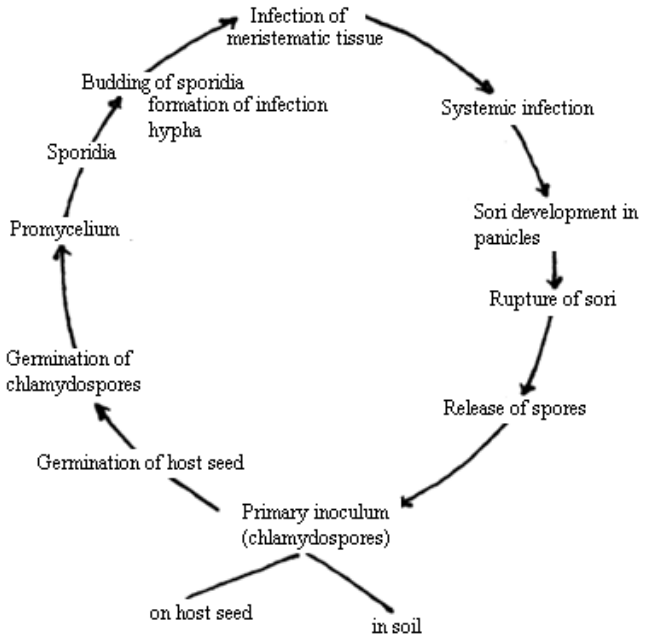

Fig. 10.14: Disease cycle of head smut of sorghum

The pathogen may be externally seed borne but the major source of infection is soil borne inoculums. Spores present in the soil cause infection. The smut spores remain viable for two years. The survival of the spores in the soil depends on soil temperature and soil moisture. Only young plants are susceptible. The fungus becomes systemic. Infection is favoured by soil temperatures of 21°-28°C. In dry soil the spores remain viable for a considerable period, at least until the following crop season. Thus crops raised in clay loam soils which have higher soil moisture than silty or sandy loam soils show less disease incidence.

Control Measures

Cultural Control:

Crop sanitation can reduce the inoculum potential. Since only few plants are affected in the field it is possible to locate and destroy the affected ear before they shed the spores.

Crop rotation is of primary importance to control the edisease.

Frequent irrigation after sowing has been found to reduce disease incidence.

Chemical Control:

Seed treatment: Disinfection of seed has been found ineffective in south India while in the USA it has given only the partial control of the disease.

Resistant varieties are available and most of these are also resistant to covered smut.

4. Long Smut of Sorghum

This has been reported in Africa (widespread) and Asia (China, India, Iraq, Israel, Pakistan, Turkey, former USSR). In India, it occurs in Tamil Nadu, Karnataka, Andhra Pradesh, Maharashtra, Madhya Pradesh and Uttar Pradesh. Losses due to this disease are usually slight. Its occurence is sporadic and confined to a few grains in an ear.

Symptoms

The presence of long smut can be discovered only by a close examination of the ears in the field. This disease is normally restricted to a relatively a small proportion of the florets which are scattered on a head. In only a few grain smut sori develop which are scattered sporadically throughout the ear. Each sorus is surrounded by healthy grains. Sori are more or less cylindrical, elongated, with tapered ends, slightly curved up to 4.0 cm long, 0.5-1.0 cm wide, with a relatively thick creamy-brown covering membrane, the peridium. The peridium splits at the apex to release black mass of spores balls among which are found several dark brown filaments which represent the vascular bundles of the infected ovary.

Pathogen

The pathogen responsible for the long smut of Sorghum is *Tolyposporium ehrenbergii* (Kuhn) Pat. Its biosynonym are *Sorosporium ehrenbergii* J.G. Kühn and *Anthracocystis ehrenbergii* (J.G. Kühn) McTaggart & R.G. Shivas.

Systematic position of pathogen

Division	Eumycota
Subdivision	Basidiomycotina
Class	Teliomycetes
Order	Ustilaginales
Family	Anthracoideaceae
Genus	*Tolyposporium*
Species	*ehrenbergii*

The spore mass is granular, black, intermixed with shreds of host tissue. The spores of the pathogen remain united in solid balls. Spore balls are many spored, permanent, rather irregular in size and shape, globose to elongated, dark brown in patches, opaque, 45 µ or less, with a maximum diameter of more than 200 µ. The spores at the surface of the ball are dark brown with the free surface having papillae. The inner spores are pale in colour, smooth, globose to subglobose or angular and 10-15 µ in diameter. The exposed surface of the spores is covered by flattened echinulations.

Spores do not have dormancy period. They germinate *in situ* by the formation of an elongated promycelium which is frequently branched. Sporidia are numerous, single or in chains. Sporidial cultures may be obtained in 1 % potato dextrose agar or carrot agar (Kamat, 1933).

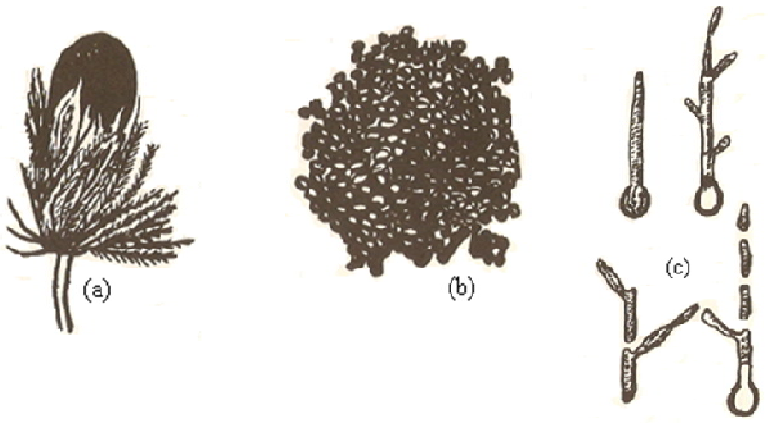

Fig. 10.15: *Tolyposporium ehrenbergii*

(a) single sorus (b) spore ball (c) germinating spores

Disease Cycle

The spore balls are soil borne. Germination of spores can occur at the temperature of 15°-36°C with optimum at 28°C.

Primary infection occurs by air borne sporidia. The spores in the soil may germinate to produce clusters of sporidia which become air borne and cause primary infection. It is believed that floral infection occurs by means of sporidia which after producing systemic mycelium enter the floral parts, producing sori about 12 to 15 days later thus expressing the disease in the head. The ovary is converted into smut sorus in the same season. Continuous cultivation of sorghum in the same field is said to increase the incidence of disease.

Secondary infection may also take place through the spores released from the smutted ears in the field.

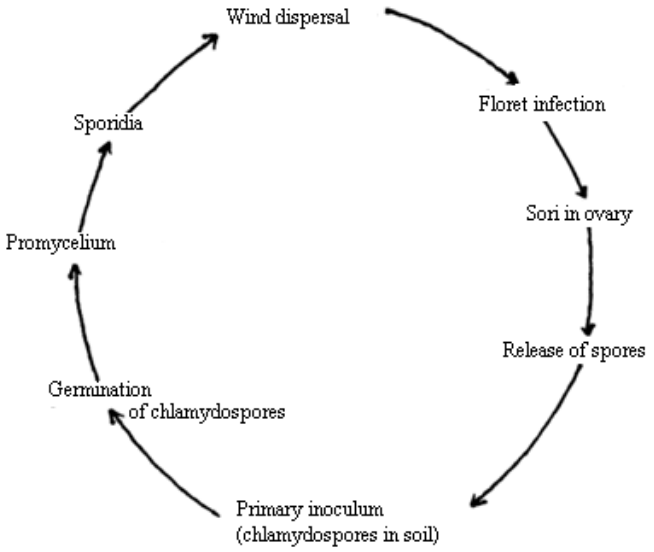

Fig. 10.16 : Disease cycle of long smut of sorghum

Control Measures:

Cultural Control:

The use of healthy seed and sanitary practices to reduce the infection potential are advisable. Crop rotation and field sanitation to kep down the buildup of inoculums should be practiced. Early sowing of the crop reduce disease incidence.

Chemical Control:

Since the inoculum is air borne seed treatment is of no use.

Table 10.2: Characteristic of four types of smut of *Sorghum*

Character	Grain smut	Loose smut	Long smut	Head smut
Pathogen	*S. sorghi*	*S. cruenta*	*T.ehrenbergii*	*S.reiliana*
Host	Stunted, heading premature	Not stunted, heading normal	Not stunted, heading normal	Not stunted, heading premature
Ear infection	All or most grains smutted	All or most grains smutted	Very few grains are infected	The entire inflorescence is converted into a big sorus
Site	Ovary	Ovary	Ovary	inflorescence
Sori	Small	Small	Long	Very large
Membrane	Rather tough and persists	Ruptures easily	Relatively thick membrane	Ruptures easily
Collumella	Short collumella present	Long collumella present	Collumella absent but 8-10 vascular strands present	Collumella absent but a network of vascular tissue present
Spores	6-7μ, surface apparently smooth	7-8μ, minutely echinulate	10-15μ, spore balls, free surface papillate	9-14μ, conspicuously echinulate
Viability of spores	More than 10 years	About 4 years	About 2 years	Upto 4 years
Spread	Externally seed borne	Externally seed borne	Air borne	Soil borne seed borne

10.6 Red Rot of Sugarcane

Red rot of sugarcane was first reported from Java (now Indonesia) by Went in 1893. During 1895-1900 the disase assumed epidemic proportion in the Godavari delta of Andhra Pradesh, India (Barber 1901). Butler (1906) published a detailed account of this disease from PUSA, Bihar and gave it the name 'Red rot'. Red rot is a serious disease of sugarcane in tropical and subtropical parts of the world. In India, the disease attacks standing canes in the field often in epiphytotic form and cause huge losses to the cultivators and sugar industry. Serious epiphytotics have occurred in U.P. and Bihar during 1939-1940, and 1946-47. Significant local losses due to the disease occur almost every year in Haryana, U.P and Bihar. In U.S.A. the disease is not serious on standing crop but causes appreciable losses through seed rot and inhibition of growth.

Fig. 10.17 : Symptoms of red rot of sugarcane: reddening of the pith with transverse blotches

Symptoms

The disease appears on all above-ground parts, but stems and midrib areas of leaves are most affected. In the early stages it is difficult to recognize the diseased plant in the field. First symptoms are seen after the rainy season when plant growth stops and sucrose formation begins. In early stage, drooping of leaves and the loss of their colour can be seen in the field. Later, the cane also starts developing the symptoms. The canes are completely rotted within and the rind loses its natural bright colour, becomes dull, longitudinally wrinkled and shrinks at the nodes. By this time the leaves begin to wither at tips and finally the entire top wither completely and droop. The sugarcane tissue reacts vigorously to the presence of the fungus and some kind of reaction sets in the host in advance of the hyphal invasion. The protoplasm changes in colour and a gummy dark red material oozes out of the cells filling the intercellular spaces.

The soluble pigment present in this ooze is absorbed by the cell walls producing the characteristic red rot appearance. Split open stems show longitudinally reddened tissues of internodes usually at the base. Reddening is most severe in the vascular bundles extending to pith. In very advance stages of the disease, the red colour may be replaced by dirty brown and white bands may not be conspicuous. Cavities filled with greyish or white mycelium are found in the pith. The juice often gives a bad odour (alcoholic odour) due to conversion of sucrose into glucose and alcohol as a result of enzymatic activity of the pathogen. Late in the season, minute velvety dark dot-like structures, acervuli develop near the nodes of diseased canes.

On the midribs of leaves, infection originates as a dark redish area which elongates rapidly forming blood-red lesions with dark margins (Fig. 27). In old lesions, the centre becomes straw-coloured, and after formation of conidia, these become covered with a powdery mass.

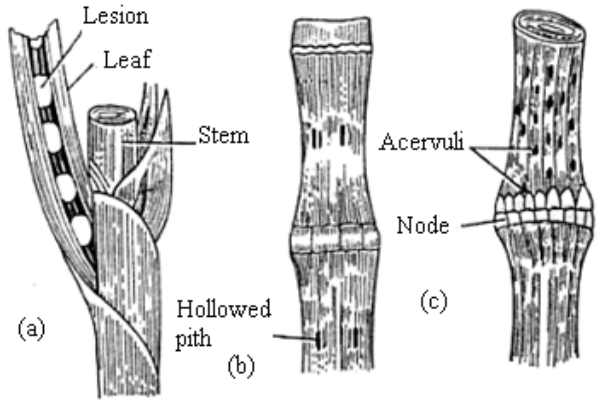

Fig. 10.18 : Red rot of sugarcane caused by *Colletotrichum falcatum*
(a) lesions on midrib of leaf (b) lesions on stem (c) acervuli

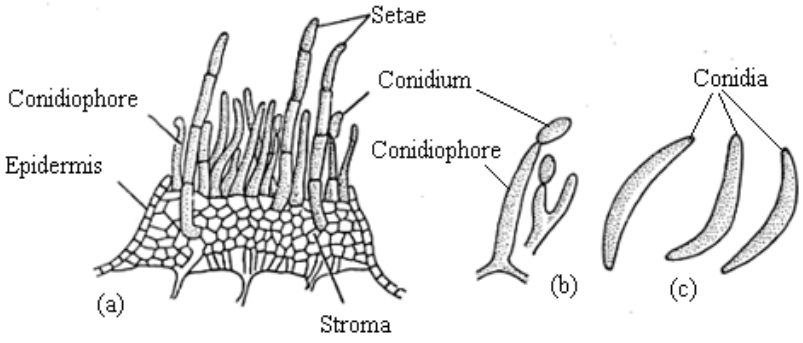

Fig. 10.19 : Somatic and reproductive structures of *Colletotrichum*
(a) Acervulus (b) Conidia on conidiophores (c) Conidia

Pathogen

The disease is caused by imperfect fungi, *Colletotrichum falcatum* Went. Its telomorph or perfect state (ascigerous state) is *Glomerella tucumanensis* (Speg). Earlier telomph of *Colletotrichum falcatum* was identified as *Physalospora tucumanensis*. Later it was identified as a saprophyte often associated with decaying leaves of sugarcane in the field.

The mycelium once inside the host grows rapidly. Hyphae are slender, branched, septate, colourless, inter-and intracelular. The hyphae in lumen of vascular bundles grow out rapidly. Eventually the sugarcane tissues react vigorously to the presence of fungus. The host cell protoplasm changes its colour and a gummy dark substance oozes out of cells filling the intercellular spaces. The soluble pigment present in this ooze is absorbed by the cell walls producing the characteristic red rot appearance.

After growing for a period within the host tissues the hyphae produce a large number of chlamydospores in the pith.

Systematic position of pathogen

	Anamorph	Telomorph
Division	Eumycota	Eumycota
Subdivision	Deuteromycotina	Ascomycotina
Class	Coelomycetes	Pyrenomycetes
Order	Melanconiales	Sphaeriales
Family	Melanconiaceae	Polystigmataceae
Genus	*Colletotrichum*	*Glomerella*
Species	*falcatum*	*tucumanensis*

Disease Cycle

Though the pathogen is mainly sett borne, the fungus may also persist in the soil on diseased clumps and dry leaves left in the field after harvest. It means that the planting material harbour the fungus and thus perpetuate the disease from season to season. The pathogen affects the cane plant from germination. Once the fungus gains entry into the host, it grows rapidly producing inter and intracellular septate mycelium. The soil borne fungus may also enter the healthy setts through cut ends, and cause early infection of the shoots. After

growing for a period within the host tissue the hyphae produce a large number of chlamydospores in the pith. These chlamydospores can survive in soil for a long time.

After the primary infection the fungal hyphae collect beneath the epidermis and form a stroma or acerculus of densly packed cells. Usually 4 sepate and 100-200 µ long hair like setae arise in and around the acervulus. These acervuli push their way through the epidermis exposing the conidia which are borne on small conidiophores. Conidiophores are small, aseptate whereas conidia are one celled, usually falcate (sickle shaped), hyaline and densely granular, possess large oil globule in the centre, frequently guttulate, 20 - 80µ x 5-7µ in size. Acervuli are formed on surface of rind as minute structures.

Secondary spread in the field may be through irrigation water, cultivation tools and wind borne inoculum. Conidia are disseminated by wind, rain or water of irrigation, raindrop splash and insects. They may settle on the leaves, germinate by producing germ tube soon in presence of water and may cause secondary infection and spread the disease to new healthy tissues of host plant. This germ tube on coming in contact with any hard surface such as soil particle produces appressorium. The appressoria are brown, oval, round or irregular in shape measuring average 15x13µ in size. This appressorium become thick walled and function like a chlamydospore. These chlamydospores can survive in soil for a long time. After growing for a period within the host tissue the hyphae produce a large number of chlamydospores in the pith.

The pathogen mainly spreads during the rainy season and if environment favours it can wipe out entire sugarcane plantation.

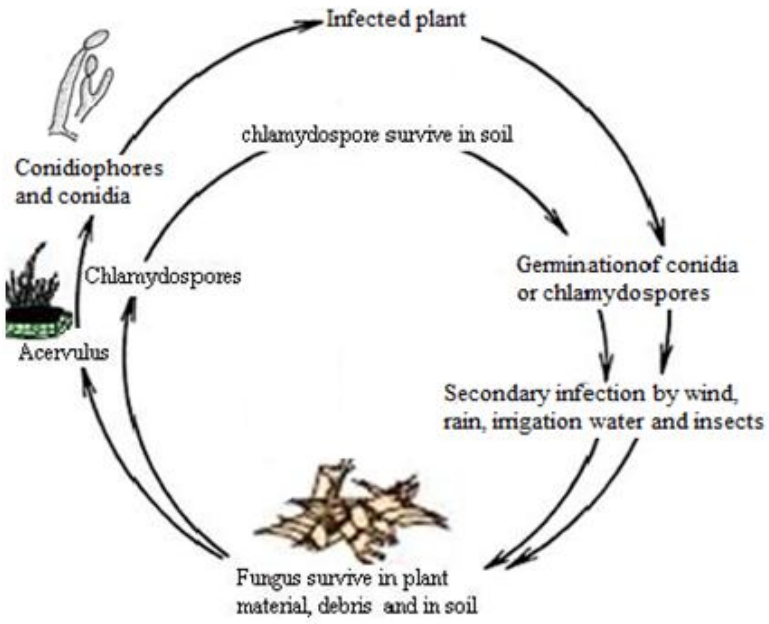

Fig. 10.20 : Disease cycle of Red rot of sugarcane

The perfect state or perithecial stage of the fungus was first reported in India by Chona and Srivastava in 1952 in cultural conditions. Later in 1953 it was reported by them from nature also at Delhi. Perithecia of *Glomerella tucumanensis* are globose, measuring 150-300µ in diameter, ostiolate, superficial with the bottom embedded in the host tissue. Asci are numerous, hyaline, clavate measuring average 56x9µ. Numerous hyaline paraphyses are present along with these asci. Each ascus contains 8 ascospores arranged basirtaly. They are single celled, hyaline, elliptical and measures average 20x 6 µ.

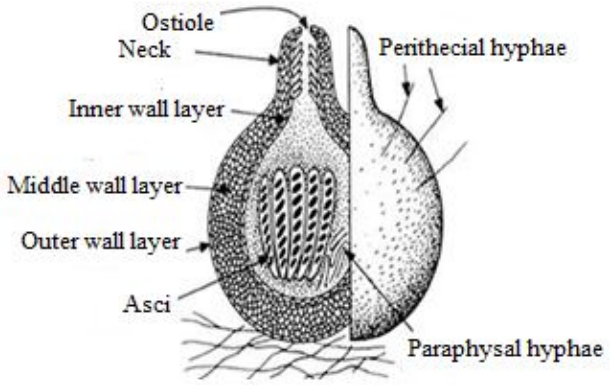

Fig. 10.21: Perithecia of *Glomerella tucumanensis*

According to the available evidence, seed sets from diseased canes are the chief means of survival and annual recurrence of the disease. When such sets are planted, shoots are invariably attacked. Once the fungus establishes, secondary spread occurs by conidia. Ratoon crops may also serve as a source of perennation and inoculum multiplication. Acervuli are also produced on the patches developing on midrib of leaves. High humidity, water-logged conditions, lack of proper cultural operations, continuous cultivation of single variety in a given area, help in appearance of disease and build up of inoculum.

Control Measures

Cultural Control

(1) Field sanitation. (2) Use of healthy sets. (3) Ratooning should be discouraged

(4) Crop rotation.

Resistant varieties

The disease is currently managed through host resistance as fungicides do not get adequate entry (conc. is less to effect eradication) into the stalk.

A large number of such varieties are evolved. A few among more recent varieties recommended as resistant to disease are, Co 846, Co 951, Co 975, Co 1007, Co 1148, Co S 109, Co 561.

Biocontrol

Application of biocontrol agents like *Trichoderma* has shown promise in the containment of the disease through antibiosis and induced systemic resistance.

10.7 Flax Rust

This is an important disease of linseed (flax). In our country also this results into considerable loss in the yield of this oilseed crop. The rust appears in February March and at the harvest time most affected plants give fired appearance in the field. Within a few days of its appearance, most of the linseed fields in a particular locality get rapidly affected. The loss in the crop yield results from reduced photosynthetic area of leaves. There occurs reduction in the seed yield and injury to fibre in flax. Oil content of the seed is also affected. In some cases 16-100 % loss in oil yield has been reported. In India, linseed being the major oilseed crop in many parts of the country, the disease has been of particular significance.

(a) (b) (c)

Fig. 10.22: Symptoms of Linseed rust (a) infected leaves bearing uredosoi or uredia (b) uredia on flax boll (c) Black telia on stem and leaves

Symptoms

The symptom of the rust usually appears in February or later although its occurrance as early as November had also been reported from central India. Disease is conspicuous in the field due to bright orange colour of the infected parts, which is due to uredia. Uredia develop on both surfaces of leaves and other aerial parts. Uredia are reddish yellow. The young uredia may be surrounded by a chlorotic zone, which later involves whole leaf that now dies prematurely. On leaves uredia are small and almost round but on stem they are irregular and elongated. Toward the harvest time affected plants get a fired appearance due to the formation of telia on the plant. Since leaves are shed early telia develop mainly on stem. They develop on leaf if infection is late and leaves are intact on the plant. Telia are most common and pronounced on stem. Telia are reddish brown in color. Very often, telia on stem are surrounded by orange yellow uredia. The telia on stem do not break the epidermis and remain covered appearing glossy.

Pathogen

The disease is caused by *Melampsora lini* (Pers.) Lev. or *Melampsora lini-usitatissimi* Kuprev, an autoecious rust i.e. all stages occuring on the same host, linseed plant. The pycnia and aecia stages are not naturally found but are obtained on plants in experimental cultures during 1940's. The host range of the fungus extends over a dozen species of genus *Linum*. In India wild linseed

(*Linum mysorens*) has been reported to harbour the fungus.

Systematic position of pathogen

Division	Eumycota
Subdivision	Basidiomycotina
Class	Teliomycetes
Order	Uredinales
Family	Melampsoraceae
Genus	*Melampsora*
Species	*lini*

Disease cycle

Pycnia, if formed are pale yellow, flask -shaped and subepidermal on the leaf and stem. They may also be diffused. They produce minute and oval to globose pycnospores. The aecia are orange-yellow, scattered on the underside of leaf, and also on stem. They lack a peridium *(caeoma* type) and have no paraphyses. Aeciospores are polygonal with thin verrucose outer wall.

Uredia are usually circular in shape, scattered or in group on both the surface of the leaves. They are elongated on stems. The uredospores are stalked, ovate, measuring 15-25x 13-18 μ in size and provided with spines on the surface (exospore). There are numerous capitate paraphyses intermingled with uredospores in each uredosorus.

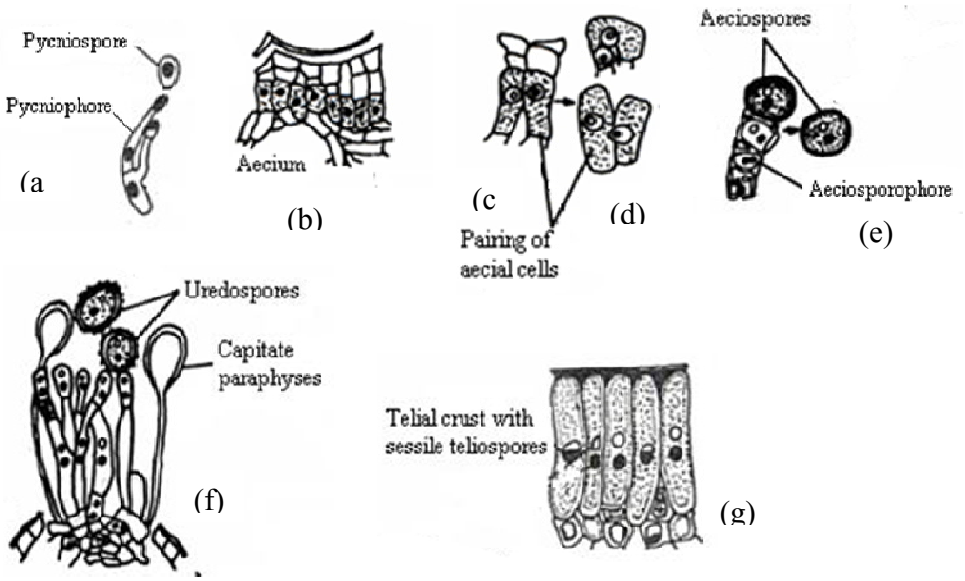

Fig. 10.23: ***Melampsora lini***

(a) pycniophore and pycniospore (b) aecium (c) paired cells at the base of aecium (d) fusion of the cells (e) chain of binucleate aeciospores (f) a part of uredium with uredospores and capitate paraphyses (g) a part of T.S. infected stem showing telium and teliospores

The telia are irregularly elongate, sub-epidermal and form solid crust on the stem. Teliospores are reddish brown and arranged in a palisade layer. They are sessile, cylindrical, one celled, 8-20x46-80 μ in size. Teliospores germinate without any dormancy period.

The uredospores of *M. lini* are sensitive to heat and are not able to survive the summer heat of India (temperature above 34-50°C). Uredospores germinate over a wide range of temperature ranging from 3 to 30°C with optimum temperature for gemination is 16-28°C. They are perhaps unable to survive during summer months in the plains. Telia are also said to be killed by these temperatures during summer. However, in hills they may survive on plant debris.

The pycnial and aecial stage were reported by Prasada (1940) on plants in experimental cultures at Shimla. Pycnial morphology and mating types have been described by Lawrence (1988). Where formed, the pycnia are pale yellow, flask shaped and subepidermal on leaf and stem. The Pycniospores are minute and ovate to globose. The aecia are orange yellow, scattered on the undersurface of the leaf, and also on the stem. They are without peridium and

have no paraphysis. The aeciospores are polygonal, 17-27μ in diameter, and have a thin verrucose outer wall.

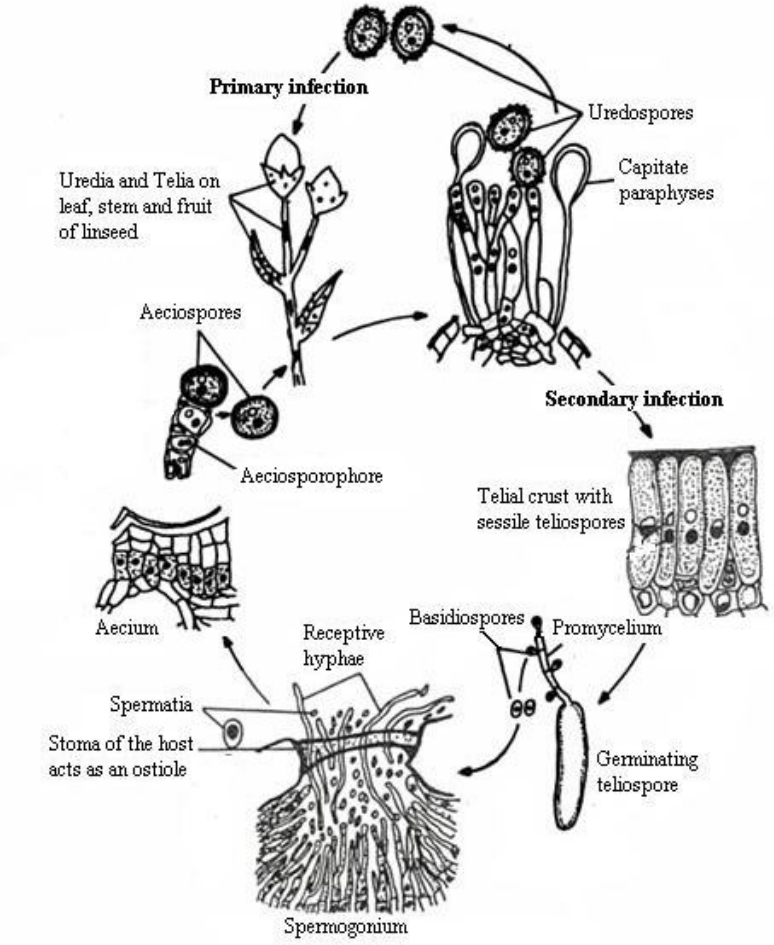

Fig: 10.24 Disease cycle; rust of linseed

In India linseed is a winter season crop and the diseased tissues are exposed to summer heat, the telia do not play any role in perennation of the pathogen from one crop to the next. Uredospores are also destroyed during summer. Perhaps the rust survives on linseed and other hosts in uredial or telial stage at high altitudes 4,000-6,000 ft. From there the uredospores might be blown down to plains by wind.

Temperature between 15-21°C is most conducive for infection and disease development.

Control Measures

Cultural Control

Sanitary precaution is useful. Diseased plant debris and weed hosts should be destructed.

Resistant varieties

Use of resistant varieties is only sure method for the management of the disease. In India, the cultivar NP (RR) 9, 10, 56, 218 etc has been cited as resistant varieties. Cultivars like LC-216, LC-255, LC-256 are resistant to all races of the pathogen prevalent in the hills.

Chemical Control

In the hills seed treatment with chemicals to inactivate the telia is suggested. Fungicide sprays and application of borax are found useful but these methods are not economical so not recommended on field scale.

10.8 Early Blight of Potato

This is a common and destructive disease of potato in India and other potato-growing areas of the world. In India the disease may cause upto 40% loss in yield of tuber. The disease appears on young plants, much earlier than late blight. This disease occurs in cold as well as warm areas, thus being serious in hills as well as plains of the country.

Symptoms

Symptoms of early blight occur on foliage and tubers of potatoes. Initially the disease appears as small, isolated, scattered pale1-2 mm brown spots on leaflets. These spots later turn to deep greenish blue colour due to mycelial growth. Under conducive environmental conditions the lesions will enlarge and are often surrounded by a yellow halo. Lower leaves are attacked first and the disease then progresses upwards. The characteristic concentric ridges with target-board effect then develop in the necrotic tissues of leaf. There is a narrow chlorotic zone around the spots. This so-called “bullseye” type lesion is highly characteristic of early blight severe attack spots coalesce to form larger necrotic areas. The leaves shrivel and fall down.

In dry weather the spots turn hard and the leaves curl. In humid weather the spots coalesce and big rotting patches may appear. Tubers may also be affected showing brown to black necrotic lesions on skin. Necrosis occurs due to the toxin - alternaric acid produced by the pathogen.

Fig. 10.25: (a) **Symptoms of early blight on Potato leaves, (b) on tuber**

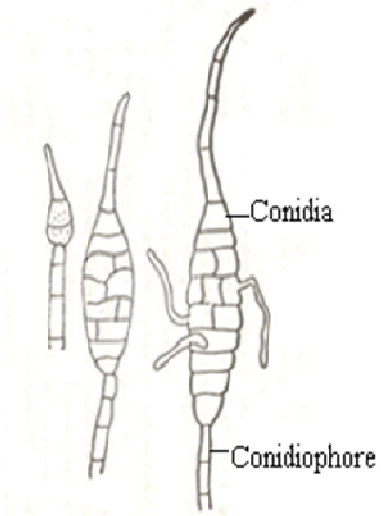

Fig. 10.26: Conidiophores and conidia of *Alternaria solani*

Pathogen

The disease is caused by *Alternaria solani* (EII and Martin) Jones and Grout. Host of the fungus is **Potato (***Solanum tuberosum***).**

Other species of *Alternaria* can also cause early blight on potato. These include *A. tomato* and *A. alternata.* This is an imperfect fungus with no sexual stage reported but later their sexual stage is reported as *Pleospora infectoria*.

The mycelium consists of septate, branched, light brown hyphae which become darker with age. Hyphae first are intercellular later penetrates the host cells and become intracellular. Conidiophones emerge through the stomata from the dead centre of the spot. They are relatively short, 50-90µ in size, dark and geniculate. Each conidiophore develops single conidia apically and singly.

The conidia are 120-296 x 12-20µ in size, beaked, muriform, dark brown and septate. They are borne singly on conidiophores from a bud formed by the

apical cell of the conidiophore. Five to ten transverse as well as few longitudinal septa are present in each conidium. In moist weather they germinate readily and 5-10 germ tubes develop from single conidium. There are several races and strains of this pathogen.

The fungus produces chlamydospores also which are formed by the differential swelling of the hyphal cells in the curly mycelium. They are single, in chain or in clusters, one celled thick walled, dark brown and measures 8-15μ in diameter.

Systematic position of pathogen

	Anamorph	Telomorph
Division	Eumycota	Eumycota
Subdivision	Deuteromycotina	Ascomycotina
Class	Hyphomycetes	Discomycetes
Subclass	-	Dothideomycetidae
Order	Moniliales	Pleosporales
Family	Dematiaceae	Pleosporaceae
Genus	*Alternaria*	*Pleospora*
Species	*solani*	*infectoria*

Disease Cycle

The mycelium remains viable in dry infected leaves for one or more year. Mycelium and conidia are said to survive in the soil on diseased plant debris. These are the sources of primary infection in next season. Tubers contaminated with mycelium or conidia are another source of primary inoculum. Infection of the lower leaves first takes place through conidia formed on soil. Infection occurs through stomata but direct penetration may also take place.

Secondary spread of disease occurs through conidia developing on primary spots. These conidia are disseminated by wind, water and insects. The disease assumes a severe state when season begins with abundant moisture or frequent rains followed by warm and dry weather.

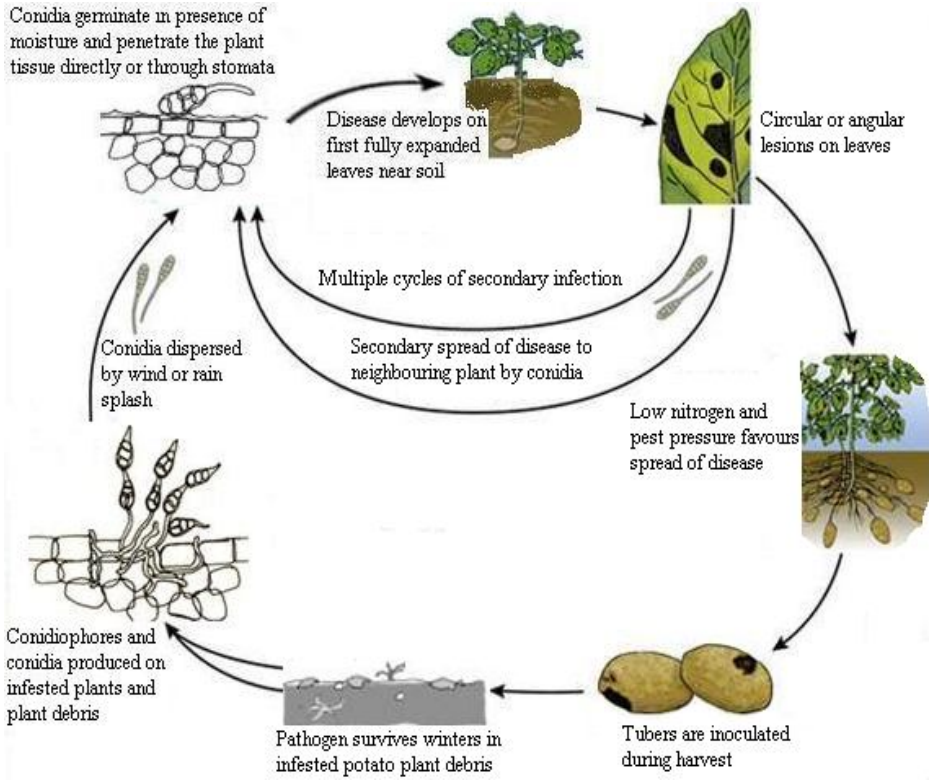

Fig. 10.27: Disease cycle of Early blight of Potato

Control Measures

Cultural Method:

Since the disease is soil-borne following methods are to be used for its control.

(1) Sanitation by destructing diseased haulms immediately after harvest.

(2) Crop rotation.

Chemical Method:

Foliage spraying with fungicides; These are to be done early, about 3-4 week old plants and to be continued throughout the period of plant growth at 10-21 days interval. Most fungicides are those used for late blight disease. These are dithane Z-78 (2 lb/100 gallons), dithane M-45 (0.2%), blitox-50 (0.25%), difolatan and captan (0.2%), Zineb (0.2%). Of these 4-5 sprays of 0.2% Zineb are found most effective.

10.9 Blight of Maize

There are three different types of blight affecting maize. Southern corn leaf blight (SCLB), Northern corn leaf blight (NCLB) and maize seedling blight. All the three types of blight are caused by different pathogens.

Fig. 10.28: Symptoms of (a) Southern corn leaf blight (SCLB), (b) Northern corn leaf blight (NCLB) and (c) Maize seedling blight

1. Southern corn leaf blight (SCLB)

The first epidemics of the Southern Corn Leaf Blight took place in the USA in 1970. A highly virulent strain called Race T appeared on maize hybrids with Texas male sterile cytoplasm. The disease development was related to the growing of maize with T-cytoplasm on large areas. In Krasnodar Territory only 70% of maize sowings were characterized as having T-cytoplasm. Since 1990 the use of cultivars with T male sterile cytoplasm has been forbidden in selection. Presently the cultivars with M and C types of sterility are used.

Symptoms

The disease affects leaves, leaf sheaths, ear, and maize grains. The lesions on leaves and ears are the main symptoms of the disease. On leaves of adult plants the greyish-red or stramineous long lesions with dark brown center appear along leaf veins, being spindle-shaped or elliptical. The length of the lesions is 40 mm, and their width is about 6 mm. The lesions can coalesce, causing death of leaves. On sheaths the lesions are brown with purple border. The length of

the lesions is 50 mm. On ears the lesions are spindle-shaped, brown with dark border. The germ part of seeds becomes dark, and the seeds lose germinating ability. Besides maize the pathogen affects sorghum.

Pathogen

Helminthosporium maydis Nisik. & Miyake. Current name Bipolaris maydis (Y. Nisik. & C. Miyake) Shoemaker. Synonym *Bipolaris maydis* (Y. Nisik. & C. Miyake) Shoemaker, *Drechslera maydis* (Y. Nisik. & C. Miyake) Subram. & B.L. Jain.

The fungus has the teleomorph *Cochliobolus heterostrophus* (Drechsler) Drechsler. It is a facultative parasite. It produces a specific T-toxin causing the same symptoms of disease as those caused by conidia.

Systematic position of pathogen

	Anamorph	Telomorph
Division	Eumycota	Eumycota
Subdivision	Deuteromycotina	Ascomycotina
Class	Hyphomycetes	Discomycetes
Subclass	-	Dothideomycetidae
Order	Moniliales	Pleosporales
Family	-	Pleomassariaceae
Genus	*Helminthosporium*	*Cochliobolus*
Species	*maydis*	*heterostrophus*

Disease Cycle

Infected seeds and corn residues are sources of the infection. Conidia produced are curved, elliptical, from light-olive to brown. The size is 25-115 x 8.5-20.6 microns.

Wind spreads the spores to long distance. The fungus develops at a wide range of temperatures from 10 to 36.C. Optimum temperature for disease development is 25-31.C. Plants are infected at 90-100% humidity. The disease only develops in zones with high humidity (where annual sum of atmospheric precipitation is 800 mm and more). At high temperatures and humidity the sowings can perish over a period of 10-14 days.

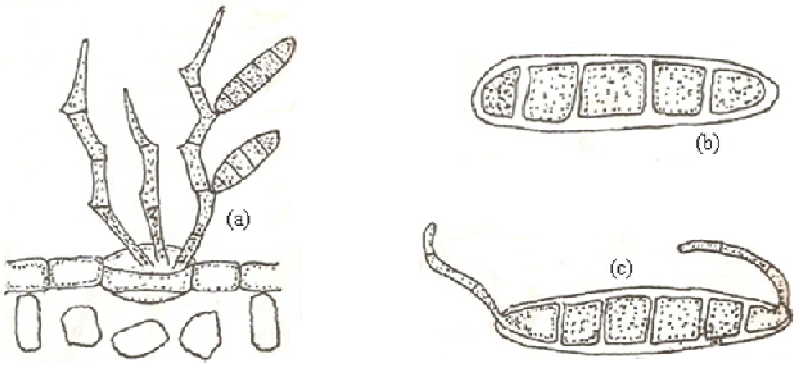

Fig. 10.29: (a) conidiophores of *Helminthosporium maydis* emerging through stomata (b) conidium (c) germination of conidium

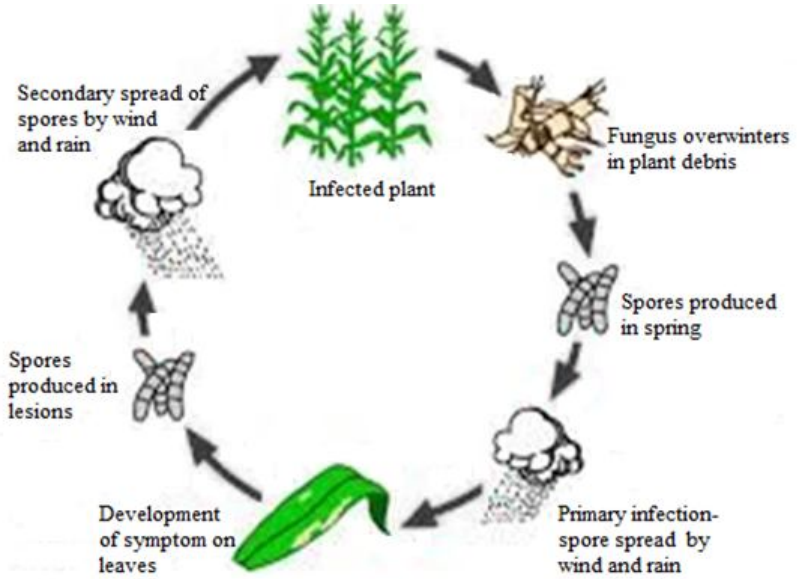

Fig. 10.30: Disease cycle of Southern Corn Leaf Blight

Control Measures

The Southern Corn Leaf Blight is a quarantine disease in Russia. To protect the maize from the disease the following measures are recommended:

Cultural control:

It is performed by removal of infected maize residues; crop rotation; removal of crop residues that contain the infection; use of disease-resistant hybrids of maize.

Chemical Control:

By the treatment of grain and sowings with fungicides.

2. Northern Corn Leaf Blight (NCLB)

Northern leaf blight has traditionally been one of the most damaging corn leaf diseases. The disease is spread throughout the west and south Ukraine, Moscow Region, West Georgia, Altai, Siberia, Primorskii Territory. It has a very high severity in the Caucasus and west Georgia, where the yield losses can reach 40-70%. In Primorskii Territory 70% of leaves infected by Northern Corn Leaf Blight disease during epidemic years can cause yield losses up to 40%. Use of resistant/tolerant hybrids has limited yield losses from this disease in commercial corn. However, in recent years the disease has increased, which may suggest a decline in tolerance levels. Significant losses continue to occur in seed corn production when highly susceptible corn inbreds are planted.

Symptoms

The disease is often confused with Bacterial Leaf Blight disease or Stewart's Wilt. Initial infection is most frequent on the lower leaves, looking like small grey-green lesions. The disease appears as long, elliptical, 2-15 cm (1-6 in.), greyish-green or tan streaks. As the disease develops, the lesions spread to all leafy structures. The lesions become cigar-shaped, brown, with light center and darker border. The length of spots is 3-7 cm and they are 0.4-1.0 cm wide. Lesions can coalesce when weather is favorable for disease development and leaves dry out. As the disease develops, individual lesions may join, forming large blighted areas. In some cases, the entire leaves may become blighted or "burned". During warm and moist weather the greenish black fungal sporulation forms on the infected leaf surface. Conidiophores are olive-green. The length of conidiophores is 150 microns, and the width is to 33 microns. They are multi-cellular, straight or slightly curved. *Exserohilum turcicum* produces spindle-shaped multi-cellular olive conidia.

Losses due to northern leaf blight are most severe when the leaves above the ear are infected at or slightly after pollination. When the disease demonstrates weak development the weight of ears decreases by 3.5%; with moderate development it decreases by 26.6%; with high development it decreases by 54.4%.

Pathogen

Exserohilum turcicum formerly known as *Helminthosporium turcicum* Pass., Boln Comiz. Agr. Parmense is the causal agent of the disease.

Biosynonyms of the fungus are *Bipolaris turcica* (Pass.) Shoemaker and *Drechslera turcica* (Pass.) Subram. & B.L. Jain

The fungus has the teleomorph *Setosphaeria turcica* (Luttr.) K.J. Leonard & Suggs. It is a facultative parasite. Telomorph synonym is *Setomelanomma turcica* (Luttr.) K.J. Leonard & Suggs.

Systematic position of pathogen

	Anamorph	Telomorph
Division	Eumycota	Eumycota
Subdivision	Deuteromycotina	Ascomycotina
Class	Hyphomycetes	Discomycetes
Subclass	-	Dothideomycetidae
Order	Moniliales	Pleosporales
Family	-	Pleoporaceae
Genus	*Exserohilum*	*Setosphaeria*
Species	*turcicum*	*turcica*

Disease Cycle

The fungus survives in corn residue as either spores or fungal strands (mycelium). The spores of the fungus are spread from the ground residue to the developing corn plant through wind or rain "splashing." Plants that become infected act as a secondary source of infection and may spread to other fields. Disease development is favoured by moderate temperatures (18°C-27°C) with prolonged periods of humid or rainy weather.

The fungus causing NCLB over-winters as mycelia and chlamydospores on corn residues. In early summer new conidia are produced, carried by wind or rain to lower leaves of young corn plants. Infection occurs by germinating conidia, when free water is present on leaf surface for 6-18 hours and the temperature is between 18 and 27^0C. Secondary infection spreads among fields by conidia formed on leaf tissues. The optimum temperature for conidia germination is 23-30^0C, optimum humidity is 90% and more.

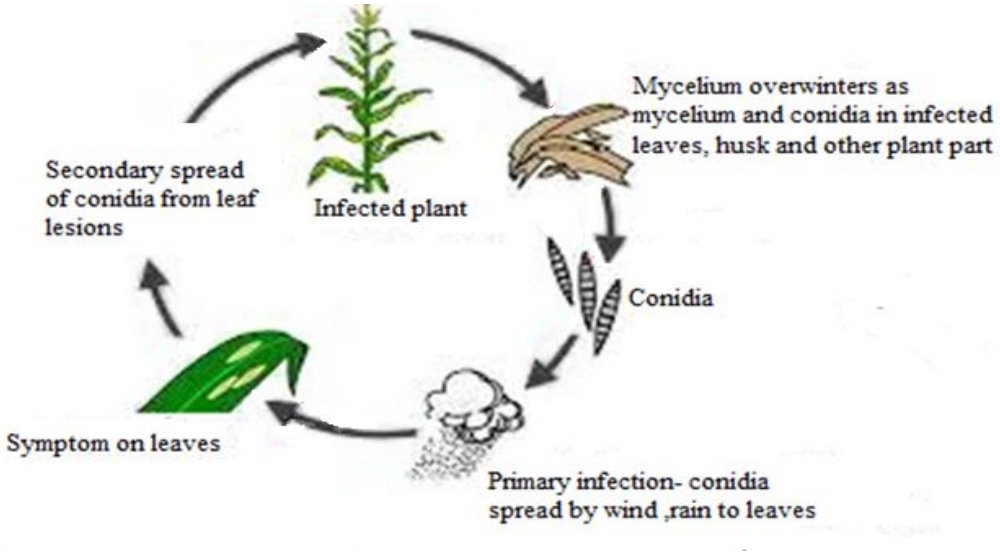

Fig. 10.31: **Disease cycle of northern leaf blight**

Control Measures

There are various races of northern leaf blight. Most of the commercial corn hybrids have resistance or tolerance to the common races. An increase in northern leaf blight symptoms in an area could indicate the potential for a new race developing and should be reported.

Cultural Control:

Crop rotation and tillage will reduce inoculum levels in surface residues. In reduced tillage systems, rotation and the use of resistant hybrids is necessary. Foliar fungicides are not usually economical in field corn but may be warranted if a susceptible hybrid is planted and disease develops early in the season.

To protect the corn from the disease the following measures are recommended: removal of infected corn residues; rotation of corn and sorghum in 1-2 years;

Chemical Control:

By the treatment of grain by fungicides;

Resistant varieties:

Disease-resistant hybrids of corn generally used.

3. Maize Seedling Blight

The disease is of worldwide distribution wherever maize is grown.

Symptoms

Seedling blight occurs when seedlings are attacked before or shortly after emergence. Thin pink or white bloom of the fungus appears on the surface of

sprouting maize. There may be brown spots on the coleoptile, roots and culms. Soon after emergence of shoots they turn brown and die. Seedlings may be killed or stunted by early infections. Surviving shoots have weakly developed root system; infected plants are characterized by delay of growth, their leaves dry, and some plants can be lodged. Damping-off often occurs when the disease is seed-borne.

Pathogen

The disease agents are mold fungi, particularly soil-inhibiting imperfect fungi *Fusarium verticillioides and Microdochium nivale* (formally known as *Fusarium nivale*). This is a facultative parasite. Telomorph of the fungus is *Gibberella avenacea* R.J. Cook. Biosynonym *Selenosporium herbarum*

Systematic position of pathogen

	Anamorph	Telomorph
Division	Eumycota	Eumycota
Subdivision	Deuteromycotina	Ascomycotina
Class	Hyphomycetes	Sordariomycetes
Subclass	-	Hypocreomycetidae
Order	Tuberculariales	Hypocreales
Family	Tuberculariacea	Nectriaceae
Genus	*Fusarium*	*Gibberella*
Species	*verticillioides*	*avenaceanivale*

Disease Cycle

The disease agents are kept in seeds, soil and on plant residues. Most infections are initiated by soil-borne spores however, infection can also be carried on or in the seed. New spores are produced on infected plants or on crop debris. Spores are spread by wind, water, cultivation and infected seed and can remain viable in the soil for several years.

The severity of seed infection strongly influences the disease development. With low levels of infection the germinating ability of seeds decreases by 14%, and by 40% with strong levels of infection.

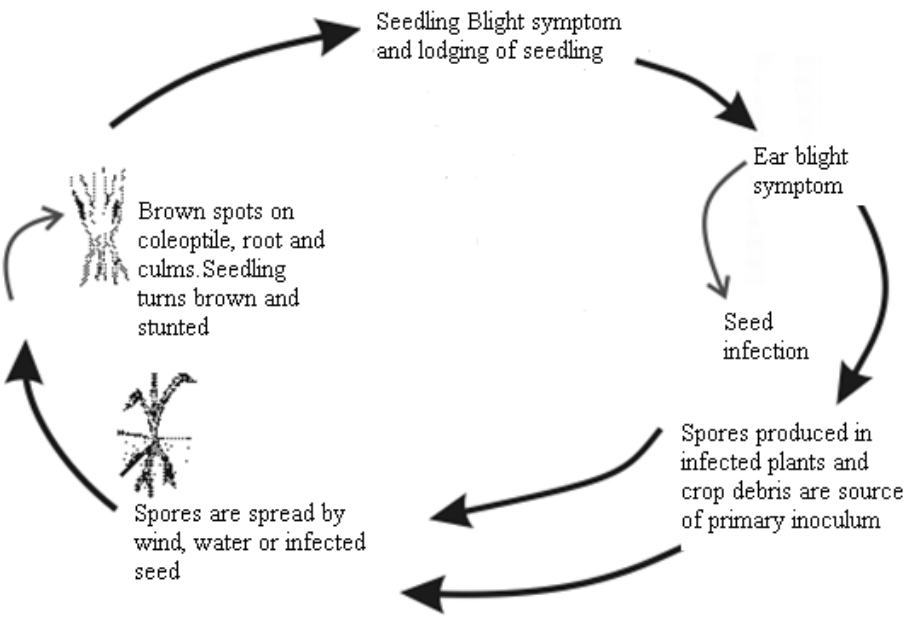

Fig. 10.32 : Disease cycle of seedling blight of maize

Disease development is enhanced by low temperatures during the period of seed sprouting, high soil moisture and acidity, very deep or shallow sowing of seeds and high density of crops.

Under unfavorable conditions the disease can cause 60-70% thinning of plants. The disease is especially harmful in zones with prolonged springs and wet weather. In these districts the seedlings can appear as early as 20-30 days after sowing.

Control Measures

Cultural Control:

To protect maize from the disease the following measures are recommended: sowing on optimal dates into warm soil, use of hybrids seed varieties that are resistant to the disease.

Chemical Control:

Fungicide seed treatment will reduce seedling blight, but will not control root rot in post-seedling plants.

10.10 Ergot: Smut of Bajra

The disease was first reported from South India (Thomas *et al.,* 1945). This disease, in its epiphytotic form, was first reported in India in 1956 from the South Satara area of Maharashtra. By 1966 the disease had become a major

limitation in the cultivation of improved hybrid bajra varieties. Severe epidemics of the disease occurred in Delhi, Uttar Pradesh, Rajasthan, Maharashtra, Karnataka, Tamil Nadu, Andhra Pradesh, and Haryana. Natarajan, *et al.* (1974) estimated the average incidence to be about 62 per cent with grain loss of about 58 per cent. The damage caused by the disease depends upon weather at the time of ear formation. The fungus causing the disease has been reported from several African countries.

In addition to India, there are reports of the incidence of ergot from several countries in Africa, for example, Gambia, Ghana, Nigeria, Rhodesia Senegal, Tanzania and Zambia.

There are several reports of human beings and cattle being poisoned in India by ergot. Presence of toxic alkaloids in the ergot adds to the importance of the disease. Shinde and Bhide (1958) reported that the mature sclerotia contain 0.42% total alkaloid as ergotoxin. Loveless (1967) stated that the pearl millet ergot sclerotia contain groups of water soluble alkaloids which are different from rye ergot (i.e. ergotoxin, ergotamine and ergometrine).

Fig. 10.33: (a) sclerotia formed in the affected spikelets (b) The 'honeydew' (conidial) stage of *Claviceps fusiformis* on an inflorescence of *Pennisetum typhoides*.

Symptoms

The disease becomes evident as small droplets of pinkish or light honey coloured fluid, the honeydew stage. This is the early stage in which honeydew like secretion flows from the affected spikelets to which insects like bees,

wasps are attracted. Later these droplets become darker, coalesce, and cover larger areas of the cob. It advanced stages, small, dark brown to black bodies called sclerotia are formed in the affected spikelets replacing grains completely. These sclerotia are the ergots which contain alkaloids responsible for ergot poisoning in animals.

Pathogen

The fungus causing ergot of pearl millet was described as *Kentrosporium microcephalum* Wallr. in 1853. It was transferred to the genus *Claviceps* by Tulasne who named it *Claviceps microcephala.* Loveless (1967) made detailed study of the fungus from specimen colleted in Africa and on the basis of spore size argued that since *Claviceps microcephala* was considered by Petch (1937) as a synonym of *Claviceps purpurea* and the measurements of pearl millet ergot fungus lie outside the range of C. *purpurea,* it should be called *Claviceps fusiformis* instead of C. *microcephala.* The identity of the pearl millet ergot fungus was further confirmed by other scientists.

The fungus shows three well marked stages in the life cycle- the sphacelia or honeydew stage, the sclerotium stage and the ascigerous stage. The honeydew stage typically occurs on the host inflorescence. The honeydew produced on the ears is full of conidia. In the initial stages macroconidia are formed and later microconidia are also present. Macroconidia are hyaline, fusiform, and broadly falcate, unicellular and measuring 10.8-21.75 x 3.2-4.35 μ, the average of 100 conidia being 16.5 x 3.8 μ. Microconidia are globular, hyaline, unicellular, and measure 2.4-10.8 x 1.2-4.8μ.

At 25° C the macroconidia germinate by producing 1-3 polar or lateral germ tubes. These germ tubes produce secondary macro- and microconidia by septation at the tip. Microconidia are in chains. Tertiary and quaternary conidia are formed from secondary conidia. The tertiary and quaternary conidia are infective.

The honeydew stage is followed by the development of sclerotia which are elongated to round, light to dark brown, hard to brittle in texture, with cavities inside which contain conidia. The sclerotial stage develops on the host and carries the fungus to the soil. The sclerotia measure 3.6-6.1 x 1.3-1.8mm. The inner pseudoparenchymatous tissue (medulla) of the sclerotium is whitish. Sclerotia germinate with seed in the soil. Older sclerotia (12 months old) germinate better than one month old sclerotia.

The ascigerous stage occurs outside the host after germination of sclerotia and production of perithecia and ascospores. Sclerotia germinate by producing 1-3 or up to 16 fleshy, purplish, 6-26 mm long stipes. The stipes bears perithecial stroma or capitulum at its tip. The stromata are globular and light to dark brown in colour and show perithecial projections on the surface. Cross-section of capitulum reveals numerous pyriform perithecia embedded in the tissue and arranged in semi-circular manner in fully developed stromata. The protruding neck has an ostiole measuring about 37 µ in diameter and showing presence of periphyses. The asci are numerous, long, cylindrical, slightly tapering towards the base, having short stalks, hyaline, thin-walled, and obtuse at the apex where there is a narrow opening. Paraphyses are not seen. Each mature ascus contains 8 ascospores which are long, filiform, hyaline, aseptate, and measure 103.2-176 x 0.4-0.5 µ. These uninucleate ascospores germinate to produce primary and secondary conidia. Nucleus from most ascospores migrates to primary conidia without division. But in some ascospores the nucleus divides once thus two primary conidia are formed.

The fungus C. *fusiformis* is reported on a number of plant species in addition to pearl millet. These include *Pennisetum purpureum, P. spicatum, P. alopecuras, P. hohenackeri. P. polystachyon. P. ruppelii. Cenchrus ciliaris and Cenchrus setigerus.*

Systematic position of pathogen

Division	Eumycota
Subdivision	Ascomycotina
Class	Pyrenomycetes
Subclass	Hypocreomycetidae
Order	Hypocreales (Clavicipitales)
Family	Clavicipitaceae
Genus	*Claviceps*
Species	*fusiformis*

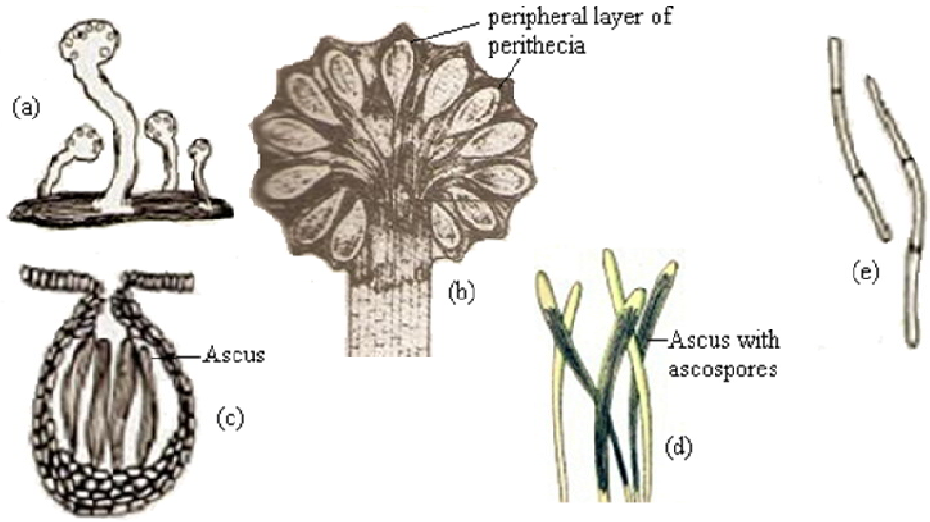

Fig. 10.34: (a) Germinating sclerotium; (b) Longitudinal section of a stroma or capitulum showing peripheral layer of perithecia; (c) L.S. of perithecia (d) Ascus; (e) Ascospores

Fig. 10.35: Conidia of *Claviceps fusiformis* (a) macroconidia (b) microconidia (c) germinating macroconidia (d) production of macro and microconidia on the tip of germ tube

Disease cycle

It occurs during the middle of September and November and when serves causes about 10 per cent damage to the yield of the crop. The conidia of C. *fusiformis* are reponed to remain viable for 13 months. These conidia are abundantly present on the sclerotial structures. The role of this type of inoculum

has now been realized in primary infection of the crop. It appears that during conversion of the honeydew stage to the sclerotial stage, sclerotial structures are of two types. Some of these structures are aggregations of hyphae and conidia and later show only myceliogenic germination. Others are true sclerotia which show carpogenic germination. Sharma and Chauhan (1982) had suggested that primary infection arises from conidia on sclerotia left on the soil surface.

Caprogenic germination of true sclerotia results in production of perithecia and ascospores for primary infection. The sclerotia are easily dispersed as admixture with the seed. When introduced into the field with seed they take about 30-45 days after rains to germinate and produce perithecia. The sclerotial germination normally coincides with the flowering of the crop. The ascospores are carried by wind currents to the fresh flowers. The secondary spread is carried out by conidia in the honeydew mainly by contact, rain and insects. Houseflies are very effective in disseminating the conidia from honeydew. On the same ear, honeydew trickles down to healthy florets. The infection of florets mainly takes place through stigma and occasionally by piercing the thin ovary wall before fertilization. It takes about 5-7 days to develop the honeydew stage and, thus 2-3 conidial generations can be completed within the anthesis period.

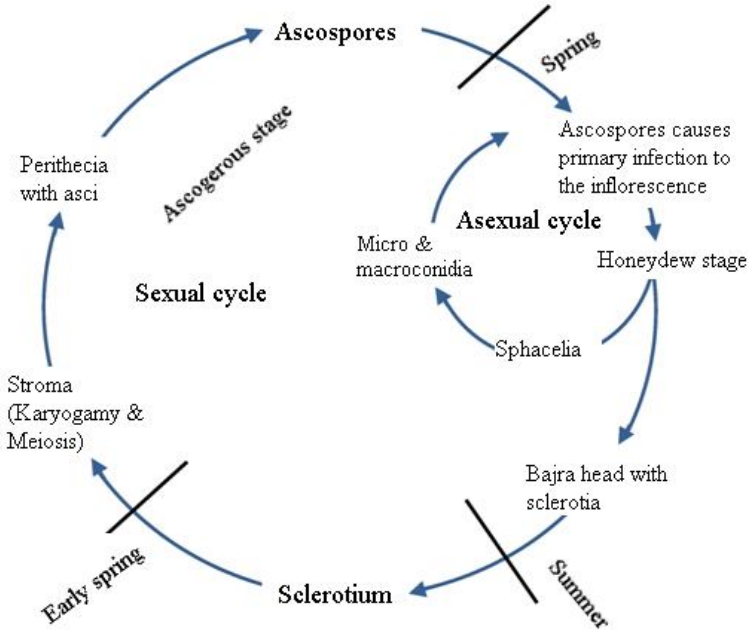

Fig. 10.36 : Disease cycle of Ergot of Bajra

According to Ramaswamy (1968) the meteorological factors affecting epidemics of bajra ergot disease are: high morning relative humidity (85-95%) during flowering and also in the evening (60-90 %) as compared to normal evening humidity of 45-50 %, cloudy weather, low sunshine, and daily light showers.

Singh, R. and Singh, S.N. (1969) conducted an experiment for one season and obtained indications that the disease is less severe in crops planted before July 30 whereas in planting done on September 15 no grain yield was obtained due to serious incidence of the disease. In fields where deep ploughing is done after crop harvest disease severity is relatively less. Heavy nitrogen application promotes disease incidence.

Control Measures

Cultural Control

Long crop rotations help in avoiding soil-borne inoculum. The most commonly recommended method of control is use of clean seed. Seeping the seed in 20--32 per cent salt solution floats the sclerotia which can be removed by hand.

According to Kulkarni (1967) sclerotia remain viable for longer time if buried deep. Therefore, repeated deep ploughing, especially during dry summer, may reduce their viability. Intercropping of pearl millet with mung bean is reported to reduce incidence of ergot disease.

The best means of managing the ergot disease in pear millet is through the use of resistant varieties. Several ergot, smut, and downy mildew resistant lines with high yield potential developed at ICRISAT are sources for resistance in breeding programmes (Thakur, *et al.,* 1988).

Chemical Control

No satisfactory chemical control method has so far been developed. Sundaram (1967) recommended sprays with Ziram or a mixture of copper oxychloride and Zineb (1 : 2, 375-450 g. a. i./ha) applied 2-3 times at 5-7 days intervals, starting just prior to earhead emergence.

Reddy *et al.* (1969) carried out a greenhouse trial where the fungus was inoculated 24 hours after a single spray of the seven fungicides was tried and an inorganic sulphur preparation (80% wettable sulphur) was found to be most effective. Ten per cent salt solution gets rid of all sclerotia and sclerotial fragments from the pearl millet.

Biological Control

Recently Mower *et al.* (1975) examined the fungal hyper parasites of *Claviceps purpurea* as potential biological control agents for wheat ergot. Nene and Singh (1976) reported that *Fusarium sambucinum* Fuckel may possibly be utilized for biological control.

10.11 Summary

Flag smut infects wheat and many grass species mainly in wheat growing areas of the world. In India, it causes heavy destruction in wheat growing states of Punjab, Haryana Madhya Pradesh and Rajasthan. Symptoms appear on all parts of shoot but leaf and leaf sheath are more affected showing flagging. *Urocystis agropyri* (Preuss) A.A. Fisch. Waldh belonging to subclass Ustilaginomycetes, order Urocystales is the causal agent of the disease flag smut.The disease is spread through teliospores which survive in soil for several years. Seed treat ment with systemic fungicides and cultural control is effective control measures.

Covered Smut of Barley is found everywhere in areas of barley cultivation. There is no symptom of disease on the host before ear emergence. In infected ears the grains appear to be covered in a thin, whitish or grey membrane which is characteristic feature of the disease. Sori in spikelets are present as blackish brown coherent smut spore mass. *Ustilago hordei (Pers.) Langerheim of class Teliomycetes of Subdivison Basidiomycotina is the pathogen.* Teliospores are globose or subglobose. The disease is seed borne which spread through harvest and seed handling operations. Primary infection occurs through sporidia produced after germination of teliospores.Cultural control by sowing seeds free of pathogenis best method. Seed traetment with systemic fungicides give excellent results.

Blast of Rice or Paddy or rotten neck of rice is common in rice growing countries and is more severe in humid areas. The disease is chiefly a foliage disease but attacks leaf sheath, rachis the joints of the culm and even the glums also. . Initial symptoms appear as white to gray-green lesions or spots, with dark green borders. Older lesions on the leaves are elliptical or spindle-shaped and whitish to gray centers with red to brownish or necrotic border. Blast is caused by the fungus *Magnaporthe oryzae,of subdivision Ascomycotina, class Discomycetes* asexual conidial stage of *which* is known as *Pyricularia oryzae* Cavar (Subdivision Deuteromycotina, class Hyphomycetes). Fungi survive on

collateral host. Conidia cause secondary spread of the disease. Field sanitation and seed treatment with Agrosan GN is effective control measures.

On the basis of symptoms and pathogen, four types of smut of Jowar (sorghum) occur; Grain smut (Kernel smut / Covered smut / Short smut), Loose smut, Head smut and Long smut. The fungus *Sphacelotheca sorghi* (Link) Clint. causes covered smut of sorghum which causes development of smut sori instead of the flower elements. The affected ovaries turn into conical porcelain white sori (spore sacs) which contain black powder consisting of millions of chlamydespores of the fungus. Thus individual grains are replaced by smut sori. The pathogen responsible for loose smut is *Sphacelotheca cruenta* (Kohn) Potter (Synonym *Ustilago cruenta* J.G. Kühn). They are shorter than the healthy plants. The spores are formed in the ovaries and floral bracts. The sorus is covered by a thin membrane which ruptures very early, exposing the spores even as the head emerges from the sheath. The pathogen responsible for heat smut of sorghum is *Sphacelotheca reiliana* (Kuhn) Clinton. The entire inflorescence is converted into a big sorus. The pathogen responsible for the long smut of Sorghum is *Tolyposporium ehrenbergii* (Kuhn) Pat. This disease is normally restricted to a relatively a small proportion of the florets which are scattered on a head.Smut of Jowar disease can be controlled by sanitation and chemical methods are used.

Red rot of sugarcane caused by imperfect fungi (subdivision-Deuteromycotina), *Colletotrichum falcatum* Went. is a serious disease of sugarcane in tropical and subtropical parts of the world. Stems and midrib areas of leaves are most affected part of the plant by this disease. The canes are completely rotted within and a gummy dark red material oozes out of the cells filling the intercellular spaces. The soluble pigment present in this ooze is absorbed by the cell walls producing the characteristic red rot appearance. The pathogen is sett borne and produces aseptate one celled, sickle shaped conidia. The disease can be controlled by cultural methods and by use of resistant varieties.

Flax rust disease is autoecious rust, conspicuous in the field due to bright orange colour of the infected parts, which is due to uredia. Later telia appear which are reddish brown in color. The disease is caused by *Melampsora lini* (Pers.) Lev. or *Melampsora lini-usitatissimi* Kuprev, belonging to sub-divisionBasidiomycotina, class teliomycetes. The rust survives on linseed and other hosts in uredial or telial stage at high altitudes from where there the uredospores might be blown down to plains by wind. Sanitary precautions and

use of resistant varieties are most effective control measures.

Early blight disease is common and destructive disease of potato in India and the world. Symptoms of early blight occur on foliage and tubers of potatoes. Initial spots are small, isolated and brown which later enlarge and are often surrounded by a yellow halo forming characteristic concentric ridges which is called "bullseye" lesions. The disease is caused by *Alternaria solani* (EII and Martin) Jones and Grout. an imperfect fungi (subdivision Deuteromycetes, class hyphomycetes). Beaked, muriform, dark brown and septate conidia are the sources of primary infection. Secondary spread of disease occurs through conidia developing on primary spots. Destruction of diseased plant, crop rotation and use of fungicide can control the disease.

There are three different types of blight affecting maize. Southern corn leaf blight (SCLB), Northern corn leaf blight (NCLB) and maize seedling blight. Southern Corn Leaf Blight caused by *Helminthosporium maydis* Nisik. & Miyake affects leaves, leaf sheaths, ear, and maize grains. Symptoms are grayish-red or stramineous long, spindle shaped or elliptical lesions with dark brown centre appearing along leaf veins. Infected seeds and corn residues are sources of the infection. Northern leaf blight disease appears as long, elliptical, 2-15 cm, greyish-green or tan streaks. Later lesions become cigar-shaped, brown, with light centre and darker border which coalesce to become large blighted areas. *Exserohilum turcicum* formerly known as *Helminthosporium turcicum* Pass. Boln Comiz. Agr. Parmense *is* the causal agent of the disease. Infection occurs by germinating conidia which are spindle-shaped multi-cellular olive coloured. Maize seedling blight disease appears on sprouting maize. Symptoms are brown spots on the coleoptile, roots and culms. Soon after emergence of shoots they turn brown and die. Soil-inhibiting imperfect fungi *Fusarium verticillioides and Microdochium nivale* (formally known as *Fusarium nivale*) are the causative agents of the disease. Spores are spread by wind, water, cultivation and infected seed that causes the infection. Infected crop removal, use of resistant varieties and treatment of grain with fungicide helps to control the disease.

Ergot of bajra becomes evident as small droplets of pinkish or light honey coloured fluid, the honeydew stage. Later these droplets become darker, coalesce, and cover larger areas of the cob. It advanced stages, small, dark brown to black bodies called sclerotia are formed in the affected spikelets replacing grains completely. *Claviceps fusiformis* belonging to subdivision Ascomycotina and class Pyrenomycetes is the causative agent of the disease.

Ascospores produced in perithecia bring about primary infection. The secondary spread is carried out by conidia in the honeydew mainly by contact, rain and insects. Crop rotation helps in avoiding soil borne inoculum. Use of disease free seeds, deep ploughing of the field, use of resistant varieties and fungicides may help to reduce the incidence of disease.

10.12 Glossary

- **Acervulus :** Flator saucer shaped aggregation of hyphae
- **Autoecious :** A fungi that can complete its entire life cycle on the same host
- **Dikaryotic :** Mycelium or spore having two sexually compatible nuclei per cell
- **Perthecium :** A globular or flask shaped sexual fruit bogy having an opening or ostiole.
- **Pycnedia :** Asexual spherical or flask shaped fruiting body lined inside with conidiophore and conidia
- **Quarantine :** control of import and export of plants to prevent spread of disease and pests
- **Sporadic :** A disease that breaks out ocassionally without being constantly destructive
- **Susceptibility :** the inability of the plant to resist the effect of a pathogen or any other factor
- Systemic **:** spreading internally throughout the plant body

10.13 Self-Learning Exercise

Section A : (Very Short Answer Type Questions)

1. What is acervulus?
2. Write the pathogen of flag smut of wheat diseases.
3. Give the systematic position of *Ustilago hordei*.
4. Write the name of telomorph of *Colletotrichum falcatum*.
5. Write the name of resistant varieties of flax.

Section B : (Short Answer Type Questions)

1. Write note symptoms of red rot of sugarcane.
2. Differentiate between four types of smut of jowar.
3. Write a short note disease cycle of ergot disease of bajra.

4. Write note characteristic symptoms of early early blight of potato.
5. Write note on disease control of coverd smut of barley.

Section C : (Long Answer Type Questions)

1. Explain the disease cycle of rusts.
2. Write detail note on Blast of Paddy.
3. Write detail note on three types of blight of maize.

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Unit-11

Bacterial Diseases-I:

Classification and General

Structure of the Unit:

11.0 Objectives

After going through this unit you will be able to understand:

- various plant pathogenic bacteria.
- the classification of plant pathogenic bacteria.
- systems and approaches of bacterial nomenclature.
- general disease symptoms caused by bacteria.
- methods to identify and differentiate various phytopathogenic bacteria.
- explain various control measures.

11.1 Introduction

In 1969 R.H. Whittaker proposed a five kingdom classification of living world. He proposed a separate kingdom Monera for the true prokaryotes which included bacteria and cyanobacteria. Although all prokaryotes possess the common chemical composition (DNA, RNA, and protein) some of them exhibit

absolutely no relationship with the majority in their structure and function. In the absence of knowledge about their ancestry, a hierarachical system of classification (families, orders, etc.) has not been possible. Major treatise on bacterial taxonomy was given in *Bergey's Manual of Determinative Bacteriology,* 1923. The classification had been considerably changing with respect to composition of taxa higher than genus (families, orders, tribes) in its successive editions. The eighth edition, published in 1974, had adopted a more rational approach but the subsequent changes in bacterial taxonomy based on nature of DNA and chemical composition of the cell and the cell-wall have had this edition also of not much value. After this edition, no further editions of this manual are being brought out. Instead a series of volumes under the title "*Bergey's Manual of Systematic Bacteriology*" has been started. The first volume (Krieg and Holt, 1984) contains such phytopathogenic bacteria as *Pseudomonas, Xanthmonas, Agrobacterium* and *Erwinia* and *MLOs.* Coryneform hacteria are given in the second Volume (Sneath, 1986).

11.2 Classification

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The eighth edition of the Bergey's Manual had divided the prokaryotes in 19 groups (parts) of which 18 parts are in the division Scotobacteria which contain

the true bacteria and mycoplasmas. These 18 parts are arranged in three classes (I,II,III). The following is a condensed form of the grouping which lists only the parts having plant pathogenic bacteria.

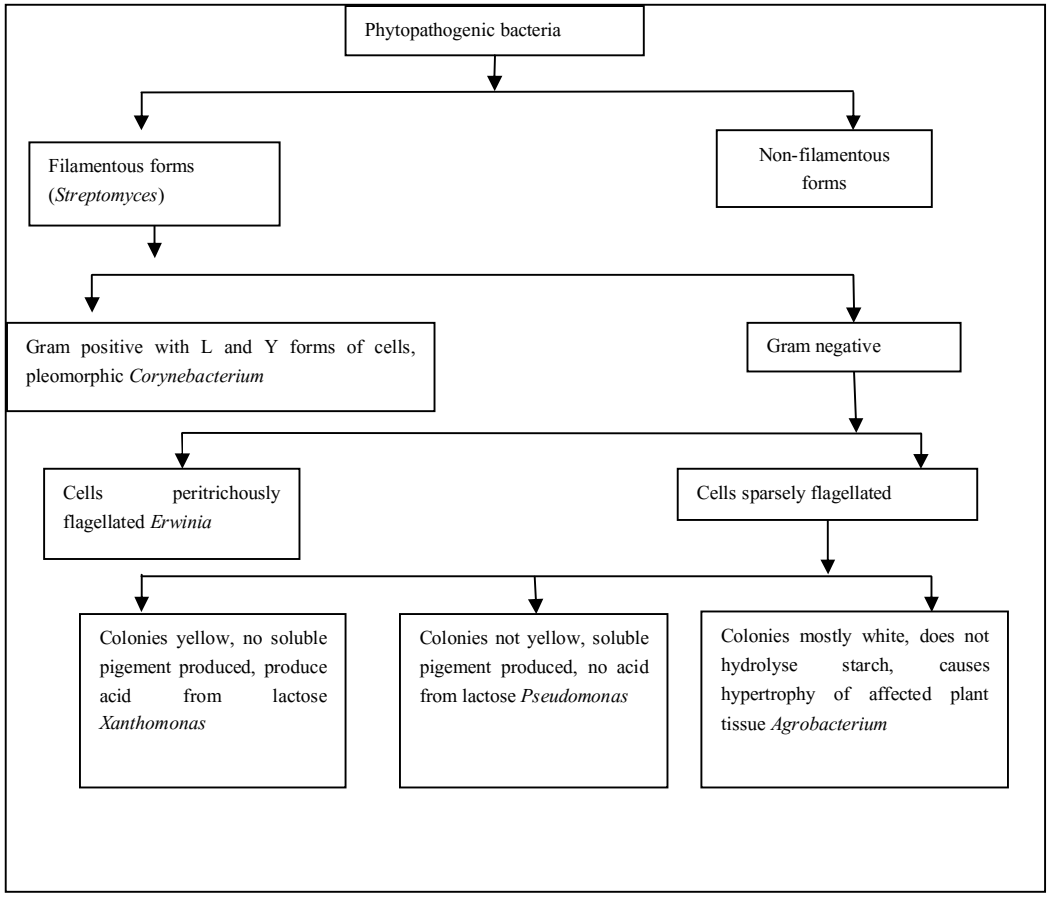
In the following table salient features of plant pathogenic prokaryotes are summarized. It will be seen that most of the important plant pathogenic genera of bacteria are Gram-negative. Only coryneform bacteria *(Clavibacter* and *Curtobacterium)* and *Streptomyces* are Gram-positive. *Bacillus* and *Clostridium* which are also Gram-positive are rarely plant pathogenic. Most phytopathogens are flagellate. Except the mycoplasmas all are rod-shaped.

Table 11.1: Classification of Plant Pathogenic Prokaryotes

Division II	Scotobacteria (indifferent to light)
Class 1	The Bacteria
Part 7	Gram-negative, aerobic *rods* and cocci. Five families with 14 genera and 6 genera of uncertain affiliation. Plant pathogens in the following families: Pseudomonadaceae (order Pseudomonadales): Genera: *Pseudomonas*. *Xanthomonas.* Rhizobiaceae: Genus *Agrobacterium.*
Part 8	Gram-negative, facultatively anaerobic rods. Two families with 17 genera and 9 genera of uncertain affiliation. Plant pathogens in the family Enterobacteriaceae. Genus *Erwinia*
Part 15	Endospore fornning rods and cocci. Genera *Clostridium* and *Bacillus*
Part 17	Gram-positive, irregular rods and filamentous bacteria. Five genera described in arbitrary sequence. One family with two genera and one order comprising 8 families and 31 genera. Plant pathogens in the following genera:

	A) Irregular rods: *Corynebacterium* (plant pathogenic genera *Curtobacterium* and *Clavibacter)* B) Filamentous bacteria: Order Actnomycetales: Family *Nocardiaceae* (genera *Rhodococcus* and *Nocardia)* and family Streptomycetaceae (genus *Streptomyces)*
Class II	Rickettsiae: obligate, intracellular pathogens of eukaryotic cells.
Part 18	Two orders, four families and II genera. Not proven plant pathogens.
Class III	Mollicutes: pleomorphic scotobacteria devoid of cell-wall; collectively known as mycoplasmas
Part 19	One order and three families. Order Mycoplasmatales Family Mycoplasmataceae Genus *Mycoplasma* with 51 named species, none plant pathogenic. Organisms resembling *Mycoplasma* in plants, not yet cultured, are known as MLO. Genus *Ureaplasma* with no plant pathogens. Family Acholeplasmataceae not plant pathogenic. Family Spiroplasmataceae with Genus *Spiroplasma* with at least three plant pathogenic species

Box 1: Key of differentiating phytopathogenic bacterial genera (after Rangaswamy,1962)



All bacteria, pathogenic or non-pathogenic are saprophytes and can be cultured on artificial media. All the plant pathogenic bacteria are rod shaped. A majority of phytopathogenic bacteria are flagellated. Amongst six genera only two genera *viz. Corynebacterium* and *Streptomyces* are Gram positive while rest are Gram negative.

11.3 Nomenclature

Since the basic category in biological system is the species, a new bacterial isolate must be identified at this level through certain universally accepted criteria avoiding need for major dependence on morphology alone. The criteria are incorporated in the *International Code of Bacterial Nomenclature*. A complete and modern description which meets these criteria includes morphology including flagellation, Gram's stain reaction, cultural, biochemical and physiological characters, serology, phage and bacteriocin typing, DNA base composition (the G + C content), DNA-DNA or rRNA-DNA homology and extensive pathogenicity and host range tests with detailed descriptions of symptoms. Chemical analysis of cell and cell walls are nowadays very

important tools of identification and classification. Any new claim for a new bacterium should always be published in the *International Journal of Systematic Bacteriology* and type culture must be deposited with some internationally recognised type culture collection.

Approved names for bacterial nomenclature

In spite of the existence of strict rules for bacterial nomenclature the establishment of new species had been on "new host-new species" concept basis and this had resulted in multiplicity of names given to bacteria that were basically the same. The approach suffered from lack of adequate modern description, bacteriological comparison with possible related species and absence of proper host range study. Some bacterial species which are capable of producing different symptoms on different hosts or variants of the same bacterium producing different symptoms on the same host were identified by different workers as different species.

In order to remove the above confusion the International Committee on Systematic Bacteriology (ICSB) took certain steps. The International Code on Bacterial Nomenclature was revised in 1976. This code provided that on January I, 1980, all names (class, order, tribe, family, genera and species) published prior to this date and included in the *Approved Lists of Bacterial Names* (Skerman, *et al.,* 1980) shall be treated as though they had been validly published for the first time on that date. Those names considered valid prior to January I, 1980 but not included in the *Approved Lists* will have no further standing in bacterial nomenclature and such names can be used for other taxa proposed in future. Although this decision helped in reducing the confusion to a great extent, it posed some problems for plant pathologists. Also, in subsequent studies, published soon after, many changes in the approved names were accepted.

Box 2: Partial list of approved names that were recommended for use according to International Committee on Systematic Bacteriology (ICSB). (Only plant pathogenic taxa are listed in alphabetical order)

Divisions:	Firmicutes, Gracilicutes
Classes:	Actinomycetes, Bacteria, Schizomycetes, Mollicutes
Orders:	Actinomycetales, Bacillales, Chlamydiales, Clostridiales, Eubacteriales, Mycoplasmatales, Pseudomonadales, Rickettsiales
Suborders:	Eubacteriineae, Pseudomonadineae
Tribes:	Erwinieae, Eubacterieae, Pseudomonadeae, Rhizobieae, Rickettsieae
Families:	Bacillaceae, Chlamydiaceae, Clostridiaceae, Corynebacteriaceae, Mycoplasmataceae, Nocardiaceae, Pseudomonadaceae, Rhizobiaceae, Rickettsiaceae, Streptomycetaceae, Spiroplasmataceae.
Genera:	The following genera which contain plant pathogenic species are recognized by the *Approved Lists* and by subsequent valid publications; *Agrobacterium, Arthrobacter, Bacillus, Chlamydia, Clavibacter, Clostridium, Curtobacterium, Erwinia, Nocardia, Pectobacterium, Pseudomonas, Spiroplasma, Streptomyces, Xanthomonas*

11.4 Symptomatology of Bacterial Diseases

The bacterial diseases of plants can be grouped under following catagories:

1. **Blights:** The invasion by bacterium leads to very rapid and extensive necrosis of the affected plant parts resulting in scorched appearance of the surface.
2. **Soft rots:** The major effect is softening of the tissue due to dissolution of the middle lamella by enzymes and disintegration of tissues is preceded by change in colour.
3. **Leaf spots:** When the specific bacterium invades leaves through stomata the necrosis of tissue around the substomatal space results in the appearance

of necrotic areas on the lamina surface. The dead tissues appear water soaked and the spots generally remain restricted in growth.

4. **Tumors and galls:** In some bacterial diseases the effect of invasion is hyperplasia and hypertrophy of the invaded tissues. As a result, tumors develop on the affected parts. The formation of root nodules in leguminous plants due to association of *Rhizobium* is a similar effect but since the association is not antagonistic the plants do not suffer. Crown gall disease caused by *Agrobacterium tumifacience* is the best known and extensively studied disease in this group.
5. **Canker:** Canker or warty outgrowths are formed on leaves, twigs and fruits. They results from necrosis of the tissue and reaction of the undamaged tissue to produce cork cells. This symptom is mostly localized.
6. **Vascular diseases:** In some bacterial leaf spots the pathogen moves into the vascular system of the leaf and become systemic. In others, the invasion from seed or underground parts is concentrated in the vascular tissues causing typical wilt of the plant by plugging the vessels and by producing toxins. Examples: bacterial wilt of tomato, potato, brinjal, cucurbits, maize and beans.
7. **Scabs:** These are corky outgrowths
8. **Local lesions:** The symptoms are in the form of lesions which are limited to a definite area of varying extent of the organ, or only to a particular part of the plant.

11.5 Methods of Identification of Bacterial Pathogen

The various phytopathogenic bacterium species are differentiated to their host specificity and range. Besides this they are also identified according to their cultural, physiological and biochemical properties.

Bergey's Manual of Determinative Bacteriology, started in 1923, had been the major treatise on bacterial taxonomy but it could serve only the purpose of identification. Most bacterial pathogens can easily be assigned to their correct genus but identification of species, subspecies, etc. is rather difficult. In eukaryotes (plants, fungi, etc.) the morphological differences are such that it is easy to compare two individuals and distinguish them from each other. On the other hand all prokaryotes within a genus look so much alike that it is impossible to distinguish them on the basis of morphology. In addition, while in eukaryotes it is easy to compare the individuals (single plant or single thallus or its parts), in the prokaryotes this is difficult because mostly it is possible to

make comparison between colonies or mass of cells and in this the presence of contaminants or variants cannot be ruled out.

Table 11.2: Summary of Bacterial Identification

Genus or trivial name	Gram's stain reaction	Flagellation	Morpho-logy	G+C Content (mole%)	Pigmenta-tion
Agrobacterium	-	Peritrichous	Rod	60-63	None
Bacillus	+	Peritrichous	Rod (endospores)	43-46	None
Clostridium	+	Peritrichous	Rod (endospores)	52-54	None
Corynebacterim	+ or ±	None or polar	Pleomorphic , V-form	53-56	None or blue, yellow or orange
Erwinia (Pectobacterium)	-	Peritrichous	Rods	50-57	None or blue, pink or yellow
Pseudomonas	_	Polar	Rods	57.7-67	None or green blue
Streptomyces	+	None	Mycelia, conidia	69-73	Variable
Spiroplasma	+	None but motile	Helical,	26	None
Xanthomonas	_	Polar	Rods	63-69	Yellow

G + C content are proportion of guanine and cytosine in the DNA molecule (genome) which is a stable character. All phytopathogenic species that were earlier in *Corynebacterium* have now been reclassified under genera

Curtobacterium, Clavibacter, Arrhrobacter and *Rhodococcus.*

Confirmation of the Pathogen: Serology, bacteriophage typing, fatty acid profiles, PCR (polymerase chain reaction), and DNA analysis are useful for identification and classification of bacterial isolates into pathovars. For example; there are distinct forms of citrus canker disease caused by various pathovars and variants of the bacterium, *X. axonopodis*. Because symptoms are generally similar, separation of these forms from each other is based on host range, cultural and physiological characteristics, bacteriophage sensitivity, serology, DNA-DNA homology and by PCR (polymerase chain reaction) analysis of genomic DNA. The latter assays demonstrate that these forms are genetically unique.

However, when such techniques are unavailable, strains of *X. axonopodis* pv. *citri* can be distinguished from other pathovars by a panel of susceptible and resistant citrus hosts.

11.6 Control Measures

In most of the bacterial diseases once the disease sets in it is very difficult to control. Thus precautionary measures play important role to control the disease than cure.

1. *Cultural Control*

Crop rotation: Proper crop rotation is one way of avoiding the soil-borne inoculum of the bacterium. Susceptible varieties should not be planted in soils known to be infested with the pathogen. For example brown rot of potato can be controlled by three-year rotation with maize, soybean and red top grass *(Agrostis alba).* Ageing of the seed for two years before sowing has also been recommended.

Intercropping: Intercropping with plants of different families have been used as a means of reducing populations of the bacterium in soil and also root to root transmission of the bacterial pathogen. Bean, maize, cowpea and sugarcane have been used as intercrops in different countries to control brown rot of potato.

Use of infection free seed: Seed should always be obtained from a disease free field or locality.

Sanitation: Removal and destruction of diseased plant debris is recommended to reduce the soil-borne inoculum. Destruction of possible alternate or collateral hosts is also essential.

At the time of harvest no diseased plant material and crop refuge should be left in the field. Infected plant residue should always be removed and burnt. Crop sanitation is necessary to avoid the introduction of infected material into the nursery stock. For example introduction of pathogenic *A. tumefaciens* strains can be avoided by thorough inspection of nursery stock for crown gall symptoms. Black leg affected plants if noticed should be dug out and burnt. Certified nursery stock should be used as seed.

In rot of sugarcane Systematic cutting down and burning of the affected shoots reduces the spread.

Deep Ploughing: Deep ploughing of the field after harvest helps to expose the soil to summer heat of May-June in the plains. In some cases Infested soil exposed to 43° C continuously for 4 days or more becomes free of the bacterium. Example; *Ralstonia solanacearum* cannot withstand desiccation and its populations in soil are considerably reduced in fields given regular ploughing.

Deep ploughing after harvest buries the infected stalks and, thus, reduces survival ability of the bacterium in soil. Pre-sowing irrigation to enable the left over seeds germinates, followed by ploughing and then planting of the main crop is followed in some countries. Crop rotation, late sowing, early thinning, good tillage and early irrigation help in reducing the disease incidence.

Moisture control: In some cases high soil moisture accumulations resulting from either a high water table or heavy rainfall usually favour development of disease eg. Bacterial wilt. Infection is reduced below 50 per cent soil moisture level. Even rain or irrigation water should not be allowed to flow from infested to healthy fields.

2. *Chemical Control*

In general, bacterial diseases of plants are very difficult to control owing to the lack of effective chemicals. The most effective alternative is the use of copper, which is potentially phytotoxic.

Soil treatment: In many bacterial diseases since the pathogen perennates in diverse soil types its control through chemicals has not been possible. Application of very high dosages of urea, toxic chemicals like sulphur, chloropicrin, etc. which are effective cannot be recommended because of the very high cost. Soil treatment with bleaching powder has also been recommended in some cases. The bleaching powder (chlorine) treatment inhibits respiratory enzyme activity of the bacteria. In some cases addition of

potash to soil helps in reducing the disease incidence.

Seed Treatment: Seed-borne inoculum can be eliminated by seed treatment. External inoculum on the seed is destroyed by delinting of seed with concentrated sulphuric acid. Seeds are immersed in acid for 10-15 min, then rinsed thoroughly by suspending in water to remove acid and finally dried and treated with organo-mercurial compounds such as Agrosan GN, Ceresan, etc. This treatment does not destroy the internally seed-borne inoculum.

Eradication of infection in seeds by soaking in solution of Agrimycin may be used as one of the control measures.

In some diseases simply soaking of seed in water for 12 hr followed by exposure to hot water at 53° C is enough to eradicate the seed-borne inoculum.

Du-Ter and Captan dissolved in dichloromethane and Blitox, Terramycin and Benlate dissolved in acetone were effective in elimination of the pathogen from seed. Antibiotics could be used, but they are expensive.

Treating seeds with antibiotics like streptomycin eradicates the internally seed-borne inoculum. The systemic fungicides Vitavax and Plantvax are more effective than antibiotics.

Hot water treatment of seed at 56° C for 10 min destroys the external as well as internal inoculum without affecting seed viability.

Antibiotics have also been recommended for tuber treatment by some scientists in blackleg disease. Antibiotics, such as Vancomycin, Aureomycin, Streptomycin and Terramycin have been tried to prevent crown gall disease.

Dipping cut tubers in mercuric chloride solution or organo-mercurial compounds like Agallol, Aretan or Emisan or in stable bleaching powder solution helps to control the blackleg disease of potato.

Foliar Spray: Foliar spray helps to check the secondary spread of the disease. Sprays of Agrimycin were recommended for preventing secondary spread.

Severity of bacterial blight in the field can be reduced and yields increased by giving 5 sprays of Agrimycin plus copper oxychloride at 12 day intervals.

Resistant varieties

The problem in developing resistant varieties is that resistance found so far is race specific. Hence, resistance to one race/biotype fails to be resistant to other races/biotypes.

In the absence of specific chemical control measures efforts have been continuing to find out resistant varieties. More than half of the 198 wild type of rice were found resistant to six races of the bacterium.

Srinivasan, *et al.* (1959) had conducted inoculation experiments on 32 varieties and 8 wild species of *Oryza* and found only the varieties BAM-9 and MTO-15 showing some degree of resistance to the disease.

Control through development of resistant varieties is possible and provides the best and most effective preventive measure. *Gossypium herbaceum* and *G. arboreum* were considered to be practically immune whereas *G.* barbedanse, *G. herbaceum var. typicum* and *G. hirsutum* were considered susceptible. Varieties known to be resistant are HC-9, BJA-592, 101-102B and Reba-B.50.

4. *Genetic-engineering techniques*

Genetic-engineering and technology are being used for the introduction of lysozyme, cecropsins and other potent antibacterial proteins derived from insects into potato as a means of augmenting resistance to bacterial wilt and other bacterial diseases but the short term probability of success is low. Attempts to control bacterial wilt through biocontrol agents are also underway. The agents are antagonistic rhizobacteria and avirulent srains of *Ralstonia solanacearum.* The mechanism involved may be induced resistance and precolonization of the rhizosphere by the antagonists.

5. Biological Control

A. tumefaciens prompted the first successful development of a biological control agent. Allen Kerr of Australia discovered the biocontrol system by isolating non-pathogenic strains of *Agrobacterium radiobacter* from disease sites and testing their ability to compete with pathogenic strains in mixed inoculations. Several non-pathogenic strains helped to reduce infection, but one strain in particular, *A. radiobacter* **strain K84,** completely prevented disease when added to wound sites at a 1:1 ratio with cells of *A. tumefaciens*.

Preventative treatment of seeds or transplants with the non-pathogenic biocontrol organism *Agrobacterium radiobacter* is a relatively inexpensive and effective means of managing the development of crown gall in commercial operations. This strain is the one that is marketed globally. It is supplied commercially on agar plates or in a peat substrate and it is used by suspending the bacterial cells in water, then dipping seeds, seedlings or cuttings in this suspension before planting. Application of this antagonist by soaking seeds or

dipping transplants can prevent infection by most strains of *A. tumefaciens* due to the production of the antibiotic agrocin 84 by strain K84 of *A. radiobacter*.

Biological control with *A. radiobacter* is only as a preventative treatment, not to cure infections, so it is applied at a high population level to protect any wound sites against pathogenic invasion.

11.7 Summary

All the prokaryotes are kept under Monera in five kingdom classification by Whittaker. Major treatise on bacterial taxonomy was given in *Bergey's Manual of Determinative Bacteriology,* 1923. In the later edition the bacterial classification was revised.

The criteria of phyto pathogenic bacteria nomenclature are incorporated in the *International Code of Bacterial Nomenclature*. These criteria includes morphology including flagellation, Gram's stain reaction, cultural, biochemical and physiological characters, serology, phage and bacteriocin typing, DNA base composition, DNA-DNA or rRNA-DNA homology, and extensive pathogenicity and host range tests with detailed descriptions of symptoms.

The bacterial diseases of plants can be grouped under blight, soft rots, leaf spots, tumours and galls, cankers, scabs and local lesions. In some bacterial leaf spots the pathogen moves into the vascular system of the leaf and become systemic. In others, the invasion from seed or underground parts is concentrated in the vascular tissues causing typical wilt of the plant by plugging the vessels and by producing toxins. Examples: bacterial wilt of tomato, potato, brinjal, cucurbits, maize and beans.

The various phytopathogenic bacterium species are differentiated to their host specificity and range. Chemical analysis of cell and cell walls are nowadays very important tools of identification and classification. Gram's stain reaction, type of flagella, morphology, pigmentation and G+C content are the basis for identification of bacteria. Types of pathogenic bacteria are confirmed by serology, bacteriophage typing, fatty acid profile, Polymerase chain reaction and DNA analysis.

In most of the bacterial diseases once the disease sets in it is very difficult to control. Thus precautionary measures are considered better to control the disease than cure. Cultural methods involving crop rotation, intercropping, deep ploughing, sanitation and use of infection free seed are important control measures. Soil treatment with bleaching powder is effective chemical control. Seed-borne inoculum can be eliminated by seed treatment. Control through

development of resistant varieties is possible and provides the best and most effective preventive measure against bacterial pathogen. Recently genetic engineering techniques are gaining importance.

11.8 Glossary

- **Bacteriophage** : A virus that infects bacteria and destroys them
- **Biological control** : Total or partial destruction of pathogen by use of other organism
- **Gall** : A swelling or outgrowth produced on the plant as a result of infection by certain pathogen
- **Genetic engineering** : The alteration or manipulation of the genetic composition of cell by various procedures such as protoplast fusion, transformation etc. in tissue culture.
- **Genome** : All genes together
- **Pathovar (*pv*)** : In bacteria, a subspecies or group of strains that can infect only plants within a certain genus or species.
- **Serology** : A method using specificityof the antigen-antibody reaction for the detection and identification of antigenic substances and the organism that carry them.

11.9 Self-Learning Exercise

Section –A (Very Short Answer Type Questions)

1. Who proposed five kingdom classifications?
2. Write the names of two pathogic Gram positive bacteria.
3. Write the name of gall forming bacteria.
4. Write key to identify *Pseudomonas*.

Section –B (Short Answer Type Questions)

1. What is the basis of bacterial nomenclature? Write note.
2. What techniques are used to confirm the bacterial identification?
3. Write a short note on symptomatology of bacterial disease.
4. Write note on disease control by genetic engineering method.

Section –C (Long Answer Type Questions)

1. Explain the nomenclature of bacteria.
2. Write detail note on Biological control of bacterial disease.
3. Write detail note on control measures of bacterial diseases.

11.10 References

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Unit-12

Bacterial Diseases-II:

Plant Diseases Caused by Bacteria

Structure of the Unit:

12.0 Objectives

After going through this unit you will be able to understand:

- various plant diseases caused by bacteria.
- various symptoms produced by bacteria on their specific host plant.
- the disease cycles of various bacterial pathogens.
- about control measures of particular disease.

12.1 Introduction

Pathogentic ability of bacteria was first recognized by Burrill in as early as 1878. He discovered that Fire blight of apple is caused by bacterium. Today it is estimated that more than 180 diseases are caused by bacteria.

The infection of bacterial to plant produces specific symptoms such as galls, canker, leaf discoloration, blight, dwarfing, rot and wilting etc. These

symptoms cause heavy loss of food crops and in some cases deteriorates the quality of the fruits and vegetables.

12.2 Brown Rot of Potato

The bacterial brown rot and wilt disease of potato is common in tropical and subtropical regions and also in some warm temperate regions of the world where temperature and moisture conditions are favourable for its development. In India, it is the most serious bacterial disease of potato accounting for 10 to 70 per cent loss in tuber yield. The disease is destructive in the mid-hills, plateau region and West Bengal. In Karnataka, Madhya Pradesh, Maharashtra and West Bengal the disease poses constant threat to the cultivation of potato. Apart from solanaceous crops like chilly, potato, tomato and eggplant the bacterium is known to attack castor, ginger, groundnut, papaya, cabbage, radish, banana and a large number of other plant species.

The disease damages the crop through direct killing of the plants in the field and through rotting of tubers in stores. The losses vary with changes in the cropping systems. For example, in Kumaon hills of north India 37 -55 per cent of potato yield is lost every year whereas up to 40 per cent in Maharashtra and up to 75 per cent in some localities of Karnataka.

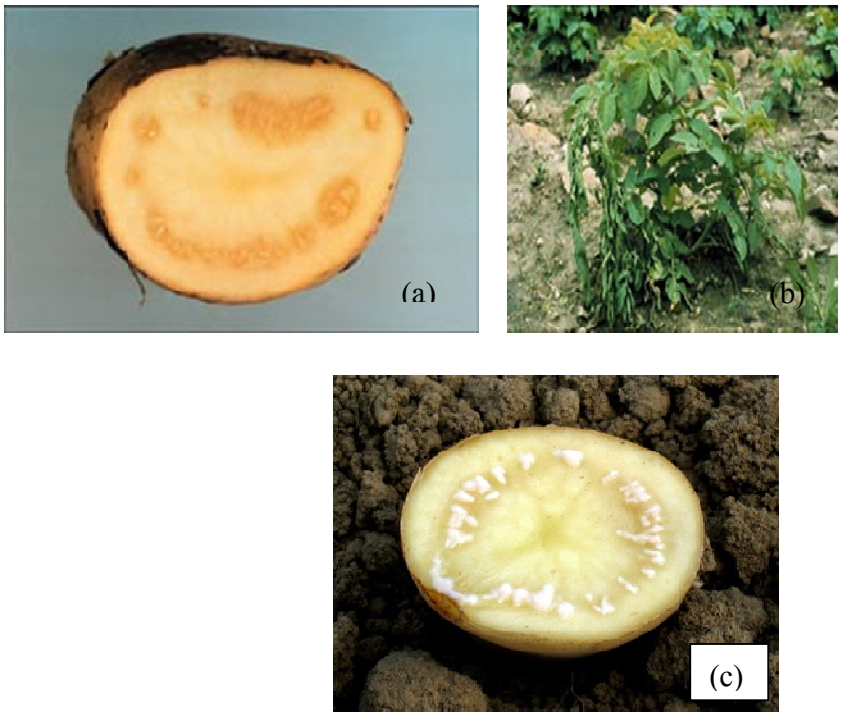

Fig. 12.1: (a) Potato tuber with brown rot; (b) Potato plant showing wilting of one stem; (c) bacterial ooze coming out of the vascular ring of the tuber.

Symptoms

The characteristic symptoms of the disease may appear at any stage and include stunting, yellowing of the lower foliage, sudden wilting and finally collapse of the entire plant. Disease may be severe in young, succulent plants and may appear as rapid wilting of leaves and collapse of stems. Initially, only one stem may wilt. Symptoms of the disease are variable. In India stunting and yellowing of lower leaves is not commonly seen. Browning of the vascular bundles may or may not be present. In the hills where bacterial wilt usually appears in July, about a fortnight after the start of the monsoon many wilted plants show stem rot at soil level. This symptom may be confused with blackleg disease of potato caused by *Erwinia carotovora*. For a quick field diagnostic identification of *R. solanacearum* and to distinguish bacterial wilt from vascular wilts caused by fungal pathogens, bacterial streaming from infected plant material can be used.

The *brown rot* refers to the browning of the xylem in the vascular bundles. Due to browning stems may appear streaked as infected vascular bundles become visible. This browning is due to production of pigment by the bacterium. The disease is also known as *ring disease* due to the fact that a brown ring is formed in the tuber due to discolouration of the vascular bundles. The skin of the infected tubers is often discoloured. In severely affected tubers the eye buds turn greyish brown, and a sticky exudate may form at the eyes or where the stolon is attached to the tuber. If the infected stems or tubers are cut across and squeezed, greyish-white bacterial ooze comes out of the vascular ring. Eventually, infected tubers left in the ground will turn into a slimy mass surrounded by a thin layer of outer tissue. Some isolates do not produce the brown pigment but cause bacterial wilt and rot of tubers. In such cases the vascular bundles and surrounding parenchyma appear water-soaked.

Pathogen

Bacterial wilt of potato is caused by *Ralstonia solanacearum* (Smith) Yabuuchi *et al*.; formerly called *Pseudomonas solanacearum* (Smith) Smith belonging to order Burkholderiales and family Ralstoniaceae. *Ralstonia solanacearum* is a widely distributed pathogen found in tropical, subtropical, and some temperate regions of the world.

Host range: The very extensive host range of the bacterium includes several hundred species representing 44 families of plants.

Primary hosts are *Lycopersicon esculentum* (tomato), *Nicotiana tabacum* (tobacco), *Solanummelongena* (aubergine), *Solanum tuberosum* (potato), *Musa paradisiaca* (banana) and *Heliconia*.

Secondary hosts are

Anthurium, Ricinus communis (castor bean), *Zingiber officinale* (ginger), *Arachis hypogaea* (groundnut),

Capsicum annuum (bell pepper), *Colocasia esculenta* (taro), *Curcuma longa* (turmeric), *Gossypium* (cotton), *Hevea brasiliensis* (rubber), *Ipomoea batatas* (sweet potato) and *Manihot esculenta* (cassava). Whereas wild hosts are *Solanum nigrum* (black nightshade), *Galinsoga parviflora* (gallant soldier), *Portulaca oleracea* (pussley) and *Urtica dioica* (stinging nettle).

It is a soilborne bacterial pathogen that is a major limiting factor in the production of many crop plants around the world. The bacterium often attacks the crop in association with root knot nematodes. This organism is the causal agent of brown rot of potato, bacterial wilt or southern wilt of tomato, tobacco, eggplant and some ornamentals and Moko disease of banana.

The pathogen species is subdivided into races based on host range. Currently, polymerase chain reaction (PCR) is the primary means of definitive identification of pathogen race. Recently, it has been suggested that *Ralstonia solanacearum* should be considered a "species complex."

Ralstonia solanacearum is a gram-negative motile rod. The rod-shaped cells measure 0.5-0.7 x 1.5-2.5 μ and are non-encapsulated. The organism grows aerobically and does not form endospores. They are motile by 1-4 polar flagella. However, virulent strains are usually not flagellated and cells are surrounded by thick polysaccharide material. The bacterium is aerobic although many strains can grow anaerobically. Two types of colonies are produced on complex media: one type is smooth, fluidous, elevated and the other is somewhat rough, dry and flat. A large number of organic compounds are utilized as carbon source by individual strains. Most isolates utilize glucose, fructose and sucrose. *R. solanacearum* is catalase and oxidase positive. Nitrates are reduced and ammonia and hydrogen sulphide are produced on specific media. In broth culture, the organism is inhibited by concentrations of NaCl greater than 2%.

The species does not characteristically utilize xylose, arabinose and arginine. Starch and gelatin are not hydrolyzed. Optimum temperatures for growth is

35°-37° C, maximum about 41° C and minimum 10° C. Thermal death point lies at about 52° C. The isolated cultures of the bacterium easily lose their virulence when grown on laboratory media.

Direct isolation of *Ralstonia solanacearum* can be obtained from plant ooze and exudates. Pure cultures are usually easily isolated. *R. solanacearum* can also be isolated from water and soil using a modified Kelman's TZC medium.

Systamatic Position of Pathogen

Kingdom: Bacteria
Phylum: Proteobacteria
Class: Betaproteobacteria
Order: Burkholderiales
Family: Burkholderiaceae
Genus: *Ralstonia*
Species: *solanacearum*

Disease cycle

Soil is considered a potential source of primary inoculum. The pathogen survives between crop seasons in soil, seed tubers and on cultivated or wild alternate host plants. The disease has been noticed even in the first plantings in newly cleared land. In cultivated soil, survival of the bacterium has been reported for a period of more than two and a half years although some reports indicate that it cannot survive in soil even for one year. Survival of up to 673 days in naturally infested soil stored in plastic bags at 4° C has been reported. According to Rangaswami and Thirunavakarasu (1964) the bacterium can survive in sterile and natural soil for over 250 days. Well drained soils with good water retention capacity, moderate to high soil temperature and low to moderate soil pH promote survival of the bacterium. In most soils infestation of *Ralstonia solanacearum* is highest in the top 30 cm of the profile but low populations have been recovered as deep as 65-75 cm.

Populations of the bacterium can survive on roots of non-host plants also. Root system of even such non-host plant as wheat, sorghum and maize can harbour low populations of the bacterium. In the north Indian hills, the potato crop is cultivated during March-September. At harvest, farmers usually leave the brown rot affected tubers in the field. Since the weather during off season (September-March) remains cool and humid, desiccation is prevented and also there is little activity of secondary invaders or antagonists. Thus, the pathogen

may survive in plant debris under such conditions. In the Nilgiri Hills the bacterium may survive on potato crops grown throughout the year. However, survival of the pathogen in diseased plant debris has been doubted on account of very hot and dry summer that follows the main potato crop season in the plains of India.

Potato tubers carry the bacterium in three ways; in vascular tissues (active infection), on the tuber surface and in lenticels. Infection through lenticel has become the most important and significant method of tuber transmission in the last two decades. In lenticels infection no symptoms develop in the tubers and the bacterium goes unnoticed. It can express itself only when the tubers are incubated at high temperatures. Tuber transmission of the pathogen is possible if infected or contaminated (or latently infected) tubers are stored for seed at moderate temperatures. Tubers carrying active infection easily rot at high temperatures and are not used for seed. Even if they are planted most of them do not germinate due to rotting in soil. Delayed harvesting of the tubers increases number of lenticel infected tubers. In the plains of India, where soil survival of the bacterium is doubtful, infected and surface contaminated (latent infection) potato seed tubers appear to be the only source of primary inoculum.

Potato tubers are infected by way of stolons. Infection in tomatoes takes place through injured roots but the bacterium can also enter the host through wounds on the stem and through stomata. Root knot nematodes aid in penetration if they attack the host before it comes in contact with the bacterium. In addition, certain gall forming insects, cut worms, white grubs and root invading parasitic fungi may also provide root injury for entry of the wilt bacterium. During the sowing operation, cutting of tubers with knives, without taking precautions, helps in contamination of healthy tubers. After entry, the pathogen moves upward through the host plant vessels and colonizes rapidly. The incubation period varies with age of the plant, environment and host variety.

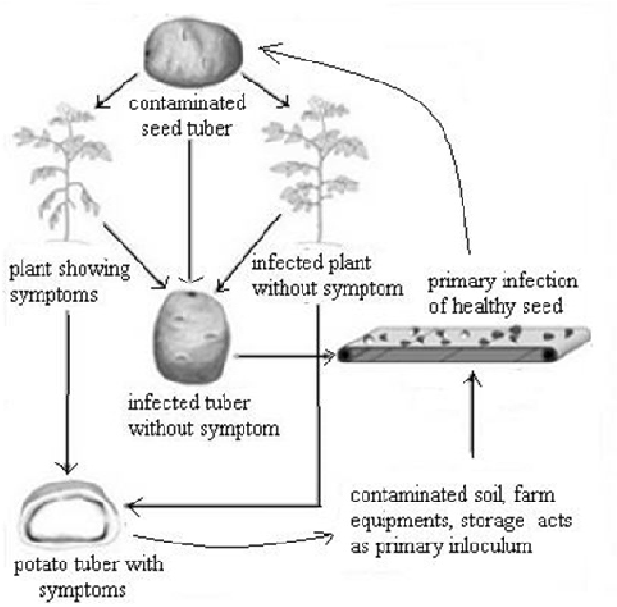

Fig. 12.2 : Disease cycle of Brown rot of potato

Temperature is the most important factor affecting the host-parasite interaction as well as survival in soil. Most rapid disease development took place at 37° C. High temperature (30°-35° C) and high soil moisture favour the disease. For wilt development caused by race 1 of the bacterium a relatively high temperature range of 28°-30° C is required. At lower temperatures infection can occur but symptoms fail to develop. Thus, at lower temperatures chances of latent infection are very high.

High soil moisture favours the disease. The disease starts its appearance from mid-July after rains have started. It is also reported that bacterial population in soil is also highest during July and August when both soil moisture and temperature are high. The incidence of wilt declines when soil moisture drops to 8-10 per cent water holding capacity and maximum/minimum temperature below 20°/15° C.

The pathogen grows over a wide range of pH. Optimum pH is 6.2 to 6.6, maximum 7.4. However, this may vary with strains. The disease occurs in diverse soil types both acidic and alkaline. It is severe in acidic soils (pH 3.6-5.0) of the Nilgiris as well as in alkaline soils (pH 7.0 to 8.0) of Madhya Pradesh. Growth of the bacterium is decreased if the clay content of soil increases. Organic matter promotes growth while inorganic fertilizers decrease it. There is decrease in disease severity with increasing age of the plants.

Control Measures

Cultural Control

Crop rotation: Proper crop rotation is one way of avoiding the soil-borne inoculum of the bacterium. Three-year rotation with maize, soybean and red top grass *(Agrostis alba)* had been found to afford considerable protection to the crop. In India, rotations consisting of potato-finger millet (mandua)-potato, potato-wheat-potato are recommended in crop rotations for management of the disease. Besides these potato-sorghum-potato, potato-maize-potato, potato-wheat-sunhemp green manure, wheat-green manure-potato have been found effective in keeping disease incidence below 3 per cent.

Intercropping has also been used as a means of reducing soil populations of the bacterium and its root to root transmission. Bean, maize, cowpea and sugarcane have been used as intercrops in different countries.

Use of infection free seed: Seed tubers should always be obtained from a disease free field or locality. The tubers obtained from a healthy crop in an endemic area should be treated with 0.02 % Streptocyclin for 30 min after giving a 5 mm deep cut. Rain or irrigation water should not be allowed to flow from infested to healthy fields.

Sanitation: At the time of harvest no diseased or rotting tubers and crop refuge should be left in the field. Infected plant residue should always be removed and burnt.

Ploughing: After harvest, the field should be ploughed to expose the soil to summer heat of May-June in the plains. *Ralstonia solanacearum* cannot withstand desiccation and its populations in soil are considerably reduced in fields given regular ploughing.

Moisture control: High soil moisture accumulations resulting from either a high water table or heavy rainfall usually favour development of bacterial wilt. Survival of the bacterium is greatest in wet but well-drained soils whereas survival is adversely affected by soil desiccation and by flooding. No infection is obtained below 50 per cent soil moisture level. Thus Rain or irrigation water should not be allowed to flow from infested fields to healthy plots.

Infested soil exposed to 43° C continuously for 4 days or more becomes free of the bacterium.

Resistant varieties

Differences in susceptibility to brown rot occur in varieties of *Solanum*

tuberosum but so far the commercial varieties of potato under cultivation in India have not been found resistant to bacterial wilt and brown rot. Races of the bacterium in India are highly virulent and exotic cultivars resistant to the bacterium lose resistance under Indian conditions. A clone of the wild potato *(S. microdontum)* was identified to carry high level of resistance to race I and 3 and biotypes II, III, and IV. Culture BRB/A-24 of S. *tuberosum* x S. *microdontum* was found resistant to the disease. Certain clones of S. *phureja* also show resistance to the disease and are being used in hybridization programmes. The problem in developing resistant varieties is that resistance found so far is race specific. Hence, resistance to one race/biotype fails to be resistant to other races/biotypes.

Chemical Control

Since the disease is a systemic vascular wilt and the pathogen perennates in diverse soil types its control through chemicals has not been possible. Application of very high dosages of urea, toxic chemicals like sulphur, chloropicrin, etc. which are effective cannot be recommended because of the very high cost. Application of stable bleaching powder in furrows at planting time (12 kg/ha) has also been recommended.

Genetic-engineering techniques

Genetic-engineering and technology are being used for the introduction of lyso-zyme, cecropsins and other potent antibacterial proteins derived from insects into potato as a means of augmenting resistance to bacterial wilt and other bacterial diseases but the short term probability of success is low. Attempts to control bacterial wilt through biocontrol agents are also underway. The agents are antagonistic rhizobacteria and avirulent srains of *Ralstonia solanacearum.* The mechanism involved may be induced resistance and precolonization of the rhizosphere by the antagonists.

12.3 Blight of Rice

The bacterial leaf blight (BLB) of rice had been known in Japan as an endemic disease since 1881. It is also known to occur in China, Taiwan, Korea, Thailand, Vietnam, Philippines, Indonesia, Malaysia, Bangladesh and Australia. The first Indian record, based on isolation and pathogenicity of the bacterium, was by Srinivasan, *et al.* (1959) from Maharashtra where it was reported to be widespread and destructive since 1951. However, the symptoms described by them conform to the bacterial leaf streak which is distinct from leaf blight both

in respect of the causal agent and the symptoms. Bhaskar *et al.*1960 appear to be the first to have described the typical bacterial leaf blight symptoms in India. The disease was considered to be localized in Maharashtra until 1963 when in Shahabad district of Bihar a severe blight, previously attributed to nutritional disorder, was identified as bacterial blight (Srivastava and Rao, 1963). Since then the disease has been reported from other parts of north India also. The disease causes much damage in the Tarai region of Uttar Pradesh where weather conditions are usually favourable for its appearance and development.

The disease is a typical vascular wilt, leaf blight being only the mild phase resulting from secondary infections. Damage is due to the partial or total blighting of leaves or due to complete wilting of the affected tillers leading to unfilled grains. In the wilt or "Kresek" phase crop may dry up completely before seed maturation. If the attack is late the loss in yield may be negligible. In India the damage has been estimated to vary from 6 to 60 per cent (Srivastava and Rao 1966). Losses up to 50 per cent were estimated by Rao and Kaufman (1971).

Symptoms

Bacterial Leaf Blight of Rice have 2 phases (a) Blight (b) "Kresek" or wilting. Symptoms of the disease vary considerably with the stage of infection and the prevailing weather conditions. The most apparent symptom is the leaf blight phase of the disease. This phase of the disease is characterized by linear, yellow to straw coloured with wavy margins, generally on both edges, rarely on one edge of the leaf. These stripes usually start from the tip and extend downwards. This is followed by the twisting of the leaf tip and rapid extension of marginal blight lengthwise crosswise to cover large areas of the leaf. On seedlings, infected leaves turn greyish green and roll up. On older plants, lesions usually develop as water-soaked to yellow-orange stripes on leaf blades or leaf tips or on mechanically injured parts of leaves. Lesions have a wavy margin and progress toward the leaf base. The blighting may extend to the leaf sheath and culms, killing the tillers or the whole culm.

In dry weather opaque and turbid drops of bacterial ooze which dry into yellowish beads can be seen on the leaf surface. These drops are washed down by rains. The glumes of seeds also get infected but the symptoms are not well defined. The blight phase of the disease usually appears 4-6 weeks after trans-planting.

Fig. 12.3: Symptoms of Bacterial Leaf Blight of Rice;

(a) Blighted leaf (b) bacterial ooze

The diseased leaves should be subjected to microscopic examination for correct diagnosis because leaf drying can be due to physiological disorders or attack of tungro virus also. If the affected portions of leaves are cut and mounted in a drop of water on a glass slide bacterial ooze can be seen as a cloudy mass at the cut ends.

The most destructive form of the disease in the tropics is the wilting of seedlings (also called "kresek") resulting from early systemic infection or from infected seed and the bacterium brought in contact with germinating seedlings. As the disease progresses, the leaves roll completely, droop, turn yellow to straw-colored and ultimately the tillers wither away wilt, leading whole seedlings to dry up and die. In severe cases the affected stool may be completely killed. The Kresek-affected tillers can be confused with stem borer injury.

According to Nayak and Reddy (1985) the disease appears in small patches in the field, each patch originating from a single infected plant. It then spreads to neighbouring plants. In the initial stages there is patchy appearance of the disease in the field due to more rapid vertical spread than horizontal spread. Later, due to large number of such patches, the field appears uniformly affected.

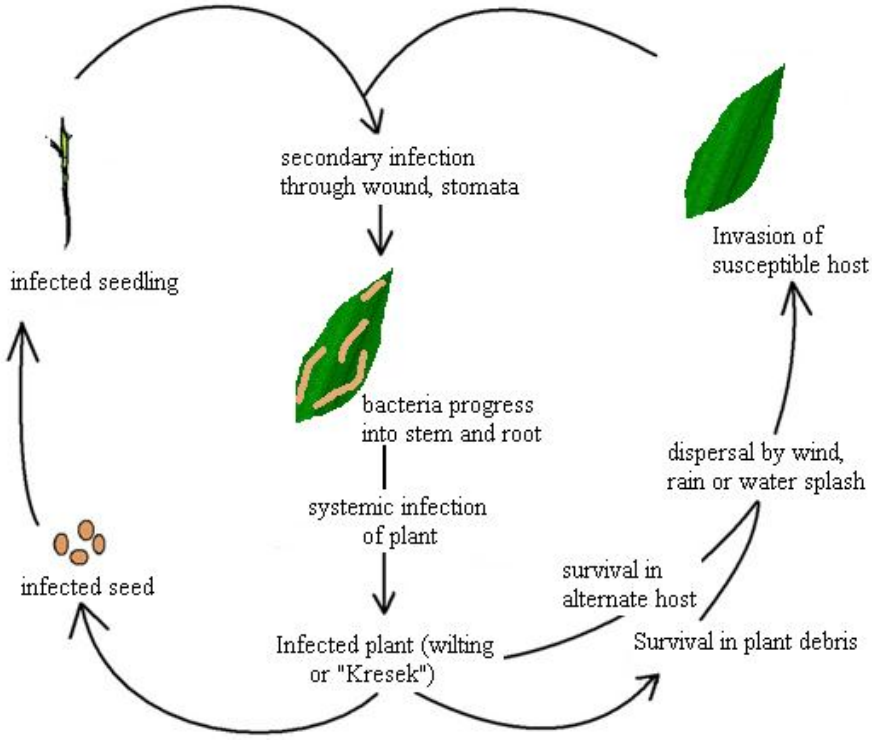

Fig. 12.4 Disease cycle of of Bacterial Leaf Blight of Rice

Pathogen

Xanthomonas campestris pv. *oryzae* (Ishiyama) Dye. The cells of the bacterium are rods measuring 0.5-0.8 x 1.0-2.0 μ in size. They are single and form no capsule. Stain reaction is Gram-negative. They are motile by a single polar flagellum. Cultural characters are similar to those of other *Xanthomonas* species. Optimum temperature for growth is 25°-30° C. Thermal death point is 53° C. Accordingly, *Xanthomonas campestris* contained more than 140 pathovars.

Systamatic Position of Pathogen

Kingdom: Bacteria
Phylum: Proteobacteria
Class: Gammaproteobacteria
Order: Xanthomonadales
Family: Xanthomonadaceae
Genus: *Xanthomonas*
Species: *campestris* pv. *oryzae*

Disease cycle

There can be several modes of perpetuation of the bacterium from season to season. The pathogen perpetuates in the rhizosphere of the grass hosts. Goto, *et al*. (1953) identified the weed hosts *Leersia oryzoides* and *L. oryzoides* var. *japonica* constitute the chief source of primary inoculums in Japan. In India also many such grass hosts have been identified. These include *Cyperus rotundus* (motha grass) C. *defformis*, *Leptocorsia acuta* and *Leersia hexandra*. According to Trimurthy and Devadath (l981a) leaves of *Paspalum scrobiculatum, Leersia hexandra, Panicum repens* and *Cyperus rotundus* harbour the bacterium up to 130-140, 120-130, 100-110 and 50-60 days, respectively, without showing blight symptoms.

Perennation of the pathogen in infected rice straw left in the field and also in the stubbles has been reported by several Japanese and Indian workers. However, this mode of survival in India is doubted. In double cropped areas, such as in south India, volunteer rice seedlings also perpetuate the pathogen under low lying field conditions. The bacterium survives in soil for only a short period though survival in soil is influenced by soil type.

Diseased wild rice growing in ponds may, thus, provide primary inoculum. Irrigation water contaminated with the bacterium flowing through field to field also provides the primary inoculum. Contact of rice leaves with the contaminated water is essential for initiation of the disease. The pathogen and its phage (virus) survive longer at 15°-20° C than at 30°-45° C and more in sterilized than in unsterilized field water. Examination of phage population can forecast onset of the disease but not disease severity.

Seed from infected crop is also one of the major sources of primary inoculum. Viability of the bacterium in naturally infected leaves and artificially inoculated seeds is longest at a very low temperature (4° C). Thus, under natural conditions viability of the bacterium seems to be for short durations at high temperatures. According to Trimurthy and Devadath (1984) X. *campestris* pv. *oryzae* may be present in 54 per cent seeds and survive for 120-180 days. Such seeds may not produce diseased plants although the bacterium can be isolated from soil around seeds suggesting that the primary inoculum moves from seeds through soil.

Secondary spread is brought about through wounds and stomata by bacterial cells disseminated by wind-borne raindrop splashes, by irrigation water or rain water coming from infested fields and by contact between diseased and healthy

leaves. The leafhopper and the grasshopper can transmit the bacterium mechanically on their body. The bacterium does not survive inside the insect body. After entry the bacterium multiplies in the intercellular spaces of the host parenchyma and is again released through stomata. The bacterium produces such phytotoxic organic acids as phenyl acetic acid, isovaleric acid, 3-methylthiopropionic acid, fumaric acid and succinic acid which have been shown to be toxic to cut rice shoots (Sreeramulu, *et al.,* 1987).

In climatic conditions of Japan, poorly drained fields along rivers or lakes and mountainous basins, excess of rainfall and humidity, floods and typhoons, higher temperature during tillering stage, low temperature with less sunshine in mid-summer and warm autumn are conducive to disease development. In India cloudy weather with a shower or high humidity is reported to be most conducive for disease development in Uttar Pradesh (Pavgi, *et al.,* 1964). Moderate amounts of rains evenly distributed during the crop season can bring about epidemics. The development of the disease is favoured by temperatures above 25° C. Symptoms never appear at temperatures below 20° C. At mean temperature range of 21.3° to 32.7°C the size of lesions is longest (Premlatha Dath, *et al.,* 1979). Combination of rainy weather, strong winds and temperatures of 20°-26° C, favour rapid spread of the disease in the field. According to Srinivasan and Singh (1983) severe Kresek development occurs when there is a combination of maximum temperature of 30°-35° C, minimum temperature of 24°-26° C, uniform high humidity of 64-84 % RH, short sunny days and heavy well-distributed rainfall.

It is commonly observed when strong winds and continuous heavy rains occur, allowing the disease-causing bacteria to easily spread through ooze droplets on lesions of infected plants. Workers have also reported that water logging favours the disease as it helps contact of leaves with the floating bacteria. Disease incidence increases with age of the crop. Heavy nitrogen application and ill-drained and shaded conditions aggravate the disease severity even in moderately resistant varieties (Devadath and Padmanabhan, 1969; Mahmood and Singh, 1970; Davadath and Rao, 1978; Devadath, *et al.,* 1987). Aggravation of disease is not due to increased nitrogen content of leaves. It is possibly due to certain metabolites liberated by the bacterium which helps the process of invasion (Pandey and Iswaran, 1982). Microbial fertilizers such as *Azotobacter* sp. and *Pseudomonas* sp. aggravate the disease. Bacterial blight can be severe in susceptible rice varieties under high nitrogen fertilization.

Control Measures

Many chemicals including antibiotics have been tested and used for the control of bacterial leaf blight of rice but under Indian conditions none has proved satisfactory. Partial control of the disease was reported in isolated experiments at various places in the country.

Seed Treatment: Srivastava and Rao (1964) claimed 95 percent eradication of infection in seeds by soaking for 12 hr in 0.025 % solution of Agrimycin (15 % streptomycin and 1.5 % oxytetracycline) plus 0.05 per cent wettable Ceresan and then transferring the seed to hot water at 52° -54° C for 30 min.

According to Nene and Singh (1967) simply soaking of seed in water for 12 hr followed by exposure to hot water at 53° C is enough to eradicate the seed-borne inoculum.

Du-Ter and Captan dissolved in dichloromethane and Blitox, Terramycin and Benlate dissolved in acetone were effective in elimination of the pathogen from seed. Soaking of infected seed at pH 3.5 for 24 hr and at pH 1.25 for 6 hr also reduced seed infection from 20 per cent to 3.4 and 3.1 per cent, respectively.

Foliar Spray: Sprays of Agrimycin were recommended by Srivastava and Rao (1964) for preventing secondary spread.

Jain, *et al*. (1966) observed that dipping the seed for 8 hr in 0.1 per cent Ceresan wet plus Streptocycline at 0.3 g in 10 litres of water had significant effect in controlling the initial infection. The spread of secondary infection could be checked to a great extent by spraying with this antibiotic (3 *g/* 100 l).

The antibiotic Streptocycline had been most commonly recommended chemical for seed treatment and foliar spray against bacterial blight of rice in India. Singh, R.A. (1975, 1976) had shown that severity of bacterial blight in the field can be reduced and yields increased by giving 5 sprays of Agrimycin plus copper oxychloride at 12 day intervals. Fungicides TF-130 and RH-893 were also highly effective.

Resistant varieties: In the absence of specific chemical control measures efforts have been continuing to find out resistant varieties. More than half of the 198 wild type of rice were found resistant to six races of the bacterium.

Srinivasan, *et al.* (1959) had conducted inoculation experiments on 32 varieties and 8 wild species of *Oryza* and found only the varieties BAM-9 and MTO-15 showing some degree of resistance to the disease.

Pavgi, *et al.* (1964) had reported variety N 22 as highly resistant. Among later varieties IR-20, IR-22 and Ratna have mild resistance.

12.4 Soft Rot of Vegetables

Bacterial soft rot of vegetables, including potato is caused by different species *Erwinia* in most parts of Europe, the USA and Canada. It is a common storage and transit disease. In potato, these bacteria not only cause soft rot of tubers but also black leg and wilt of plants in the field. In India, the disease was investigated in some detail by Hingorani and Addy (1953) and the characteristic of the causal organism established with special reference to properties of the causal organism.

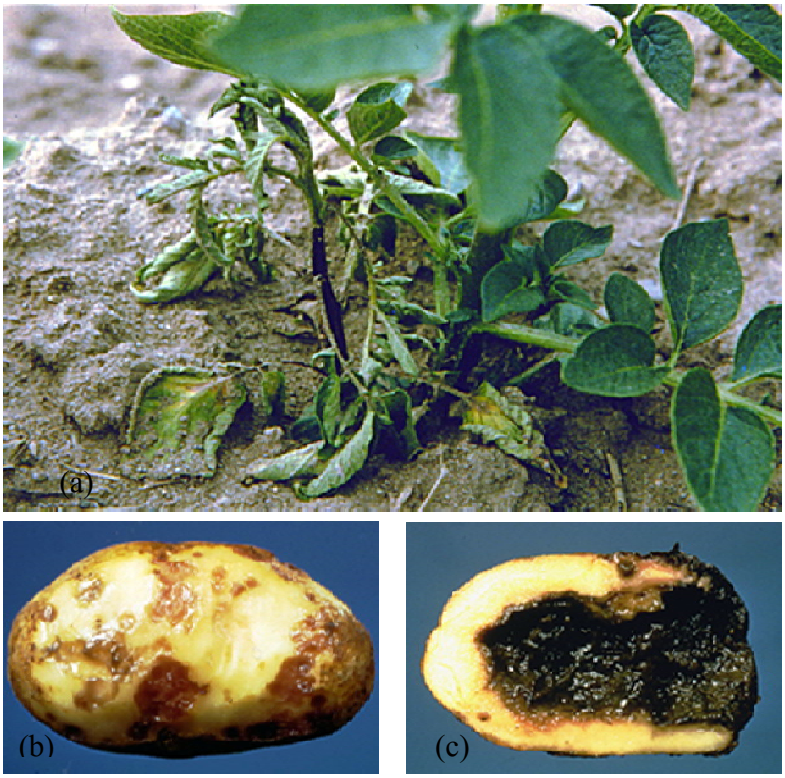

Fig. 12.5 : (a)The blackened stem and wilted leaves are typical of the potato blackleg disease. (b) in poor storage environment, blackleg bacteria present on the surface of tubers,(c) rotting of inner perimedullary region of the potato tuber.

Symptoms

The disease appears sporadically in the field. The plant turns pale green or yellow and soon wilts and ultimately dies. In potato, the typical "black leg" is characterized by a striking brown black or jet black colour of the stem at the soil level. This discolouration usually starts from the old seed tuber. The cortical tissues may shrivel and rot. The plants may achieve normal height but usually they remain dwarfed and stunted. Instead of spreading normally, the branches and leaves show a tendency to grow upwards. The foliage turns light green or yellow, with a slight metallic luster and soon wilts. Curling of leaves similar to that caused by potato leaf curl virus may also be found. Sometimes, young seedlings arising from diseased seed tubers are destroyed before or soon after emergence. As the disease progresses, the entire tuber may decay or the rot may remain partially restricted to the inner perimedullary (or parenchymal) tissue, that is, the tissue inside the vascular ring.

When infection occurs late and tubers have developed, they carry the bacterium to the storage godowns. In a poorly managed potato storage environment, blackleg bacteria present on the surface of tubers can cause extensive decay. When tubers are kept in storage under damp conditions, decay sets in and the tubers are transformed partly or wholly, slowly or rapidly, into a soft decayed pulpy mass which is held together only by the corky epidermis which is not attacked by the parasite. When a tuber showing the rot is cut open the colourless putrified mass turns pink red on exposure to air, rapidly becoming brownish red to brown black with a watery mass of bacterial ooze accompanied by sulphurous odour.

Pathogen

The bacterial species involved in black leg and soft rot of potato are *Erwinia carotovora* subsp. *carotovora* (Jones) Bergey, *et al.* and *Erwinia carotovora* subsp. *atroseptica* (van Hall) Dye. The former is considered the main cause of the disease in India while the latter is reported to be the main cause of potato black leg in temperate regions. *Erwinia aroideae* (Townsend) Bergey, *et al.* that had been earlier described as a distinct species associated with soft rot is synonym of *E. carotovora* subsp. *carotovora.* These species are also known as *Pectobacterium carotovorum* var. *carotovorum* and *P. carotovorum* var. *atrosepticum.*

Chakravarti and Hedge (1971) reported that *Xanthomonas compestris* and *Erwinia atroseptica* were often the cause of soft rot of cabbage in Rajasthan.

The rod-shaped cells of *Erwinia* species measure 0.5-1.0 x 1.0-3.0 μ and are motile by peritrichous flagella. They are Gram-negative, facultative anaerobes and possess strong pectolytic activity. They produce acid from fructose, glucose and sucrose. Gas production is relatively weak or absent. Optimum temperature for growth is 27° to 30° C and maximum temperature for growth varies with isolates. While the subspecies *carotovora* has a maximum temperature for growth at 37°-40° C the subspecies *atroseptica* does not grow at temperatures above 35° C. Both species are tolerant to erythromycin. The G + C content of the DNA of both subspecies ranges from 51 to 53 mol per cent.

Systamatic Position of Pathogen

Kingdom: Bacteria
Phylum: Proteobacteria
Class: Gammaproteobacteria
Order: Enterobacteriales
Family: Enterobacteriaceae
Genus: *Erwinia*
Species: *carotovora* subsp. *carotovora atroseptica*

Disease cycle

Contaminated tubers when used as seed serve as one source of primary inoculum in the field. The soft rot causing species of *Erwinia* are not seed-borne but in potato the bacteria are present in rotting tubers or in tubers which have been contaminated in the stores. Low populations of the bacterium exist in soil, especially when protected in pre-colonized crop debris or as epiphytes in rhizoplane of weed plants, even before potato is planted. Studies have revealed that populations of the bacteria rapidly decline in soil when rotting tubers and plants have been removed.

Insect hosts and vectors are common on culled potato haulms and rotting tubers around potato fields and help in transmission of the bacteria. Infection of tubers occurs through bruises, sun scald, insect wounds, and nematode punctures. *E. carotovora* subsp. *atroseptica* can live in the body of all stages of the seed corn maggot, *Hylemya platura,* and may persist in the intestinal tract of both adult flies and larvae. The fruit fly *(Drosophila melanogaster)* is another insect host and vector. When the soft rot bacteria enter the wounds they feed and multiply at first on the liquids released by the broken cells on the wounded surface.

Rapid multiplication leads to production of increasing amounts of pectolytic and cellulolytic enzymes which breaks down the pectin and cellulose in the middle lamella and cell walls causing maceration of tissues. In moist fields lenticels of tubers are very much enlarged and, if temperature is high, they provide the maximum opportunity for infection. When cut tubers are planted, high soil moisture combined with lack of oxygen hinders cork formation and so helps in infection by the bacteria which can thrive in low oxygen conditions. When tubers are infected the bacteria enter the vascular bundles and by means of pectolytic enzymes bring about the soft rot condition. In a growing plant the bacteria become systemic causing browning of vascular bundles along the shoot, down to the stem base and newly formed tubers.

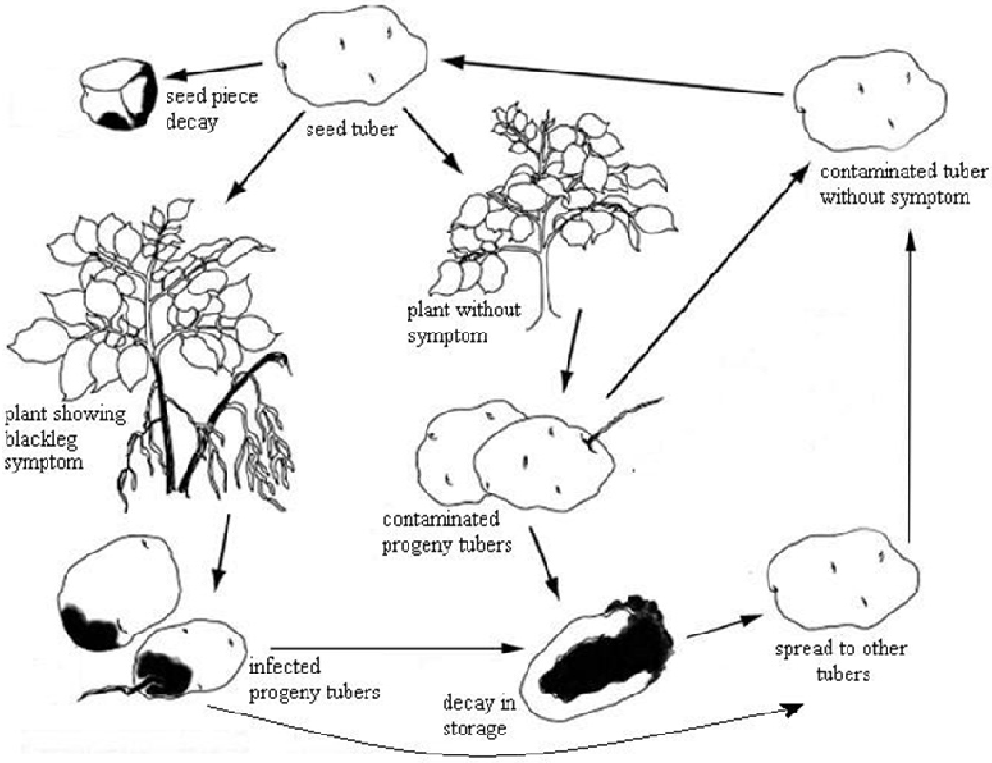

Fig. 12.6 : Disease cycle of soft rot and black leg disease of potato

Incidence of soft rot is negligible in cold storages provided proper low temperature is maintained throughout. Level and source of nitrogen also influence the soft rot incidence in tubers. Heavy nitrogenous manuring of the crop increases the disease incidence. High calcium in tubers enhances structural integrity of cell wall which impedes growth of the bacterium through tissues thus making tubers resistant to soft rot.

Control Measures

Cultural control

Only healthy tubers should be used for seed. When cut tubers are used for seed the cut pieces should be kept at low temperatures (12°-15° C) in a dark room for about 4 days. This allows cork formation and avoids entry of bacteria from soil.

Shallow planting, avoiding early planting when soil moisture and temperature are high, avoiding injury to tubers during tillage and keeping the field well aerated are other precautionary measures.

Black leg affected plants are noticed they should be dug out and burnt.

Before storage the tubers should be washed with chlorinated water. The stores should be kept dry and well aerated and maintained constantly at low temperature. Periodical culling of rotten tubers from the store reduces chances of contamination of healthy tubers.

Chemical control

Alternatively, the cut tubers should be dipped in mercuric chloride solution or organo-mercurial compounds like Agallol, Aretan or Emisan or in stable bleaching powder solution.

Antibiotics have also been recommended for tuber treatment by some scientists. According to Saini and Parashar (1980) stable bleaching powder gives better control than antibiotics.

Soil treatment with bleaching powder (12.5 kg/ha) in furrows is more effective than tuber treatment (500 to I 000 ppm). The bleaching powder (chlorine) treatment inhibits respiratory enzyme activity of the bacteria.

Increasing calcium content of soil and its uptake by plants is recommended as a method of reducing soft rot.

12.5 Crown Gall Disease

Crown gall disease of stony fruits has been known in the USA since 1870. The disease has been reported from the USA, South Africa, New Zealand, Australia, Asia, Canada and Europe. Crown gall can be found most often on stone fruit and pome trees as well as brambles and several species of ornamental plants.

The disease gains its name from the large tumour-like swellings (galls) that typically occur at the crown of the plant, just above soil level. Although it reduces the marketability of nursery stock, it usually does not cause serious

damage to older plants. Nevertheless, this disease is one of the most widely known, because of its remarkable biology. Basically, the bacterium transfers part of its DNA to the plant, and this DNA integrates into the plant's genome, causing the production of tumours and associated changes in plant metabolism.

Cavara from Italy isolated a bacterium from stem galls on grapes and proved its pathogenicity but the bacterium was correctly identified by Smith and Townsend (1907). In India, the occurrence of crown gall was reported by Singh (1943). Durgapal (1971) in Simla, Himachal Pradesh, isolated the organism and established its pathogenicity. In India, it is so far observed on cherry, plum and peach. The disease occurs on apple, peach, apricot, almond, grape, rose, pear, cherry and plum.

Symptoms

Small outgrowths appear initially as small swellings on the root or stem near the soil line and occasionally on aerial portions of the plant. When young the galls often resemble the callus tissue that results from wounding and are soft, spherical, white or flesh-coloured. The galls vary in size from 7 mm to 100 mm in diameter. On woody stems, the galls are hard and corky. As tumors become older, their shape becomes quite irregular, and they turn brown or black. They are generally knobby and knotty and become more cleft as they grow older. Tumors may be connected to the host surface by only a narrow bit of tissue, or may appear as a swelling of the stem, not distinctly separate. The affected plants may become stunted with chlorotic leaves. This is primarily a disease of the parenchyma. Infection results in the rapid proliferation of cells. Unlike other galls, the crown gall is an example of non self-limiting tumour. Several tumors may occur on the same plant and may rot from the surface of the plant completely or partially, possibly developing repeatedly in the same area season after season.

Additional symptoms include stunting, chlorotic leaves and plants may be more susceptible to adverse environmental conditions and secondary infection.

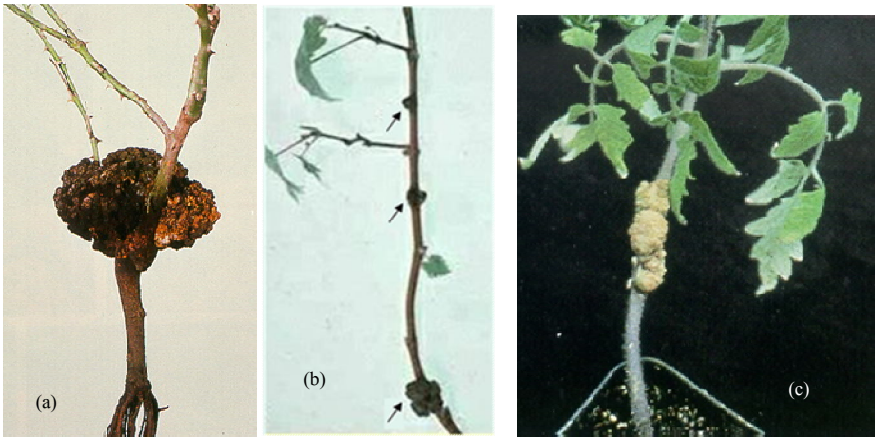

Fig. 12.7 : (a) gall formed on the stem of a rose bush. (b) A series of galls (arrowheads) along a branch of a grapevine (c) on tomato plant

Pathogen

Agrobacterium tumefaciens (E.F. Smith and Towns) Conn. causes crown gall disease of a wide range of dicotyledonous (broad-leaved) plants, especially members of the rose family such as apple, pear, peach, cherry, almond, raspberry and roses. Biosynonym is *Rhizobium.* A separate strain, termed biovar 3, causes crown gall of grapevine. *Agrobacterium tumefaciens* is cosmopolitan in distribution and has wide host range affecting dicotyledonous plants in more than 60 different plant families.

A. tumefaciens is a Gram-negative, non-sporing, motile, rod-shaped bacterium, closely related to *Rhizobium* which forms nitrogen-fixing nodules on clover and other leguminous plants. These bacteria grow aerobically, without forming endospores. The cells are rod-shaped and motile, having one to six peritrichous flagella. Cells are 0.6-1.0 μ by 1.5- 3.0 μ and may exist singly or in pairs.

A. tumefaciens can be effectively isolated for identification from gall tissue, soil or water. Optimal gall tissue for isolation is white or cream-colored from a young, actively growing gall. In culture on carbohydrate-containing media, cells produce large amounts of extracellular polysaccharides, giving colonies a voluminous, slimy appearance. The bacterium can grow on relatively simple medium but its isolation from the soil requires selective media (Patel, 1926).

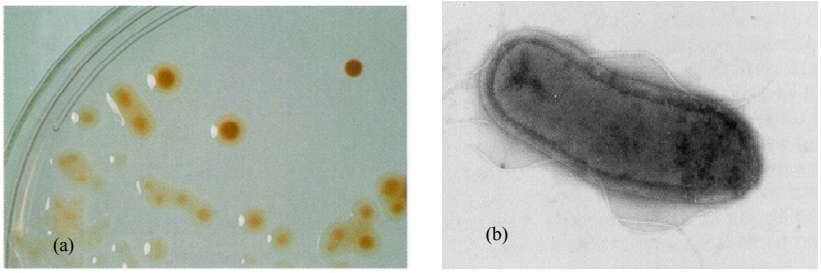

Fig. 12.8 : (a) *A. tumefaciens* colony in culture (b) Single cell

The bacterium consists of a heterogenous group of strains. Strains of *Agrobacterium* are classified in three **biovars** based on their utilisation of different carbohydrates and other biochemical tests. The differences between biovars are determined by genes on the single circle of chromosomal DNA. Biovar differences are not particularly relevant to the pathogenicity of *A. tumefaciens*, except in one respect: biovar 3 is found worldwide as the pathogen of gravevines. But this is almost certainly because biovar 3 has been spread around the world in vegetative cuttings of vines, not by natural mechanisms.

Presence of *A. tumefaciens* cells in a sample plant does not necessarily dictate the existence of the crown gall-inciting strain in the sample. Only cells containing a specific plasmid (the T_i plasmid) can cause disease. *A. tumefaciens* strains lacking the plasmid live as rhizosphere-inhabiting bacteria without causing disease.

Systamatic Position of Pathogen

Kingdom: Bacteria
Phylum: Proteobacteria
Class: Alphaproteobacteria
Order: Rhizobiales
Family: Rhizobiaceae
Genus: *Agrobacterium*
Species: *tumefaciens*

Recently, a reclassification of the species of *Agrobacterium* has been undertaken by use of ribosomal RNA sequencing as a taxonomic tool. The resulting nomenclature places the former species, *A. tumefacians* biovar 1, *A. radiobacter* biovar 1, and *A. rhizogenes* biovar 1, within the new taxon: *Agrobacterium tumefaciens.*

The unique mode of action of *A. tumefaciens* has enabled this bacterium to be used as a tool in plant breeding. Any desired genes, such as insecticidal toxin

genes or herbicide-resistance genes, can be engineered into the bacterial DNA and thereby inserted into the plant genome.

Disease Cycle

Pathogenic strains of *A. tumefaciens* may live saprophytically in soil for up to two years. The bacterium is soil borne. *Agrobacterium tumefaciens* is found commonly on and around root surfaces - the region termed the rhizosphere - where it seems to survive by using nutrients that leak from the root tissues. When a nearby host plant is wounded near the soil line by insect feeding, transplant injury or any other means the bacterium chemotactically moves into the wound site and between host cells. In natural conditions, the motile cells of *A. tumefaciens* are attracted to wound sites by chemotaxis.

These bacteria then stimulate the surrounding host cells to rapidly and irregularly divide. The mechanism this bacterium uses to parasitize plant tissue involves the integration of some of its own DNA into the host genome resulting in changes in plant metabolism. The tumour-inducing principle transforms the normal plant cells to tumour cells.

The bacterium insert a piece of its own DNA into the host cell's chromosomes, causing overproduction of cytokinins and auxins which are plant growth regulators and opines which serve as nutrients for the pathogen. The resulting tissue is undifferentiated with a white or cream color and cells may have one or more nuclei. This tissue continues to enlarge and a tumor is formed on the root or stem of the plant, depending on original wound site. The bacteria occupy the intercellular spaces around the periphery of the gall and are not found in the center of the enlarging tumor. The tumor is not protected by an epidermis, leaving the tissue susceptible to secondary pathogens, insects and saprophytes. Degradation of the tumor by secondary invaders causes brown or black discoloration and releases *A. tumefaciens* cells back into the soil to be carried away with soil or water, or remain in the soil until the next growing season. In perennial plants, part of the infected tissue may remain alive and inhabited by *A. tumefaciens*, which, even if the tumor has sloughed off, can persist to cause a new tumor the following season in the same place.

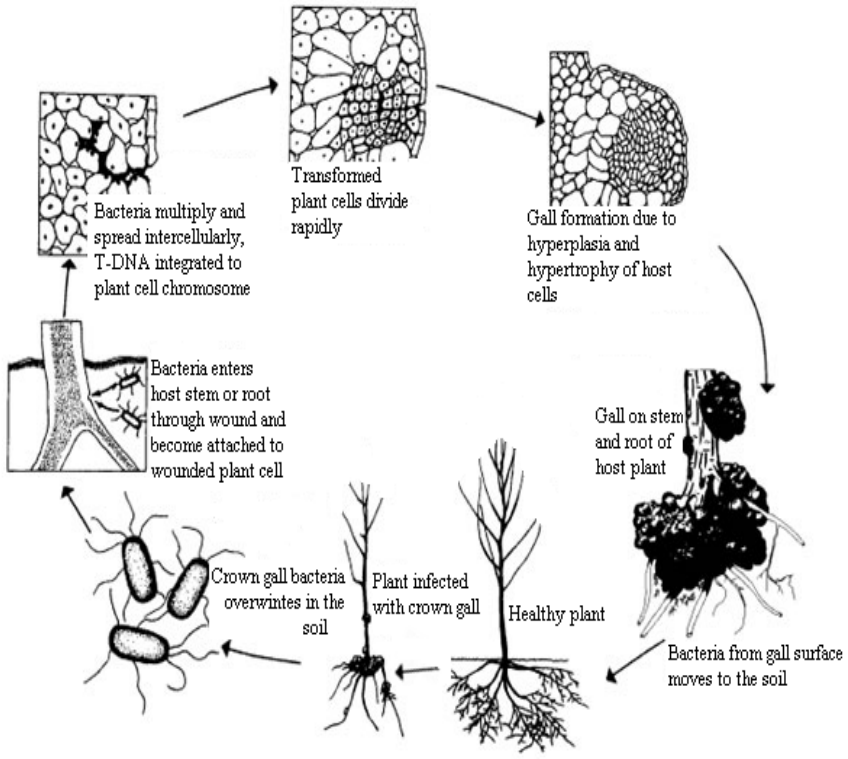

Fig. 12.9: Disease cycle of Crown gall disease caused by *A. tumefaciens*

Control Measures

Cultural Control

Crop sanitation is necessary to avoid the introduction of infected material into the nursery stock.

Introduction of pathogenic *A. tumefaciens* strains can be avoided by thorough inspection of nursery stock for crown gall symptoms. Susceptible varieties should not be planted in soils known to be infested with the pathogen. These soils should be planted in a monocotyledonous crop like corn or wheat for several years. Nursery stock should be certified crown gall-free and should be budded rather than grafted. If the threat of crown gall exists, all practices that wound tissue should be avoided and chewing insects should be controlled.

Chemical Control

In general, bacterial diseases of plants are very difficult to control owing to the lack of effective chemicals. The most effective alternative is the use of copper, which is potentially phytotoxic.

Antibiotics could be used, but they are expensive. Antibiotics, such as Vancomycin, Aureomycin, Streptomycin and Terramycin have been tried to

prevent crown gall disease.

Some curative properties are exhibited by a commercially available mixture of 2,4-xylenol and metacresol in an oil-water emulsion when painted directly on established tumors. But this is rarely used due to labor and time constraints.

Biological Control

A. tumefaciens prompted the first successful development of a biological control agent. Allen Kerr of Australia discovered the biocontrol system by isolating non-pathogenic strains of *Agrobacterium radiobacter* from disease sites and testing their ability to compete with pathogenic strains in mixed inoculations. Several non-pathogenic strains helped to reduce infection, but one strain in particular, *A. radiobacter* **strain K84,** completely prevented disease when added to wound sites at a 1:1 ratio with cells of *A. tumefaciens*.

Preventative treatment of seeds or transplants with the non-pathogenic biocontrol organism *Agrobacterium radiobacter* is a relatively inexpensive and effective means of managing the development of crown gall in commercial operations. This strain is the one that is marketed globally. It is supplied commercially on agar plates or in a peat substrate and it is used by suspending the bacterial cells in water, then dipping seeds, seedlings or cuttings in this suspension before planting. Application of this antagonist by soaking seeds or dipping transplants can prevent infection by most strains of *A. tumefaciens* due to the production of the antibiotic agrocin 84 by strain K84 of *A. radiobacter*.

Biological control with *A. radiobacter* is only as a preventative treatment, not to cure infections, so it is applied at a high population level to protect any wound sites against pathogenic invasion.

12.6 Angular Leaf Spot of Cotton

Angular leaf spot also known as black arm disease is the most serious bacterial disease of cotton. The disease was first reported from Alabama state of U.S.A. in 1891 and is now found in all major cotton growing regions of the world including South America, Russia, Sri Lanka, China, Australia, Egypt, Sudan etc and other African countries. In India the disease was first observed in Tamil Nadu in 1918 and is now known to occur in almost all cotton growing areas of Maharashtra, Madhya Pradesh, Andhra Pradesh and Uttar Pradesh. Severe epidemics of the disease were reported during 1948-1952 in Tamil Nadu which resulted in rejection of many very promising cotton varieties of all the four species of *Gossypium.* Annual losses due to this disease vary from 5 to 25 %.

Symptoms

The disease has four distinct phases depending on the plant part affected: angular leaf spot (leaf infection), black arm (stem infection), boll rot (boll infection) and seedling blight (seedling infection).

The earliest symptom of the disease is seedling infection which is seen in the cotyledons of germinating seeds. Later the bacterium attacks all aerial parts of the plant at different stages of plant growth. Minute, water-soaked spots appear on the under surface of the cotyledons as earliest symptom. These water-soaked spots increase in diameter, turn brown to black and form irregular patches distorting the shape of the cotyledons causing them to dry and wither. The disease spreads to new leaves formed and the seedlings may ultimately collapse and die.

In the second stage or leaf infection stage which is the most common and conspicuous symptom is angular leaf spot which begins with dark-green, water-soaked spots, initially more clearly visible on the underside of the leaf lamina; the spots are angular in shape, being delimited by the smaller veins. These spots increase in size, become angular, bound by small veinlets of the leaf and turn brown to black. Sometimes, large patches are formed due to coalescing of a number of small spots leading to death and shedding of leaves. The infection may spread to petioles causing them to collapse. In the affected areas large quantities of bacteria slime are exuded which form a dry film on the brown lesions. Sometimes infection on the leaf occurs as water-soaked tissue, which later turns black, on either side of the main veins. This is referred to as vein blight or Black vein and can occur together with, or occasionally in the absence of, angular leaf spot

In older plants, lesions on stem, petioles, and fruiting branches are dark brown to sooty black. The affected stems show cracks and gummosis. The lesions can girdle the main branches causing them to break, with the loss of leaves and fruiting branches. This phase of the bacterial blight syndrome is known as blackarm because of the blackened appearance of the affected petioles and branches.

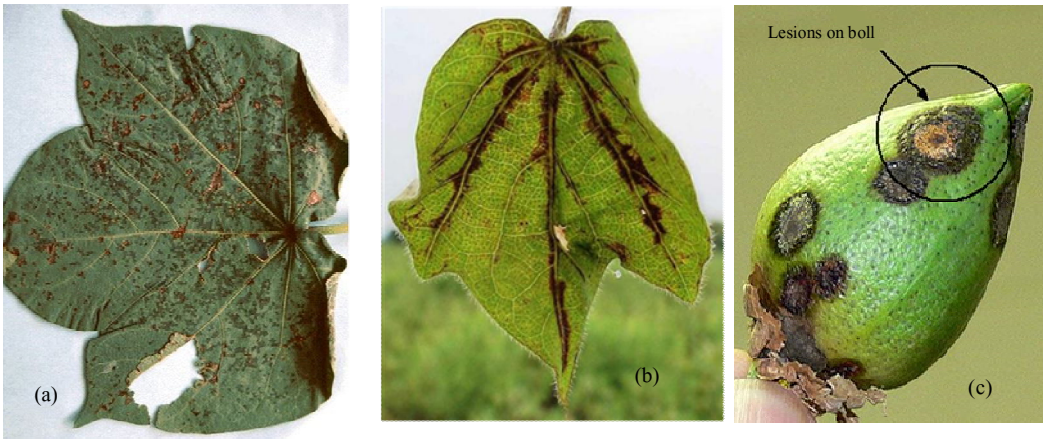

Fig. 12.10: Symptoms of angular leaf spot of cotton; (a) water soaked lesions on leaf, (b) vein black symptom & (c) boll rot

On the bolls or fruits the disease is characterized by the appearance of water-soaked lesions on the surface (boll rot stage). Bacterial boll rot begins as roughly spherical water-soaked spots on the boll surface which can expand to >1 cm in diameter on susceptible cultivars. These lesions turn brown and finally black and are invariably sunken. Young infected bolls fall down prematurely. If they mature, lint is of not much commercial value. The bacterium within the boll passes along the fibers and infects the seed externally. It may also reach the interior of the seed either through micropyle or through punctures.

The Pathogen

The bacterium *Xanthomonas campestris* pv. *malvacearum* (Smith) Dye is the causal agent of angular leaf spot disease of cotton. (synonym *Xanthomonas axonopodis pv. malvacearum*)

There are 32 races of the pathogen which vary in their pathogenicity on different species of *Gossypium.* In India, 26 of these races have been identified. Race 10 is reported to be the most common in India.

The bacterium is rod-shaped measuring 1.3-2.7 x 0.3-0.6 μ. The bacterium is non endospore forming, encapsulated, Gram-negative and motile by a single polar flagellum. Growth is aerobic. Colonies on beef agar are pale yellow, round, thin, raised, smooth and shining. Optimum temperature for growth in culture varies from 25° to 32° C. Maximum temperature for growth is 42° C and minimum 6° C Thermal death point is 50° C.

Systamatic Position of Pathogen

Kingdom:	Bacteria
Phylum:	Proteobacteria
Class:	Gammaproteobacteria
Order:	Xanthomonadales
Family:	Xanthomonadaceae
Genus:	*Xanthomonas*
Species:	*campestris* pv. *malvacearum*

Disease cycle

The main source of primary inoculum is seed. The infected seeds lying dormant in the field and germinating in the crop season prior to the main crop also serve as source of primary inoculum. The bacterium may be present as slimy mass on the fuzz or inside the seed. On germination of such seeds, the bacterium moves to cotyledons and then maintains a resident population on the first and second leaves but not on the third leaf. In favourable weather the inoculum from this source is spread to new leaves and further spread continues. Leaves are infected mainly through stomata. Secondary spread is through wind splashed rain and dew.

Infected cotton bolls, leaves and twigs present on soil surface also form an important source of carryover of the bacterium. The bacterium may remain alive in dried leaves for 17 years and in dry or moist soil for 8 days at 21 °-33° C. However, infected stalks buried in moist soil cause death of the bacterium.

Primary infection is favoured by a temperature of 30° C. High humidity and moderate temperature (28° C) favour development of the disease. Soil temperature and moisture at the time of sowing and a few days after are important. Secondary infection is better at 35° C. Presence of moisture is very important for secondary infection during the first 48 hours. Highest progress of the disease occurs when there are frequent rains followed by cloudy weather. Dry and hot weather retards development of the disease.

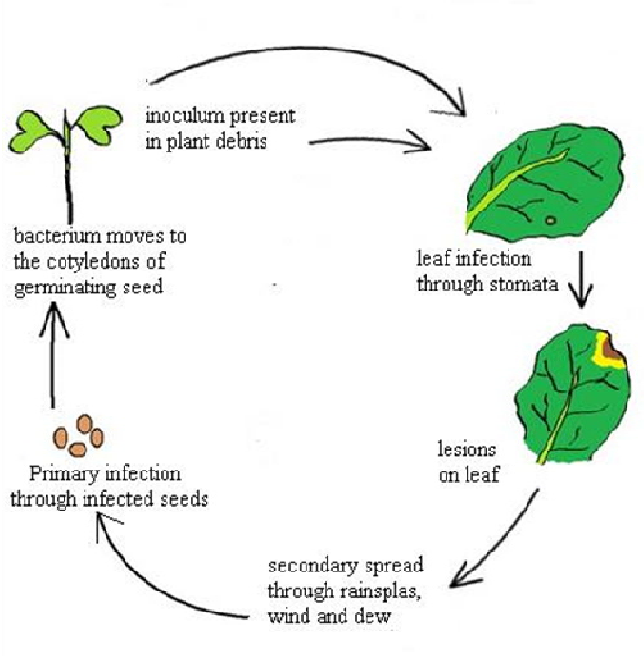

Fig. 12.11: Disease cycle of Angular leaf spot of cotton

Thurbaria thespesoides, Eriodendron anfructosum, Jatropha curcas and *Lochnera pusilla* are reported to be collateral hosts of the bacterium but their role in disease cycle is not yet established.

Control Measures

Cultural control

Sanitation: Removal and destruction of diseased plant debris is recommended to reduce the soil-borne inoculum. Destruction of possible alternate or collateral hosts is also essential.

Cultural practices: Deep ploughing after harvest buries the infected stalks and, thus, reduces survival ability of the bacterium in soil. Pre-sowing irrigation to enable the left over seeds germinate, followed by ploughing and then planting of the main crop is followed in some countries. Crop rotation, late sowing, early thinning, good tillage and early irrigation help in reducing the disease incidence.

The bacterium is known to live in the seed for about a year or so and, therefore, ageing of the seed for two years before sowing has also been recommended.

Chemical control

Seed-borne inoculum can be eliminated by seed treatment. External inoculum on the seed is destroyed by delinting of seed with concentrated sulphuric acid. Seeds are immersed in acid for 10-15 min, then rinsed thoroughly by

suspending in water to remove acid and finally dried and treated with organo-mercurial compounds such as Agrosan GN, Ceresan, etc. This treatment does not destroy the internally seed-borne inoculum.

Treating seeds with antibiotics like streptomycin eradicates the internally seed-borne inoculum. The systemic fungicides Vitavax and Plantvax are more effective than antibiotics.

Hot water treatment of seed at 56° C for 10 min destroys the external as well as internal inoculum without affecting seed viability.

The secondary spread can be checked by regular sprays with copper fungicides (0.2-0.3 %). First spray is given when the crop is 5-6 weeks old. In all, 3-6 sprays, depending on the severity of the disease, are given at 15 days interval. Mathur, *et at.* (1973) had reported that seed treatment with Agrimycin (3 g/40 kg seed) and its spray (25 ppm) are most promising in controlling the black arm of cotton. In Haryana, Chauhan, *et at.* (1983) could effectively control bacterial blight of cotton by 3 sprays of a mixture of Agrimycin (0.01 %) + Blitox (0.2 %) at 40-50 days, 70-80 days and 85-90 days after planting.

Addition of potash to soil helps in reducing the disease incidence.

Control through development of resistant varieties is possible and provides the best and most effective preventive measure. *Gossypium herbaceum* and *G. arboreum* were considered to be practically immune whereas *G.* barbedanse, *G. herbaceum var. typicum* and *G. hirsutum* were considered susceptible. Varieties known to be resistant are HC-9, BJA-592, 101-102B and Reba-B.50.

12.7 Red Strip of Sugarcane

The disease is common in all the sugarcane growing areas of the world. In India it was first reported in 1933. Since then its occurrence has been recorded in Bihar, Uttar Pradesh, Punjab, Maharashtra and Tamil Nadu. In certain areas, as in Bihar, the disease causes considerable damage to young plants.

Symptoms

The disease first appears as water-soaked elongated streaks which soon become chlorotic and carry dark-red stripes which are 0.5-1 mm in width and 5-100 mm or more in length. Sometimes two or more strips coalesce to form larger bands. The lower half of the leaf is more affected than the tip. When young shoots are affected symptoms of top rot appear. The growing point of the shoot shows many dark-red stripes with water-soaked appearance and undergoes rotting. The disease proceeds downwards killing the terminal buds and the leaves.

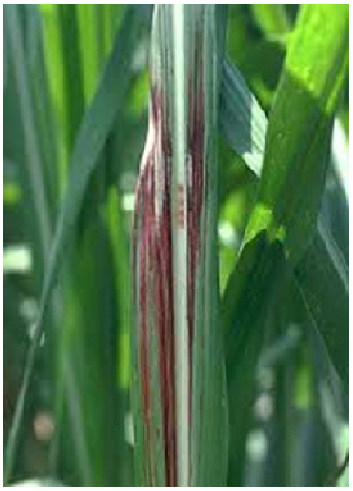

Fig. 12.12: Symptoms of red stripe of sugarcane

Pathogen

Pseudomonas rubrilineans (Lee, *et al.)* is the causal agent of the disease. The bacterium is a rod measuring 0.7 x 1.6 μ and occasionally forming chains. It produces no endospores and no capsule. It is motile by 1-3 polar flagella. Gram-stain reaction is negative. The bacterium is facultative anaerobe. Colonies on beef extract agar are light buff to yellow, opalescent, small, smooth, glistening. Thermal death point is 51 °-52° C.

Systamatic Position of Pathogen

Kingdom: Bacteria
Phylum: Proteobacteria
Class: Betaproteobacteria
Order: Burkholderiales
Family: Comamonadaceae
Genus: *Pseudomonas*
Species: *rubrilineans*

Disease cycle

The pathogen remains viable in the soil and infected plant residues. Use of setts from diseased plants also serves as a source of primary inoculum in new fIelds. Besides this, the bacterium also survives on sorghum, pearlmillet, maize, fingermillet and other species of *Saccharum*. Several grasses, including ragi and bajra, have been reported to be infected by the bacteria and these hosts may also play a role in the perpetuation and spread of the pathogen. These plant acts

as collateral host for the bacterium. Collateral hosts may serve as agents to carry over of the bacterium from season to season. The secondary spread is mainly through rainsplash, irrigation water and insects.

Infection starts through wounds and through stomata. The bacterium is confined to young tissues of the host. It rapidly multiplies in the parenchymatous tissues and moves to vascular bundles. The bacterial slime on the leaves is spread by wind-splashed rain drops which act as secondary source of infection.

Continuous ratooning and prolonged rainy weather with low temperature (25^0 C)

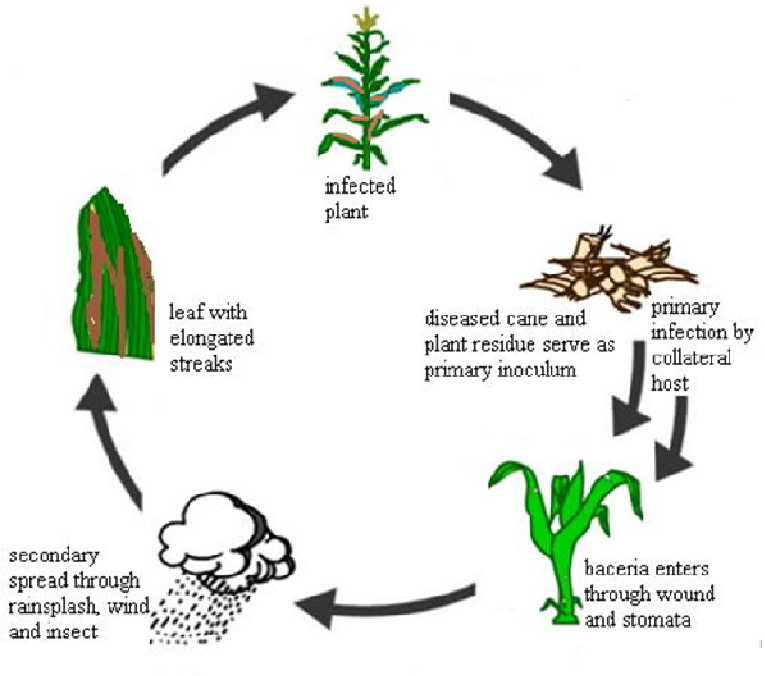

Fig. 57: Disease Cycle of Red strip of sugarcane

Control Measures

Once the disease sets in it is very difficult to control. Systematic cutting down and burning of the affected shoots reduces the spread. Use of resistant cultivars is the best method to avoid this disease. CO cultivars 6805, 7202, 7321, 7537, 7642 and 8005 and cv. Ponda are reported to be resistant.

12.8 Summary

The bacterial brown rot of potato is common in tropical and subtropical regions of the world. The disease damages the crop through direct killing of the plants

in the field and through rotting of tubers in stores. The characteristic symptoms of the disease may appear at any stage and include stunting, yellowing of the lower foliage, sudden wilting and finally collapse of the entire plant. Due to browning of the xylem in the vascular bundles stems may appear streaked. The disease is also known as *ring disease* due to the fact that a brown ring is formed in the tuber due to discolouration of the vascular bundles. Bacterial wilt of potato is caused by *Ralstonia solanacearum* (Smith) which is a gram-negative motile rod. Bacteria is soil borne having extensive host range. Soil is the source of primary inoculum because pathoge survive in seed tubers. High soil moisture favour the disease.Crop rotation, use of infection free seeds, sanitation and moisture control are cultural control of the disease. Resistant varieties and genetic-engineering and technology are also used.

Bacterial Leaf Blight of Rice has 2 phases (a) Blight (b) "Kresek" or wilting symptoms. The disease is a typical vascular wilt, leaf blight being only the mild phase resulting from secondary infections. Symptoms of leaf blight is characterized by linear, yellow to straw coloured with wavy margins, generally on both edges, rarely on one edge of the leaf. Damage is due to the partial or total blighting of leaves or due to complete wilting of the affected tillers leading to unfilled grains. In the wilt or "Kresek" phase crop may dry up completely before seed maturation. G-negative, rod shaped uniflgallate bacterium *Xanthomonas campestris* pv. *oryzae* (Ishiyama) Dye.is the pathogen. Many grass species and diseased wild rice growing in ponds may constitute the chief source of primary inoculum. Secondary spread is brought about through wounds and stomata. Seed traeatment and foliar spray with Agrimycin, treatment with hot water, seed treatment with streptocycline can control the disease.

Bacterial soft rot of vegetables is caused by different species *Erwinia*. In this disease plant turns pale green or yellow and soon wilts and ultimately dies. In potato, the typical "black leg" is characterized by a striking brown black or jet black colour of the stem at the soil level. As the disease progresses, the entire tuber may decay or the rot. The bacterial species involved in black leg and soft rot of potato are *Erwinia carotovora* subsp. *carotovora* (Jones) Bergey, *et al.* in India. The bacterium is rod shaped, Gram-negative, facultative anaerobe and are motile by peritrichus flagella. Contaminated seed tubers serve as one source of primary inoculum. Infection of tubers occurs through bruises, sun scald, insect wounds, and nematode punctures. Sanitation, treating cut tubers in mercury chloride or organo murcurial compounds can help to reduce the

incidence of disease.Soil treatment with bleaching powder is found more effective control.

Crown gall disease gains its name from the large tumour-like swellings (galls) that typically occur at the crown of the plant, just above soil level. When young the galls often resemble the callus tissue that results from wounding and are soft, spherical, white or flesh-coloured. As tumors become older, their shape becomes quite irregular, and they turn brown or black. The affected plants may become stunted with chlorotic leaves. *Agrobacterium tumefaciens* (E.F. Smith and Towns) Conn. causes crown gall disease of a wide range of dicotyledonous plants, especially members of the rose family. *A. tumefaciens* is a Gram-negative, non-sporing, motile, rod-shaped bacterium. The bacterium gain entry into plant trough wounds near soil line. Crop sanitation, use of antibiotics such as Vancomycin, Aureomycin, Streptomycin and Terramycin is useful control measures of the disease. Preventative treatment of seeds or transplants with the non-pathogenic organism *Agrobacterium radiobacter* has been suggested as biocontrol.

Angular leaf spot disease has four distinct phases depending on the plant part affected: angular leaf spot (leaf infection), black arm (stem infection), boll rot (boll infection) and seedling blight (seedling infection). Minute, water-soaked spots appear on the under surface of the cotyledons as earliest symptom. These water-soaked spots increase in diameter, turn brown to black and form irregular patches distorting the shape of the cotyledons causing them to dry and wither. In the second stage or leaf infection stage symptom is angular leaf spot which begins with dark-green, water-soaked spots are formed. These spots increase in size, become angular, bound by small veinlets of the leaf and turn brown to black.. Sometimes black lesions appear on either sides of vein. This is referred to as vein blight or Black vein. Black lesions can girdle the main branches or petiole causing them to break. This phase is known as blackarm. Boll rot stage is characterized by the appearance of water-soaked lesions on the surface of the boll. The bacterium *Xanthomonas campestris* pv. *malvacearum* (Smith) Dye is the causal agent of angular leaf spot disease of cotton. The bacterium is rod shaped, non endospore forming, encapsulated, Gram-negative and motile by a single polar flagellum. The infected seeds lying dormant in the field and germinating in the crop season prior to the main crop also serve as source of primary inoculum. Infected cotton bolls, leaves and twigs present on soil surface also form an important source of carryover of the bacterium. Sanitation, seed treatment with with organo-mercurial compounds and antibiotics are

effective control measures.

Red stripe of sugarcane disease first appears as water-soaked elongated streaks which soon become chlorotic and carry dark-red stripes. The lower half of the leaf is more affected than the tip. When young shoots are affected symptoms of top rot appear. The growing point of the shoot shows many dark-red stripes with water-soaked appearance and undergoes rotting. The disease proceeds downwards killing the terminal buds and the leaves. *Pseudomonas rubrilineans* is the causal agent of the disease. The bacterium is a rod, nonendospore forming and motile with 1-3 polar flagella. The bacterium is facultative anaerobe and Gram-negative. Use of setts from diseased plants also serves as a source of primary inoculum in new fIelds. Besides this, the bacterium also survives on sorghum, pearlmillet, maize, fingermillet, other species of *Saccharum* and several grasses. Infection starts through wounds and through stomata. Systematic cutting down and burning of the affected shoots reduces the spread. Use of resistant cultivars is the best method to avoid this disease.

12.9 Glossary

- **Incubation period :** The period of timebetween penetration of host by a pathogen and the first appearance of symptoms on the host.
- **Plasmid :** Aself replicating, extra-chromosomal, hereditary circular DNA found in many bacteria.
- **Resistant :** Possessing qualities that hinder the development of a given pathogen.
- **Serology :**A method using specificity of the antigen-antibody reaction for the detection and identification of antigenic substances and the organisms that carry them.

12.10 Self-Learning Exercise

Section A : (Very Short Answer Type Questions)

1. Who recognized pathogenic ability of bacteria?
2. Write the pathogen of brown rot of potato.
3. What is “Kresek” phase of blight of rice?
4. What is "black leg"?
5. Write the systematic position of *Agrobacterium tumefaciens.*

Section B : (Short Answer Type Questions)

1. Write note symptoms of Bacterial Leaf Blight of Rice.
2. Write the disease cycle of soft rot of vegetables.
3. Write a short note disease cycle of Crown gall disease.
4. Write note characteristic symptoms of Angular leaf spot disease of cotton.
5. Write note on pathogen of Red stripe of sugarcane.

Section C : (Long Answer Type Questions)

1. Explain the disease cycle of soft rot and black leg disease of potato.
2. Write detail note on Crown gall disease.
3. Write detail note on bacterial leaf blight of rice.

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Unit-13

Viral Disease- I:

Classification and General

Structure of the Unit:

13.0 Objectives

After going through this unit you will be able to:

- Understand various viruses as disease causing agent on plants.
- Explain disease symptoms.
- Understand and explain various methods of transmission of viruses.
- Explain disease control measures.

13.1 Introduction

Viruses are more dangerous than fungi and bacteria as because of their chemical nature and special type of parasitism; it is much more difficult to control viruses.

Beijerinck (1898) was the first to use the term virus .The term 'virus' had long been used for any slimy liquid poison, venom, or infectious matter.With advances in knowledge of the nature of virus particles more precise concepts about viruses emerged. According to Mathews (1981) virus is a set of one or

more template molecules normally encased in a protective coat or coats of protein or lipoprotein, which is able to organize its own replication only within suitable host cells. Bos (1983) defined virus as an infectious agent often causing disease, invisible with the light microscope (submicroscopic), small enough to pass through a bacterial filter, lacking a metabolism of its own, and depending on a living host cell for multiplication.. According to **Bawden (1964)** "Viruses are obligatory parasitic pathogens with dimensions of less than 200 milli-microns (mμ). They are submicroscopic, infective entities that multiply only intracellularly and are potentially pathogenic'.

At present viruses are considered as living organism and widely acceptable definition of virus may be given as under:

'Viruses are strictly intracellular and potentially pathogenic entities with an infectious phase, possessing only one type of nucleic acid (RNA or DNA) multiplying in the form of nucleic acid only and unable to grow or undergo binary fission'. Viruses are small packages of host-alien genetic information of one type (RNA or DNA), either in one strand or in a few segments encapsulated together or separately and enclosed in a coat of one or more types of protein, sometimes with an extra coat (envelop) and some other constituents. Modern concept about the plant viruses is that 'viruses are nucleo-proteins in nature behaving as living organisms *in vivo* and as chemicals or molecules *in vitro*.

13.2 Classification of Viruses

- In the early 1930s standardization of names attracted the attention of botanists and micro-biologists. Originally, virus names were *common names* or *vernacular names*
- James Johnson in 1927 proposed that the viruses should be named only after their host, not symptoms, since the latter are variable on different hosts infected with the same virus.
- In 1937, K.M. Smith proposed Latin names like *Nicotiana virus* 1 for Tobacco virus 1.
- In 1939, P.O. Holmes had proposed a binomial system for virus nomenclature.
- In 1970, H.P. Hansen proposed another system based on the fundamental characters of the virus.

- The requirement for a sound classification system for plant viruses become imperative and this requirement wasmet by the appointment of a committee to investigate virus taxonomy at the International Congress of Microbiology held in Moscow in 1966. The committee so constituted became known as International Committee on Taxonomy of Viruses (1CTV).The International Committee on Taxonomy of Viruses (ICTV) has framed 20 rules for virus nomenclature. Among others it recognizes common names, their international meaning and use of existing names.
- ICTV database (ICTVdb)- A centralized repository of virus information
- ICTV collect information from databases around the world.
- Facilitated the job of accurate identification and diagnosis of new and important virus diseases.
- Until recently, many viruses were not associated with specific taxa and most plant viruses were categorized into groups rather than families or genera.
- As more sequence data have accumulated, more viruses have been placed in newly described or existing taxa,
- And the "group" designation has given way to categorization into families or genera of existing families.
- Some viruses are still are not associated with named families, but with the current taxonomic framework solidly in place, this number is being reduced with each report of the ICTV.

Rules of orthography changed in 1999 to require italics, but no true binomial – only modified binomial e.g.

- Family *Reoviridae*
- Genus *Orbivirus*
- Species *Bluetongue virus* (24 named strains: BTV-1 to BTV-24)
- In 2000 7th report of the ICTV was published
- 56 families, 9 subfamilies, 233 genera and 1550 virus species
- Includes retrotransposons, satellites, viroids, prions
- In 2005 8th report of the ICTV was published

As a result of work of this committee and its plant virus sub-committee, a system of plant virus classification was introduced based on characteristics of virus particle morphology, type and quantity of nucleic acid, genome structure and type of vector. This classification was widely accepted by most plant virologist.

Table -13.1 : Virus classification approved by ICTV (Francki, *et al.*, 1991)

Classification	Genome properties*	Number of viruses **
GROUPS		
a) *Rod-shaped rigid particles*		
1 . Tobamovirus	ssRNA (1)	14
2. Tobravirus	ssRNA (2)	3
3. Furovirus	ssRNA (2-4)	11
4. Hordeovirus	ssRNA (3)	4
b) *Filamentous particles*		
1. Capillovirus	ssRNA(l)	4
2. Carlavirus	ssRNA(l)	56
3. Closterovirus	ssRNA(l)	22
4. Potexvirus	ssRNA(I)	39
5. Potyvirus	ssRNA(lor2)	153
6. Tenuivirus	ssRNA (4)	7
c) *Isometric parnicles*		
1. Carmovirus	ssRNA(l)	17
2. Luteovirus	ssRNA(l)	21
3. Sobemovirus	ssRNA(l)	16

4. Tombusvirus	ssRNA (1)	12
5. Tymovirus	ssRNA (1)	19
6. Maize chlorotic dwarf virus	ssRNA(l)	3
7. Marafivirus	ssRNA (1)	3
8. Necrovirus	ssRNA(I)	4
9. Parsnip yellow fleck virus	ssRNA(l)	3
10. Comovirus	ssRNA (2)	13
1 1 Dianthovirus	ssRNA (2)	3
12. Nepovirus	ssRNA (2)	36
13. Fabavirus	ssRNA (2)	3
14. Pea enation mosaic virus	ssRNA (2)	1
15. Bromovirus	ssRNA (3)	6
16. Cucumovirus	ssRNA (3)	4
17. Caulimovirus	dsDNA(l)	17
18. Cryptovirus	dsDNA (2)	31
d) *Quasi-isometric to Bacilliform particles*		
1. Itarvirus	ssRNA (3)	20
2. Alfalfa mosaic virus	ssRNA (3)	1
e) *Geminate particles*		
1. Geminivirus	ssDNA(l or 2)	
Subgroup I		10
Subgroup 11		5
Subgroup III		33

f) *Bacilliform particles*		
1. Badnavirus (Commelina yellow mottle virus)	dsDNA(l)	14
FAMILIES		
Rhabddoviridae	(-) ssRNA (1)	
Subgroup A		9
Subgroup B		4
Subgroup C (non-enveloped particles)		4
Unclassified species		68
Bunyaviridue	(-)ssRNA (3)	
Tospovirus genus		
(Tomato spotted wilt virus)		1
Reoviridae	dsRNA (10-12)	
Phytoreovirus genus		3
Fijivirus genus		3
Unnamed genus		2

Current Classification of Plant Viruses

Plant viruses have recently been classified into families and genera. This classification is now recognized by the International Code of Nomenclature and Taxonomy of Viruses. A partial list is as under :

Table 13.2 : Current Classification of Plant Viruses

Type of genome	Family	Genus	Type species
ssDNA	*Geminiviridue*	*Mastrevirus*	Maize streak virus
		Curtovirus	Beet curly top virus
		Begomovirus	Bean golden mosaic virus
ssDNA	-	*Nanpvirus*	Subterranean clover stunt virus
Reverse transcribing	*Caulimoviridae*	*Caulimovirus*	Cauliflower mosaic virus
		"SbCMV-like"	Soybean chlorotic motile virus
		"CsVMV-like"	Cassava vein mosaic virus
		"PVCV-like"	Petunia vein yellow mottle virus
		Budnavirus	Commelina yellow mottle virus
		"RTBV-like"	Rice tungro baciliform virus
Reverse transcribing	*Pseudoviridae*	*Pseudovirus*	Saccharomyces cerevisiae Tyl virus
Reverse transcribing	*Metaviridae*	*Metavirus*	Saccharomyces cerevisiae Ty3 virus
ds RNA	*Reoviridae*	*Phytoreovirus*	Wound tumor virus
		Fijivirus	Fiji disease virus
		Oryzavirus	Rice ragged stunt virus

ds RNA	*Partutvirdiae*	*Alphacryptovirus*	White clover cryptic virus 1
		Betacryptovirus	White clover cryptic virus 2
ds RNA	-	*Varicosavirus*	Lettuce big-vein virus
(-) ss RNA	*Rhabdoviridae*	*Cytothabdovirus Nucleorhabdovirs*	Lettuce necrotic yellow virus Potato yellow dwarf virus
(-) ss RNA	*Bunyaviridae*	*Tospovirus*	Tomato spotted wilt virus
(-) ss RNA	-	*Tenuivirus*	Rice stripe virus
(-) ss RNA	-	*Ophiovirus*	Citrus psorosis virus
(+) ss RNA	*Bromoviridae*	*Bromovirus*	Brome mosaic virus
		Cucumovirus	Cucumber mosaic virus
		Alfamovirus	Alfalfa mosaic virus
		Ilarviruss	Tobacco streak virus
		Oleavirus	Olive latent virus 2
(+) ss RNA	*Closteroviridae*	*Closterovirus*	Beet yellow virus
		Crinivirus	Lettuce infectious yellows virus
(+) ss RNA	*Comoviridae*	*Comovirus*	Cowpea mosaic virus
		Nepovirus	Tobacco ringspot virus
		Fabavirus	Broad bean wilt virus 1
(+) ss RNA	*Luteoviridae*	*Luteovirus*	Barley yellow dwarf

			virus-PAV
		Polerovirus	Potato leafroll virus
		Enamovirus	Pea enation mosaic virus-1
(+) ss RNA	*Polyviridae*	*Potyvirus*	Potato virus Y
		Rymovirus	Ryegrass mosaic virus
		Bymovirus	Barely yellow mosaic virus
		Macluravirus	Maclura mosaic virus
		Ipomovirus	Sweet potato mild mottle virus
		Tritimovirus	Wheat streak mosaic virus
(+) ss RNA	*Sequiviridae*	*Sequivirus*	Parship yellow fleck virus
		Wajkavirus	Rice tungro spherical virus
(+) ss RNA	*Tombusviridae*	*Tombusviria*	Tomato bushy stunt virus
		Carnovirus	Carnatioon mottle virus
		Necrovinis	Tobacco nercosis virus A
		Machlomovirus	Maize chlorotic mottle virus
		Dianthovirus	Carnation ring spot virus
		Avenavirus	Oat chlorotic stunt

			virus
		Aureusvirus	Pothos mosaic virus
(+) ssRNA	-	*Tobravirus*	Tobacco rattle virus
(+) ss RNA	-	*Tobamovirus*	Tobacco mosaic virus
(+) ss RNA	-	*Hordeivirus*	Barley stripe mosaic virus
(+) ss RNA	-	*Furovirus*	Soil-borne wheat mosaic virus
(+) ss RNA	-	*Pomovirus*	Potato mop-top virus
(+) ss RNA	-	*Pecluvirus*	Peant clump virus
(+) ss RNA	-	*Benyvirus*	Beet necrotic yellow vein virus
(+) » RNA	-	*Sobemovirus*	Southern bean mosaic virus
(+) ss RNA	-	*Marafivirus*	Maize rayado fino virus
(+) ss RNA	-	*Umbravirus*	Carrot mottle virus
(+) ss RNA	-	*Tymovirus*	Turnip yellow mosaic virus
(+) ss RNA	-	*Idaeovirus*	Raspberry bushy dwarf virus
(+) ss RNA	-	*Ounniavirus*	Ourmia melon virus
(+) ss RNA	-	*Potexvirus*	Potato virus X
(+) ss RNA	-	*Carlavirus*	Carnation latent virus
(+) ss RNA	-	*Foveavirus*	Apple stem pitting virus
(+) ss RNA	-	*Allexvirus*	Shallot virus X

(+) ss RNA	-	*Capillovirus*	Apple stem grooving virus
(+) ss RNA	-	*Trichovirus*	Apple chlorotic leaf spot virus
(+) ss RNA	-	*Vitivirus*	Grapevine virus A
(+) ss RNA	*Pospiviroidae*	*Pospiviroid*	Potato spindle tuber viroid
		Hostuviroid	Hop stunt viroid
		Cocadviroid	Coconut cadang-cadang viroid
		Apscaviroid	Apple scar skin viroid
		Coleviroid	Coleus blumei viroid 1

13.3 Symptomatology of Viral Diseases

Symptoms of Virus Plants Diseases

Viral diseases of plants exhibit a variety of symptoms.

1. **Mosaic**: The most common symptoms is mosaic. As a result of viral infection white or yellow colour spots appear on leaf surface which are known as mosaic Interspersion of various degrees of chlorosis with the normal green colour of the leaf results in a mosaic with yellow and green. When there is a uniform reduction of chlorophyll, the symptom is referred to as the yellows type.
2. **Vein clearing and vein banding**: These symptoms are appear in systemically infected leaves. Vein clearing occurs before mottle or mosaic clearing or chlorosis of the tissue in or immediately adjacent to the veins. Vein banding consists of broader bands of green tissue in that portion, set off by chlorosis or necrosis in the interveinal parenchyma.
3. **Ring spots** : These symptoms appear in the localized spots and consists of various types of chlorosis and necrosis. They may be circular chlorotic areas and are called chlorotic ring spots. In necrotic ring spots, necrosis

may appear in rings alternating with normal green. The centers of either types of spots may eventually become necrotic. Ex. Tobacco ring spot.

4. **Necrosis**: In both the yellow and mosaic diseases, necrosis appears in various forms. The most drastic effect is to kill cells, tissues or the whole plant. Necrosis is not only confined to chlorophyll containing tissues but also can develop on chlorophyll less leaves, hypocotyls or even roots.
5. **Wilt**: In certain virus infected plants, the outer whorls of leaves droop and become yellow. The inner whorls also become pale yellow. The leaflets soon turn brown and start drying up from their tips. The leaves shed in quick succession. The leaves become smaller and stunted in size. Root growth is also reduced considerably.
6. **Stunning**: It may affect all parts of plant, involving a reduction in the size of the leaves, flowers and fruits. Shortening of the petioles and internodes takes place as in bean yellow mosaic virus. The extent of stunting varies according to when infection occurs in the development stage of the plan. Sometimes some parts of plants may be more stunted
7. **Changes in leaf form** : In mosaic disease there is often uneven growth of leaf lamina. Leaves become curled, brittle and show puckering (prominences and depressions). There may be upwards and inward rolling of leaf, as in potato leaf roll. In some the lamina is extremely reduced giving a 'fern leaf' effect as in TMV or CMV on tomato.
8. **Outgrowths**; Abnormal growth is common in many viral infections. On the underside of the leaves of infected plants vein swellings and foliar outgrowths and are generally formed as in tobacco leaf curl disease. Tumours or swellings are formed on stems and roots in sweet clover infected with wound tumour virus.
9. **Flower breakings**: Due to viral infections variegation in flower colour is very common. This is called breaking of flower colour as in tulip breaking. Similar symptoms are produced by turnip mosaic virus in Wall flower.
10. **Fruit abnormalities:** A variety of symptoms may develop on the fruits in certain viral infections as mottling on Cucumber and papaya a due to mosaic type of viruses and star crack in apples and plum are due to plum pox virus.

Internal Symptoms:

Several cytological and histological abnormalities can be observed within the virus affected plants. The histological deviations though seen in tissues, are of purely cytological origin. Many viruses affect vascular tissues. The xylem elements of grape vine infected with grape vine fan leaf virus contains lignified strands known as endocellular cordons. In yellowing viruses tyloses are formed. The phloem cells degenerate or die and callose deposition occurs in the phloem sieve plates. One virus affects particular organelles. They may induce formation of marginal vesicles in the chloroplasts, modify mitochondria and aggregate them to form inclusion bodies. The inclusion bodies may be crystalline and amorphous. The amorphous structures are in the shape of 'pin wheels' and 'cat-o-nine tails'. These bodies occur in the infections by flexous rod-shaped viruses of the potato virus group.

13.4 Transmission of Viruses

The various methods of transmission of plant viruses are as follows:

1. **Seed Transmission:** Viruses may be externally seed borne as in tomato, cucumber, etc. or internally seed borne in testa, endosperm and/or embryo as in barley, cowpea, bean (bean mosaic), etc. The internally seed borne viruses are more effective than the externally seed borne ones.
2. **Transmission by Grafting:** Since viruses are intimately associated with the living cells of the host, it is rather easy for their transmission through grafting between living cells of virus infected and virus-free plants. In fruit and ornamental trees where grafting is the normal method of propagation, transmission of virus by grafting becomes a means of natural transmission.
3. **Transmission by Vegetative Propagation:** Viruses are very commonly perpetuated in the vegetative organs of perennial plants (fruit trees). When such plants are virus-infected all the vegetative parts used for their propagation also *become* virus-infected. As such, viruses are readily transferred from locality to locality in virus-infected nursery stock, bulbs, tubers (leaf roll of potato) and roots. Hence the infected perennials are the common reservoirs for perennating of many viruses.
4. **Transmission by Parasitic Phanerogams.** Species of *Cuscuta* when parasitizing virus-infected host plants sends haustoria into the host tissue and thereby receives virus infection. The same virus-infected species, of *Cuscuta* when extends its stem to parasitize other plants, the virus may be

transmitted, to such plants through the newly formed penetrating) haustoria. *Cuscuta* thus functions as the transmitting agent.

5. **Transmission by Insects.** Most viruses are transmitted by insects. The insects responsible for, the transmission of viruses either possess mouth parts adapted for biting or stylets for piercing and sucking. The sucking insects are the usual insect vectors. But the principal insect vectors are: thrips, plant bugs, leafhoppers, white flies, aphids and coccids.

   There is specificity of certain insects for particular viruses. Some viruses may be carried mechanically on the mouth parts of the insects and the latter remain viruliferous for a period of only a few minutes to a few hours. These are known as **non- persistent viruses.** They are rapidly lost by the vector, usually after a short period of feeding. The non-persistent viruses are carried to the first plant and rarely to the second if the feeding period is of some hours' duration. Again some vectors may not transmit the viruses to a healthy plant until some time has elapsed after they have fed upon the diseased plant. These are **persistent viruses.** The persistent viruses retain infectivity for a long period of time and there is delay in the development of infective power. In such case it is possible that the virus is ingested by the insect and is later transmitted through the body into the saliva, by which channel it eventually reaches the next host plant. The delay in the development of infective power—the **latent period** also known as the **period of incubation** varies greatly with the different viruses. These viruses may also multiply within the body of the vectors. The persistent viruses are not transmitted to the first two or three plants but to all the others for a considerable period. The longest latent period, so far discovered, is that of a strawberry virus known as 'Virus 3' transmitted by the aphid *Capitophorus fragariae* Theob., which takes 10 to 19 days.

6. **Transmission by Mechanical Means:** Transmission by this means consists of transference of sap from a virus-infected plant to a healthy plant by artificial or natural means. Since infection through 'natural openings, like stomata is rather rare, mechanical transmission often involves wounding of the host tissue for the easy entrance of the virus from host to host. Viruses transmitted by mechanical means are usually in high concentration in the plant. Some viruses can spread from a diseased plant to a healthy one by contact of the leaves brought about by the wind. Cultivation procedures and the movement of animals may play some part in the spread of viruses. The tobacco mosaic virus is transmitted very rapidly by rubbing the extracted

juice of a diseased plant over the leaf of a healthy tobacco plant. By this process hairs or epidermal cells are sufficiently wounded to bring about infection.

Some viruses may spread below ground by mechanical contact between the roots of infected and healthy plants.

Usually viruses of mosaic group are most readily transmitted by mechanical means.

7. **Soil Transmission:** The soil-borne viruses infect host through root system. These viruses do not usually persist in the soil more than a few months at the most. The viability of the soil-borne viruses, however, depends largely on the soil texture. Roots of infected perennial hosts serve as permanent reservoirs of soil-borne viruses.
8. **Transmission by Mites:** Eriophyid mites transmit several viruses. The big-bud mite, *Phytopus ribis* transmits virus that causes disease of *Ribes.* Mites cannot fly and presumably spread viruses by crawling from plant to plant or more likely, by being dispersed by wind.
9. **Transmission by Nematodes:** Nematodes belonging to the genera *Xiphinema, Longidorus* and *Trichodorus* transmit a number of viruses. Spread might result from systemic root infection of plants with extensive root systems; the roots could thus be made available to nematodes feeding at some distance from the original site of infection. The nematodes feed on the epidermal cells near the root tip and acquire virus.
10. **Transmission by Fungi:** Several viruses including those causing big-vein diseases of lettuce and tobacco necrosis are transmitted by *Olpidium* and *Synchytrium* which infect plants. The virus is borne internally by the zoospores of the fungus when they are developed in the virus infected host.
11. **Pollen Transmission: C**ases of dissemination of viruses through pollen grains are few in comparison with other means. Common example is bean mosaic virus.
12. **Transmission through weeds:** Weeds serve as collateral hosts for transmission of sugarcane mosaic virus.

13.5 Control Measures

Many of the methods described below are used for general plant disease control and can be used. Plant virus control is still mainly based on preventive and sanitary practices.

1) **Selection of seed:** Although majority of the plant viruses are not true seed-borne it is better to select seeds from disease- free localities, field or plants.
2) **Cuttings, bulbs and tubers:** Majority of viruses of vegetatively propagated crops are carried by the planting material. In such cases, care should be taken to select the planting material from plants that are not infected. However, this method will not be successful when symptoms are not visible on the plant and when secondary infection through insect vectors is common.
3) **Tuber indexing:** This method is used to select virus-free potato tubers for multiplication on a large scale. When sufficient seed tubers have been produced in this way they are distributed to growers.
4) **Protection against insect victors:** This is one of the important steps in the prevention of spread of plant virus diseases during growth of the crop. In order to prevent insect vectors from reaching the diseased or healthy plants or to reduce their population build up proper spray or soil application of insecticides should be undertaken. Use of trap crops to attract and destroy the insects and use of barriers and repillant mulches are also recommended.
5) **The host-free period:** The method is a voluntary effort made by all the growers in an area in which they agree to eliminate all host plants of the disease for a period. At the end of this period fresh planting is started with disease-free seed and plants.
6) **Roguing:** *Viral diseases are best managed by this method.* Regular removal of all diseased plants is very effective in reducing the amount of initial pathogen inoculums and further spread of disease.
7) **Destruction of weed hosts:** It is well known that some viruses are carried in weeds. Destruction of such weeds is essential for managing disease. Every possible precaution should be taken to keep the field and its surroundings free from these weeds.

8) **Resistant varieties:** The development of resistant varieties is the most satisfactory method of plant disease control but comparatively few virus diseases have been controlled in this manner. Sometimes wild species of plants possess good tolerance to viruses and many other diseases. These wild species sources can be used to develop resistance in cultivated plants.

9) **Temperature treatments:** When disease is transmitted through cuttings, bulbs and grafts this method is used. Sereh disease of sugarcane has been controlled by immersing the cuttings in water at a temperature of 52° C for 30 min. Sugarcane mosaic has also been controlled by treating cuttings at 53°-54° C. Immersion of setts in water at 52° C for 20 min. kills the virus of chlorotic streak of sugarcane. Similar results have been obtained with many virus diseases of fruit trees.

10) **Immunization:** Although cross protection has been proved in case of many plant diseases, especially the viral diseases, the method has not been very successful for field scale disease management

13.6 Summary

Viruses can neither be seen nor can be grown on artificial media. The symptoms Produced are similar to those of nutritional deficiencies. But viruses are always infectious while inanimate agent is non-infectious. Important viral diseases include mosaic, vein banding and vein clearing, ring spots, necrosis, stunting, outgrowths etc. The name virus (Latin vim* = venom of poisonous fluid) was given by Pasteur to the causative agents of infectious diseases, Adolph Mayer (1885), a Dutch scientist, and D.J. Ivanowsky (1892), a Russian scientist, recognized certain microbes as causative agent of mosaic disease of tobacco

In 1927, Johnson proposed a system for naming and grouping plant viruses. The requirement for a sound classification system for plant viruses become imperative and this requirement was-met by the appointment of a committee to investigate virus taxonomy at the International Congress of Microbiology held in Moscow in 1966.The original meaning of "virus" can still be detected in the commonly used adjective, 'virulent', meaning extremely poisonous, venomous, or malignant. Viruses can neither be seen nor can be grown on artificial media. The symptoms produced are similar to those of nutritional deficiencies. But viruses are always infectious while inanimate agent is non-infectious. Hence, in artificial culture media no pathogen is obtained and the disease is infectious it can be a viral disease. Viral diseases of plants exhibit a variety of symptoms.

Important viral diseases include mosaic, vein banding and vein clearing, ring spots, necrosis, stunting, outgrowths etc.

13.7 Glossary

- **Agglutination :** A serologicl test in which viruses or bacteria suspended in a liquid collect into clumps.
- **Chlorosis :** Yellowing of green tissue due to chlorophyll destruction.
- **Circulative viruses :** Viruses that are acquired by their vectors through their mouthparts, accumulate internally, then are passed through their tissues and introduced into plants again through the mouthparts of the vectors.
- **Damping off :** Destruction of seedlings near the soil line, resulting in the falling of seedlings on the ground (characteristic infection by *Phythium spp.)*
- **Disease :** Any disturbance of plant that interferes with its normal structure, function, or economic value.
- **Disinfectant :** A physical or chemical agent that frees a plant or organ from infection.
- **Dissemination :** Dispersal of inoculums from its source to healthy plants.
- **Eradicant :** A chemical substance that destroys a pathogen at its source.
- **Fumigant :** A toxic gas or volatile substance that is used to disinfest certain areas from various pests.
- **Hypoplasia :** Under development of a tissue or plant due to decreased cell division.
- **Hypotrophy :**Under development of a tissue or plant due to abnormally reduced cell enlargement.
- **Infection :** Invasion, or condition caused by endoparasites.
- **Infectious disease :** A disease that is caused by a pathogen which can spread from a diseased to a healthy plant.
- **Inoculum :** The pathogen that can cause disease.

- ***in -vitro* :** Out side the host.
- ***in -vivo* :** In the host.
- **Latent virus :** A virus that does not induce symptoms in its host.
- **Leaf spot :** A self – limiting lesion on a leaf.
- **Mosaic :** Symptom of certain viral diseases of plants characterized by intermingled patches of normal and light green or yellowish colour.
- **Necrosis :**The death of cells or of tissues.
- **Ring spot :** A circular area of chlorosis with a green centre ; symptom of many virus diseases.
- **Sanitation :** The removal and burning of infected plant debris.
- **Symptom :** The external and internal alterations of plant as a result of a disease.
- **Transmission :** The transfer of virus from one plant to another.
- **Tumour :** A malignant overgrowth of tissue.
- **Virus :** An ultramicroscopic obligate parasite which consists of nucleic acid and protein.

13.8 Self -Learning Exercise

Section A : (Very Short Answer Type Questions)

Fill in the blanks

1. In 1939, _____ had proposed a binomial system for virus nomenclature**.**
2. Viruses have protein coat made up of _____.
3. Viruses can pass through _____ filters.
4. The International Congress of Microbiology held in Moscow in _____.
5. _____was the first to use the term virus
6. A viroid consists of a very short strand of _____ without any protective coat

Section B : (Short Answer Type Questions)

1. Write short note on transmission of viral disease.
2. Write short note on the International Committee on Taxonomy of Viruses (ICTV).
3. Explain following :
   (a) Wilt (b) Chlorosis (c) Hypotrophy

Section C : (Long Answer Type Questions)

1. Give an account of classification of viruses.
2. Give an account of symptoms of viral diseases in plants.
3. Write an essay on the control measures of viral diseases.

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Unit-14

Viral Disease -II:

Plant Diseases Caused by Viruses

Structure of the Unit:

14.0 Objectives

After going through this unit you will be able to:

- Understand various viruses as disease causing agent on plants
- Explain disease symptoms
- Differentiate and can compare the distinguishing symptoms produced by various viruses in plants
- Explain disease control measures

14.1 Introduction

A virus is a nucleoprotein that multiplies in living cells and also has ability to cause disease in plant and animals. Plant viruses differ from all other plant pathogen in many respect and cause typical symptoms on diseased plants. Dwarfing and stunting of the entire plant and reduction in yield occur in almost all viral diseases They are transmitted from plant to plant in a number of ways.

Management of disease caused by viruses can be done by spraying insecticides to control the insect vector population.

14.2 Potato Virus X (PVX)

The distribution of this virus is associated to potato cultivation, particularly in Europe. It infects many hosts including vegetables and ornamentals Because of its being carried symptomlessly and spreading readily through contact, the PVX incidence may reach 40-50% in an uncared crop. PVX can cause yield losses above 10-15%, the extent varying according to the strain, weather and the cultivars. It infects all cultivated solanaceous and other plants, including weed.

Casual organism

Potato Virus X (PVX) is of Potexvirus genus. Mechanical transmission of this virus is very easy, for instance by tools, cloths. It might also be transmitted through chewing insects such as grasshoppers.

Symptoms

Majority of the strains of PVX remain symptomless in common potato cultivars. At times, this virus causes mild or impereceptible mosaic, which is very faint and transient.

Some bands of dark green tissue can be observed along the veins ("vein banding"), there may also be ring spots, or necrotic bright grey spots.

More damage is sometimes observed when there is a co-infection with PVY. PVX infection results in numerous, large amorphous inclusions that can be observed under light microscope.

Electron microscopy of such inclusions show virus particles interspersed between alternating layers of curved or rolled laminate inclusion components.

Control Measures

- Sanitation-workers should disinfet their hands with soap and water during agricultural operations.
- Spray of 0.5-1.0% tannic acid.
- The best method of control is to grow resistant varieties**.**
- lant early. All infected plants shold be rouged out as early as observed.

14.3 Potato Virus Y (PVY)

Potato virus Y, is the second most important among potato viruses and yield losses range up to 60%.

Symptoms

Its symptoms vary widely according to the strain, variety and environment.

Severe or rugose mosaic, bunching and twisting of leaves, stunting are the important symptoms of PVY strains.

Some PVY strains cause severe mosaic and veinal necrosis, PVY is very mild on potatoes All the PVY strains are flexuous rods of different lengths.

PVY induce pinwheel inclusions in the systemically infected tissues of potato plants which can be seen electron microscopically

Pathogen

Potato virus Y (Potexvirus genus)

A large number of aphid vectors are known for PVY but the important ones for are *Myzus persicae, Macrosiphum euphorbiae* and *Aphis gossypii.* Spread of PVY mainly occurs through diseased tubers or the viruliferous vector aphids. These viruses are non-persistently aphid-borne.

Control Measures

Control of the mosaics is normally affected by clonal selection combined with serological testing at all stages for production of the nucleic/foundation seed stocks. The ideal approach for control of potato mosaics is to adopt an integrated schedule of practices as follows:

- Start with reliable virus-free seed tubers.
- Use resistant varieties, if possible.
- Apply weedicide(s) and adopt blind earthling up.
- Control aphid population using systemic (soil+foliar) insecticides.
- Plant early. All infected plants shold be rouged out as early as observed.

14.4 Tomato Ring Mosaic

The virus causing the common mosaic of tomato is known as Tomato mosaic virus (tobamovirus group). Lycopersicon virus 1 is a synonym. There are many strains of the virus producing different symptoms and often these have been

described as different diseases. These strains are tomato aucuba mosaic, tomato enation mosaic, yellow ringspot strain and tomato rosetted strain.

Pathogen

Tomato mosaic virus (tobamovirus group)

Lycopersicon virus 1 is a synonym.

The tomato mosaic virus is very stable and can persist in dry contaminated soil, in infected tomato debris, on or in the seed coat. The virus is transmitted readily from plant to plant by mechanical mean

Fig.13.1 Tomato Ring Mosaic

Symptoms

Generally, the symptoms of tomato mosaic are influenced by various factors such as variety of tomato, virus strain temperature, day length, light intensity, plant age.

On tomato, virus infection causes light and dark green mottled areas on the leaves

The leaf shows rough appearance due to sunken of green areas.

Stunting of young plants is common and leaves distorted to "fern leaf" or tendril shape but mottling may be slight.

In extreme cases almost the entire lamina of old or new leaves becomes pale yellow to white, with scattered small islands of green which stand up as blisters

Certain strains of the virus can cause a mottling, streaking and necrosis of the fruits

Seedling infection may kill the plants.

Fruits are fewer, undersized and often deformed.

In some cases there is necrosis of stem, petioles, leaves and fruits.

Plant is not killed but growth is retarded.

The virus induces pollen sterility which results in low fruiting and low yield (Giri and Mishra, 1992).

14.5 Bunchy Top of Banana

Bunchy top of Banana is the most serious disease in banana .Banana bunchy top disease is widely distributed along banana growing countries It is a viral disease caused by the virus *Banana* virus – I, producing a cluster of leaves at the apex forming a rosette in the diseased plant. The Banana bunchy top disease was first reported from Fiji in 1879. In India, the disease was recorded for the first time in 1940 from Kerela State

Symptoms

The disease may occur and show the symptoms of bunchy top at any stage of plant growth.In badly infected plant leaves are ilustered together at the top of the pseudostem to form a ***rosette*** appearance.

The appearance of irregular, brown ***streaks*** along the ***secondary veins*** are the first external symptom**.**The midrib portion on the undersurface of the leaf shows ***chlorotic streaks.***

On leaf petiole brown streaks are also produced.The leaf size becomes ***smaller*** and ***narrow.***In the diseased plant, ***leaves are clustered*** at the apex forming a ***rosette.***

The infected plant shows ***stuned growth.***Usually the infected plants do not bear any fruits. The phloem of the infected plant is disorganized to some extent

The roots show delay.

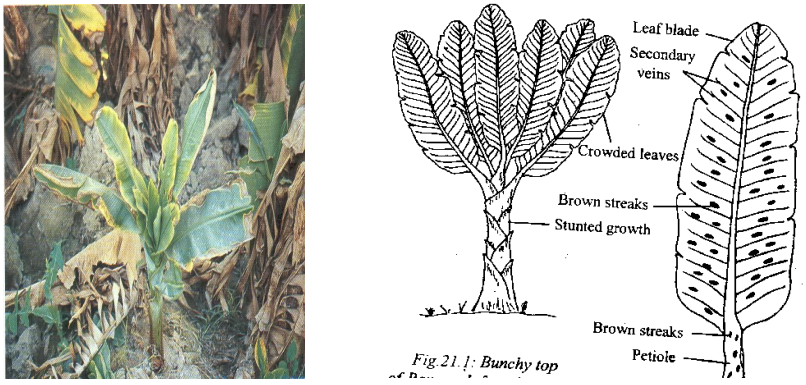

Fig. 14.1: Bunchy top of Banana Infected plant showing rosette appearance

Pathogen

This disease is caused by a virus namely *Bunchy top virus – I* or *Banana Virus – I* or *Musa virus-I.*

The virus is being transmitted by a aphid ***vector*** *Pentalonia nigronervosa* Coq

Dissemination

In the infected plant, the virus is present in all the parts including the ***rhizomes*** and the ***suckers***. The aphid acquires the virus in a feeding period of 24 hours and transmits the virus to the healthy plants. The aphid (vector) remains virulferous for about 13 days after the ***acquisition*** of virus. The aphid usually attacks the host plant around the basal portion of the ***Pseudostem***. The insect may carry the disease to long distances.

Control Measures

- The diseased plants should be thoroughly and carefully dug out with suckers and destroyed by ***burning.***
- Exclusion of diseased suckers and plants has been recommended.
- The infected plants along with the aphids should be killed by pouring 50ml of ***kerosene*** on each plant.
- The virus particles can be killed by injecting the ***herbicides agrokone*** (or) ***2, 4-D*** (2,4-dichlorophenoxy acetic acid) or MCPA (2-Methyl-1-4 chlorophenoxy acetic acid) in the diseased plant.
- Disease free ***suxkers*** or ***certified virus free suckers*** should be used for planting.
- ***Strict control*** on the entry of banana plants from other states and movement of suckers within the state must be checked for infection.

No resistant varieties have been evolved.

14.6 Tobacco Necrosis

Probably Tobacco necrosis virus (TNV) is distributed worldwide.

Pathogen

Tobacco necrosis virus (TNV) is of Necrovirus genus .

The virus is transmitted by the aquatic fungus *Olpidium brassicae*. This virus is spread worldwide, particularly in Europe, although not extensively in tobacco. It infects many hosts including vegetables and ornamentals.

Symptoms

Brown necrotic spots appear near the veins, they may be coalescing. Occasionally young seedlings die.

On mature plants, the symptoms are mainly located on the lower leaves.
Sometime it causes poor yield or distorted fruits, delayed fruit ripening, and non uniform fruit color.
The symptoms are very dependent on the age of the infected plant, the environmental conditions, the virus strain, and the genetic background of the host plant.

Control Measures

- To avoid transmitting the virus from an infected plant to healthy plants, the watering hose or watering can should not be allowed to make contact with the plants.
- Care should be taken to dispose of dead leaves and old plants because dry, infected leaves can be blown around the greenhouse as 'dust' which can subsequently infect healthy plants if they are wounded.
- Inoculation of a mild strain of the virus onto young plants can protect them from subsequent infection by more severe strains.This is a well documented control strategy, called "cross protection".
- The best method of control is to grow resistant varieties**.**
- During the growing season, infected plants should be dug up, bagged, and removed from the field.

14.7 Summary

Plant viruses differ from all other plant pathogen in many respect and cause typical symptoms on diseased plants. Dwarfing and stunting of the entire plant and reduction in yield occur in almost all viral diseases They are transmitted from plant to plant in a number of ways. Management of disease caused by viruses can be done by spraying insecticides to control the insect vector population. Potato virus X (PVX) is of Potexvirus genus .It infects many hosts including vegetables and ornamentals. The virus causing the common mosaic of tomato is known as Tomato mosaic virus (tobamovirus group). Bunchy top of Banana is the most serious disease in banana .Banana bunchy top disease is widely distributed along banana growing countries. Tobacco necrosis virus (TNV) is of Necrovirus genus. The virus is transmitted by the aquatic fungus *Olpidium brassicae*.

14.8 Glossary

- **Chlorosis :** Yellowing of green tissue due to chlorophyll destruction.
- **Disease :**Any disturbance of plant that interferes with its normal structure, function, or economic value.
- **Disinfectant :** A physical or chemical agent that frees a plant or organ from infection.
- **Dissemination:** Dispersal of inoculums from its source to healthy plants.
- **Infection :** Invasion, or condition caused by endoparasites.
- **Mosaic :** Symptom of certain viral diseases of plants characterized by intermingled patches of normal and light green or yellowish colour.
- **Necrosis :** The death of cells or of tissues.
- **Ring spot :** A circular area of chlorosis with a green centre ; symptom of many virus diseases.
- **Sanitation :** The removal and burning of infected plant debris.
- **Symptom :** The external and internal alterations of plant as a result of a disease.
- **Transmission :** The transfer of virus from one plant to another.

14.9 Self -Learning Exercise

Section A : (Very Short Answer Type Questions)

Fill in the blanks

1. The virus causing the common mosaic of tomato is known as _____.
2. Potato virus X (PVX) is of _____ genus.
3. Tobacco Necrosis Virus (TNV) is transmitted by the aquatic fungus _____.
4. The Banana bunchy top disease was first reported from _____.

Multiple Choice Questions

5. Tobacco Necrosis disease of is caused by:

   (i) Fusarium (ii) Tobacco necrosis virus (TNV)

(iii) Bacterium (iv) Cercospora

6. *Banana Virus – I* is transmitted by:

(i) Water (ii) *Aphid* (iii) Virus X (iv) Fruit fly

Section B : (Short Answer Type Questions)

1 Which is the casual organism of the Bunchy top of Banana?

2 Write down the symptoms of Tobacco Necrosis.

3 Write down the control measures of Bunchy top of Banana disease.

Section C : (Long Answer Type Questions)

1 Write an essay on the control measures of Potato virus X &Y (PVX&PVY) diseases in plants.

2 Write down the causal organism, symptoms and control measures of Mosaic of tomato disease.

3 Write down the causal organism, symptoms and control measures of Tobacco Necrosis.

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Unit-15

Phytoplasmal Diseases

Structure of the Unit:

15.0 Objectives

After going through this unit you will be able to:

- Understand various Phytoplasma or Mycoplasma as disease causing agent on plants
- Explain Phytoplasmal disease symptoms
- know the distinguishing symptoms produced by various Phytoplasma or Mycoplasma in plants
- Explain disease control measures

15.1 Introduction

Phytoplasma or Mycoplasmas were first discovered by Pasteur (1843) while studying the causative agent of pleuropneumonia in cattle (bovinepleuropneumonia). They were designated as PPLO (pleurapneumonia-like organisms). In 1898, two French microbiologists, Nocard and Roux, were successful in obtaining pure cultures of these microorganisms in media

containing serum. They observed that these organisms could produce disease when inoculated in healthy cattle.

15.2 General Account of Phytoplasma

Nowak (1929) placed these organisms in the genus *Mycoplasma,* which belongs to the class Mollicutes of the order Mycoplasmatales and are characterized by having no cell wall but they do have a confining unit membrane. Mycoplasmas like organisms are now called as phytoplasma. They are pleomorphic in shape and are usually confined in phloem or xylem cells. They are too small to be seen in the light microscope (0.1 to 1.0 *p m* in diameter) and are readily seen in the electron microscope. Mycoplasmas (phytoplasma) reproduce by binary fission and are resistant to penicillin but susceptible to tetracyclin. Plant MLOs contain ribosomal RNA and DNA in the form of a coil in their nuclear region. The presence of both RNA and DNA distinguishes them from plant viruses. They are not normally transmitted by sap but are often transmitted by leaf hoppers and occasionally by plant hoppers or psyllids. In recent years various MLOs have been cultured on artificial media. Aster yellows, papaya bunchy top, little leaf of bringal and phyllody of sesame are common diseases incited by MLOs (= phytoplasma)

Structure of Mycoplasmas

- Due to absence of true cell wall these organisms are highly plastic and readily deformable; hence mycoplasmas are irregular and variable in shape.
- The cells may be coccoid, granular, pear-shaped, cluster-like, ring-like or filamentous.
- The cells are small, ranging in diameter between 0.3 and 0.9 m viruses.
- A unit of lipoprotein cytoplasmic membrane, (7.5-10 μm thick) is present which cover these organisms.
- Ribosomes and nucleoplasm-like structure are present in cytoplasm.
- Though genetic material is composed of both DNA and RNA. Mycoplasmas may be the simplest form of life capable of independent growth and metabolism.
- Mycoplasmas are Gram-negative.
- They are usually non-motile, some forms, however, show gliding movements.
- They reproduce by budding or binary fission.

- They are sensitive to oxytetracycline, streptomycin, erythromycin and chloramphenicol.
- Chemically, they are more close to bacteria than viruses.

15.3 Phytoplasmal Diseases

15.3.1Symptoms of Phytoplasmal Diseases

More than 80 plant diseases are reported from various parts of the world due to mycoplasma like bodies. The symptoms include abnormalities in floral and vegetative parts i.e., virescence and phyllody, yellowing, proliferation of axillary buds, reduction in leaf lamina, general stunting etc.

1. **Phyllody**: It is most common in sesamum and manifests in the flowering season. The floral parts are transformed into green leafy structures. The sepals are transformed into leaf-like structures and the corolla, stamens and carpels turn green and leafy. The ovary is also malformed into elongated structure. The leaves are reduced in size, internodes are shortened, the plant is stunted and branching is abnormal, resulting in a plant malformed beyond recognition.
2. **Spikes** : Spike disease occurs in sandal trees of all ages. It is of two types: Rosette and Pendular type. Rosette spike is more common and is commonly referred to as spike or spike disease. The characteristic symptom is the whole shoot looks like spike bearing four rows of spiked bristles. There is extensive reduction in the size of the plant.
3. **Little leaf:** Little leaf is widespread in brinjal. The plants show extreme reduction in the size of leaves and nodes giving a bushy appearance. A large number of axillary buds are stimulated to grow into short branches with small leaves.
4. **Grassy shoot disease** : It is a serious disease of sugarcane. It is characterized by the production of numerous, thin tillers from the base of affected plants. Premature and excessive tillering gives a crowded grass like appearance of the clump. The tillers bear pale yellow, thin, narrow leaves resembling grasses. The affected clumps are stunted and exhibit a varying degree of loss of chlorophyll ranging from total green to white.

15.3.2 Sesamum of Phyllody

Sesamum (*Sesamum indicum* L) is an seed crop grown in India,Japan, China,Burma,and the southern Mediterranean regions and in parts of

Africa.Phyllody is one of the most important & destructive disease of *Sesamum.* Sesamum of Phyllody is a Mycoplasma *Disease which occurred every year in India* .In India, it is found in all those regions, wherever the *Sesamum* crop is grown.

Symptoms

The symptoms of sesamum phyllody may be in the flowering stage of plant.

The floral parts are transformed into green leafy structures and grow profusely.
Sepals, petals, stamens and pistils are transformed into green leafy structures.
The veins of the leafy structures are thick and prominent.
Ultimately , all floral parts become phylloid and sterile.
The leaves of affected plants become chlorotic and reduced in size with vein clearing symptoms.
Due to shortened internodes and abnormal branching the whole plant becomes dwarf and stunted.

Pathogen

The disease is caused by a mycoplasma. It was first reported to be graft transmitted (B.P. Pal and Puskar Nath, 1935) and in nature by *Orosius albicintus* (R.S. Vasudeva and H.S. Sahambi, 1956). Sahambi (1970) reported the pathogen vector relationship and a wide host range. Prasad (1978) reported association of rickettsia like bodies in the phloem cells of sunnhemp inoculated with sesamum phyllody pathogen. It is still to be confirmed whether these bodies are rickettsia or MLO's.

Control Measure

- This disease can be controlled to some extent by spraying the plants with, some effective insecticide to check the transmission of mycoplasma.
- All the known varieties of sesamum are susceptible to the disease.

15.3.3 Sandal Spike Disease

Sandal *(Santalum album* L.) is one of the most economically important forest trees in India. This species is confined to India and Indonesia. It is confined to only Karnataka, Kerala and Tamil Nadu. Spike disease is the most destructive of the few diseases that attack this plant.

Symptoms

The common symptom is 'rosette spike' which is characterized by severe reduction of leaf size and shortening of the internodes. This results in the crowding of leaves on the branches.

All such leaves stand out stiffly on the branches like spikes. In advanced stages of disease, just before the death of the tree, leaves become yellowish and finally reddish.

The flowers of the infected trees show phyllody. The ends of the roots and haustorial connections with the host plant are either damaged or die.

The other form of the disease, known as 'Pendulous Spike' is characterized by continueus apical growth of shoots without proportionate thickening of shoot resulting in drooping habit of the shoot.

Pathogen

This disease was thought to be caused by a 'Virus', later since 1969, electron microscopic examinations have confirmed that the disease is caused by a mycoplasma like organisms which are found in the sieve tubes of leaves and twigs showing symptoms. Remission of symptoms and disappearance of MLO after tetracycline treatment have further confirmed mycoplasmal etiology of the disease.

The spike disease is transmitted through root contacts and injects vectors. The leaf hoppers *Nephotethix virescens* and *Moonia albimasculata* seem to be involved in the disease transmission.

Control Measures

Treatment with tetracycline and systemic fungicides like Benlate alone or in combination resulted in temporal control of the disease for 50-100 days and the disease reappears. Resistant varieties need to be developed.

15.4 Summary

Phytoplasma or Mycoplasmas were first discovered by Pasteur (1843). Nowak (1929) placed these organisms in the genus *Mycoplasma,* which belongs to the class Mollicutes of the order Mycoplasmatales. More than 80 plant diseases are reported from various parts of the world due to mycopmsma like bodies.Symptoms of phytoplasmal diseases include viresecnce and phyllody, yellowing, proliferation of axillary buds, reduction in leaf lamina, general stunning etc. Phyllody is one of the most important & destructive disease of

Sesamum which occured every year in India and found in all those regions, wherever the *Sesamum* crop is grown. Spike disease is the most destructive of the few diseases that attack **Sandal *(Santalum album* L.)** plant.

15.5 Glossory

- **Control :** Prevention of, or reduction of loss from plant disease.
- **Host :** Any plant attacked by a parasite.
- **Mycoplasma :** A genus of highly pleomorphic, gram-negative, aerobic or facultatively anaerobic bacteria that lack cell walls, including the pleuropneumonia - like organisms and other species.
- **Phyllody :** The abnormal transformation of a floral structure into a foliage leaf.
- **Rosette :** Disease symptom with stems shortened to produce a bunchy growth habit.
- **Virescence :** state or process of becoming green, especially the abnormal development of green coloration in plant parts, such as flowers, that are normally not green.

15.6 Self -Learning Exercise

Section A : (Very Short Answer Type Questions)

Fill in the Blanks:

1. Phytoplasma or Mycoplasmas were first discovered by _____.
2. Phyllody sesamum disease is a _____.
3. Phyllody is one of the most destructive disease of _____in India.
4. The spike disease is transmitted through _____.

Section B : (Short Answer Type Questions)

1. Who is the casual organism of the Spike disease of sandal?
2. Who is the casual organism of the Seasame phyllody?
3. Write down the symptoms of Seasame phyllody.
4. Write down the control measures of Spike disease of sandal.

Section C : (Long Answer Type Questions)

1. Write note on phytoplasmas diseases in plants.
2. Describe the characteristics of phytoplasmas.

3. Write an essay on the control measures of phytoplasmas diseases in plants.
4. Write down the causal organism, symptoms and control measures of spike disease of sandal.

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Unit-16

Nematode Diseases-I:

Plant Pathogenic Nematodes

Structure of the Unit:

16.0 Objectives

After going through this unit you will be able to:

- Understand history of pathogenic Nematode
- Explain Classification & Identification of plant nematodes
- Understand morphology & anatomy of Nematodes
- Differentiate and can compare the distinguishing symptoms produced by nematodes in plants.
- Explain disease control measures.

16.1 Introduction

The nematodes belong to the Phylum Nematode of the animal kingdom. They resemble to roundworms and are natural fauna of soil and water. Free living nematodes are found in deserts, hot springs, lakes and even polar seas, but water is essential for their survival. They may be free living or parasitic on plants and animals including man. Plant parasitic nematodes are soil inhabitants. Three forms of nematode are found:

(A)**Saprophytic forms—** These nematodes help in breaking down organic matter and are useful for increasing soil fertility.

(B)**Predaceous forms—**These nematodes feed on fungi, algae and even on other nematodes, thus, they are useful in biological control of different disease causing fungi algae and phytophagous nematodes,

(C)**Phytophagous forms—**These namatodes feed on plants and are disease causing agents.The phytophagous forms of nematodes may be endo parasite or ectoparasite. They may be migratory or sedentary (remaining non-migratory while feeding) in nature.

16.2 Pathogenic Nematode

16.2.1 History of Nematodes

Members of the phylum Nematoda (round worms) have been in existence for an estimated one billion years, making them one of the most ancient and diverse types of animals on the earth (Wang *et al.* 1999). They are thought to have evolved from simple animals some 400 million years before the "Cambrian explosion" of invertebrates able to be fossilized (Poinar 1983). The two nematode classes, the Chromadorea and Enoplea, have diverged so long ago, over 550 million years that it is difficult to accurately know the age of the two lineages of the phylum (Figure 1).

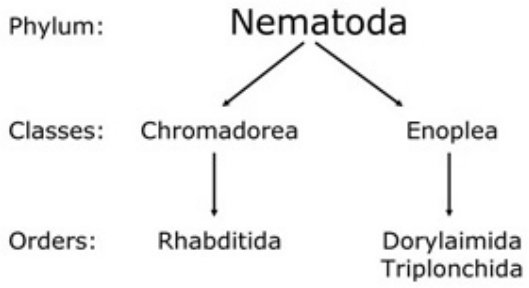


Fig. 16.1 : Two Lineages of the Phylum Nematoda

Nematodes are multicellular animals in the group Ecdysozoa, or animals that can shed their cuticle. Also included in this group with nematodes are insects, arachnids and crustaceans. In contrast to some of their relative invertebrates, nematodes are soft-bodied. Thus, very few nematodes have been fossilized (22 species from 11 genera) and exactly what ancestral nematodes looked like remains unknown. While we do not know the morphology of the first nematodes, it is probable that they were microbial feeders in the primordial oceans. The oldest known fossil nematodes are only 120-135 million years old; by then nematodes had diversified to feed on microbes, animals and plants (Poinar *et al.* 1994, Manum *et al.* 1994). The oldest fossil nematodes are found in amber and are commonly associated with insects. This is probably due to the fact that tree sap, which fossilizes to make amber, captures and preserves insects and their associated nematodes much more easily than an animal- or a nematode-infested portion of a plant. Much of what we know about the evolution of nematodes is inferred from the comparative anatomy of existing nematodes, trophic habits, and by the comparison of nematode DNA sequences (Thomas *et al.* 1997, Powers *et al.* 1993). Based upon molecular phylogenic analyses, it appears that nematodes have evolved their ability to parasitize animals and plants several times during their evolution (Blaxter et al. 1998). One point is clear; nematodes have evolved to fill almost every conceivable niche on earth that contains some amount of water. Nematodes are extremely abundant and diverse animals; only insects exceed their diversity. Most nematodes are free-living and feed on bacteria, fungi, protozoans and other nematode (40% of the described species); many are parasites of animals (invertebrates and vertebrates (44% of the described species) and plants (15% of the described species).

Nematodes were noted early in human history because some serious human diseases are caused by relatively large vertebrate-parasitic nematodes. Some of these nematodes were first described in the ancient Chinese scientific literature as early as 2700 B.C. (Maggenti, 1981). Since plant parasitic nematodes often are small and subterranean, there are not many ancient references to phytoparasitic nematodes. One interesting observation suggests that phytoparasitic nematodes were known in antiquity (235 B.C.) because the ancient Chinese symbol for a soybean root-infesting organism resembles in shape an adult female soybean cyst nematode (Noel, 1992). The first described plant parasitic nematodes were discovered in wheat seeds by Needham (1743). Not until the identification of root-knot nematodes on cucumber by Berkeley

(1855) and cyst nematodes causing "beet-tired" disease on sugar beets by Schacht (1859), did plant nematology begin to emerge as an important scientific discipline. Nathan A. Cobb, the "Father of US Nematology," pioneered agricultural nematology as a USDA scientist in the early 1900's. The use of soil fumigation to reduce nematode populations and increase crop yields in the 1940's (Carter) demonstrated that nematodes were significant crop pathogens and ushered in the "chemical era" for nematode management in production agriculture. For a review of the history of plant nematology see the book "General Nematology "by Armand Maggenti (1981); see also the nematode history web sites in Table 1. Today plant parasitic nematodes are recognized as major agricultural pathogens and are known to attack plants and cause crop losses throughout the world. Some estimates suggest they cause 77 billion dollars of damage worldwide each year (Sasser and Freckman 1987). As the full extent of damage caused by plant-parasitic nematodes is recognized by agricultural scientists, the study of the biology of plant-parasitic nematodes will become increasingly important.

Pathogenic Effect of Nematode Infestation

The effects of nematode infestation are as follows:

1. **Cellular hypertrophy and hyperplasia—**Such as in knots and galls produced by them.
2. **Selective stimulation of growth—**Few selective tissues may be stimulated for abnormal growth resulting in proliferation of organs.
3. **Necrosis—**Infestation may result in death of the tissue.
4. **Abnormal cell division** may be manifested. Suppression of mitosis is common in case of root manifestation.

16.2.2 Classification of Plant Parasitic Nematodes

All nematodes belong to the phylum *Nemata* (Nematoda) of the Animal Kingdom. They are classified into class, order, suborder, superfamily, family and sometimes subfamily, genus and species. The phylum is divided into two classes: Secernentea and Adenophorea. Except the Dorylaimida group of plant parasitic nematodes all phytonematodes belong to the class Secernentea, especially the order Tylenchida, The plant parasitic nematodes of common occurrence in this order are subdivided as follows:

Order: *Tylenchida*

Suborder: Tylenchina

Superfamily: Tylenchoidea

Family: Tylenchidae

Subfamily: Anguininae

Genera: *Angaioa, Dityhnchus*

Family: Hoplolaimidae

Subfamily: Hoplolaiminae

Genus *Hoplolaimus*

Subfamily: Rotylenchinae

Genera: *Rotylenchus, Helicotylenchus*

Subfamily: Rotylenchulinae

Genus: *Rotylenchulus*

Family: Tylenchorhynchidae

Subfamily: Tylenchorhynchinae

Genus: *Tylenchorhynchus*

Family: Pratylenchidae

Subfamily: Pratylenchinae

Genus: *Pratylenchus*

Superfamily: Heteroderoidea

Family: Heteroderidae

Subfamily: Heteroderinae

Genera: *Hcterodera, Globodera*

Family: Meloidogynidae

Genus: *Meloidogyne*

Superfamily: Criconematoidea

Family: Criconematidae

Subfamily: Paratylenchinae

Genus: *Paratylenchus*

Family: Tylenchulidae

Subfamily: Tylenchulinae

Genus: *Tylenchulus*

Suborder: Aphelenchina

Family: Aphelenchidae

Genus: *Aphelenchus*

Family: Aphelenchididae

Genus: *Aphelenchoides*

Order: *Dorylaimida*

Suborder: Dorylaimina

Superfamily: Dorylaimoidea

Family: Longidoridae

Subfamily: Longidorinae

Genera: *Longidorus, Paralongidorus*

Subfamily: Xiphineminae

Genus: *Xiphinema*

Suborder: Diphtherophorina

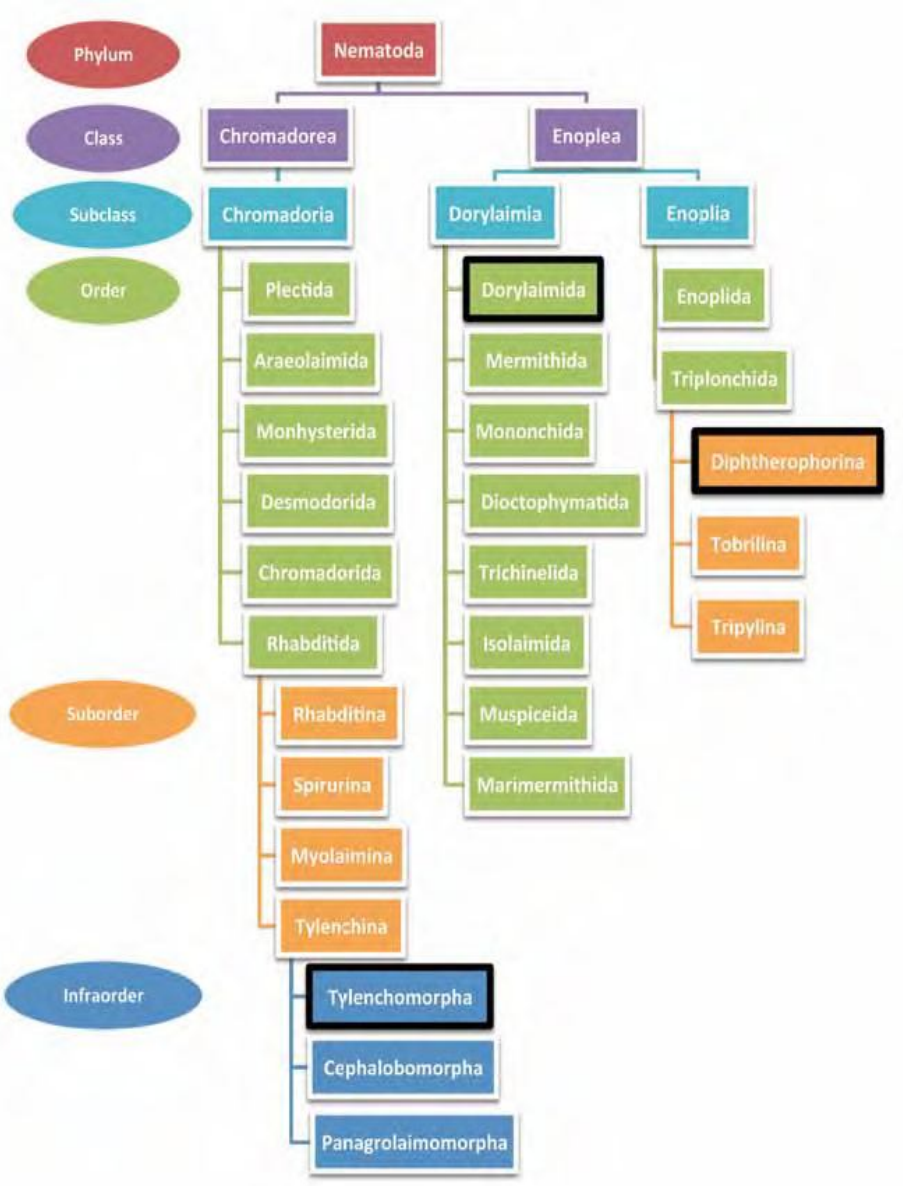
Superfamily; Trichodoroidea

Family: Trichodoridae

Genera: *Trichodorus, Paratrichodorus*

De Ley and Blaxter (2002) provided a new classification system mainly based on molecular phylogenetic results and additional morphological analyses. The scheme for this classification would be:

III. Nematode classification


Phylum
Class
Subclass
Order
Suborder
Infraorder
Nematoda
Chromadorea
Enoplea
Chromadoria
Dorylaimia
Enoplia
Plectida
Araeolaimida
Monhysterida
Desmodorida
Chromadorida
Rhabditida
Dorylaimida
Mermithida
Mononchida
Dioctophymatida
Trichinelida
Isolaimida
Muspiceida
Marimermithida
Enoplida
Triplonchida
Diphtherophorina
Tobrilina
Tripylina
Rhabditina
Spirurina
Myolaimina
Tylenchina
Tylenchomorpha
Cephalobomorpha
Panagrolaimomorpha

The major nematode orders that plant-parasitic nematodes belong to are Rhabditida, Dorylaimida, and Triplonchida.

Order Rhabditida

Infraorder Tylenchomorpha

Superfamily Aphelenchoidea

Family: Aphelenchidae

Family: Aphelenchoididae

Family: Paraphelenchidae

Superfamily Criconematoidae

Family: Criconematidae

Family: Hemicycliophoridae

Family: Tylenchulidae

Superfamily Myenchoidea

Family: Myenchidae

Superfamily Sphaerularioidea

Family: Allantonematidae

Family: Anguinidae

Family: Iotonchiidae

Family: Neotylenchidae

Family: Parasitylenchidae

Family: Sphaerulariidae

Family: Sychnotylenchidae

Superfamily Tylenchoidea

Family: Atylenchidae Family: Meloidogynidae

Family: Belonolaimidae Family: Pratylenchidae

Family: Dolichodoridae Family: Psilenchidae

Family: Ecphyadophoridae Family: Telotylenchidae

Family: Heteroderidae Family: Tylenchidae

Family: Hoplolaimidae Family: Tylodoridae

Order Triplonchida

Suborder Diphtherophorina

Superfamily Diphtherophoroidea

Family: Diphtherophoridae

Family: Trichodoridae

Suborder Tobrilina

Superfamily Prismatolaimoidea

Family: Prismatolaimidae

Superfamily Tobriloidea

Family: Pandolaimidae

Family: Rhabdodemaniidae

Family: Tobrilidae

Family: Triodontolaimidae

Suborder Triplonchida

Family: Bastianiidae

Family: Odontolaimidae

Suborder Tripylina

Superfamily Tripyloidea

Family: Onchulidae

Family: Tripylidae

16.2.3 Identification

The common plant-parasitic nematode genera are fairly easy to identify to that level using a standard compound microscope. Identification of nematodes to the species level often requires detailed morphological analysis, growth of the nematode on different host plants, or DNA or isozyme analysis. Common morphological features used in nematode identification include the mouth cavity (presence or absence and shape of a stylet), the shape and overlap of the pharyngeal glands with the intestine, size and shape of the nematode body at the adult stage, size of the head, tail and number and position of ovaries in the female. More subtle characters may include number of lines on the nematode's cuticle or the presence or absence of pore-like sensory organs. In females the reproductive organs are used as traits for identification because the number of ovaries and the position of the vulva in the female nematode's body are easily

seen under the light microscope. Male nematodes have one or two testes and they are easily identified by the presence of spicules. Spicules are copulatory structures that are used during mating to guide the sperm into the vagina of the female nematode

16.3 Morphology and Anatomy of Nematodes

1. Plant parasitic nematodes are slender, cylindrical filiform and tapering at each end, motile and 0.2 to 10 mm in length. In some genera such as Meloidogyne, female forms may be pear or lemon or kidney shaped.
2. Nematodes are triploblastic (having three layers), bilaterally symmetrical, unsegmented animals with single cavity (non-coelomic).
3. The body of a nematode is covered by an impermeable cuticle which may be smooth or marked by different kinds of sculpturing.
4. Beneath the cuticular layer is a sub-cuticular layer followed by the muscular layer.
5. Nematodes usually undergo four moults from egg to adult and at each stage they cast off the cuticle getting larger each time.
6. The mouth opening is usually surrounded by lips bearing sensory organs which are at the anterior end.
7. The mouth is followed by a mouth cavity or stoma.
8. Below the stoma is the esophagus (pharynx) followed by intestine and a rectum terminating into a ventral terminal or sub-terminal anus in females, or a cloacal opening in males.
9. At the posterior (opposite) end there is an anus.
10. There is no segmentation of the body.
11. The nematodes have excretory as well as nervous system but lack organs for circulation and respiration.
12. The nervous system is not well-defined in plant parasitic nematodes. However, the nerve ring encircling the resophagus is easily identifiable
13. The sexes are usually separate.
14. Females are always larger than males.
15. Numerically, the males and females of plant parasitic nematodes exist mostly in equal numbers
16. The males are slender and worm-like while the females swell to become pyriform, lemon-shaped, or saccate.
17. The male reproductive system consists of testis and vas deferens and is a single tube, sometimes two tubes, opening directly into the rectum forming the cloaca.

18. Mature females possess a vulva which is situated at the ventral side in the middle of the body.
19. After copulation the body of a female becomes filled with elliptical and hyaline eggs in a Jelly-like sac.
20. The mature eggs are usually oval, elliptical or rounded. The morphology varies according to life habit of the nematode
21. The number of eggs produced by each female varies from 20-30 eggs (in *Aphelenchoides ritzemabosi)* to more than 2800 eggs in *Meloidogyne.* The rate of egg laying per day may be 24 eggs (in *Meloidogyne* at 22° C) to only 2 eggs in *Aphelenchoides ritzemabosi*
22. Eggs differentiate into larvae, remaining inside the mother they are hatched as larvae.
23. The second stage larvae hatch out to develop into adults after several moltings.
24. All nematodes undergo four molts from the juvenile to the adult phase of their life cycle. They have four juvenile stages and an adult stage. In many nematodes the first molt usually occurs in the egg and it is the second-stage juvenile that hatches. While all nematodes undergo four molts, molting is not required for growth of the nematode
25. Temperature, moisture, aeration, soil texture, organic matter, rhizosphere and cultural practices are factors affecting the population and parasitism of the soil inhabiting nematodes.

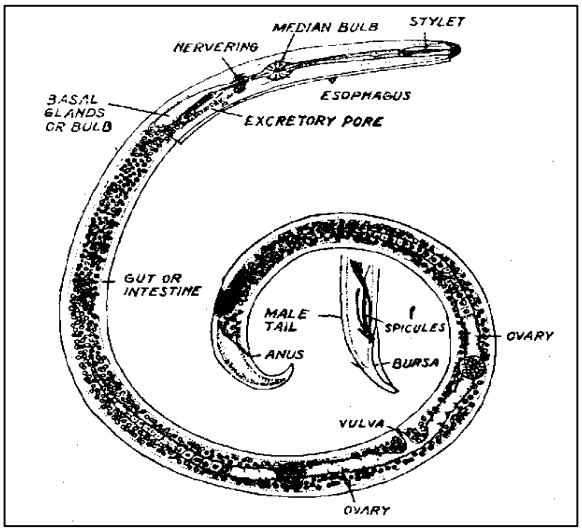


Fig. 16.2 A

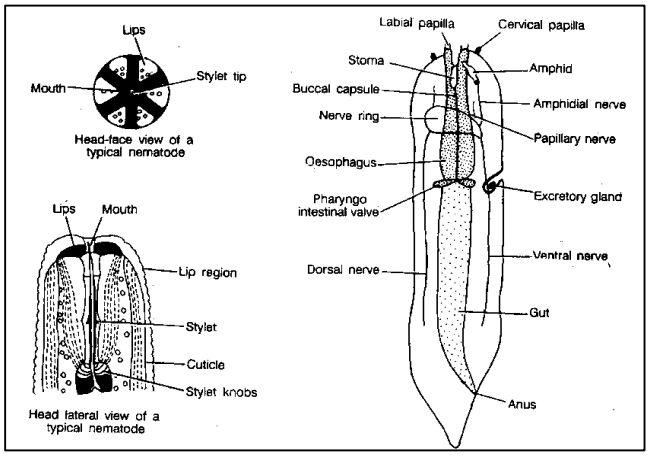


Fig. 16.2 B

Fig. 16.2 A-B : Structure of Typical Nematode

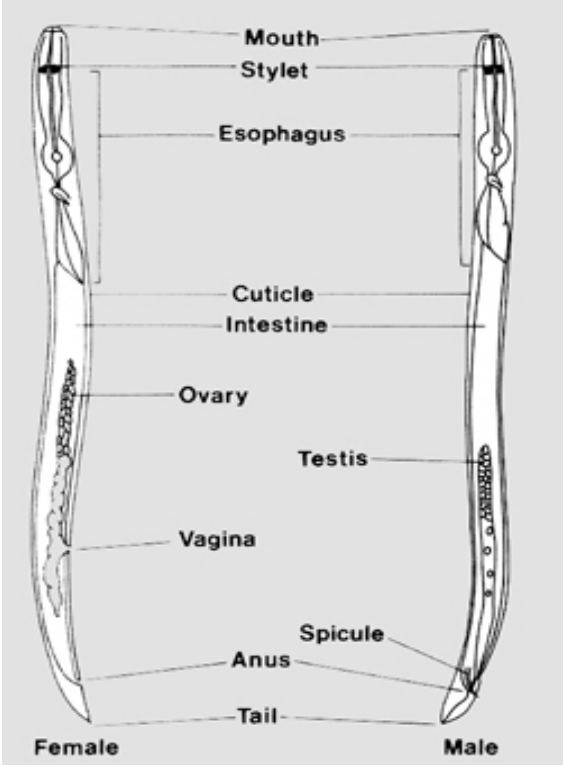


Fig. 16.3 : Female and male Nematode (Internal structure)

Table -16.1 : Some Important Nematode Diseases of Plants

Common Name	Latin Name	Host Range
Ufra of rice nematode	*Ditylenchus angustus*	Rice
Root knot nematodes	*Meloidogpie exigua*	Coffee
	M. Javonica	Sugarcane, Limabeans Gardenbalsom.
	M. hapla	Potato, tomato, celery
	M. arenaria	Groundnut, wheat, barley, corn etc.
	M. incognita var *acrita*	Jute, cotton, etc.
	M. brevicauda	Tea
Potato rot nematode	*Ditylenchus destructor*	Potato, sugar beet
Wheat ear cockle nematode Root lesion nematode Burrowing nematode Golden nematode of potato Sugar beet nematode	*Anguina tritici*	Wheat
	Pratylenchus spp.	Tea
	Rodopholus similis	Banana citrus
	Heterodera rostochiensis	Potato, tomato, bringal
	Heierodera schachtii	Sugarbeet, Brassica sp.

Life cycle of Wheat Seed Gall Nematode *Anguina tritici*

Some species of *Anguina* are known, and all of them cause formations of galls on seeds, leaves and other above ground parts of plants. The pathogen *Anguina tritici* is a large nematode about 3.2 mm. long and 120 m μ in diameter. The nematode lays its eggs and produces all its larval stages and the adults in seed galls. The symptoms appear on plants in all growth stages. Infected seedlings are more or less severely stunted and show characteristic rolling, twisting, curling and wrinkling of the leaves. Stems are often enlarged near the base. Diseased heads are shorter and thicker than healthy ones. Mature galls are shed off more readily than kernels. Galls are hard dark rounded and shorter than the normal.

The seed gall nematodes over winter as second stage larvae in seed galls. Galls fallen to the ground or sown with the seed releases infective second stage larvae during war moist weather.

16.4 Symptoms of Nematode Diseases

Nematodes infect both root and shoot systems of the plant. Root symptoms appear as hypertrophy, necrosis or abnormal growth.

1. **Root knots or Root galls**: These are enlargements of the roots caused by the feeding of the nematodes. The swellings vary in size from 1mm to more than 2cm. The feeding of nematodes induces the formation of 'giant cells' in the host tissue and cell division is stimulated. This leads to the formation of galls of various sorts. In the cells of susceptible plants a chain of events starting with nuclear and nucleolar enlargement, followed by cell wall breakdown, synchronous mitosis and incorporation of adjacent cells leads to the formation of highly specialized syncytia or giant cells. This syncytium is induced and maintained by continuous stimulus from the nematodes.
2. **Root lesions:** By nematode feeding, portions of roots are discolored and often collapsed. These portions vary in size from being as minute as to be almost invisible to the named eye to lesions girdling the whole root. Necrotic lesions are caused by toxic salivary secretions injected during the feeding of nematodes.
3. **Excessive root branching:** This is caused by the formation of numerous short lateral branches of roots in the vicinity of nematode injury.
4. **Root rots** : When nematode infections are accompanied by plant pathogenic or saprophytic bacteria and fungi, root rots occur.
5. **Injured root tips** : When the nematodes feed on or near the root tips, growth is stopped.

Nematodes feeding ectoparasitically at the root tips, cell division in the apical meristem is suppressed and results in the formation of short roots as in *Trichodorus*. Nematode root infections are usually accompanied by non-characteristic symptoms in the above ground parts of plants such as reduced growth, symptoms of nutrient deficiencies, yellowing of foliage, excessive wilting in hot or dry weather, reduced yields and quality of products.

16.5 Control Measures

Plant parasitic nematodes can be controlled by several methods. The nematode control aims to improve growth, quality and yield by keeping the nematode population below the economical threshold level. The control measures to be adopted should be profitable and cost effective. It is essential to calculate the cost benefit ratio before adopting control measures.

The nematode control methods are

1. Regulatory control
2. Cultural control
3. Physical control
4. Biological control

1. Regulatory Control

Regulatory control of pests and diseases is the legal enforcement of measures to prevent them from spreading or having spread, from multiplying sufficiently to become intolerably troublesome. The principle involved in enacting quarantine is exclusion of nematodes from entering into an area which is not infested, in order to avoid spread of the nematode Quarantine principles are traditionally employed to restrict the movement of infected plant materials and contaminated soil into a state or country. Many countries maintain elaborate organizations to intercept plant shipments containing nematodes and other pests. Diseased and contaminated plant material may be treated to kill the nematodes or their entry may be avoided. Quarantine also prevents the movement of infected plant and soil to move out to other nematodes free areas.

Plant Quarantine in India

The Destructive Insects and Pests Act, 1914 (DIP) was passed by the Government of India which restricts introduction of exotic pests and disease into the country from abroad. The agricultural pests and disease acts of the various states prevent interstate spread of pests within the country. The rules permit the plant protection advisor to the government of India or any authorizes officer to undertake inspection and treatments. Strict regulations have been made against *G. rostochiensis*, the potato cyst nematode and *Rhadinaphelenchus cocophilus*, the red ring nematode of coconut. Domestic quarantine regulations have also been imposed to restrict the movement of

potato both for seed and table purposes in order to present the spread of potato cyst nematode from Tamil Nadu to other states in India.

2. Cultural Control

Cultural nematode control methods are agronomical practices employed in order to minimize nematode problem in the crops.

Selection of healthy seed material

In plants, propagated by vegetative means we can eliminate nematodes by selecting the vegetative part from healthy plants. The golden nematode of potato, the burrowing, spiral and lesion nematodes of banana can be eliminated by selecting nematode free plant materials. The wheat seed gall nematode and rice white tip nematode can be controlled by using nematode free seeds.

Time of planting

Certain pathogenic nematodes are inactive during the winter months because low temperature inhibits their activities. Nematode life cycle depends on the climatic factors. Adjustment of the time of planting helps to avoid nematode damage.

Fallowing : Fallow is the practice of keeping land free of all vegetation for varying periods by frequent tilling of the soil by disking, plowing hansowin or by applying herbicides to prevent plant growth**.** Preferably after ploughing leaving the field without cultivation, helps to expose the nematodes to sunlight and the nematodes die due to starvation without host plant. This method is not economical.

Deep summer ploughing

During the onset of summer, the infested field is ploughed with disc plough and exposed to hot sun, which in turn enhances the soil temperature and kills the nematodes.

For raising small nursery beds for vegetable crops like tomato and brinjal seed beds can be prepared during summer, covered with polythene sheets which enhance soil temperature by 5 to 10˚C which kills the nematodes in the seed bed. This method is very effective and nematode free seedling can be raised by soil solarization using polythene sheets.

Manuring

Raising green manure crops and addition of more amounts of farm yard manure, oil cakes of neem and castor, pressmud and poultry manure etc

enriches the soil and further encourages the development of predacious nematodes like *Mononchus* spp. and also other nematode antagonistic microbes in the soil which checks the parasitic nematodes in the field.

Flooding

Flooding of fields to control nematodes is not widely accepted. Flooding can be adopted where there is an enormous availability of water. Under submerged conditions, anaerobic condition develops in the soil which kills the nematodes by asphyxiation. Chemicals lethal to nematodes such as hydrogen sulphide and ammonia are released in flooded condition which kills the nematodes.

Trap cropping

Two crops are grown in the field, out of which one crop is highly susceptible to the nematode. The nematode attacks the susceptible crop. By careful planning, the susceptible crop can be grown first and then removed and burnt. Thus the main crop escapes from the nematode damage.

Antagonistic crops

Certain crops like mustard, marigold and neem etc have chemicals or alkaloids as root exudates which repell or suppress the plant parasitic nematodes. In marigold (*Tagetes* spp.) plants α – terthinyl and bithinyl compounds are present throughout the plant from root to shoot tips. This chemical kills the nematodes. In mustard allyl isothiocyanate and in pangola grass pyrocaterchol are present which kills the nematodes.Such enemy plants can be grown along with main crop or included in crop rotation.

Removal and destruction of infected plants

Early detection of infested plants and removal helps to reduce nematode spread. After harvest the stubbles of infested plants are to be removed. In tobacco, the root system is left in the field after harvest. This will serve as a inoculum or the next season crops. Similarly in *D.angstus* the nematode remains in the left out stubbles in the field after harvest of rice grains. Such stubbles are to be removed and destroyed and land needs to be ploughed to expose the soil.

Use of resistant varieties

Nematode resistant varieties have been reported from time to time in different crops. Use of resistant varieties is a very effective method to avoid nematode damage. Nemared, Nematex, Hisar Lalit and Atkinson are tomato varieties resistant to *M. incogntia* . The potato variety Kufri swarna is resistant to *G. rostochiensis*.

3. Physical Control

It is very easy to kill the nematodes in laboratory by exposing the nematodes to heat, irradiation and osmotic pressure etc., but it is extremely difficult to adopt these methods in field conditions. These physical treatments may be hazardous to plant or the men working with the treatments and the radiation treatments may have residual effects

Heat treatment of soil

Sterilization of soil by allowing steam is a practice in soil used in green house, seed beds and also for small area cultivation. Insects, weed seeds, nematodes, bacteria and fungi are killed by steam sterilization. In such cases steam is introduced into the lower level of soil by means of perforated iron pipes buried in the soil. The soil surface needs to be covered during steaming operation. Plastic sheets are used for covering. In the laboratory and for pot culture experiments autoclaves are used to sterilize the soil.

Hot water treatment of planting material

Hot water treatment is commonly used for controlling nematodes. Prior to planting the seed materials such as banana corms, onion bulbs, tubers seeds and roots of seedlings can be dipped in hot water at $50^0 – 55^0$ °C for 10 minutes and then planted.

Irradiation

Irradiation also kills the nematode. Cysts of *G. rostochiensis* exposed to 20,000 Ɣ contained only dead eggs and at 40, 000 Ɣ exposure, the eggs lost their contents. *Ditylenchus myceliphagus* in mushroom compost exposed to Ɣ rays between 48,000 to 96,000 Ɣ inactivated the nematodes. UV light also kills the nematodes. But this irradiation is not practically feasible under field conditions.

Osmotic pressure

Feder (1960) reported 100% nematode mortality when sucrose or dextrose was added to nematode infested soil @ 1 to 5% by weight. But these methods are not practical and economical.

Washing process

Plant parasitic nematodes are often spread by soil adhering to potato tubers, bulbs and other planting materials. Careful washing of such planting material helps to avoid the nematodes in spreading in new planting field. Washing

apparatus for cleaning potato and sugar beet tubers are commercially developed and are being used in many countries.

Seed cleaning

Modern mechanical seed cleaning methods have been developed remove the seed galls from normal healthy wheat seeds.

Ultrasonics

Ultrasonic have little effect on *Heterodera* spp. The use of this ultrasonics is not practically feasible.

4. Biocontrol agents

A comprehensive list of measures were provided, including a range of methods to Along with crop rotation, some biocontrol agents can be used, which will not give immediate results but over a period of 3 or more crops, will establish itself well enough to render the soil suppressive to nematodes.

The three biocontrol agents are:

- *Paecilomyces lilacinus*, a nematophagus fungus, now commercially available in a spore formulation.
- *Pochonia clamydosporia*, is also a fungus. No products are available in the market , but it can be cultured on a compost/grain medium in a small scale and is suitable for small holdings
- *Pasteuria penetrans* a form of bacteria that multiplies inside the rootknot nematode. There are a few cultural methods to be adopted, that can prevent the spread of root knot.

5. **Chemical control**

Several general purpose fumigants like Methyl bromide, Ethylene dibromide' 1,3 dichloropropene extra give excellent control of nematodes in soil. The efficacy is related to their high volatility at ambient temperatures. All fumigants have low molecular weights and occur as gases or liquids. As they volatilize, the gas diffuses through the spaces between soil particles; nematodes living in these spaces are killed.

Root-knot typically occurs in patches, always introduced from external sources. Hence agricultural implements used in the affected areas should be washed before moving to a clean area. By simply hosing down all parts which have soil

sticking to it (including tractor wheels) with water within the infested area, the spread can be prevented.

Later (September 2011), University of Arkansas Press Release recommended pelletised chicken litter. It has 4% nitrogen, 2% phosphorus and 3% potassium, and also contains calcium, magnesium and iron. It was used with good results for sweet potato, especially in reducing the effects of soil nematodes.

16.6 Summary

The nematodes belong to the Phylum Nematoda of the animal kingdom. They resemble to roundworms and are natural fauna of soil and water. Plant parasitic nematodes are soil inhabitants. Three forms of nematode are found (A) Saprophytic form (B) Predaceous forms (C) Phytophagous form.Nematodes are simple animals, often only containing 1000 cells or less. In the middle to posterior of the nematode are the reproductive organs. Nematode species often have both males and females, but it is not uncommon for plant nematodes to reproduce asexually by parthenogenesis Plant-parasitic nematodes occur in all sizes and shapes. The typical nematode shape is a long and slender worm-like animal, but often the adult animals are swollen and no longer even resemble worms. Plant-parasitic nematodes range from 250 um to 12 mm in length, averaging 1 mm to about 15-35 μm in width. While nematodes may look dramatically different, they all share some common features. Nematodes often look segmented because of the numerous annulations (accordion-like transverse grooves) on the cuticle that allow the nematode to bend without kinking but in fact nematodes are unsegmented and have no replication of body parts throughout the worm. Developmentally, nematodes are triploblastic, containing three body layers (ectoderm, mesoderm and endoderm) in the embryo. Nematodes have a body cavity that is not totally surrounded by mesoderm, so they are pseudocoelomic. They have four juvenile stages and an adult stage. In many nematodes the first molt usually occurs in the egg and it is the second-stage juvenile that hatches. While all nematodes undergo four molts, molting is not required for growth of the nematode. Nematodes do not have a skeleton, but they do have a hypodermis which functions as a flexible support for their muscles.. Nematodes have no defined respiratory or circulatory systems; they depend on diffusion of water, gasses and metabolites in and out of their semi-permeable body walls and internal transport by mixing of the pseudocoelomic fluid as the nematode moves. The largest nematode found thus far was more than 7 meter long and 1 cm in diameter.. Nematodes have a sophisticated

nervous system and sensory organs to help them find their host plant, to locate specific plant cell types, and to mate and reproduce.

16.7 Glossary

- **Disease :** Any disturbance of plant that interferes with its normal structure, function, or economic value.
- **Disease cycle :** The chain of events involved in disease development, including the stages of development of the pathogen and the effect of the disease on the host.
- **Egg :** A female gamete. In nematodes, the first stage of the life cycle containing a zygote or a larva.
- **Hypotrophy :** Under development of a tissue or plant due to abnormally reduced cell enlargement.
- **Incubation period :** Period between infection and appearance of symptoms induced by parasitic organisms.
- **Larva :** The life stage of a nematode between the embryo and the adult.
- **Nematocide :** A chemical compound that kills nematodes.
- **Nematode :** Microscopic, wormlike animals that live as saprobes in water or soil, or as parasites of plants and animals.
- **Pathogen :** An organism capable of producing disease.

16.8 Self -Learning Exercise

Section A : (Very Short Answer Type Questions)

1. Nematode penetrate in plant tissues with the aid of

   (a) Crvicial (b) Labial papilla
   (c) Stylet (d) Saliva

2. Yellow ear rot of wheat is caused-by

   (a) *Heterodera* (b) *Anguina*
   (c) A and B (d) *Corynebacterium*

Fill in the blanks :

3. Nematodes are_____ symmetrical.

4. Only a few plant nematodes are _____ parasites.
5. Root knot nematodes have _____host range.
6. *Anguna tritici* alone causes _____.
7. Root-knot nematodes can easily be controlled by _____.

Section B : (Short Answer Type Questions)

1. Define Nematode.
2. Write the name of those disease which caused by *Heterodera avenae* in barley**.**
3. Write any five symptomes of nematode disease.
4. Write note on classification of nematodes.
5. What is pathogenic effect of nematode infestation ?

Section C : (Long Answer Type Questions)

1. Write short notes on the following:
   (i) Plant pathogenic nematode
   (ii) Pathogenic effect of nematode infestation
2. Describe life cycle of *Angurina tritici* in detail.
3. Describe Morphology and Anatomy of Typical Nematode.
4. Write a detail note on Cultural Control Method.

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Unit-17

Nematode Diseases-II:

Plant Diseases Caused by Nematodes

Structure of the Unit:

17.0 Objectives

After going through this unit you will be able to:

- Differentiate and compare the distinguishing symptoms produced by nematodes in plants
- Explain control measures of plant disease caused by namatodas

17.1 Introduction

Plant parasitic nematodes are microscopic round worms that feed on and damage plants. The most common serious nematode is the root-knot nematode found throughout the country (Fourgani and Edongali, 1989) with a very wide host range among all the cultivated crops in the country. All plant parasitic nematodes have a stylet at the anterior end which can be protruded and used like a hypodermic needle to penetrate the plant cells. The most striking feature of nematode distribution and damage within a field is the irregularities of infested areas, damaged crops will appear as irregular patches or streaks that

may vary in size, shape and number. These variations usually reflect the compounding of nematode stress (as a function of population density, feeding behaviour, damage potential, host response etc.) on a plant by such other factors as physical soil differences, irrigation and drainage patterns. The root-knot nematodes are most important pest of Wheat ,Barley ,Vegetable like Tomato, Brinjal crop.

17.2 Molya Disease of Wheat and Barley

In India, this Molya disease of wheat and barley was first reported from Punjab in 1957. Its widespread occurrence in Rajasthan was described by Prasad, *et al* (1959) and Swarup and Singh (1961). The disease has now spread to neighbouring areas of Haryana and Delhi also.

Symptoms

In newly infested fields the disease occurs in small patches of 2-3 feet diameter. Every year with continuous cultivation of cereals in the same field these patches gradually increase in dimension until the whole field gets infested.

Infected crop looks stunted showing symptoms similar to those of nutritional deficiency. The Infected plants are dwarfed and pale.

Leaves are discoloured to yellow and often become reddish from the tip.

Tillering is also markedly reduced and badly affected plants fail to produce earheads.

In severe cases the whole root system becomes dwarfed and matted.

There are no long roots penetrating into the deeper soil layers so that the affected plants become very susceptible to drought conditions.

As the infection advances, slight swellings near the root tips can be seen.

This condition is found generally within 4-6 weeks after sowing.

Pathogen

The nematode causing the molya disease is known as *Heterodera avenae* (Woll.) Filipjev. The females usually vary between 0.55 and 0.75 mm in length and about two-thirds as wide as long. They have paired ovaries. Cysts are typically lemon-shaped and contain 230-250 larvas. Development of larva take place in cysts and after attaining its full development the embryo in the egg undergoes the first molt giving rise to the second stage larva. Under suitable conditions, the larvae escape via the vulva and other apertures in the cyst wall. The free living second stage larvae, thus released, migrate through the soil in search of suitable host plants.

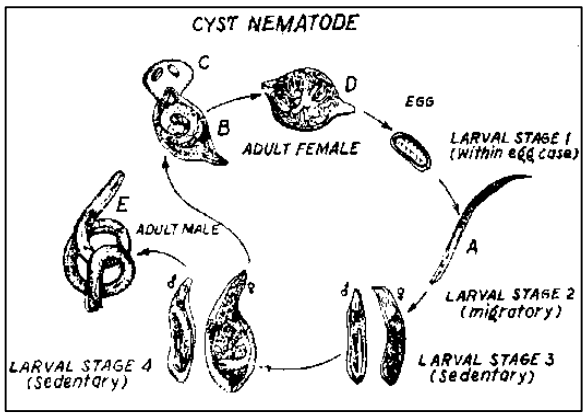

Fig. 17.1 : Life cycle of a cyst nematode

Control Measures

- **Cultural Method:** Fallows and crop rotation
- **Resistant Varieties:** None of the wheat varieties has been found resistant to the cereal cyst nematode. However, in barley resistance is reported. Field trials have shown that growing of a resistant barley variety in one season reduces the cyst population to a level where a normal wheat crop can be raised in the next season.
- **Chemical Control:** Soil fumigation with volatile chemical or treatment with non-fumigant nematicides achieve only a partial control of the nematode in soil In absence of these fumigants, systemic non-fumigant nematcides have been tried.
- **Biological Control:** Some fungi such as *Verticillium chlamydosporium, Paecilomyces lilacinus, Catenaria auxiliaris and C. vermicola* parasitize the eggs, larvae and cysts of the nematode.

17.3 Ear Cockles of Wheat

It was first noticed in England in the year 1743. Now it is reported from all the wheat growing regions of the world. In India nematode is a problem. The losses due to ear cockle disease had been as high as 30-70 per cent in the U.S.A. In India the disease accounts for a loss of 1-3 per cent every year which may be as high as 50 per cent in individual fields (Vasudeva and Hingorani, 1952). The nematode is often present in association with the yellow ear rot *(tundu or tannan* disease) caused by the bacterium *Clavibacter tritici.* flour or seed.

Symptoms

The base of wheat seedling or stem gets enlarged.

Leaves of the infected seedling get twisted and crinkled.

The infected ears are shorter and broader with very short or no awns on the glumes.

The infected ears are greener than normal healthy ears.

Grains in the affected ears are partially or completely replaced by cockles (galls) that are hard, dark brown or black.

These stony structures, which are generally 3-5 x 2-3 mm, vary in size from region to region.

They are filled with nematode larvae which can be seen by soaking the galls in water and then macerating them.

More than 1000 to 30000 larvae are found in each gall.

Pathogen

Anguina tritici (Steinbuch, 1799) Filipjev, 1936.

The adult female is 2.64-4.36 mm long The middle portion is swollen while anterior and posterior portions are slender. Mature female is obese and immobile.

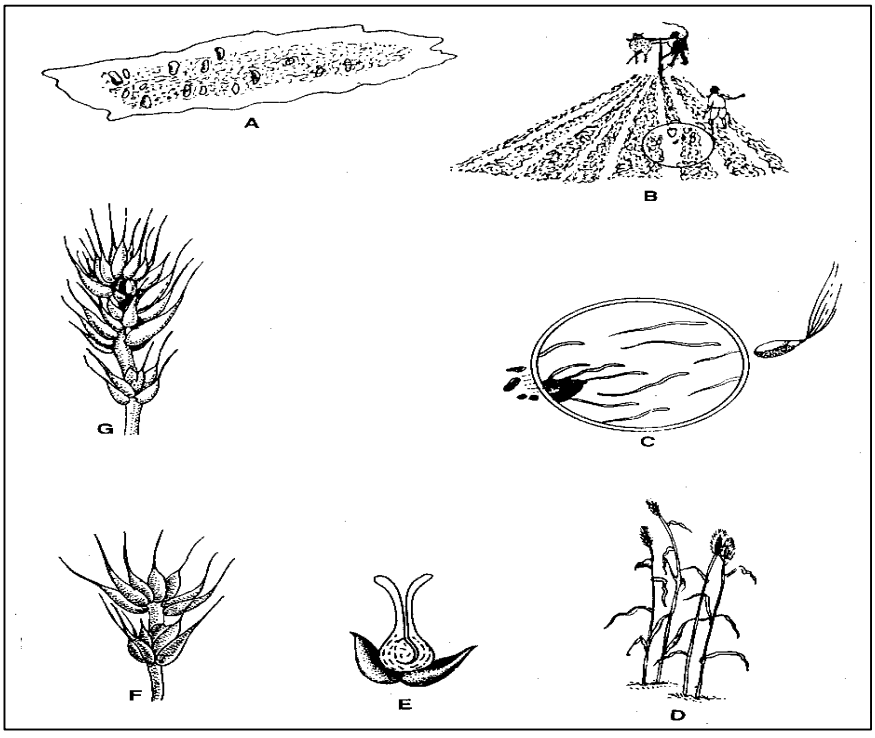

Fig. 17.2 : Different stages of nematode disease in wheat.

A: infected grains with wheat seed; B: sowing of infected grains with seed; C: nematodes coming out from infected grain in soil and infecting the seedling; D: infected plants with ears; E: infected ovary of wheat; F-G: infected grains in ears

Control Measures

- Use only gall-free seed from a healthy crop.
- By winnowing or using a sieve of proper size the galls should be removed from seeds.
- By soaking the seed in 2 per cent to 5 per cent common salt, galls can also be removed. But before sowing these seeds should be washed by plain water 2 to 3 times and then dried.
- In fields where the disease has once appeared cultivation of wheat should be stopped for 2-3 years.
- Granular nematicides such as Nemaphos (Zinophos), aldicarb or thionazin can be used for soil treatment when the soil infestation is heavy.

17.4 Root-Knot Disease of Vegetables

17.4.1 Root Knot Disease of Tomato

Symptoms

Due to of the presence of gall or knots on the roots the root-knot disease of tomato is very distinctive. The above-ground symptoms consist chiefly in foliage colour paler than normal. The affected plant remain-stunted and wilting is caused.

Fig. 17.3 : Root knot of tomato *(Meloidogyne javanica)*

Pathogen

Meloidogyne javanica (Treub.) Chitwood

Large and small roots contain swellings which vary from rounded galls to elongated spindles.

Sometimes other soil-borne fungi like species of *Fusarium* may become involved, which results in a more complex disease which kills the plant.

Disease cycle

The female nematodes having numerous eggs in their body are present in great numbers in the root galls. These survive in the soil and leaf debris. Root infection is caused by larvae when crop is sown in infested soil. The nematodes multiply rapidly within the roots and cause disease.

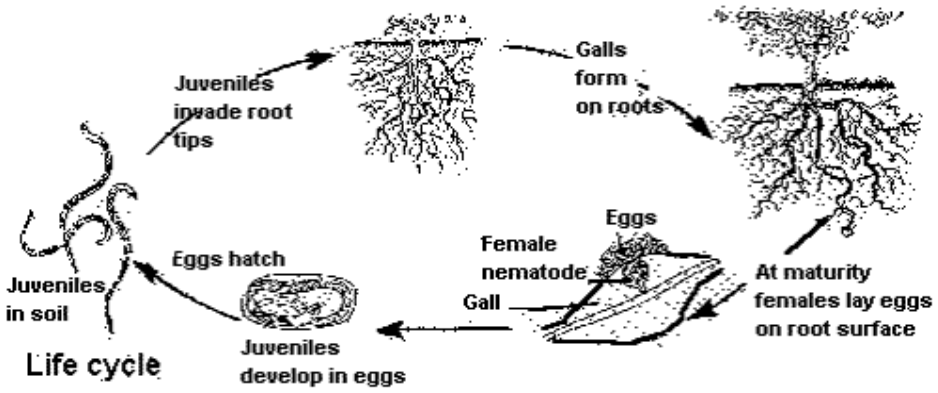

Fig. 17.4 : Life-cycle of Root knot nematode

Control Measures

- Soil fumigants should be used. For this purpose the soil of the fields should be fumigated with DD fumigant, nemagan, nematox, vapam, nemaphos, etc.
- The other nematode susceptible weeds should be eradicated.
- The resistant varieties should be grown.

17.4.2 Root Knot Disease of Brinjal

Root knot nematode, *Meloidogyne* sp., is a soil dwelling, endo- root parasite distributed worldwide. It attacks almost all plants known from nursery stage. Severity of root knot disease in brinjal caused by *Meloidogyne* spp.

Symptoms

The nematode infestation on the root appears as tiny tubercles but heavy and localized infestations stimulate excessive cell division leading to gall formation. The plants develop slowly and appear stunted if the infestation has been early and severe.

The leaves are yellowish green to yellow, tend to droop and wilt suddenly.

The main roots and the laterals in all cases bear spherical to elongated galls.

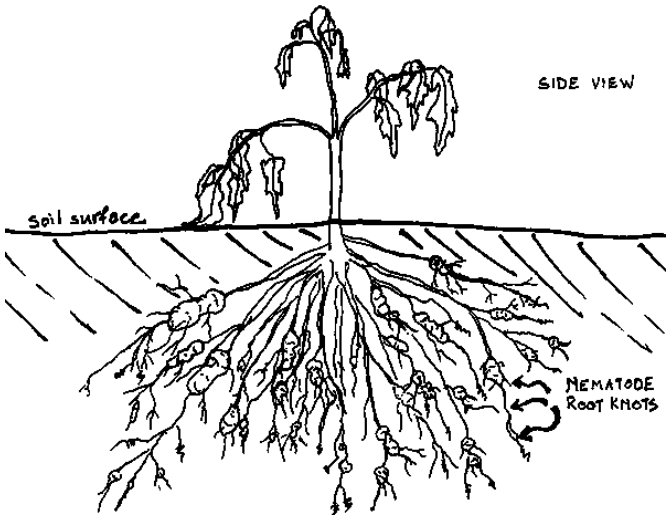

Fig. 17.5 : Root knot of Brinjal

Pathogen

Meloidogyne spp.

Disease cycle

The female nematodes having numerous eggs in their body are present in great numbers in the root galls. These survive in the soil and leaf debris. Root infection is caused by larvae when crop is sown in infested soil. The nematodes multiply rapidly within the roots and cause disease.

Control Measures

- Use only gall-free seed from a healthy crop.
- Remove the galls from seeds by winnowing or using a sieve of proper size.
- Galls can also be removed by soaking the seed in 2 per cent to 5 per cent common salt. When salt water is used, make sure that the treated seed is washed by plain water 2 to 3 times and then dried before sowing.
- In fields where the disease has once appeared cultivation of wheat should be stopped for 2-3 years.

When the soil infestation is heavy granular nematicides such as Nemaphos (Zinophos), aldicarb or thionazin can be used for soil treatment.

17.5 Summary

Among the three most economically damaging genera of plant-parasitic nematodes on horticultural and field crops root-knot nematodes (*Meloidogyne* spp.) is one of them . They are obligate parasites of the roots of thousands of plant species, including monocotyledonous and dicotyledonous, herbaceous and woody plants and are distributed worldwide. More than 60 species are present in this genus, with some species having several races. Nematodes cannot be cultured on artificial media but are always visible in or on the affected parts. They cause root knots or root galls, root lesions, excessive root branching etc.

17.6 Glossary

- **Disease :** Any disturbance of plant that interferes with its normal structure, function, or economic value.
- **Disease cycle :** The chain of events involved in disease development, including the stages of development of the pathogen and the effect of the disease on the host.
- **Egg :**A female gamete. In nematodes, the first stage of the lifecycle containing a zygote or a larva.
- **Epidemic :** A widespread and severe outbreak of a disease.
- **Eradication :** Control of plant disease by eliminating the plants that carry the pathogen.
- **Incubation period :** Period between infection and appearance of symptoms induced by parasitic organisms.
- **Larva :** The life stage of a nematode between the embryo and theadult.
- **Nematocide :** A chemical compound that kills nematodes.
- **Nematicide :** Microscopic, wormlike animals that live as saprobes in water or soil, or as parasites of plants and animals.
- **Pathogen :** An organism capable of producing disease.
- **Rosette :** Short, bunchy habit of plant growth.

- **Rot:** The softening, discolouration, and disintegration of a succulent plant tissue as a result of fungal or bacterial infection.
- **Root-Knot Nematode :** Any of several small plant-parasitic nematodes (genus *Meloidogyne*) that cause root knot.
- **Russet :** Brownish roughened areas on fruits skin.
- **Sanitation :** The removal and burning of infected plant debris.unfavorable environmental conditions.
- **Sterilization:** The elimination of pathogens from soil by means of heat or chemicals.
- **Symptom :** The external and internal alterations of plant as a result of a disease.

17.7 Self-Learning Exercise

Section A : (Very Short Answer Type Questions)

Fill in the blanks

1. Root-knot disease of tomato is a _____ disease.
2. Root-knot disease of vegetable is a _____ disease.
3. Root knot disease in brinjal caused by _____ spp.
4. The nematode causing the molya disease is known as _____.
5. Molya disease of wheat and barley was first reported from_____ in 1957.

Section B : (Short Answer Type Questions)

1. Which is the casual organism of the root-knot disease of tomato?
2. Write the name of those disease which caused by *Heterodera avenae* in barley.
3. Write any two symptomes of root-knot disease of brinjal.
4. Write note on disease cycle of Root knot nematode of brinjal.

Section C : (Long Answer Type Questions)

1. Write short note on Root knot disease of brinjal.
2. Write down the causal organism and symptoms of Molya disease of wheat and barley

3. Write down the causal organism, symptoms and control measures of ear cockle disease of wheat .

17.8 References

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Unit 18

Non-Parasitic Diseases

Structure of the unit:

18.0 Objectives

After going through this unit you will be able to understand:

- Various non-parasitic diseases
- Diseases due to deficiency of different elements
- Diseases due to excess of different inorganic pollutants
- Control measures

18.1 Intorduction

Plants can be damaged by infectious microbes such as fungi, bacteria, viruses, and nematodes. They can also be damaged by non-infectious factors, causing problems that can collectively be termed "abiotic diseases" or "abiotic disorders" like soil, water etc.

Unfavorable soil properties, fertility imbalances, moisture extremes, temperature extremes, chemical toxicity, physical injuries, air pollution, genetic defects and other problems are examples of abiotic disorders that can reduce plant health and even kill plants. Plants are also damaged by deficiencies of macro & micronutrients and due to excess of some chemical compounds liks O_3, PAN, SO_2, Hf etc. This unit is deals with the various types of Non-parasitic diseases.

18.2 Plant Diseases due to Abiotic Factors

Soil Structure

Soil structure determines the soil's ability to hold water, nutrients, and oxygen and make them available to plants. The most common issue related to soil structure is compaction, which results in inadequate pore space for root growth. Clay soils, with their smaller particle size, have naturally smaller pore space and are at high risk for becoming severely compacted. Compaction can occur from a variety of sources including traffic (particularly heavy farming or construction equipment), raindrop impact, tilling operations (plow layer) and minimal crop rotation. Reduced water availability is an obvious consequence of compaction as runoff occurs more frequently in a compacted soil and available pore space to hold water is limited. However, low oxygen availability for root respiration can also be a serious consequence of restricted soil pore spaces.

Soil pH

Soil pH is the measure of the H^+ ion activity in the soil solution. A high amount of H^+ activity results in an acidic soil condition, while low activity results in a predominance of OH^- activity, leading to alkaline soil. Although some plant species have preferences for more extreme acidic or alkaline soil conditions, it is generally regarded that a slightly acidic pH range of 6-7 is most favorable for plant growth. Soil pH outside of this range can have a dramatic impact on the solubility and therefore availability of plant nutrients. Soil pH below 5.5 generally results in low availability of calcium (Ca), magnesium (Mg), and phosphorus (P) and increased solubility of aluminum (Al), iron (Fe) and boron (B). High levels of these three nutrients in low soil pH are common and can induce toxicity symptoms in plants. Soils with pH levels above 7.8 have a high availability of Ca and Mg at the expense of P, B, Fe, manganese (Mn), zinc (Zn) and copper (Cu). Plants grown in these alkaline soils often have deficiency symptoms to these nutrients.

Moisture extremes

Water is an important requirement for growth and survival of plants. Water needs of plants can vary greatly based on the species and environment. If water requirements are not adequately met for any given species, the plant's physiology and biochemistry are affected. Both water deficiency and excess can cause injury to plants. Sometimes the injury is short lived (acute several hours) or long lived (chronic days or weeks). Plants may recover from short term injury, but as the duration increases, the likelihood of recovery decreases due to sustained negative effects on overall plant function and growth.

Deficiencies in Available Water

Low water status in plants can occur either as a short term or chronic deficiency. A short term deficit of water might result in only minor effects on the plant such as wilted leaves or shoots. These symptoms may be temporary and occur during the warmest part of the day when transpiration rates are highest. Once the environmental stresses are relieved (i.e., air temperature declines, supplemental irrigation is applied, a rain event occurs, etc.) the symptoms of low water status may disappear.

High Temperature Damage

Some plant species can be very sensitive to high temperatures. In plants adapted for cooler climates, physiological changes can result in response to excessively high temperatures. For example, shoots and/or roots may stop growing if high temperatures prevail for an extended period of time. Roots may die. If high temperatures are coupled with low soil moisture, plants may exhibit scorching on the margins of the leaves, premature leaf drop, and in severe cases entire plant death. Sometimes physiological changes result in abnormal color or growth habits. For example, when geraniums (*Pelargonium spp.*) are subjected to temperatures above 95°F (35°C), newly forming leaves becomes "bleached" or white in color. Another common occurrence is the effect of high temperatures on pollination. Many food crops species are highly subject to poor pollination during periods of high temperatures.

Low Temperature Damage

Damage from low temperatures generally develops because ice crystals form in plant cells resulting in damage to cell membranes and organelles. Dehydration or low water status can also occur as a result of low temperatures. Many plants native to tropical regions can be injured by chilling injury (e.g., damage occurs above 32°F 0°C) and killed if subfreezing temperatures occur for long periods of time. Other plants may be better adapted for cold environments and not

experience damage until temperatures are at or below freezing (32°F 0°C). For example, Bermuda grass is adapted for the southern zones of the United States but slightly farther north Bermuda grass has an extended period of winter dormancy but also can be killed due to sustained subfreezing winter temperatures.

Chemical Injuries (Phytotoxicity)

Numerous agricultural chemicals are used in plant production and maintenance – herbicides, insecticides, fungicides and plant growth regulators. Always read the pesticide label before use. These materials are applied to protect plants from pests and improve plant health, but exposure to inappropriate products, overly high rates, or certain product mixtures can cause severe problems. When using a material or a mixture for the first time it is best to test on a small scale first.

1. **Herbicides :** Herbicides are used to control weeds, but exposure to non-target plants can be devastating. Herbicides may be nonselective, affecting all plants, or selective, affecting certain plant groups (e.g., broadleaf plants or grasses). They may be even more selective within those groups. Herbicide modes of action vary and include plant growth regulation (hormone mimics), amino acid biosynthesis inhibitors, lipid biosynthesis inhibitors, membrane disruptors and respiration inhibitors. These modes of action can affect photosynthesis, root growth, shoot growth, and other aspects of plant growth.

   Some herbicides cause root stunting or swelling and could be confused with damage from nematodes. Others herbicides cause necrotic/chlorotic spots or blotches that could be confused with a foliar disease. Some herbicides cause mottled colors, distortion or vein banding that could be confused with a viral disease. For example, the phenoxy herbicide 2,4-D, a synthetic auxin, causes distortion in grapes, cotton, tomatoes and many other plants, which could be confused with a virus disease. Diuron can cause discoloration along the veins in grapevine which could be confused with a virus disease or a nutritional problem.

2. **Fungicides and Insecticides :** While fungicides and insecticides are designed to protect plants from diseases and arthropod pests, inappropriate rates or tank mixes can cause problems. Some fungicides have well documented phytotoxic effects. Copper materials can cause tissue bronzing. Some cultivars of certain crops, such as grapes, are particularly sensitive to copper or S. Mixtures of some pesticides can cause damage, such as combining captan or sulfur with certain oils,

especially in hot weather. Fungicide labels and extension publications often mention known incompatibilities.

Plant Growth Regulators

Plant growth regulators (PGRs) are chemicals that alter the physiology of plants, affecting flowering, elongation, root growth, and other functions. They are utilized in the production of many types of crops. For example, in some fruit crops, PGRs are used to reduce vegetative growth, thin fruit to appropriate levels and improve fruit quality. In ornamentals, gibberellin inhibitors are applied as foliar sprays or substrate drenches to reduce shoot elongation, making plants stronger, more compact and of overall higher quality. In turfgrass, cell elongation inhibitors are used to reduce mowing frequency and cell division inhibitors are used to reduce seed head production.

Unfortunately, PGRs also can have negative effects. For example, foliar applications of chlormequat chloride, a gibberellin inhibitor, can cause temporary chlorosis in ornamentals. If applied at the wrong rate, the wrong time, or under certain environmental conditions (e.g., high light or high temperature), the consequences can be severe, with long lasting discoloration, stunted growth, or other symptoms. Certain fungicides also have negative growth regulating effects, which may be stronger in particular species or cultivars or more severe in hotter weather.

Deicing Salts

Deicing salts used to lower the melting point of ice on roadways and sidewalks can cause significant damage to surrounding trees, lawn, and landscape plants. For example, sodium chloride salt (NaCl) separates into sodium (Na) and chloride (Cl) when dissolved in water, both of which are toxic to plants in high amounts. Sodium impacts nutrient availability (particularly Mg and K) by competing at the root exchange sites. The disassociated chloride ion is also taken up by plants and accumulated in leaves where it can reach toxic levels.

Salt absorbs water and also changes the osmotic potential of the soil solution so that water flows out of the root instead of in. A "burn" symptom is the result of the combination of these factors and is generally noted as a chlorotic scorch symptom of leaf blades, with or without a well-defined lesion margin. Salt toxicity is usually reported, and first observed, in the spring after snow melt. Diagnosis of salt injury is greatly aided by knowing the proximity of the plant to a road or sidewalk surface and observation of a decrease in plant symptoms as distance increases from the salt source.

Mechanical Injury

Mechanical damage comes in many forms such as storms, misuse of equipment, or animal activity. Most of this type of injury is linked to a specific event and symptom expression can generally be traced back to the time of the event. Symptoms of broken limbs, flattened tree tops or torn bark on tree trunks are easy indicators of mechanical damage. However, subtle symptoms of a general decline can develop when the root system is affected. Mechanical injury can cause opens wounds on plants that can be used as points of entry for pathogens, lead to a slow decline, or kill them outright.

Unusual Plant Growths

Other symptoms that can be mistaken for plant diseases but are actually abiotic in origin are variegated plants, sloughing bark and odd growths. Plants that are propagated because they have variegated leaves are very common in the horticultural industry. For example, variegated hostas are commonly planted in many gardens. The white tissue in the leaves of these plants lacks chlorophyll. This condition is the result of a genetic mutation. These plants are generally produced vegetative because the trait is not stable through the process of typical seed production. Variegated plants tend to be smaller than their solid green counterparts because they produce less chlorophyll.

In trees, bark that is unusually rough or that separates from the wood may be of concern. If this is a new event, then concern is probably warranted. If the tree loses its bark every year then this is more likely a normal occurrence. Sycamore trees tend to slough their bark off in early summer following a period of rapid growth. Sometimes the bark gradually falls off and sometimes appears to blow off within a couple of days. This process is normal and does not affect the health of the tree.

Bumpy bark, burls, and other odd plant growths often look like symptoms of infectious diseases. However, burls, lignotubers and other growths on trees are usually the result of an injury. Burls frequently can be small or grow to a very large size. The bark covers the burl and remains intact rather than appearing cracked or sunken. Lignotubers frequently hold a cluster of buds that sprout rapidly in the spring. These gnarled growths look odd but they don't seem to have a major impact on the health of the tree.

Animal Damage

Although not technically "abiotic", damage by mammals and birds is included here as another example of injuries that could be confused with disease

symptoms. In addition, animal damage can predispose plants to diseases by weakening the plant and providing entry points for pathogens through wounds.

Feeding by insects and arthropods is important to overall plant health but is not covered here. During cold winters with abundant snowfall, voles and rabbits will feed on shrubs and young or thin barked trees. Small plants can be clipped off at the height of the snow pack and extensive feeding can result in girdling and death of the plant. Habitat modification, protective barriers and live trapping are the best options for managing this type of damage.

Animals such as porcupines and squirrels can damage trees by removing bark. Small twigs that have been clipped may be visible on the ground. In urban landscape trees, squirrel damage is minimal in most years. During stressful years, however, squirrels can strip enough bark to cause branch dieback in the tree. Individual trees can be protected by placing a 2 foot wide collar of metal hardware cloth located about 6 feet off the ground. Live traps can also be used to remove squirrels.

Deer browse on buds and young stems as they feed which can result in a bushy appearance of some plants. This may affect the aesthetic quality of the plant. More damaging are male deer that cause extensive limb breakage when they rub the velvet from their antlers. This damage can permanently disfigure and weaken trees. Fencing and repellants are the primary management strategies for preventing this type of injury. Deer also feed on many agronomic and horticultural crop plants and can be a nuisance for many farmers. On tree trunks, multiple holes in a straight line, oriented either vertically or horizontally, are indicative of sapsucker damage. Yellowbellied sapsuckers are in the woodpecker family. As with other woodpeckers, they feed on insects, but their brush like tongue allows them to feed on tree sap as well. Sapsuckers feed on many ornamental and fruit tree species, including pine, maple, birch, hemlock, Atlas cedar, mountain ash, as well as cherry and apple.

Heavily damaged trees will be less vigorous and more prone to environmental stress, disease and insect problems. Yellow bellied sapsuckers are protected by both state and federal law under the Migratory Bird Treaty Act, so it is illegal to kill them. Using barriers, repellants or scare tactics may help to reduce their feeding activity.

Some Non-infectious diseases

1. **Blossom drop :** Affected blossoms dry, turn brown, fail to produce fruit, and may fall from their small stems (pedicels). Blossom drop has

been associated with environmental stresses and improper soil fertility that inhibit flower pollination. Prolonged periods of high temperatures (above 90°F) and wind during the day are associated with blossom drop. However, night temperatures may be most important for effective flower pollination. Night temperatures outside the range of 55 to 70°F inhibit effective pollination and promote blossom drop. Excessive nitrogen fertilization encourages lush vegetative growth and inhibits flower production and/or pollination.

Control: Following recommended fertilizer rates for tomatoes reduces blossom drop, but if excessive heat and wind develop during flowering, little can be done. Heat-tolerant cultivars have recently been developed which may reduce blossom drop caused by high temperatures. Shading and overhead watering can reduce temperature, but their value decreases under prolonged periods of high temperature.

2. **Blossom-end rot :** Blossom-end rot is an easily recognized disorder of tomato. Fruit are most commonly affected when they are about half grown. Symptoms begin as small, tan, water-soaked area near the blossom end of the fruit. The spot enlarges, darkens, and becomes sunken and leathery. Affected fruit often ripen prematurely and are prone to invasion from secondary, fruit-rotting pathogens.

Fig. 18.1: Blossom-end rot

Blossom-end rot is a complex disorder thought to be caused by a localized calcium deficiency in the blossom end of the fruit. Calcium is required in large amounts in the growing parts of plants when rapidly growing fruit lack adequate calcium, tissue breakdown results. Calcium is water-soluble and moves with the plant's water stream. Under hot and windy conditions, water movement and use is rapid. Since fruit do not

transpire water as efficiently as leaves, calcium availability to the outer areas of fruit becomes limiting. Thus high temperatures and wind, widely fluctuating water availability and drought stress promote blossom-end rot. Conversely, excessive soil moisture for prolonged periods can damage the root system and its ability to uptake calcium. Soil deficiency in calcium is rarely a direct cause of blossom-end rot in Oklahoma. However, excessive growth due to too much nitrogen fertility can also promote blossom-end rot.

Control: Recommended soil fertility practices for tomato production in Oklahoma should be followed. Use of nitrate forms of nitrogen fertilizer, rather than ammonium forms which can interfere with calcium uptake, may reduce blossom-end rot. Highly susceptible tomato varieties such as Roma should be avoided. Maintain adequate and uniform levels of soil moisture though irrigation, particularly during periods of drought and high temperatures. Drip irrigation along with the use of plastic or organic mulches facilitates the maintenance of a uniform and non-fluctuating soil moisture profile. In the unlikely event that a soil test report indicates a calcium deficiency, applications of agricultural lime or gypsum should be made prior to transplanting. Foliar sprays with soluble forms of calcium are of little value because of poor absorption and movement to fruit where it is needed.

3. **Cat face :** Cat face is a term describing misshapen fruit with scars on the blossom end. Affected fruit are often kidney shaped, but can also be distorted into other shapes. Bands of brown scar tissue may form between swollen areas of the fruit. Cavities or indentations extend deep into the fruit. Affected fruit ripen unevenly and market quality is reduced. Catface occurs most often on the first-formed fruits. Tomato varieties with very large fruit are most susceptible. Catface is the result of abnormal development of the pistil of the flower, which eventually forms the fruit. Cold temperatures (below 58°F) during flower development and bloom and exposure to hormone-type, phenoxy herbicides such as 2,4-D are known to cause catface.

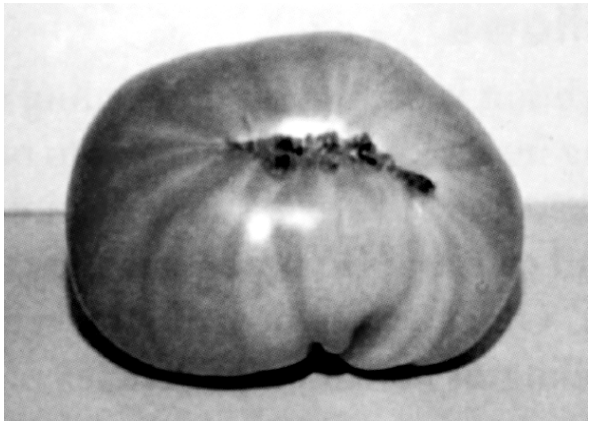

Fig. 18.2 : Cat face

Control: Little can be done to control catface other than avoiding highly susceptible, large-fruited varieties and keeping phenoxy herbicide away from tomatoes.

4. **Sunscald :** Mature green and breaker-stage (first pink) fruit exposed to direct sunlight frequently develop sunscald. Affected fruit develop white, bleached areas on exposed areas (usually the shoulders of the fruit), which become obvious during ripening. Sunken areas with a paper-like surface later develop.

   Control: Maintaining healthy foliage is important in protecting developing fruit from sunscald. Controlling foliar fungal and bacterial diseases, as well as spider mite damage is essential in preventing premature defoliation. Caged tomatoes suffer less sunscald than stalked tomatoes or those allowed growing on the ground. Artificial shading may be beneficial where fruit exposure to direct sunlight is expected.

5. **Yellow shoulder :** Yellow shoulder, also referred to as heat ripening or persistent green shoulder, is a ripening disorder of tomato fruit. The shoulders of affected fruit remain green or eventually turn yellow, but not red. The entire shoulder or an irregular patch may be affected. Internal areas of the upper fruit remain hard and white. The cause of yellow shoulder is not fully understood. Apparently fruit exposed to high temperatures during fruit maturation and ripening express this disease. Tomato varieties vary in susceptibility to yellow shoulder, with those having dark green shoulders being more susceptible than uniform ripening varieties.

   Control: The best control is achieved through the use of varieties with resistance to yellow shoulder. During periods of high temperatures, a lower incidence of yellow shoulder can be expected when fruit are picked at the breaker stage (first pink color) and are allowed to ripen at room temperature.

18.3 Plant Nutrients and Deficiencies Diseases

Damage from excessive macronutrient levels can occur in crop and ornamental plants as the result of over application of fertilizers or manures. Nitrogen toxicity is most typical under hot, dry conditions and plants turn an overly deep shade of green. Lesions often occur on the stems of annual seedlings and these can be confused with canker diseases. Similarly, twisting and distorting of mature tomato plants that experience ammonium toxicity may appear similar to symptoms caused by viruses. Ammonium toxicity can be a problem in greenhouse soils because of the lack of specific microorganisms that convert ammonium to nitrite and then to nitrate.

18.3.1 Nitrogen

Symbol: N; available to plants as nitrate (NO_3^-), and ammonium (NH_4^+) ions. Nitrogen (N) deficiency is a major limitation for non-leguminous agricultural plants, and use of N fertilizer for crop and ornamental plants is higher than any other single macro-or micronutrient. Nitrogen is important for the production of chlorophyll, the pigment that makes plant tissues green. Plants deficient in N typically have a pale yellow color (chlorosis) as a result of reduced chlorophyll production. Nitrogen is also a vital element for many other plant physiological processes as a component of proteins. Therefore, plants that are deficient in N may also appear stunted and display poor vigor. Because N is highly mobile in the plant, N deficiency is usually observed first in the older leaves of the plant. These older leaves may senesce while younger shoots near the apical meristem remain healthy. Nitrogen deficiencies are common in non-legume crops because N is quickly leached out of the soil once it is converted into NO_3 by soil microbes. Plants can absorb N in two ionic forms, NO_3^- and NH_4^+. Soil amendments that are commonly used to provide N to plants include a variety of synthetic fertilizers in addition to cover crops and compost applications. Nitrogen deficiencies may also result from infection by root pathogens such as rootknot nematodes (*Meloidogyne* spp.). Nitrogen deficiencies can cause increased susceptibility to certain leaf pathogens such as *Alternaria solani,* while excessive plant N levels may result in increased susceptibility to other pathogens such as *Botrytis cinerea*, or *Rhizoctonia solani*.

Nutrient functions

1. N is biologically combined with C, H, O, and S to create amino acids, which are the building blocks of proteins. Amino acids are used in forming

protoplasm, the site for cell division and thus for plant growth and development.

2. Since all plant enzymes are made of proteins, N is needed for all of the enzymatic reactions in a plant.
3. N is a major part of the chlorophyll molecule and is therefore necessary for photosynthesis.
4. N is a necessary component of several vitamins.
5. N improves the quality and quantity of dry matter in leafy vegetables and protein in grain crops.

Deficiency symptoms

1. Growth is retarded due to lack of cell division and cell enlargement thus shows stunted growth.
2. Chlorophyll contents are lowered and leave turn yellow in color.
3. It affects respiration, photosynthesis, amino acid synthesis, protein synthesis and nucleic acid metabolism.
4. Cellular proteins get depleted.
5. Induces dormancy and senescence, shedding of leaves, reduction of flowering are the common symptoms. Fruits remain immature or remain small even if they are mature.

18.3.2 Boron

Boron (B) is a micronutrient that is essential for cell wall formation and rapid growing points within the plant, such as reproductive structures. Interestingly, while higher plants require B, animals, fungi and microorganisms do not need this nutrient.

Boron is found to be antagonistic to the uptake of potassium and a few other cations, but favors the absorption of calcium. Boron is very effective in inducing reproductive structures in *Marchantia* and inducing germination of pollen tubes. It is also implicated in taking part in fat metabolism, photosynthesis, phosphate metabolism, etc. But the most significant role played by boron is in the translocation of photosynthates, particularly the transportation of sucrose across membrane.

More than 90 percent of B is found in cell wall structures. B deficiencies generally stunt plant growth by reducing cell wall extension at the growing point. Younger leaves show symptoms first, which indicates B is not readily translocated in the plant. Deficiency symptoms may include reduced flowering, thickened, curled, chlorotic leaves, and soft or necrotic spots in fruits and

tubers. Boron deficiencies are more pronounced during drought periods, when root activity is restricted.

Nutrient functions

1. B is necessary in the synthesis of one of the bases for RNA formation and in cellular activities.
2. B has been shown to promote root growth.
3. B is essential for pollen germination and growth of the pollen tube.
4. B has been associated with lignin synthesis, activities of certain enzymes, seed and cell wall formation, and sugar transport.

Crops remove B from the soil, in the form of boric acid H_3BO_3. Several factors influence B availability in the soil and uneven distribution within the soil is common. Organic matter is the most important reservoir for B. Extreme hot, dry conditions and extreme cold conditions may decrease organic matter decomposition and reduce the release of B into the soil solution. Plant availability is good over a wide range of pH, from 5.0 to 7.5. Boron is mobile in the soil and is subject to leaching. Leaching is of greater concern in sandy soils and/or in areas of intensive irrigation or high rainfall.

Deficiency symptoms

1. Generally, B deficiency causes stunted growth, first showing symptoms on the growing point and younger leaves. The leaves tend to be thickened and may curl and become brittle.
2. In many crops, the symptoms are well defined and crop-specific, such as:

• Peanuts: hollow hearts

• Celery: crooked and cracked stem

• Beets: black hearts

• Papaya: distorted and lumpy fruit

• Carnation: splitting of calyx

• Chinese cabbage: midribs crack, turn brown

• Cabbage, broccoli, and cauliflower: pith in hollow stem

3. Death of the stem tip and root tips is a significant symptom.
4. Heart rot of sugar beet, browning of cauliflower, top sickness of tobacco, hardness in citrus fruits are common.
5. Decreases the production of flowers.
6. Abnormal tillering, cutting, brittleness symptoms are not uncommon.

Table 18.1 : Deficiency Symtoms of Macronutrients

Nutrient	Deficiency Symptoms	Comments	Fertilizer Sources
MACRONUTRIENTS **Replace macronutrients in soils regularly (at least once per growing season)**			
calcium (Ca)	New leaves (top of plant) are distorted or irregularly shaped. Causes blossom-end rot.	Desert soils and water generally have plenty of calcium, so deficiency problems are rare. Excessive calcium can limit the availability of other nutrients.	Anything with the word "calcium"; also gypsum.
nitrogen (N)	General yellowing of older leaves (bottom of plant). The rest of the plant is often light green.	Most plants absorb nitrogen in the form of ammonium or nitrate. These forms readily dissolve in water and leach away.	Anything with the words "ammonium," "nitrate," or "urea." Also manures.
magnesium (Mg)	Older leaves turn yellow at edge leaving a green arrowhead shape in the center of the leaf.	Plants absorb magnesium as an ion (charged particle), which can be readily leached from soil. May be readily leached from soil if calcium is not present.	Anything with the word "magnesium"; also Epsom salts (magnesium sulfate).
phosphorus (P)	Leaf tips look burnt, followed by older leaves turning a dark green or reddish-purple.	Plants absorb phosphorus in the form of phosphate. This form dissolves only slightly in water, but pH strongly affects uptake.	Anything with the words "phosphate" or "bone." Also greensand.
potassium (K)	Older leaves may wilt, look scorched. Interveinal chlorosis begins at the base, scorching inward from leaf margins.	Plants absorb potassium as an ion, which can be readily leached from soil. Desert soils and water generally have plenty of potassium, so deficiency problems are rare.	Anything with the words "potassium" or "potash."
sulfur (S)	Younger leaves turn yellow first, sometimes followed by older leaves.	Plants absorb sulfur in the form of sulfate. This readily leaches from the soil. Sulfur may acidify the soil (lower the pH).	Anything with the word "sulfate."

Table 18.1 : Deficiency Symtoms of Micronutrients

MICRONUTRIENTS *Replace when deficiency symptoms are evident.*			
boron (B)	Terminal buds die, witches' brooms form.	Plants absorb boron in the form of borate. Problems are seen in intensely cropped areas.	Anything with the words "borax" or "borate."
copper (Cu)	Leaves are dark green, plant is stunted.	Plants absorb copper as an ion. Arizona soils have plenty of copper, so problems are rare.	Anything with the words "copper," "cupric," or "cuprous."
iron (Fe)	Yellowing occurs between the veins of young leaves.**	Plants absorb iron as an ion through their foliage as well as their roots. Uptake is strongly affected by pH. Chelated iron is readily available for use by the plant, other forms of iron may be tied up in the soil.	Anything with the word "iron chelate."
manganese (Mn)	Yellowing occurs between the veins of young leaves. Pattern is not as distinct as with iron. Palm fronds are stunted and deformed, called "frizzle top." Reduction in size of plant parts (leaves, shoots, fruit) generally. Dead spots or patches.	Plants absorb manganese as an ion through their foliage as well as their roots.	Anything with the words "manganese" or "manganous." Often required with zinc application.
molybdenum (Mo)	General yellowing of older leaves (bottom of plant). The rest of the plant is often light green.	Plants absorb molybdenum in the form of molybdate. Problems are rare in Arizona soils but are occasionally seen on legumes where it mimics nitrogen deficiency.	Anything with the words "molybdate" or "molybdic."
zinc (Zn)	Terminal leaves may be rosetted, and yellowing occurs between the veins of the new leaves.	Plants absorb zinc as an ion through their foliage as well as their roots. High pH may limit availability.	Anything with the word "zinc."

18.3.3 Zinc

Zinc (Zn) is taken up by plants as the divalent Zn cation. (Zn^{2+})It was one of the first micro-nutrients recognized as essential for plants and the one with most commonly limiting yields. Although Zn is required in small amounts, high yields are impossible without it.

Zinc is heavily involved in enzyme systems that regulate the early growth stages, and is vital for fruit, seed and root system development, photosynthesis, formation of plant growth regulators and crop stress protection. Further, Zn is a team player with nitrogen (N), phosphorus (P) and potassium (K) in many plant development processes.

Soils require Zn in very small amounts compared with N or K. Only about a half pound of Zn is needed per acre for high yield (180 bushels per acre) corn production. Sixty bushel wheat needs about 0.28 pound of Zn per acre. Yet, lack of Zn can limit plant growth, just like N or K, if the soil is deficient or crop uptake is restricted.

In addition to being an essential component of various enzyme systems for energy production, Zn is required in protein synthesis and growth regulation. Zinc deficient plants also exhibit delayed maturity. Since zinc is not mobile in the plant, Zn deficiency symptoms occur mainly in new growth. This lack of mobility in plants suggests the need for a constant supply of available zinc for optimum growth.

Nutrient functions

1. Zn is required in the synthesis of tryptophan, which in turn is necessary for the forma-tion of indole acetic acid in plants.
2. Zn is an essential component of several metallo-enzymes in plants (variety dehydrogen ases) and therefore is necessary for several different functions in plant metabolism.
3. The enzyme carbonic anhydrase is specifically activated by Zn.
4. Zn has a role in RNA and protein synthesis.

Deficiency symptoms

1. Interveinal chlorosis occurs on younger leaves, similar to Fe deficiency. However, Zn deficiency is more defined, appearing as banding at the basal part of the leaf, whereas Fe deficiency results in interveinal chlorosis along the entire length of the leaf.
2. In vegetable crops, color change appears in the younger leaves first. The new leaves are usually abnormally small, mottled, and chlorotic.
3. In citrus, irregular interveinal chlorosis occurs with small, pointed, mottled leaves. Fruit formation is significantly reduced.
4. In legumes, stunted growth with interveinal chlorosis appears on the older, lower leaves. Dead tissue drops out of the chlorotic spots.
5. Short internodes (rosetting) and a decrease in leaf size.

6. Chlorotic bands along the midribs of corn, mottled leaves of dry beans and chlorosis of rice.
6. Loss of lower bolls of cotton and narrow, yellow leaves in the new growth of citrus also have been identified as symptoms of Zn deficiency.
7. Delayed maturity also indicates Zn deficient plants.

18.4 Diseases due to Excess of Inorganic Pollutants

18.4.1 Ozone

Ozone is the most important, plant-toxic air pollutant worldwide. It is an active form of oxygen that causes a variety of symptoms, including tissue collapse, interveinal necrosis and markings on the upper surface of leaves known as stipple (pigmented yellow, light tan, red brown, dark brown, red, black, or purple), flecking (silver, or bleached straw-white), mottling, chlorosis or bronzing bleaching, and a marginal rolling and scorching of leaves on lilac. Growth is stunted. Flowering and bud formation are depressed. Affected leaves of certain plants, such as citrus, grape, and pines, both wither and crop prematurely. Conifers frequently show a mottled green and yellow to brown and tip burn, or a yellow to brown or orange-red flecking, banding and reddish-brown dieback of the needles. Susceptible white pines range from stunted to dwarfed and chlorotic. The injury pattern in small grains and forage grasses generally occurs as a scattering of small, yellowish or white-to-tan flecks on one or both leaf surfaces. The flecks later may merge to form larger, bleached-white to yellowish dead areas.

Ozone usually attacks nearly mature leaves first, progressing to younger and older leaves. Young plants generally are the most sensitive to ozone; mature plants, relatively resistant. Ozone-killed tissues are readily infected by certain fungi (for example, *Botrytis*).

The injury pattern on the foliage is initially observed on older mature leaves near the crown or center of the plant, often progressing with time to the younger foliage. The yellowing of the plant centers in rows of watermelon is quite distinctive and can give fields an obviously striped pattern of alternating yellow and green bands. This type of injury on watermelon can be referred to as "center of the crown dieback."

In contrast, injury on muskmelons is typically much less severe and is visible at a later stage of plant development. Irrigated plants will promote greater symptom development if the cultivar is sensitive compared with drought-stressed plants. Ozone injury on watermelons generally appears in mid to late

July prior to fruit maturation. Ozone injury on beans appears as bronzing on the upper leaf surface and as the problem progresses necrotic lesions are formed that coalesce and become reddish brown.

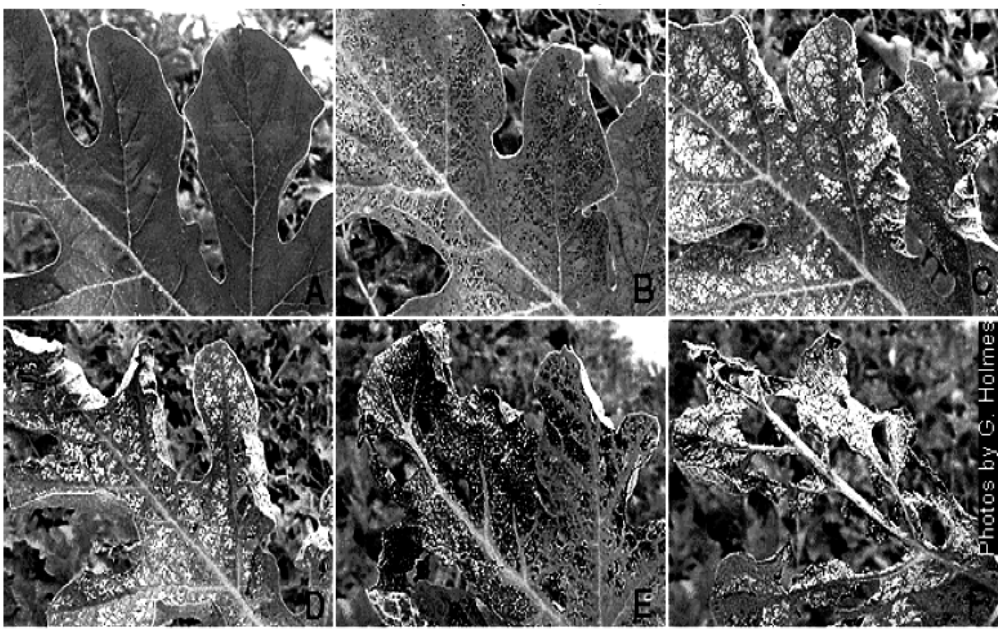

Fig. 18.3 : Progression of ozone damage on watermelon foliage

Ozone is brought down from the stratosphere by turbulence in strong vertical down-drafts during severe electrical storms; more important, it is produced when sunlight reacts with nitrogen oxides and hydrocarbons formed by refuse burning and combustion of coal or petroleum fuels, especially the exhaust gases from internal-combustion engines. When oxidant levels in the air are high, more than 90 percent is ozone. These levels usually are at their highest point from around 11 a.m. to 5 p.m. and relatively low at night.

18.4.2 PAN (Peroxyacetyl Nitrate)

The most important, plant-toxic oxidant, next to ozone, is PAN. It is formed by oxides of nitrogen reacting with unsaturated hydrocarbons (simple olefins) in the presence of light. Like ozone, PAN is produced when sunlight reacts with various exhaust gases from motor vehicles and industries. PAN causes a collapse of tissue on the lower leaf surface of numerous plants. The typical leaf marking is a glazing, bronzing, or silvering that commonly develops in bands or blotches. On some plants, such as petunia, Pinto bean, tomato and tobacco, the collapse may be through the entire thickness of the leaf blades. In grasses, the collapsed tissue has a bleached appearance, with tan-to-yellow, transverse

bands. Conifer needles turn yellow. Early maturity or senescence, chlorosis, moderate to severe stunting and premature leaf drop may also occur. PAN is most toxic to small plants and young leaves. The very young and most mature leaves are highly resistant.

Fig. 18.4 : PAN damaged Milkweed leaves

Concentration-Typical damage to susceptible plants occurs with PAN at levels of 0.01 to 0.05 ppm for an hour or more. Plant injury requires light before, during, and after exposure. Injury is increased by any factor contributing to maximum plant growth. PAN is best known in the Los Angeles basin area, with injury occurring on vegetation from Seattle to San Diego. Little is known about the concentration of PAN in the Midwest or the eastern United States, although it has been reported on a few plants. PAN is unstable, particularly at temperatures above 90°F (32°C).

18.4.3 SO_2 (Sulfur Dioxide)

The exposure of succulent, broadleaved plants to SO_2 (and its byproduct, sulfuric acid) usually results in dry, papery blotches that are generally white to tan or straw-colored and marginal or interveinal. On some species, chronic injury results in brown to reddish-brown or black interveinal blotches. Both the upper and lower leaf surfaces are affected. The leaf veins normally remain green. Chlorosis (yellowing) and a gradual bleaching of the surrounding tissues are fairly common. Injured grass blades develop light tan to white streaks on either side of the mid vein. A tan to reddish-brown dieback or banding occurs on conifer leaves, with adjacent chlorotic areas. Growth suppression, reduction in yield and heavy, premature defoliation may also occur. Full-grown and nearly full-grown leaves and young plants are most susceptible to SO_2. Young and old leaves are usually less sensitive.

Concentration-The degree of injury increases as both the concentration of sulfur dioxide and/or the length of exposure increases. Sensitive plants are injured by exposures as low as 0.5 parts per million (ppm) for 4 hours, or 0.25

ppm for 8 to 24 hours. Plants are most sensitive to SO_2 during periods of bright sun, high relative humidity and adequate plant moisture during late spring and early summer when plants are making the most rapid growth.

18.4.4 HF (Hydrogen Fluoride)

The typical injury by gaseous (primarily hydrogen fluoride (HF) and silicon tetra fluoride (SiF4) or particulate fluorides is a yellowish mottle to a wavy, reddish-brown or tan "scorching" at the leaf margins and tips of broadleaved plants, and a "tip burn" of grasses and conifers. A narrow, chlorotic to dark-brown band often occurs between living and dead tissue. Citrus, poplar, sweet cherry and corn foliage exhibit a chlorotic mottling, streaking, or blotching prior to the development of the typical "burned" area. Leaves and fruits, such as apple, apricot, citrus, fig, peach, plum, and prune, may fall prematurely.

Injured areas in stone-fruit leaves may become brittle and drop out, leaving shot-holes.

The young, succulent growth is most easily injured. Fruits may soften or become necrotic at the blossom end. Fluoride-contaminated forage that is eaten by cattle or sheep may cause fluorosis. Fluorides (compounds containing the element fluorine) are produced by combustion of coal and by glass, aluminum, steel, pottery, brick, and tile, as well as ferro-enamel, cement, fiberglass and ceramic industries. They are also produced by refineries, metal ore smelters and phosphate fertilizer factories.

Concentration- Accumulated leaf-fluoride concentrations of 10 to 150 ppm often result in injury to sensitive plants, although resistant cultivars and species of plants will tolerate leaf concentrations of 500 to 4000 ppm or more without visible injury. A 4-week exposure of susceptible gladiolus to an air concentration of 0.0001 ppm, or less than 24 hours at 10 parts per billion (ppb), produced leaf concentrations of 150 ppm and definite tissue necrosis. There is a wide variation among cultivars or clones of the same plant in their susceptibility to fluorides: for example, apricot, begonia, corn, gladiolus, grape, peach, ponderosa and white pines, roses and sweet potato. The extent of tissue damage is related to the dosage and the quantity of accumulated fluoride.

18.5 Control Measures

Air pollution can be controlled by different methods depending on the source and the pollutant. The different methods are:

One of the major causes of air pollution is the automobiles. The fuels being used should be lead free as this will reduce the level of lead in the atmosphere.

The carburetor should be cleaned regularly and good quality fuel should be used. This reduces the smoke emission from the exhaust pipes of the vehicles. Efforts to introduce vehicles running on alternate sources (for example solar energy) of energy should be made. These methods will go a long way in reducing the occurrence of photochemical smog.

The industrial pollution is best controlled at source. The polluting gases should be passed through filters and other devices such as cyclone collectors, scrubbers, precipitators, etc. so that the particulate matter is removed before the waste gases are released out. The toxic gases should be detoxified.

The domestic and industrial smoke producing units should have long chimneys to take the polluting gases far above and then disperse over a larger area. They should also invest in solar cookers or bio gas. The pollution by sulphur dioxide is mainly due to coal based industries. Alternate non-sulphur containing fuel must be used. It is also possible to remove the sulphur from the fuel before use.

There are many plant species like the neem (*Azadirachta indica*), bel (*Aegle marmelos*), gulmohur (*Delonix regia*), etc. that clean the atmosphere. More trees of such types should be planted.

For effective control and prevention of air pollution it is important to educate people and create public awareness about the ill effects of air pollution.

The following are some methods that may be adopted to control pollution on a large scale:

Combustion: Pollutants in the form of organic gases or vapors can be burnt to convert them into water vapour and relatively less harmful products, such as carbon dioxide.

Absorption: The gaseous effluents may be made to pass through scrubbers or absorbers. These contain a suitable liquid absorbent, which removes or modifies one or more of the pollutants present in the gaseous effluents making it comparatively harmless.

Adsorption: The gaseous effluents are passed through porous solid adsorbents kept in suitable containers. The organic and inorganic constituents of the effluent gases are trapped at the interphase of the solid adsorbent. Adsorbents hold (molecules of a gas or liquid or solute) to its surface, causing a thin film to form.

Methods to Control Particulate Emissions: Particulate emissions may be controlled by using mechanical devices that generally work on the basis of the following:

Gravity: In this process, the particles settle down by gravitational force. Sudden changes in the direction of the gas flow causes the particles to separate out due to greater momentum.

Fabric Filters: The gases containing dust are passed through a porous medium, which is usually woven fabric. The particles present in the gas are trapped and collected in the filters. The gases freed from the particles are then discharged.

Electrostatic Precipitators: When a gas or an air stream containing aerosols in the form of dust, fumes or mist, is passed between two electrodes, then, the aerosol particles get precipitated on the electrode.

The following practices also help in controlling air pollution:

Better designed equipment and smokeless fuels must be used in hearths in industries and at home.

Automobiles should be properly maintained and must adhere to emission control standards. (Bharat II or Euro II).

More trees should be planted along roadside. Renewable energy sources, such as wind, solar energy, ocean currents, must be tapped to fulfill energy needs.

Tall chimneys should be installed for vertical dispersion of pollutants.

18.6 Summary

There are so many non-parasitic diseases, which doesn't spread from one plant to another but can harm plants to a great extent.

Plants require various micro and macro nutrients from the immediate environment and when there is deficiency of any of these elements, the consequences usually drops the yield and make plants prone to various pathogens.

Different inorganic pollutants like ozone, SO_2, HF and various organic pollutants adversely affect plants morphologically and physiologically and may retard the growth.

18.7 Glossary

- **Ozone:** A very important constituent of our atmospheric layer (Ozone layer). Benefits the flora and fauna by inhibiting the access of U.V. rays but equally destructive when found in stratosphere.
- **PAN:** Peroxy acetyl nitrate is an organic pollutant and involved in photochemical smog.

18.8 Self-Learning Exercise

Section A : (Very Short Answer Type Questions)

1. Write full form of PAN.
2. Mention name of Inorganic Pollutants responsible for plant diseases.
3. Write down any 5 non-parasitic diseases of plants.
4. Name any one herbicide, causes plant diseases.
5. Who is responsible for temporporary chlorosis in ornamentals?

Section B : (Short Answer Type Questions)

1. Write a short note on followings-
   (i) Cat face disease
   (ii) Yellow shoulder disease
   (iii) Blossom drop disease
2. Write down a note on plant disease due to B deficiency.
3. Brifly explain the Nitrogen defiency diseases of plant.

Section C : (Long Answer Type Questions)

1. What do you mean by non-parasitic diseases? Explain in detail.
2 Describe various types of elemental deficiencies in plants.
3 Describe the ill effects of Ozone and PAN on healthy plants.
4 What are the controlling measures for air pollutants?

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Unit-19

Plant Galls-I: Classification and Anatomy

Structure of the Unit:

19.0 Objective

The main objectives of this unit are to study:

- a brief account of insect induced plant galls of Rajasthan specially Pongamia leaf gall and Ziziphus stem gall.
- Classification and anatomy of galls.

19.1 Introduction

Galls or cecidia are outgrowths on the surface of lifeforms. Plant galls are abnormal outgrowths of plant tissues and can be caused by various parasites, from fungi and bacteria, to insects and mites. Plant galls are often highly organized structures and because of this the cause of the gall can often be determined without the actual agent being identified. This applies particularly to some insect and mite plant galls. In pathology, a gall is a raised sore on the skin, usually caused by chafing or rubbing.

The meristems, where plant cell division occurs, are the usual sites of galls, though insect galls can be found on other parts of the plant, such as the leaves,

stalks, branches, buds, roots, and even flowers and fruits. Gall inducing insects are usually species-specific and sometimes tissue-specific on the plants they gall.

Plant Galls

Two important definitions can be cited about galls (i) "Galls are pathologically developed cells, tissues and organs of plants which have developed due to hypertrophy (over growth) and hyperplasia (excessive cell division) under the influence of parasitic organisms" or (ii) "Gall is a product of interspecific association between a plant and another organism characterized by the plant reacting with growth which is abnormal". Mechanical irritation, wounds, chemicals like mutagenic agents, various amino acids, excessive indole-3-acetic acid (IAA) and other plant growth hormones can also induce the formation of galls.

Gall arises on all classes of plants like fungi, algae, mosses, ferns, gymnosperms, dicotyledons and monocotyledons. Galls also arise on underground and aerial roots, shoot axis, petiole, stipules, leaf blade, leaf veins, vegetative and flower buds, inflorescence axis, bracts, flowers and fruits. The common galls are induced both by vegetable and animal parasites like virus, bacteria, fungi and even higher plants like *Viseum*. The galls induced by vegetable parasites are called Phylocecedia. The animals that induce galls are called cecidozoa. The common cecidozoa belong to protozoa, nemathelminthes, Acarina and insects. The galls induced by animal parasites are known as Zoocecidia. Some of the best known Zoocecidia are caused by Acarina like Eriophyes and by diverse groups of insects like Thysanoptera, Aleurodoidea, Psyllidae, Aphidoidea (Homoptera), Hymenoptera and Diptera etc. The branch of biology which deals with the study of galls is called Cecidology. In our country Ramakrishna (1920, 1928) generated interest in galls and gall workers. The work of Sunder Raman (1924), Saksena (1942) and Mani (1948) has developed a separate biological discipline.

19.2 Insect Induced Plant Galls of Rajasthan

Insect galls are the highly distinctive plant structures formed by some herbivorous insects as their own microhabitats. They are plant tissue which is controlled by the insect. Galls act as both the habitat and food source for the maker or the gall. The interior of a gall can contain edible nutritious starch and other tissues. Some galls act as "physiologic sinks", concentrating resources in the gall from the surrounding plant parts. Galls may also provide the insect with

physical protection from predators. Some most important insect induced plant galls are described briefly in follows:

19.2.1 *Pongamia* Leaf Galls (Anatomy)

It is a typical pouch gall, the vast majority of pouch galls develop on the upper surface of the leaf and the cecidozoa are naturally situated on the underside. A wide range of shape and external character are met with pouch galls. These galls are fleshy or hard, with thick walls and often have the ostiole more or less completely obliterated several of them have also fleshy or other out growths projecting from the wall into the cavity. The galls are remarkable for the total inhibition of differentiation of the normal tissues. The gall epidermis on the outer surface is continuous with that of the upper surface of the leaf and has often a conspicuously thicker cuticle than the normal leaf. The outermost layer of cells of the gall tissue is sometimes characterized by distinct thickening of cell walls but the cells are generally closely packed together. Chlorophyll is only sparingly present in these cells, particularly in younger galls. The deeper cells generally tend to increase in size, so that the bulk of the gall tissue is composed of large sized, thin walled parenchyma cells with considerable intercellular shapes or closely packed. The innermost layer of cells lining the gall cavity are small sized and closely packed. Numerous vascular bundles scattered in the gall tissue are connected with those of veins. These vessels have distinct xylem and phloem, the latter being directed to the inner side of the gall in other words to the gall cavity.The epidermis lining the gall cavity is very profoundly modified and is typically composed of rather large cells, with thin cuticle or without cuticle. Stomata are naturally never developed. Thick walled erect or curved hairs are commonly found especially near the ostiole opening. Large, plasma rich, thin walled stumpy and spirally twisted nutritive hair are found in the pouch gall cavity.

Fig.19.1: The pouch galls of *Eriophyes cheriani* on the leaf of *Pongamia glabra* Vent.

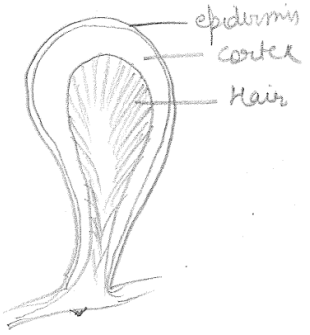

Fig. 19.2 : Anatomy of *Pongamia* leaf

19.2.2 Stem Galls on *Zizyphus*

Eriophyes cornuus (Acarina) induces shoot axis galls on *Zizyphus jujuba* (Rhamnaceae). Galls are irregularly globose, lobed, rugose or tuberculate, hard yellow to reddish brown crowded on the branches attaining sizes ranging from 25 m.m. to 50 mm in diameter (fig.3). No true epidermis but the gall surface has irregular, naked parenchymatous cells. The mass of the gall may consist wholly of parenchyma with scattered vascular elements. When old, the gall becomes brittle and readily crumbles into a black dust. Numerous mites feed externally on the surface of the gall in between the crevices of the tubercles.

This is one of the most commone gall occurring throughout India. New galls are abundant during the dry weather but in South India, the gall may be found throughout the year. Galls arise equally on young as well as older branches. Shoot galls are also induced by unknown diptera which appear on the tender ranches as a swollen curved fleshy stipular thorn. *Eriophyes* species induce shoot axis galls on *Zizyphus maritiana* which are circular disc shaped, deciduous, hollow, hemispherical epidermal emergences, sometimes irregular and scattered in large number on tender branches. Rarely several galls agglomerate into a single mass, stretching along the whole length of the tender branch. When mature, the gall cracks open and fall off as more or less clean disc exposing a circular scar on the branch. Gall cavity crowded with numerous pinkish brown mites. Gall normally occurs in Indo-Gangetic plains.

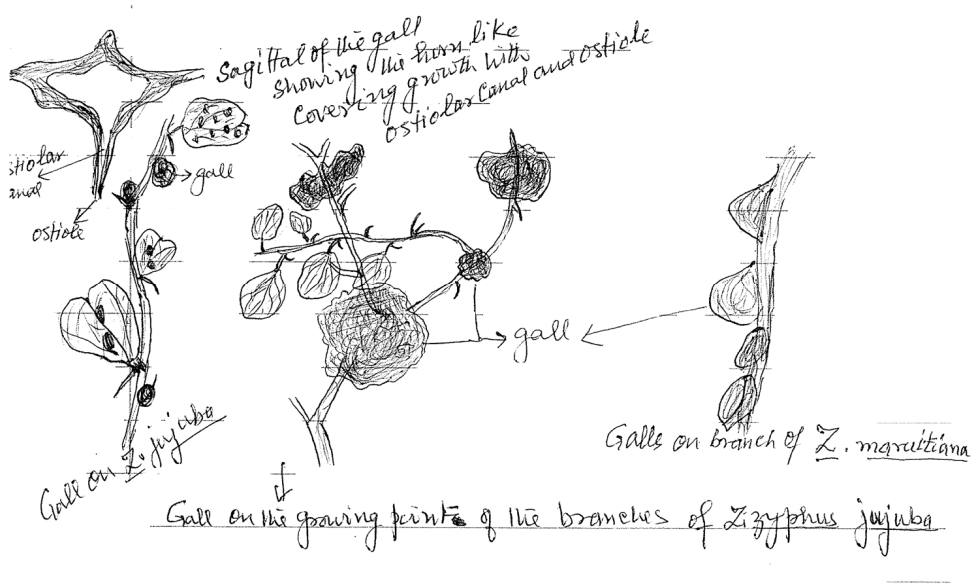

Fig. 19.3 : Stem Galls on *Zizyphus*

19.2.3 Classification and Anatomy of Gall

Classification

Mani (1964) classified the galls into two (1) Limited (Galls tissue are not much different from the normal plant. This includes galls induced by nematodes, insects mites and fungi. (ii) Unlimited (Degree of abnormal growth is not controlled by the nature of plant but by inciting factor. Braun (1969) mentioned two types of abnormal galls: (i) self-limiting tumors which possess definite pattern of growth and (ii) non self limiting tumors in which cells proliferate irregularly and are transplantable. A large number of Cecidozoa stimulate the host tissues to initiate many kinds of galls on the respective host plants.

Galls are found on all groups of plants, the greatest bulk of galls from India occur on plants of the family Leguminosae. Monaceae, Lauraceae, Combretaceae, Anacardiaceae, Cucurbitaceae, Rhamnaceae, Euphorbiaceae, Salvadoraceae, Polygonaceae, Umbelliferae, are dominant gall bearing plants in India. The Himalayas offer exceptional opportunities for the collector of galls in India. Many interesting galls however be found almost anywhere in waste grounds having wild weeds, gardens, cultivated and abandoned fields and forests. West and Northwest Himalayas are rich in endemic galls. Insect and mite induced galls (Zoocecidia) are very widespread in arid and semi arid zones of India, especially Rajasthan. In Rajasthan Zooeecidia are wide spread almost in all districts specially Jaipur, Sikar, Jodhpur, Kota, Udaipur, Bikaner, Jhunjhunu, Alwar and Bharatpur. Few galls occur almost throughout the year;

most galls are found only during and immediately after the monsoon rains and the hot weather is a unpromising period for the gall collector in India.

Galls of various stages of their development may be collected from the plants and are fixed in FAA, for at least 24 hrs and after washing thoroughly with water can be preserved in 70% alcohol for study. Galls can also be examined using Nicon Stereoscopic Zoom microscope while still attached to the plant.

Anatomy of Galls

The structure of galls depends primarily on the origin on which they are formed and the gall inducing organism. At time different gall makers give rise to extremely different type of galls (for example *Pongamia* leaf gall and *Ziziphus* stem gall).

Most of the galls are spherical, oval, Pyriform, fusiform, cylindrical, conical, disc like etc. The gall may be stalked or sessile. Sometimes they have long stalks, longer than the galls body. Galls ranges in colour from green, yellowish, green, yellow pink, violet or deep reddish brown. The colour differs with galls to galls and changes with their age. Size of galls varies from fraction of a mm to several cms. in diameter. Some galls are generally membranous and some may be very large, solid or fleshy. Generally a gall may have one or more cavities in which gall maker lives. This cavity is known as gall cavity / larval cavity (chamber). It may be situated in basal, central and peripheral position. The gall cavity may be connected to outside by a narrow passage or may be completely closed. Various morphogenic / structural types of galls are found in leaf gall as well as stem gall of *Ziziphus*.

1. **Filz galls:** Simplest galls in which the gall maker is always external. There is a heavy outgrowth of hair these hair like outgrowth are the epidermal cells these galls are generally formed on leaves but may also arise on petiole.
2. **Folds and roll galls:** These gall found in leaf blade down wards (epiphyllous/hypophyllous) associated with the affected parts. Most of the fold and roll galls become enormously swollen, spony, fresh on woody and fibrous. The folding and roling of the leaf results in enclosing the gall maker.

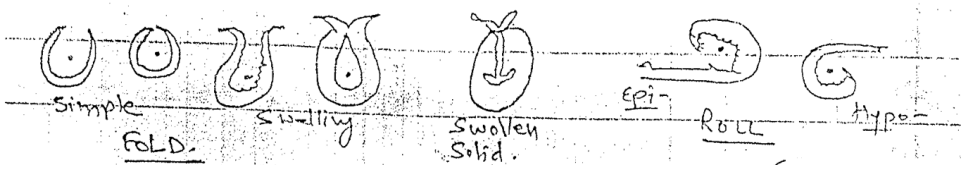

Fig. 19.4 : Folds and roll galls

3. **Pouch gall**

The pouch galls arise because of intense outarching of leaf blade. The arched part generally becomes swollen. The bulging and invagination takes place in such a way that the gall maker is enclosed within the cavity of the gall. The pouch galls are typically hollow. It is remarkable that the cecidozoa is in the gall and is really outside the plant tissue. These galls range from simple cup like out pocket to oval, globose, cylindrical horn shaped, sessile or stalked, simple or lobed. The cavity of the pouch gall is often filled with hairy processed freshly emergences in complete or complete out growth of walls.

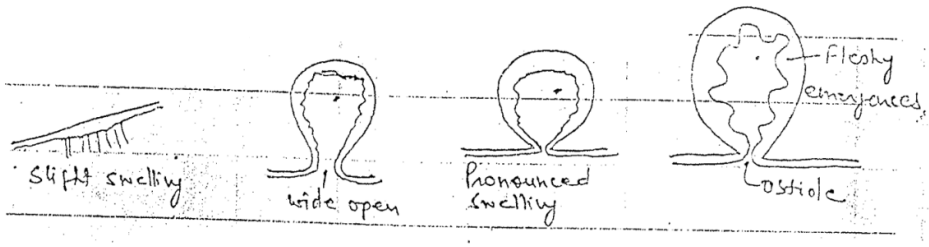

Fig. 19.5 : Pouch gall

4. **Krebs galls:** They are characterized by their enarmous growth in thickness. The cecidozoa remain permanently fixed externally on the gall epidermis. A localized cell proliferation results in the formation of the galls.

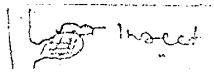

Fig. 19.6 : Krebs galls

5. **Covering galls**

In these galls the externally situated gall maker induce the tissue to grown around so that finally it is enclosed with the gall. The tissue around the cecidozoa become fused and ostiole is formed.

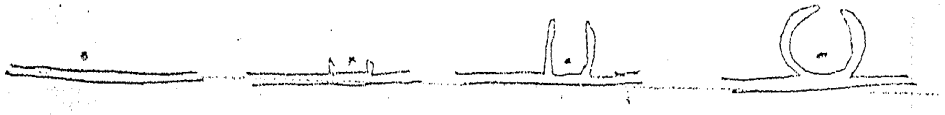

Fig. 19.7 : Covering galls

6. **Lysenchyme galls:** In these galls the cecidozoa is inside the gall cavity which is formed because of dissolution of cells. The cell lining the lumen of the cavity proliferate to give rise to the characteristic swelling of the galls. Example. Cynipidae

19.3 Summary

Galls are abnormal vegetive growth resulting from the work of insects, between the apparently normal and decidedly abnormal and as a consequence it is difficult to establish a satisfactory distinction between plant galls and deformations not worthy of classification under this term. Specially in the last two and a half decades, interest in studying galls and their inducers has grown throughout the world: several new species of gall inducing insects that live on specific host plants have been determined and described, greater volume of information explaining subtle physiological and morphogenetic processes in galls is known than before, unique patterns in the distribution of gall inducing insects and their host plants have the evolution of insect phytophagy, but also that of the interacting plants and insects.

19.4 Glossary

- **Bud galls:** Many deformations originate in buds. They vary from a plainly aborted bud to large swelling developed from such parts.
- **Cecidomyia**. This is a general term applied to any species referable to the gall midges or Itonididae and which can not be readily assigned to more closely defined genera.
- **Erineum.** These are the hairy or pile-like growths upon leaf surfaces produced by species of plant mites or *Eriophyidce.*
- **Flower galls**. Aborted or deformed flowers or masses of flowers afford suitable conditions for many gall insects.
- **Leaf gall**. This term applies to all deformations definitely associated with leaves.
- **Galls**. Outgrowths on the surface of lifeforms.

19.5 Self-Learning Exercise

Section A : (Very Short Answer Type Questions)

1. What is gall?
2. What is bud gall?
3. What is erineum?
4. Define flower gall.

Section B : (Short Answer Type Questions)

1. Write short note on leaf gall.
2. What is cecidozoa?
3. What is hypoplasia?

Section C : (Long Answer Type Questions)

1. Write the notes on classification of insect gall.
2. Write a note on insect gall of Rajasthan.

19.6 References

- Mani, M.S., Plant Galls of India (First Edition). Macmillian India, New Delhi, 1974, p. 323.
- Ananthakrishnan, T.N. and Raman, A. Thrips and Gall Dynamics. Oxford and IBH, New Delhi, 1989, p. 120.
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Unit-20

Plant Galls-II:
Mechanism and Physiology

Structure of the Unit:

20.0 Objectives

The main objective of this unit is to study:

- a brief account of the mechanism and physiology of galls

20.1 Introduction

IAA indole acetic acid (IAA) auxins are key factor in galls as well. An imbalance arising because of the stress induced by the physical action (wounding sucking) and salivary secretion either triggers new growth because of synthesis of growth promoters or enhances the vulnerability of plant cells to growth promoters that are already present at that site. A majority of gall inducing insects stimulates the host plant tissue to develop into galls by their feeding action.

20.2 Mechanism and Physiology of Galls

The precise mechanism by which different species of insects produce remarkably unique galls is still being debated by Cecidologists. The initiation of insect gall is typically associated with oviposition by the adult or by the feeding of early larval stages. Depending on the type of insect and whether it has chewing or piercing / sucking mouth parts, a variety of salivary fluids may be injected into the plant tissue. The ability to induce gall presupposes a functioning interaction of endogenous secretion of the gall former and the

physiological characteristics of the host plant i.e. only the suitable host plants an unimpaired gall formation may occur which ensures the development of the larva. According to Hori (1992), salivary fluid of bugs and beetles may include amino acids auxin (IAA) and various plant digesting enzymes such as pectinases, cellulases and proteases. The precise mechanism by which these chemicals induce cell division and morphogenesis is very complicated and varies with different species and even with different type of plants. Existing evidence suggests that most insects probably utilize both specific and general chemicals in their overall induction of a unique gall (Norris, 1979). There is no doubt that tumour formation is a result of a series of physiological changes in the host tissues which takes place as a result of association of the organism at one or the other stage of disease development.

Ceeidogenesis is a common phenomenon involving recanalisation and reorientation of plant development and such growth activities result in the insect becoming partially or completely enclosed, so that the gall insects grow, mature and reproduce within the gall. An interaction between the offensive stimuli involving growth substances released by insect and the defensive response by plants appears to be the hall mark of gall production (Rosenthal and Janzen, 1979). Symbiotic association between insects and microbes to produce gall is also recognised (Krishnamurthy, 1984).

Anders (1958, 1961) demonstrated the presence of amino acids in the saliva of aphid *Phylloxera vastatrix* causing gall on *vitis.* Combination of Tryptophane, histidine and valine was found principal Cecidogenic agent in the saliva of the aphid. However Anders suggested that aphid saliva did not contain amino acids but only the prolease enzyme. It was only during feeding by the insect that the saliva was injected into the plant and proteolytic enzymes of the saliva degraded the plant proteins to form amino acid. The Cecidogenic arise within the plant cell itself and are specific to the plant, it organs and its cells (Mani, 1964). Sme worker have achieved success in gall induction by insect part or their secretion (Martin, 1942; Anders, 1958; Kant and Patni, 1995). Many workers have reported enzymes in saliva of gall producing insects. It is known that some Cecidogenic insects contain the phytohormone IAA in pertinent tissues e.g. salivary gland of *Coccids* by Allen, 1957.

Biosynthesis of IAA from tryptophane has also been demonstrated in the salivary gland of Cecidogenetic insects. The current information strongly suggests that IAA is the principal chemical inducer of Cecidogenesis. Specific mixture of amino acids, peptides and proteins also occurs in pertinent tissues of

gall forming insects, specific enzymes are also involved (Miles, 1968; Norris, 1979). On the other hand a fluid secreted along with the ovipositor during the egg-laying process is presumed to be responsible for gall induction. The growth of gall was found to be stopped when the larva was removed (MeCalla *et al.,* 1962). The precise mechanism of Cecidogenesis the factors involved in gall development still need satisfactory answer.

The Miles hypothesis explains that salivary components (e.g. amino acids) are similar in both gall-inducing and other (free-living, but plant-feeding) insects. IAA precursors (e.g. tryptophan) in insect saliva are of negligible quantities compared with those found in plant systems. Moreover, the suggestion and salivary proteinases are utilized to release plant bound IAA is disputable, because the saliva of gall inducing insects (e.g. aphids) does not include proteinases. Vigorous uptake of oxygen occurs in the gall tissues; such an uptake coupled with 'wounding' (consequent to feeding action) stimulates plant bound auxin activity. Use of oxygen in plant tissues under insect attack will be so intense that IAA oxidase activity, which regulates accumulation of IAA, is deprived of oxygen. Such an oxygen deprivation enhances the synthesis and accumulation of IAA at insect feeding sites, triggering growth at the vinvoled meristems. Salivary oxidases could be playing a role in the disruption of the IAA oxidase pathway a supposition to be established.

The Principal Gall-Producers

The ability to produce plant galls has developed to a great extent in one section of the mites and among a number of widely different insects, though in certain groups of the letter, it is much more common than in others. The principal gall-producers are found among the plant mites, the plant lice, including the jumping plant lice, the gall midges and the gall wasps, the first and the last two being largely gall-producers. The tiny insects known as trips are found in many galls in the tropical and subtropical regions.

The plant or gall mites are microscopic in size, usually pale yellowish or nearly transparent, the larger, species being visible to the naked eye as little more than minute specks. These mites are easily distinguished from related forms by the slender, more or less pear-shaped body, usually with numerous transverse microscopic ridges or lines, and the presence of but four legs, - most full grown mites have eight legs. The plant mites, as suggested by their name, occur upon plants, some living freely upon the leaf surface, many in dense, felted masses of

plant hairs known as erineums, others in buds, as the filbert bud gall mite, and still others may be found in a variety of pouch or pocket galls, one of the most common bing the maple bladder gall. These last are really bulgings or distentions of the leaf surface, and might be classed as extreme – forms of hairy leaf patches or crineums. Mite galls on leaves may be recognized by the tiny, usually hairy orifices. Many of the gall mites winter in buds or under bud scales. There are a large number of species, many undescribed. They occur upon a great variety of plants, and exhibit no such, preference for certain plant genera or plant families as is found among some plant lice, the gall midges and the gall wasps.

The aphids or plant lice are represented by a number of gall makers. They are all soft bodied insects with well developed, sucking mouth parts. And when full-grown or adult, possess four membranous wings. These insects are small, usually 1/8 of an inch in length or less. There are several genera which commonly produce galls, though this habit is by no means, so general as in the case of the plant mites, the gall midges and the gall wasps. One gall producer, notably the *Adelges* on spruce, is best known because it produces the common cone gall upon Norway spruce. This particular species breeds-year after year on the Norway spruce, though related forms have a more complicated life history, and in the case of the Sitka spruce gall on Colorado blue spruce, the alternate generation is a woolly plant louse which frequently occurs in great numbers on the needles of the Douglas fire.

Injurious Gall Insects

A large proportion of the gall producers are of relatively little importance, though pests of the first magnitude may be found in this miscellaneous assemblage.

The fruit grower, to his sorrow, has learned of the destructive possibilities of the pear leaf blister mite, and in eastern New England and in central New York State, a few already have had experience with the leaf-curling midge of the apple. The pear midge is another pest which is of importance to the fruit grow.

Wheat growers know only too well the destructive possibilities of the Hessian fly, and in the earlier days, they were quite familiar with losses caused by the wheat midge. The clover midge prevents the growing of clover seed in large areas. These insects have exacted heavy tribute from the fruit grower and the farmer.

The gall wasps are relatively unimportant from an economic standpoint, largely because they restrict themselves to such a great extent to oaks which are, generally speaking, not readily injured and ordinarily riot hold in such high esteem as the ornamental of the greenhouse or the garden. By far the greater proportion of the gall insects occurring on oak are of negligible importance. The relatively few species, such as, those producing large, irregular galls upon the smaller branches, may, cause serious injury and kill good sized limbs or, in some cases, most of a tree. Such attacks are ordinarily restricted to an individual tree or a group of trees, and as a rule these conditions develop only after the insects have been able to take advantage of a series of favourable seasons or conditions, as the case may be. The numerous unsightly galls, so abundant on some oaks, are by no means, the product of one season. They remain for years upon a tree and consequently a superficial examination leads to an erroneous conclusion as to the degree of infestation and the amount of injury. This is especially true of the knotty oak galls on willow oak, a common tree in portions of the south.

Galls Value

Some insect galls are commercially valuable, though not to nearly the same extent as m earlier years; certain oak galls have long been used in the manufacture of ink, particularly the more permanent writing fluids. The most important for this purpose is the Eurasian *Cynips gallea tinctorice,* variously known as the Aleppo, gall, turkey gall, levant gall, gall nut, gall of commerce and ink marble. It contains 65% of tannic acid and is mostly used for dying wool and skins. The Knoppern or acorn gall produced by *Cynips quercus-calycis* contains 50% of tannic acid and is next in importance to the Aleppo gall.

It is probable that a number of American oak galls could be utilized to advantage were it not for the high cost of labour in this country. A gall produced by *Cynips theoplirastea* was used by the Greeks as a fuel in lamps. Insect galls are used ii medicine on account of their astringent properties and a few have served as articles of food, notably that of a species of Aylax on *Salvia pomijera a*, which forms an article of commerce in the Near East, and the somewhat common catmint gall, *Aylax glechoma.* Both are said to have an agreeable taste and the sweet odor of the host plant. The insect producing the letter is well established in this country. Other galls are known to be edible and

it is possible that they could be turned to good account when the insects are usually abundant.

Production of Galls

Plant galls and the insects which produce them have aroused lively have, been attempts to prove a reciprocal relation in that the plant as well as he insect benefits. There may be a few cases where this is true, but ordinarily there are no benefits, but rather, injury to the host plant.

The causes which produce plant galls have also been the subject of much speculation and study. It is evident that the gall is not produced except as a portion of a plant may be affected by some agency, in most cases, the gal insect. The effect of the producer upon the plant may be largely mechanical, as in the case of cambium borers which, by girdling a stem or branch, interrupt the flow of sap and the incidental mechanical disturbance results in the local production of many cells-really wound tissue and an enlargement known as a plant gall. There are relatively few deformities of plants produced in this manner.

A number of gall insects occupy cells in apparently normal stems or twigs and produce little or no enlargement of the infested part of the plant. There are a series of gall midges which develop in apparently normal willow twigs, several species of gall wasps which are found under similar conditions in oak twigs and a few which occur in apparently normal herbaceous stems.

A large number of comparatively simple plant galls result from local irrigation. It may be in the egg of certain sawflies; it may be the effect of the young or larvae of other gall makers. It can not be explained as a purely mechanical injury. Most of the galls in this groups are characterized by local swellings and a greater or less deformation of the affected part of the plant, though the distortion is not great enough to conceal the nature of the part affected, and usually the deformation itself varies to a marked extent in size or shape – sometimes both, dependent in large measure upon the degree of infestation.

The general structure is analogous to that of a seed, the gall maker, occupying the place of the seed, the nutritive coasts next to the gall chamber being analogous to those adjacent to or originally connected with the embryo of the seed, and they in turn are surrounded by protective and sometime fantastically ornamental outer layers. These might be termed gall buds. There may be real significance in the lobed splitting of the hickory leaf stem gall with its

suggestion of the more perfectly divided shuck of the hickory nut. There is much of this same type of structure to be seen in a number of hickory leaf galls produced by other species of Phylloxera. The hickory nuts, as well as many other nuts or seeds, ripen and naturally free themselves from the part of the plant on which they develop. The same thing is found in a number of galls, as, for example, the hickory tube gall, produced by a gall midge and ripening and falling in much the same way as a plant seed. This is found also in considerable series of oak galls. The Jumping "Flea seeds" of western oaks are in a way "insect needs", organisms developed as a blind response of plant cells to continuous or repeated stimulation by an insect.

It can not be held that the production of such galls benefits the plants upon which they occur. These adventitious "gall buds" make nutrition levies the vigor of the host plant in the production of a growth following the general developmental lines of a seed with its protective coasts and yet controlled or manipulated into a nearly independent organism for the benefit of an insect.

Biology or Life History of Gall Producers

Plant Mites : The plant mites, really not insects, have a relatively simple life history as illustrated by the habits of the pear bister mite, a tiny pest which winters under the bud scales, attacks the young leaves as they begin to appear, and continues to breed throughout much of the season if tender foliage is available. This general life circle is typical for many of the mites which produce leaf deformations such as pouch or pocket galls, and the long series of pile or velvety deformations known as erineums and due to an excessive development of plant hairs. The filbert bud mite differs in that it attacks the leaf tissues while in the bud and prevents development after the infested buds have attained approximately twice their normal size. In early spring, literally thousands of the minute, yellowish mites may be found in the blasted buds.

Plant Lice : A number of the gall producing plant lice or aphids have complicated life histories, the grape phylloxera, for example, produces galls upon the leaves and there is also a subterranean form on root galls. This insecf is exceedingly injurious to the European or wine grape, although comparatively harmless to our native grapes. The related phylloxeras are well known on account of the numerous leaf galls on hickory early in the season and at least one produces galls on pecan throughout the growing season. One of the most injurious species, well known on account of the large, irregular galls on the leaf stem and young twigs, winters as inconspicuous, dull yellowish eggs upon

rough places in the bark, the eggs hatching and the young phylloxeras entering the partly opened buds and producing galls before the leaves issue from the buds. The gall producing species of *Addles* have a complicated life history, ordinarily migrating from one food plant to another. This is true of the insect producing the Sitka spruce gall, which has an alternate series of generations of woolly aphids on foliage of the Douglas fir. The insect producing the cone gall on Norway spruce occurs only on spruce, there being no alternate host plant. The species of *Pemphigus,* well known because of the conspicuous leaf stem galls on poplar, migrate to summer "hosts, returning to the poplars in the fall. This is also true of several of the gall producing aphids occurring on elm and is seen in two species producing characteristics galls upon witch hazel, these migrating for the summer to birch and in one case; the maker of the spiny witch hazel gall, produces gall like ridges on the leaves of birch, with a most interesting series of diverse appearing generations on this latter host. We have, among the gall producing plant lice, both alternations of series of generations and alternations of host plants.

Gall Midges : The long series of gall midges present great diversities in life histories. Many winter in the gall and issue there from in the spring. A, few, desert the galls in the spring and enter the soil just prior to changing to the adult, and a great many species issue from the galls, especially the softer leaf and fruit tissues, drop to the earth and, remain in some cases for the greater part of the season and even to the following spring before producing adults. The life cycle may vary in length from a few weeks, as in the case of the Hessian fly, to a full year for a large proportion of the species. A considerable series of gall midges do not produce plant deformities. They live in dead or decaying organic matter, and in one group, *Miastor* and its allies, we have a most interesting phenomenon known as pedogenesis, or the production of maggots by maggots through a series of generations, the normal papal and adult stages and the production of eggs being eliminated for a period. These insects are found on various trees under bark in an incipient stage of decay.

The production of living *maggots* ovoviviparousness, by the female of the Indian *Thurauia chiljeansis* has been recorded by M.S. Mani the Indian museum at Calcutta.

Insect and Fungous Galls

A considerable proportion of the peculiar growths found upon plants are caused by insects and yet a number are clue to fungous growths, notably the black knot

upon plum and the cedar apple upon red cedar with its peculiar ceder-rust spot which develop upon apple foliage.

All interesting fact is that a number of galls produced by insects were in carlir years supposed to be caused by fungus. This is particularly true of the numerous blister-like thickenings on the leaves of solidago and aster, known for many years to botanists as *Rhytisma splidagmii* and *Rhytismast* is respectively, and by them supposed to be caused by a fungus which produces very similar spots upon the foliage of other plants. More recently it is developed that the flower-like gall upon cypress, likewise attributed by botanists to a fungus, proves to be the work of a gall midge, though the somewhat bell-shaped, flower-like enlargement is most, suggestive of fugous growth.

Plant Galls in Rajasthan

- **Family – Tamarescaceae**

***Tamarix articulata* Vahl.**

Eriopyes Sp. (Acarina)

Flower gall : Irregularly subglobose, solid fleshy or spongy, complex agglomerate swelling of the whole flowers and parts of even the inflorescence axis, from or grey, often with irregular fleshy tubercles and gummy and surgery exudations on the surface, attracting hordes of ants. The galls is composed of parenchyma cells, with irregular tortuous fleshy galleries and interspaces, into which project a number of fleshy emergences. The gall mites occur in large number in the interspaces. This is a covering gall, characterized by total tissue fusion size 25-30 mm in diameter.

The gall is frequently tunneled by the larva of a unknown Lepidoptera (moth) that cats away the entire flesh and leaves only the leathery skin intact as an empty bag. The moth larva then purpates inside this empty bag and emerges as adult through a performed circular hole leaving the empty cavity filled with fecal pellets by the larva and silken webbing, compare two other criophyid galls on the same plant described from Egypt, Eritrea, Moracco, Algeria and Turkey by houard.

Distribution: Rajasthan and Western U.P.

- **Family – Malvaceae**

***Hibiscus solendra* L. Herit**

Synchytrium sp. (fungus)

Leaf gall : Hypophyllous, deep reddish-brown blister-like, hemispherical, epidermal, solid out growth of tangentially elongate cells. Size 1-2 mm in diameter.

Distribution. Bund Bareta (Bharatpur, Rajasthan).

- **Family – Sterculiaceae**

Melhania futteporensis Munro

Unknown cecidomyiidae (Diptera)

Leaf gall : Semi-lenticular, atrial, discoid galls, visible equally on both sides of the leaf blade, as many as 20 galls on a single leaf and arising mostly close to one of the larger veins or the mid-rib; brownish or reddish-brown, smooth, finely pubescent, semi-persistent indehiscent, bilocular, osfiole hypophyllous, only rarely epiphyllous, on a narrow, short subcyclindrical or conical chimney – shaped fleshy prolongation of covering outgrowth enclosing a moderately large atrium; the atrium is lined largely by dead cells, surrounded outside by a few layers of parenchyma cells, elongated paralled to the axis of the chimney; the gall cavity propex is large, depressed-oval, central and surrounded by a thick zone of closely packed scterenchyma cells, within a peripheral zone of larger parenchyma cells. A part of the palisade tissue sometimes partially differentiated, but with the cells characteristically greatly elongated. The seat of the cell proliferation is in the spony parenchyma. Gall cavity with a single orange-red midge larva. Pupation in the gall, after prolonged larvae augspic which in the instance extended to over two years. The dry leaves, with the mature galls, fall off the plant to the ground at the end of growing season and remain in the forest floor litter till the next mansoon with the first monsoon rain, the larval diapause is broken and is followed by pupation, the adult midge emerges after a week synchronous with unfolding of new leaves on the plant above the ground. Size of gall is 4mm in diameter and 2 mm high.

Distribution: Bund Baretha (Bharatpur, Rajasthan).

- **Family – Anacardiaceae**

Mangifera indica

Unknown midge

Hypophyllous eruptive gall, sparsely found on the leaf; small rized, soft. The gall developing initially inside the leaf tissue, erupts by rupturing the epidermis of the leaf, leaving the remnants of its at the base. Larval cavity, light brown with a watery translucent appearance. The gall collapses soon after its removal from the plant.

Distribution – Jaipur, Madra.

- **Family – Cucurbitaceae**

***Luffa acutangula* Roxb.**

Bimba nenuae Grover (Diptera)

Stem gall : Fusiform, elongate or ovoid or sometimes also irregularly carrot shaped slightly curved, sometimes twisted, solid, smooth but obscurely costate, yellowish – green, fleshy but hard, indehiscent, persistant, turnescence of tender branches, with 4 or 5 irregularly elongate axial and central larval chambers, surrounded by sclerenchyma cells cell. Size 50 mm long and 10-15 mm thick.

Distribution – Rajasthan and western U.P.

20.3 Summary

In short, it can be summarized that a high degree of specialization and diversification is continuing to occur between gall inducing insects and their host plants.

20.4 Glossary

- **Bud galls :** many deformation originate in buds. They vary from a plainly aborted bud to large swelling developed from such parts.
- **Cecidomyia :** This is a general term applied to any species referable to the gall midges or Itonididae and which can not be readily assigned to more closely defined genera.
- **Erineum :** These are the hairy or pile-like growths upon leaf surfaces produced by species of plant mites or *Eriophyidce* .

- **Flower galls :** Aborted or deformed flowers or masses of flowers afford suitable conditions for many gall insects.
- **Leaf gall :** This term applies to all deformations definitely associated with leaves.
- **Galls :** Outgrowths on the surface of life forms.

20.5 Self-Learning Exercise

Section A : (Very Short Answer Type Questions)

1. What is rolling gall?
2. What is pouch gall?
3. What is covering gall?
4. What is IAA?

Section B : (Short Answer Type Questions)

1. What is role of IAA oxidase activity?
2. What is gall?

Section C : (Long Answer Type Questions)

1. Describe the physiology and mechanism of plant gall.

20.6 References

- Mani, M.S., Plant Galls of India (First Edition). Macmillian India, New Delhi, 1974, p. 323.
- Ananthakrishnan, T.N. and Raman, A. Thrips and Gall Dynamics. Oxford and IBH, New Delhi, 1989, p. 120.
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