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34631- MICROBIOLOGY AND PLANT PATHOLOGY

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SYLLABI – BOOK MAPPING TABLE

34631- MICROBIOLOGY AND PLANT PATHOLOGY

	SYLLABI	MAPPING IN BOOK
	BLOCK-1: SCOPE OF MICROBIOLOGY	
UNIT I	Introduction to Microbiology- Scope of Microbiology- Evolution into Science- Characterization of microorganisms.	**1-8**
UNIT II	Classification of Microorganisms- Archea and Bacteria- Characteristics of Bacteria- Morphology -Ultra Structure.	**9-15**
UNIT III	Nutrition – Growth – Reproduction- Bacterial Cultural and Cultural characteristics- Economic importance of bacteria	**16-27**
	BLOCK 2: VIRUS AND MYCOPLASMAS	
UNIT IV	Virology – General features- Classification of virus- Characteristics – Ultra structure.	**28-34**
UNIT V	Virus- Isolation – Purification –Chemical nature- Replication- Transmission- Virions.	**35-46**
UNIT VI	Prions- Phytoplasma (Including mycoplasma).	**47-60**
UNIT VII	Microbial products- Antibiotics- Enzymes- Human Diseases caused by bacteria and viruses.	**61-80**
	BLOCK 3: PLANT PATHOLOGY	
UNIT VIII	Introduction to Plant Pathology- History – Causal agents responsible for Plant diseases.	**81-94**
UNIT IX	Types of Plant diseases – Methods of studying plant diseases – Koch's postulates – Symptoms of plant diseases.	**95-113**
UNIT X	Etiology – Plant diseases epidemics – plant diseases forecasting – Diseases triangle – Diseases cycle.	**114-127**

BLOCK 4: HOST- PATHOGEN INTERACTIONS AND PLANT DISEASES

CONTENTS

BLOCK 1: SCOPE OF MICROBIOLOGY

BLOCK 2: VIRUS AND MYCOPLASMAS

UNIT – IX TYPES OF PLANT DISEASES

BLOCK 4: HOST- PATHOGEN INTERACTIONS AND PLANT DISEASES

BLOCK 1: SCOPE OF MICROBIOLOGY

UNIT I INTRODUCTION TO MICROBIOLOGY

NOTES

1.1 INTRODUCTION

Microbiology is the study of microorganisms, those being unicellular (single cell), multicellular (cell colony), or acellular (lacking cells). Microbiology encompasses numerous sub-disciplines including virology, parasitology, mycology and bacteriology. Eukaryotic microorganisms possess membrane-bound cell organelles and include fungi and protists, whereas prokaryotic organisms—all of which are microorganisms—are conventionally classified as lacking membrane-bound organelles and include Bacteria and Archaea. Microbiologists traditionally relied on culture, staining, and microscopy. However, less than 1% of the microorganisms present in common environments can be cultured in isolation using current means.Microbiologists often rely on molecular biology tools such as DNA sequence based identification, for example 16s rRNA gene sequence used for bacteria identification. Viruses have been variably classified as organisms; they have been considered either as very simple microorganisms or very complex molecules. Prions, never considered as microorganisms, have been investigated by virologists, however, as the clinical effects traced to them were originally presumed due to chronic viral infections, and virologists took search—discovering "infectious proteins". The existence of microorganisms was predicted many centuries before they were first observed, for example by the Jains in India and by Marcus Terentius Varro in ancient Rome. The first recorded microscope observation was of the fruiting bodies of moulds, by Robert Hooke in 1666, but the Jesuit priest Athanasius Kircher was likely the first to see microbes, which he mentioned observing in milk and putrid material in 1658. Antonie van Leeuwenhoek is considered a father of microbiology as he observed and experimented with microscopic organisms in 1676,

NOTES

using simple microscopes of his own design. Scientific microbiology developed in the 19th century through the work of Louis Pasteur and in medical microbiology Robert Koch. Plant pathology (also phytopathology) is the scientific study of diseases in plants caused by pathogens (infectious organisms) and environmental conditions (physiological factors).Organisms that cause infectious disease include fungi, oomycetes, bacteria, viruses, viroids, virus-like organisms, phytoplasmas, protozoa, nematodes and parasitic plants. Not included are ectoparasites like insects, mites, vertebrate, or other pests that affect plant health by eating of plant tissues. Plant pathology also involves the study of pathogen identification, disease etiology, disease cycles, economic impact, plant disease epidemiology, plant disease resistance, how plant diseases affect humans and animals, pathosystem genetics, and management of plant diseases.

1.2 OBJECTIVES

- To impart knowledge of the basic principles of bacteriology, virology, mycology, immunology and parasitology including the nature of pathogenic microorganisms, pathogenesis, laboratory diagnosis, transmission, prevention and control of diseases common in the country.
- Acquire requisite skill in the use and care of basic microbiological equipment; performance of basic laboratory procedures in microbiology and parasitology; proper collection and forwarding of microbiological and parasitological specimens to the laboratory.
- To maintain an interest in the study of medical microbiology, immunology and parasitology by appreciating its role in promotive.
- Preventive and curative medicine with special reference to the microbial and parasitic diseases common in Myanmar.
- To establish essential habits of continuing self-learning through critical reading and evaluation of information in the above mentioned fields.
- To demonstrate development of sound attitudes in relation to the role of medical microbiology in clinical and community medicine.
- To appreciate the professional and personal development in respective field.

1.3 INTRODUCTION TO MICROBIOLOGY

A microbe, or microorganism, is a microscopic organism that comprises either a single cell (unicellular); cell clusters; or multicellular, relatively complex organisms. The study of microorganisms is called microbiology, a subject that began with Anton van Leeuwenhoek's discovery of microorganisms in 1675, using a microscope of his own design. Microorganisms are very diverse; they include bacteria, fungi, algae, and protozoa; microscopic plants (green algae); and animals such as rotifers and planarians. Some microbiologists also include viruses, but others consider these as nonliving. Most microorganisms are unicellular, but this is not universal, since some multicellular organisms are microscopic. Some unicellular protists and bacteria, like Thiomargarita namibiensis, are macroscopic and visible to the naked eye. Microorganisms live in all parts of the biosphere where there is liquid

water, including soil, hot springs, on the ocean floor, high in the atmosphere, and deep inside rocks within the Earth's crust. Most importantly, these organisms are vital to humans and the environment, as they participate in the Earth's element cycles, such as the carbon cycle and the nitrogen cycle. Microorganisms also fulfill other vital roles in virtually all ecosystems, such as recycling other organisms' dead remains and waste products through decomposition. Microbes have an important place in most higher-order multicellular organisms as symbionts, and they are also exploited by people in biotechnology, both in traditional food and beverage preparation, and in modern technologies based on genetic engineering. Pathogenic microbes are harmful, however, since they invade and grow within other organisms, causing diseases that kill humans, animals, and plants. Although many microorganisms are beneficial, many others are the cause of infectious diseases. The organisms involved include pathogenic bacteria, which cause diseases such as plague, tuberculosis, and anthrax. Biofilms —microbial communities that are very difficult to destroy—are considered responsible for diseases like bacterial infections in patients with cystic fibrosis, Legionnaires' disease, and otitis media (middle ear infection) They produce dental plaque; colonize catheters, prostheses, transcutaneous, and orthopedic devices; and infect contact lenses, open wounds, and burned tissue. Biofilms also produce foodborne diseases because they colonize the surfaces of food and food-processing equipment. Biofilms are a large threat because they are resistant to most of the methods used to control microbial growth. Moreover, the excessive use of antibiotics has resulted in a major global problem since resistant forms of bacteria have been selected over time. A very dangerous strain, methicillin-resistant Staphylococcus aureus (MRSA), has wreaked havoc recently. In addition, protozoans are known to cause diseases such as malaria, sleeping sickness, and toxoplasmosis, while fungi can cause diseases such as ringworm, candidiasis, or histoplasmosis. Other diseases such as influenza, yellow fever and AIDS are caused by viruses. Microorganisms are present everywhere on earth which includes humans, animals, plants and other living creatures, soil, water and atmosphere.

1.4 SCOPE OF MICROBIOLOGY

Microbes can multiply in all three habitats except in the atmosphere. Together their numbers far exceed all other living cells on this planet. Microorganisms are relevant to all of us in a multitude of ways. The influence of microorganism in human life is both beneficial as well as detrimental also. For example microorganisms are required for the production of bread, cheese, yogurt, alcohol, wine, beer, antibiotics (e.g. penicillin, streptomycin, chloromycetin), vaccines, vitamins, enzymes and many more important products. Microorganisms are indispensable components of our ecosystem. Microorganisms play an important role in the recycling of organic and inorganic material through their roles in the C, N and S cycles, thus playing an important part in the maintenance of the stability of the biosphere. They are also the source of nutrients at the base of all ectotropical food chains and webs. In many ways all other forms of life depend on the microorganisms. Microorganisms also have harmed humans and disrupted societies over the millennia. Microbial diseases

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undoubtedly played a major role in historical events such as decline of the Roman Empire and conquest of the new world. In addition to health threats from some microorganisms, many microbes spoil food and deteriorate materials like iron pipes, glass lenses, computer chips, jet fuel, paints, concrete, metal, plastic, paper and wood pilings.There is vast scope in the field of microbiology due to the advancement in the field of science and technology.The scope in this field is immense due to the involvement of microbiology in many fields like medicine, pharmacy, diary, industry, clinical research, water industry, agriculture, chemical technology and nanotechnology. The study of microbiology contributes greatly to the understanding of life through enhancements and intervention of microorganisms. There is an increase in demand for microbiologists globally. Genetics: Mainly involves engineered microbes to make hormones, vaccine, antibiotics and many other useful products for human being. Agriculture: The influence of microbes on agriculture; the prevention of the diseases that mainly damage the useful crops.Food science: It involves the prevention of spoilage of food and food borne diseases and the uses of microbes to produce cheese, yoghurt, pickles and beer. Immunology: The study of immune system which protect the body from pathogens.Medicine: deals with the identification of plans and measures to cure diseases of human and animals which are infectious to them.Industry: it involves use of microbes to produce antibiotics, steroids, alcohol, vitamins and amino acids etc. Agricultural microbiology – try to combat plant diseases that attack important food crops, work on methods toincrease soil fertility and crop yields etc. Currently there is a great interest in using bacterial or viral insect pathogens as substitute for chemical pesticides.

Microbial ecology – biogeochemical cycles – bioremediation to reduce pollution effects.Food and dairy microbiology – try to prevent microbial spoilage of food and transmission of food borne diseases such as botulism and salmonellolis. Use microorganisms to make foods such as cheese, yogurt, pickles and beers.Industrial microbiology – used to make products such as antibiotics, vaccines, steroids, alcohols and other solvents, vitamins, amino acids and enzymes.Microbial physiology and Biochemistry – study the synthesis of antibiotics and toxins, microbial energy production, microbial nitrogen fixation, effects of chemical and physical agents on microbial growth and survival etc. Microbial genetics and Molecular biology – nature of genetic information and how it regulated the development and function of cells and organisms. Development of new microbial strains that is more efficient in synthesizing useful products.Genetic engineering – arisen from work of microbial genetics and molecular biology. Engineered microorganisms are used to make hormones, antibiotics, vaccines and other products. New genes can be inserted into plants and animals.

1.5 EVOLUTION INTO SCIENCE

The central argument of Darwin's theory of evolution starts with the existence of hereditary variation. Experience with animal and plant breeding had demonstrated to Darwin that variations can be developed that are "useful to man." So, he reasoned, variations must occur in nature that is

favourable or useful in some way to the organism itself in the struggle for existence. Favourable variations are ones that increase chances for survival and procreation. Those advantageous variations are preserved and multiplied from generation to generation at the expense of less-advantageous ones. This is the process known as natural selection. The outcome of the process is an organism that is well adapted to its environment, and evolution often occurs as a consequence. Natural selection, then, can be defined as the differential reproduction of alternative hereditary variants, determined by the fact that some variants increase the likelihood that the organisms having them will survive and reproduce more successfully than will organisms carrying alternative variants. Selection may occur as a result of differences in survival, in fertility, in rate of development, in mating success, or in any other aspect of the life cycle. All of these differences can be incorporated under the term differential reproduction because all result in natural selection to the extent that they affect the number of progeny an organism leaves.Darwin maintained that competition for limited resources results in the survival of the most-effective competitors. Nevertheless, natural selection may occur not only as a result of competition but also as a result of some aspect of the physical environment, such as inclement weather. Moreover, natural selection would occur even if all the members of a population died at the same age, simply because some of them would have produced more offspring than others. Natural selection is quantified by a measure called Darwinian fitness or relative fitness. Fitness in this sense is the relative probability that a hereditary characteristic will be reproduced; that is, the degree of fitness is a measure of the reproductive efficiency of the characteristic. Biological evolution is the process of change and diversification of living things over time, and it affects all aspects of their lives—morphology (form and structure), physiology, behaviour, and ecology. Underlying these changes are changes in the hereditary materials. Hence, in genetic terms evolution consists of changes in the organism's hereditary makeup. Evolution can be seen as a two-step process. First, hereditary variation takes place; second, selection is made of those genetic variants that will be passed on most effectively to the following generations. Hereditary variation also entails two mechanisms—the spontaneous mutation of one variant into another and the sexual process that recombines those variants to form a multitude of variations. The variants that arise by mutation or recombination are not transmitted equally from one generation to another. Some may appear more frequently because they are favourable to the organism; the frequency of others may be determined by accidents of chance, called genetic drift.

1.6 CHARACTERIZATION OF MICROORGANISMS

Bacteria are prokaryotic unicellular organisms. The bacteria may be spherical, rod-like, spirally coiled or filament like. They lack organized nucleus, but possess a rigid cell wall comparable to that found in plants. The average size of a bacterium is around 2 µm. The bacteria may be spherical, rod-like, spirally coiled or filament like. Certain bacteria may occur in more than one form. Gram-positive and Gram-negative bacteria: Based on the response to Gram's stain, the bacteria are grouped as Gram

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+ve or Gram -ve. By Gram's stain, several distinguishing features of bacteria can be identified. For instance, Gram +ve bacteria possess single-layered cell wall while Gram -ve bacteria have a double-layered one. Aerobic, anaerobic and facultative bacteria: On the basis of respiration (i.e. response to O2), the bacteria are grouped into three categories. These bacteria require O2 for their growth e.g. Pseudomonas sp, Mycobacterium sp. Anaerobic bacteria: These bacteria do not require O2 to obtain energy, and to grow. The presence of O2 is toxic to them e.g. Peptococcus sp. Facultative bacteria, the bacteria that can grow in both aerobic and anaerobic conditions are regarded as facultative bacteria, e.g. Shigella sp, Salmonella sp. Nutritional aspects of bacteria: Based on their nutrition, the bacteria are categorized as autotrophic or heterotrophic. These bacteria are capable of synthesizing their own food from inorganic substances. They are capable of higher plants in this aspect. Autotrophic bacteria utilize different hydrogen compounds (not H2O as in the case of higher plants). These include hydrogen, ammonia, hydrogen sulfide and methane e.g. Hydrogenomonas sp, Nitrosomonas sp, Methanomonas sp. Heterotrophic bacteria: These bacteria cannot synthesize their own food, and are therefore dependent on the outside source. Heterotrophs are of two types—sporophytes and parasites. Sporophytes obtain their food from sources of animal or plant origin. These include organic remains like corpses, animal excreta, meats, fruits and various other products of plant and animal origin. Sporophytes secrete digestive enzymes that break the complex organic molecules into simpler and easily absorbable forms.

These heterotrophic bacteria are useful for the disposal of sewage, cleansing of leather, and manufacture of certain compounds (alcohols, organic acids). Sporophytes can also spoil foods and damage soils (by de-nitrification). Parasites are the bacteria that obtain their food from living organisms, namely the hosts. They may be either harmless (non-pathogenic) or harmful (pathogenic) to the hosts. E. coli is a good example of non-pathogenic bacteria which has a symbiotic relationship in the human intestine. The pathogenic bacteria may cause serious diseases either by destroying the host's cells or releasing toxins e.g. Clostridium tetani.

1.7 CHECK YOUR PROGRESS

1. Who is the father of microbiology?
2. What is Evolution?

1.8 LET US SUM UP

The advent of new technologies and growing recognition of the enormous degree of microbial diversity has revolutionized our understanding of microbiology as a discipline. Microbiology is moving into a new era that focuses less on specific organisms and more on the processes and mechanisms that link them. Nature Reviews Microbiology embraces this new era by encompassing the discipline in its broadest sense. We take an integrated approach to microbiology, bridging fundamental research and its clinical, industrial and environmental applications to create a single information resource for all who share an interest in microbial life. Nature Reviews Microbiology publishes the

highest-quality reviews and perspectives highlighting important developments in our understanding of bacteria, archaea, viruses, fungi and protozoa, their interaction with their environments, how these organisms are harnessed in human endeavour and their impact on society. Also featured are timely summaries of significant research papers, as well as monthly updates on the latest developments in microbial genomics, post-genomic biology and infectious diseases. In line with our ongoing ambition to overcome the traditional barriers between bacteriology, virology, mycology and archaeal and protozoan biology, articles are tailored to appeal to microbiologists of every persuasion and at every level. The scope of the journal encompasses, but will not be limited to, the following fields pertaining to bacteria, archaea, viruses, and fungi.

1.9 UNIT – END EXERCISES

1. What is scope of Microbiology?
2. What is Microbiology and importance?

1.10 ANSWERS TO CHECK YOUR PROGRESS

1. Currently, we are in the era of Microbiology. Microorganisms are recognized as the basic research tools as they help to understand the chemical and physical basis of life as they are the dominant group of living organisms in the biosphere and are actively involved in our day to day activities. Microbiology primarily paves the way to analyze the biochemical and genetic background of living things. Moreover as microbes are the excellent models for understanding the cell functions and as they play an important role in the field of medicine, agriculture and industry that assures human welfare, microbiology is considered as one of the vital branch of science with entire promising scopes. Microbiology is not just one small subject to be explored. It has nearly six major branches. They are as follows; Agricultural Microbiology deals with soil nutrient cycling by microbes, microbial decomposition of organic wastes, plant associated microbes that enhance soil fertility, etc. Food Microbiology covers information about the microbes involved in food spoilage, food borne diseases, commercial food products prepared using microbes, etc. Industrial Microbiology explores the utility of microbes in the production of antibiotics, enzymes, alcoholic beverages, fermented food products, etc. Medical Microbiology deals with the studies related to the microbes that cause diseases, their diagnostic and preventive measures, drug designing, etc. Aquatic Microbiology deals with water purification and biological degradation of wastes in aquatic ecosystems by microbes. Aero Microbiology talks about the microorganisms prevalent in air, their abundance and beneficial or harmful issues. Exomicrobiology is all about the exploration of life in outer space. Geochemical Microbiology analyses the microbial life and their contribution to coal, oil and gas formation areas. As each branch of microbiology have got their specialization that contributes to the development of science and technology, always microbiology is crowned as innovate, an evergreen branch of biology that has wider scopes for the emerging scientists to be explored. We are living in the world of microbes without which life will not be trouble-free and comfy.

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2. Microbiology is the study of organisms and agents that are generally too small to be seen clearly by the unaided eye. These organisms include viruses, bacteria, algae, fungi, and protozoa. Understanding of how a cell works have come through the study of microorganisms. However, microbiology also is an applied science, helping agriculture, health and medicine and maintenance of the environment, as well as the biotechnology industry. Microorganisms are vital in our everyday lives.

1.11 SUGGESTED READINGS

1. Microbiology Paperback – 20 Apr 2001 by Pelczar, Jr., Michael (Author)
2. A Textbook of Microbiology Paperback – Jan 2013 by D K Maheshwari (Author), R C Dubey (Author)

UNIT – II CLASSIFICATION OF MICROORGANISMS

2.1 INTRODUCTION

Bacterial taxonomy is the rank-based classification of bacteria. In the scientific classification established by Carl von Linné, each distinct species is assigned to a genus using a two-part binary name (for example, Homo sapiens). This distinct species is then, in turn, placed within a lower level of a hierarchy of ranks. These ranks range in ascending scale from family to suborder, and upward to order, subclass, class, division/phyla, kingdom and domain. In the currently accepted scientific classification of Life, there are three domains of microorganisms: the Eukaryotes, Bacteria and Archaea, The different disciplines of study refer to them using different terms to speak of aspects of these domains, however, though they follow similar principles. Thus botany, zoology, mycology, and microbiology use several different conventions when discussing these domains and their subdivisions. In zoology, for example, there are type specimens, whereas in microbiology there are type strains.

2.2 OBJECTIVES

- Outline the factors that play a role in the classification of bacterial taxonomy
- Describe bacterial structure: colony morphology, cell shape, growth patterns.
- To distinguish how various growth media will affect colony growth.
- To be able to determine bacterial species based upon macroscopic examination.
- To be able to differentiate between the three general morphological types of bacteria.
- To be able to differentiate between Gram positive and negative bacteria.

2.3 CLASSIFICATION OF MICROORGANISMS

Historical Challenges of Classification

Despite there being little agreement on the major subgroups of the Bacteria, gram staining results were commonly used as a classification tool. As an example, Prokaryotes share many common features, such as

NOTES

lack of nuclear membrane, unicellular, division by binary-fission and generally small size. Until the advent of molecular phylogeny the Kingdom Prokaryotae was divided into four divisions, a classification scheme still formally followed by Bergey's manual of systematic bacteriology.The various species differ amongst each other based on several characteristics determined by gram staining, which allowed their identification and classification. Major groups of this system include Gracilicutes (gram negative); Firmacutes (gram positive); Mollicutes (gram variable, e.g. Mycoplasma); and Mendocutes (uneven gram stain, "metlynogenic bacteria" now known as the Archaea).

Molecular Classification

In the Molecular era of classification, Carl Woese, who is regarded as the forerunner of the molecular phylogeny revolution, argued that the bacteria, archaea, and eukaryotes represent separate lines of descent that diverged early on from an ancestral colony of organisms. However, a few biologists argue that the Archaea and Eukaryota arose from a group of bacteria. In any case, it is thought that viruses and archaea began relationships approximately two billion years ago, and that co-evolution may have been occurring between members of these groups. It is possible that the last common ancestor of the bacteria and archaea was a thermophile, which raises the possibility that lower temperatures are "extreme environments" in archaeal terms, and organisms that live in cooler environments appeared only later. Since the Archaea and Bacteria are no more related to each other than they are to eukaryotes, the term prokaryote's only surviving meaning is "not a eukaryote", limiting its value. With improved methodologies it became clear that the methanogenic bacteria were profoundly different and were erroneously believed to be relics of ancient bacteria. Thus, though Woese identified three primary lines of descent the Archaebacteria, the Eubacteria and the Urkaryotes, the latter now represented by the nucleocytoplasmic component of the Eukaryotes. These lineages were formalized into the rank Domain (regio in Latin) which divided Life into 3 domains: the Eukaryota, the Archaea and the Bacteria. This scheme is still followed today.In 1987 Carl Woese divided the Eubacteria into 11 divisions based on 16S ribosomal RNA (SSU) sequences, which with several additions are still used today.

2.4 ARCHAEA – AND BACTERIA

Archaea and Bacteria are two kinds of microorganisms that fall under the category of Prokaryotes. However, all archaea and bacteria are not prokaryotes. Earlier archaea were classified as bacteria, but now it's outdated as it's been found out that they both have different biochemistry and different evolutionary history. Archaea and Bacteria do not possess membrane found organelles or nucleus. They have similar size and shape. Archaea: They are single-celled organisms that comprise of cells with distinct properties that makes them unique from the other two domains of life namely Eukaryota and Bacteria. They use numerous source of energy and display a diverse array of chemical reactions in metabolism. Based on reactions they are categorized into nutritional groups. That is either dependent on carbon sources and energy. One group of archaea uses

sunlight as a source of energy. They are termed as phototrophs. But any of these organisms do not possess oxygen-generating photosynthesis.The other of archae uses inorganic compounds as a source of energy, namely ammonia or sulphur. They either include anaerobic methane oxidizers, nitrifiers, and methanogens. This reaction involves two compounds where one compound acts as an electron acceptor and the other as an electron donor. The energy that is released during the reaction releases ATP – adenosine triphosphate through chemios. It is one of the same basic processes that can be found in some of the eukaryotic cells. Bacteria: They are a single-celled organism that usually lives in a diverse environment. Bacterial DNA called the nucleoid is a twisted thread-like mass that flows free. They even possess a cellular structure that executes a range of circular functions that involves transfer of energy to the transportation of proteins. Bacteria consist of plasmids, which are a circular piece of DNA. Bacterial cells consist of the inner cell membrane and an outer cell wall. Wherein some of the bacteria do not possess cell walls such as mycoplasmas. In some cases, bacteria may consist of a third protective outer layer in a cell called capsule. Pili cover surfaces.

Table 2.1: Difference between Archaea and Bacteria

Basis	Archaea	Bacteria
Reproduction and growth	Asexual reproduction by the process of fragmentation, budding and binary fission	Asexual Reproduction. Eubacteria produces spore to stay latent for several years.
Cell Membrane	Pseudopeptidoglycan	Lipopolysaccharide or Peptidoglycan
Metabolism Activity	Methanogenesis	Autotrophy, Aerobic and Anaerobic Respiration, Fermentation and Photosynthesis.
RNA	Consists of three RNA	Consists of single RNA
Thriving Habitat	Can sustain in extremely harsh environment such as oceans, hot springs, marshlands, hot springs and gut of humans	They are generally found in soil, organic matter, earth's crust, water, bodies of animals and plants, radioactive wastes, hot springs etc.

2.5 CHARACTERISTICS OF BACTERIA

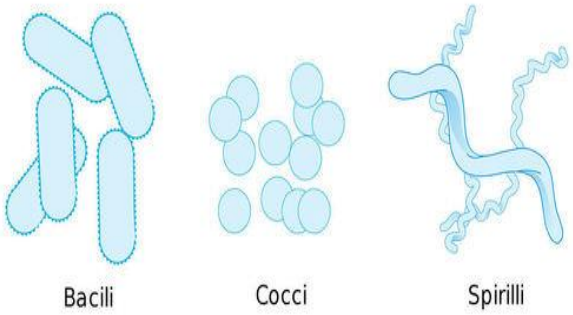
Morphology

Bacteria are the most successful organisms on the planet. They lived on this planet for two billion years before the first eukaryotes and, during that time, evolved into millions of different species. Size and Shape: Bacteria are so small that they can only be seen with a microscope. When viewed under the microscope, they have three distinct shapes. Bacteria can be identified and classified by their shape:

Bacilli are rod-shaped.

NOTES

Cocci are sphere-shaped.
Spirilli are spiral-shaped.
Like eukaryotic cells, bacterial cells have:
Cytoplasm, the fluid inside the cell.
A plasma or cell membrane, which acts as a barrier around the cell.
Ribosomes, in which proteins are put together.
DNA. By contrast though, bacterial DNA is contained in a large, circular strand. This single chromosome is located in a region of the cell called the nucleoid. The nucleoid is not an organelle, but a region within the cytoplasm. Many bacteria also have additional small rings of DNA known as plasmids.



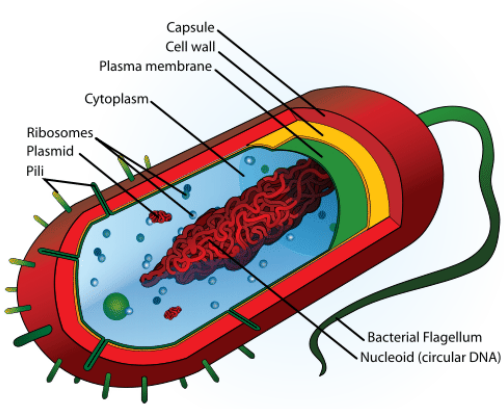


Figure 2.1: Diagrammatic representation of Bacteria

2.6 ULTRASTRUCTURE OF BACTERIA

Bacteria lack many of the structures that eukaryotic cells contain. For example, they don't have a nucleus. They also lack membrane-bound organelles, such as mitochondria or chloroplasts. The DNA of a bacterial cell is also different from a eukaryotic cell. Bacterial DNA is contained in one circular chromosome, located in the cytoplasm. Eukaryotes have several linear chromosomes. Bacteria also have two additional unique features: a cell wall and flagella. Some bacteria also have a capsule outside the cell wall. Bacteria are surrounded by a cell wall consisting of peptidoglycan. This complex molecule consists of sugars and amino acids. The cell wall is important for protecting bacteria. The cell wall is so important that some antibiotics, such as penicillin, kill bacteria by

preventing the cell wall from forming.Some bacteria depend on a host organism for energy and nutrients. These bacteria are known as parasites. If the host starts attacking the parasitic bacteria, the bacteria release a layer of slime that surrounds the cell wall. This slime offers an extra layer of protection. Flagella help bacteria move (Fig. 2.1). As the flagella rotate, they spin the bacteria and propel them forward. It is often said the flagella looks like a tiny whip, propelling the bacteria forward. Though some eukaryotic cells do have flagella, flagella in eukaryotes are rare (Fig. 2.2).

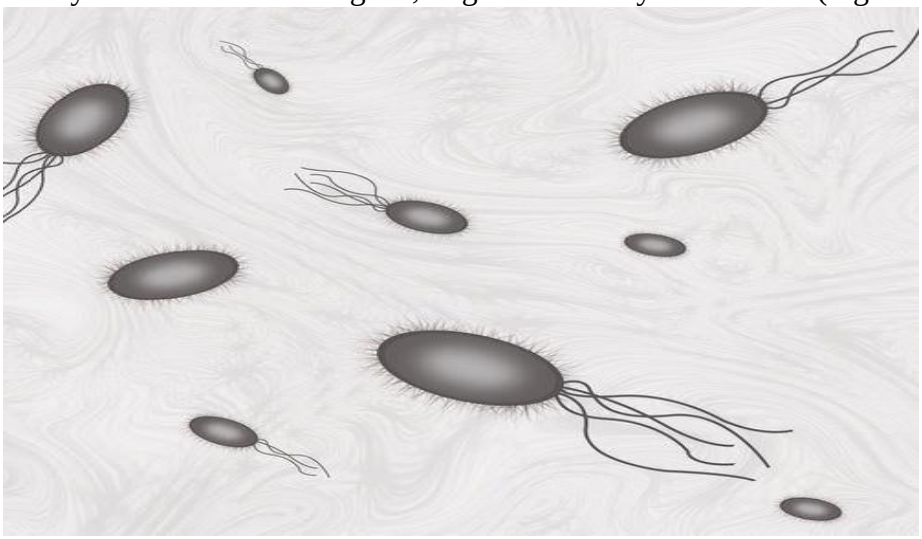


Figure 2.2: The flagella facilitate movement in bacteria. Bacteria may have one, two, or many flagella or none at all.

2.7 CHECK YOUR PROGRESS

1. What is Archaea?
2. What are the different types of bacteria?

2.8 LET US SUM UP

Bacteria are unicellular organisms that have a variety of sizes, shape, and envelope structures. The minimal requirements are cytoplasm, a cell membrane that surrounds the cytoplasm, and a DNA chromosome. A few have internal structures such as vacuoles and storage bodies but none have true organelles. The cell envelope can be as simple as a single membrane. However, it is generally a multilayered structure that includes a cytoplasmic membrane, a cell wall, and additional structures exterior to the cell wall. The nature of the bacterial envelope determines whether the strain is a Gram positive, Gram negative, or acid fast organism. Gram positive bacteria have thick cell walls, Gram negative bacteria thin cell walls plus a second exterior membrane, and acid fast bacteria have a thin cell wall plus a thick layer of specialized lipids. Cell walls function as exoskeletons that define the overall cell shape. Outer membranes protect Gram negative cells from detergents and enzymes but limit permeability. They contain specialized channel-forming proteins that provide the means for small molecules to diffuse across this barrier. The outer leaflet of the outer membrane contains lipopolysaccharides. Lipopolysaccharides are unique glycolipids that form a barrier that protects the cell from hydrophobic agents. They are endotoxins, highly immunogenetic, and the

NOTES

surface components recognized by serotyping antibodies. Bacteria can have a variety of surface appendages. These include flagella for cell movement, fimbriae for adherence, and pili for genetic exchanges. Many bacteria surround themselves with a thick polysaccharide coat (the glycocalyx) in the form of a capsule or as slime. This layer helps protect the cells from dehydration and contributes to the pathogenicity of many pathogens.

2.9 UNIT – END EXERCISES

1. What is function and structure of the bacterial cells?
2. Describe between Archeae and bacteria?

2.10 ANSWERS TO CHECK YOUR PROGRESS

1. Prokaryotic cells are not as complex as eukaryotic cells. They have no true nucleus as the DNA is not contained within a membrane or separated from the rest of the cell, but are coiled up in a region of the cytoplasm called the nucleoid. Prokaryotic organisms have varying cell shapes. The most common bacteria shapes are spherical, rod-shaped, and spiral. Using bacteria as our sample prokaryote, the following structures and organelles can be found in bacterial cells: Capsule - Found in some bacterial cells, this additional outer covering protects the cell when it is engulfed by other organisms, assists in retaining moisture, and helps the cell adhere to surfaces and nutrients. Cell Wall - The cell wall is an outer covering that protects the bacterial cell and gives it shape. Cytoplasm - Cytoplasm is a gel-like substance composed mainly of water that also contains enzymes, salts, cell components, and various organic molecules. Cell Membrane or Plasma Membrane - The cell membrane surrounds the cell's cytoplasm and regulates the flow of substances in and out of the cell. Pili (Pilus singular) Hair-like structures on the surface of the cell that attach to other bacterial cells. Shorter pili called fimbriae help bacteria attach to surfaces. Flagella - Flagella are long, whip-like protrusion that aids in cellular locomotion. Ribosomes - Ribosomes are cell structures responsible for protein production. Plasmids - Plasmids are gene carrying, circular DNA structures that are not involved in reproduction. Nucleiod Region - Area of the cytoplasm that contains the single bacterial DNA molecule. Prokaryotic cells lack organelles found in eukaryoitic cells such as mitochondria, endoplasmic reticuli, and Golgi complexes. According to the Endosymbiotic Theory, eukaryotic organelles are thought to have evolved from prokaryotic cells living in endosymbiotic relationships with one another. Like plant cells, bacteria have a cell wall. Some bacteria also have a polysaccharide capsule layer surrounding the cell wall. It is in this layer where bacteria produce biofilm, a slimy substance that helps bacterial colonies adhere to surfaces and to each other for protection against antibiotics, chemicals, and other hazardous substances. Similar to plants and algae, some prokaryotes also have photosynthetic pigments. These

light-absorbing pigments enable photosynthetic bacteria to obtain nutrition from light.

2. Describe between Archeae and bacteria?

Basis	Archaea	Bacteria
Reproduction and growth	Asexual reproduction by the process of fragmentation, budding and binary fission	Asexual Reproduction. Eubacteria produces spore to stay latent for several years.
Cell Membrane	Pseudopeptidoglycan	Lipopolysaccharide or Peptidoglycan
Metabolism Activity	Methanogenesis	Autotrophy, Aerobic and Anaerobic Respiration, Fermentation and Photosynthesis.
RNA	Consists of three RNA	Consists of single RNA
Thriving Habitat	Can sustain in extremely harsh environment such as oceans, hot springs, marshlands, hot springs and gut of humans	They are generally found in soil, organic matter, earth's crust, water, bodies of animals and plants, radioactive wastes, hot springs etc.

2.11 SUGGESTED READINGS

1. Ananthanarayan and Paniker's Textbook of Microbiology Tenth edition with booklet Paperback – Jun 2017

2. Textbook of Microbiology Paperback – 2019 by C.P. Baveja (Author)

NOTES

UNIT – III NUTRITION

3.1 INTRODUCTION

Bacterial requirements for growth include sources of energy, "organic" carbon (e.g. sugars and fatty acids) and metal ions (e.g. iron). Optimal temperature, pH and the need (or lacks of need for oxygen) are important. Bacteria can be classified into the following type according to the basis of their ability to synthesize essential metabolism. Nutrition is the science that interprets the interaction of nutrients and other substances in food in relation to maintenance, growth, reproduction, health and disease of an organism. It includes food intake, adsorption, assimilation, biosynthesis, catabolism and excretion. The diet of an organism is what it eats, which is largely determined by the availability and palatability of foods. For humans, a healthy diet includes preparation of food and storage methods that preserve nutrients from oxidation, heat or leaching, and that reduces risk of food borne illnesses. In humans, an unhealthy diet can cause deficiency-related diseases such as blindness, anemia, scurvy, preterm birth, stillbirth and cretinism, or nutrient excess health-threatening conditions such as obesity and metabolic syndrome and such common chronic systemic diseases as cardiovascular disease, diabetes, and osteoporosis. Undernutrition can lead to wasting in acute cases, and the stunting of marasmus in chronic cases of malnutrition.

3.2 OBJECTIVES

- Define essential mineral elements
- Compare micro and macronutrients – their role deficiency symptoms in plants
- Discuss the mechanisms of absorption of elements
- Describe nitrogen metabolisms in relation to plants
- Define various mode of plant nutrition

3.3 NUTRITION – GROWTH

Every organism must find in its environment all of the substances required for energy generation and cellular biosynthesis. The chemicals and elements of this environment that are utilized for bacterial growth are referred to as nutrients or nutritional requirements. Many bacteria can be grown the laboratory in culture media which are designed to provide all the

essential nutrients in solution for bacterial growth. Bacteria that are symbionts or obligate intracellular parasites of other cells, usually eukaryotic cells, are (not unexpectedly) difficult to grow outside of their natural host cells. Whether the microbe is a mutualist or parasite, the host cell must ultimately provide the nutritional requirements of its residents. Many bacteria can be identified in the environment by inspection or using genetic techniques, but attempts to isolate and grow them in artificial culture have been unsuccessful. This, in part, is the basis of the estimate that we may know less than one percent of all prokaryotes that exist. At an elementary level, the nutritional requirements of a bacterium such as E. coli are revealed by the cell's elemental composition, which consists of C, H, O, N, S. P, K, Mg, Fe, Ca, Mn, and traces of Zn, Co, Cu, and Mo. These elements are found in the form of water, inorganic ions, small molecules, and macromolecules which serve either a structural or functional role in the cells.

Major elements, their sources and functions in bacterial cells

Element	% of dry weight	Source	Function
Carbon	50	organic compounds or CO2	Main constituent of cellular material
Oxygen	20	H2O, organic compounds, CO2, and O2	The constituent of cell material and cell water; O2 is electron acceptor in aerobic respiration
Nitrogen	14	NH3, NO3, organic compounds, N2	Constituent of amino acids, nucleic acids nucleotides, and coenzymes
Hydrogen	8	H2O, organic compounds, H2	The main constituent of organic compounds and cell water
Phosphorus	3	inorganic phosphates (PO4)	The constituent of nucleic acids, nucleotides, phospholipids, LPS, teichoic acids
Sulfur	1	SO4, H2S, So, organic sulfur compounds	The constituent of cysteine, methionine, glutathione, several coenzymes
Potassium	1	Potassium salts	Main cellular inorganic cation and cofactor for certain enzymes
Magnesium	0.5	Magnesium salts	Inorganic cellular cation, cofactor for certain enzymatic reactions
Calcium	0.5	Calcium salts	Inorganic cellular cation, cofactor for certain enzymes

NOTES

			and a component of endospores
Iron	0.2	Iron salts	Component of cytochromes and certain nonheme iron-proteins and a cofactor for some enzymatic reactions

Trace elements are metal ions required by certain cells in such small amounts that it is difficult to detect (measure) them, and it is not necessary to add them to culture media as nutrients. Trace elements are required in such small amounts that they are present as "contaminants" of the water or other media components. As metal ions, the trace elements usually act as cofactors for essential enzymatic reactions in the cell. One organism's trace element may be another's required element and vice-versa, but the usual cations that qualify as trace elements in bacterial nutrition are Mn, Co, Zn, Cu, and Mo.Carbon and Energy Sources for Bacterial Growth.In order to grow in nature or in the laboratory, a bacterium must have an energy source, a source of carbon and other required nutrients, and a permissive range of physical conditions such as O2 concentration, temperature, and pH. Sometimes bacteria are referred to as individuals or groups based on their patterns of growth under various chemical (nutritional) or physical conditions. For example, phototrophs are organisms that use light as an energy source; anaerobes are organisms that grow without oxygen; thermophiles are organisms that grow at high temperatures.All living organisms require a source of energy. Organisms that use radiant energy (light) are called phototrophs. Organisms that use (oxidize) an organic form of carbon are called heterotrophs or (chemo) heterotrophs. Organisms that oxidize inorganic compounds are called lithotrophs.The carbon requirements of organisms must be met by organic carbon (a chemical compound with a carbon-hydrogen bond) or by CO2. Organisms that use organic carbon are heterotrophs and organisms that use CO2 as a sole source of carbon for growth are called autotrophs.
Major nutritional types of prokaryotes

Nutritional Type	Energy Source	Carbon Source	Examples
Photoautotrophs	Light	CO2	Cyanobacteria, some Purple and Green Bacteria
Photoheterotrophs	Light	Organic compounds	Some Purple and Green Bacteria
Chemoautotrophs or Lithotrophs (Lithoautotrophs)	Inorganic compounds, e.g. H2, NH3, NO2, H2S	CO2	A few Bacteria and many Archaea
Chemoheterotrophs or Heterotrophs	Organic compounds	Organic compounds	Most Bacteria, some Archaea

Almost all eucaryotes are either photoautotrophic (e.g. plants and algae) or heterotrophic (e.g. animals, protozoa, fungi). Lithotrophy is unique to procaryotes and photoheterotrophy, common in the Purple and Green Bacteria, occurs only in a very few eucaryotic algae. Phototrophy has not been found in the Archaea, except for non-photosynthetic light-driven ATP synthesis in the extreme halophiles.

3.4 REPRODUCTION

In binary fission, single cell divides into two equal cells (Fig. 3.1). Initially the bacterial cell reaches a critical mass in its tructure and cellular constituents.

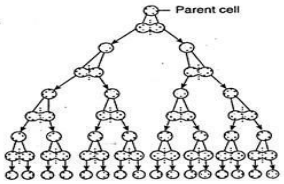


Figure 3.1: Binary fission results in rapid multiplication of bacterial cell

The new double stranded DNA molecule i.e., incipient nuclei, are then distributed into two poles of the dividing cell (no spindle formation takes place like mitotic division). A transverse septum develops in the middle region of the cell, which separates the two daughter cells (Fig.3.2).

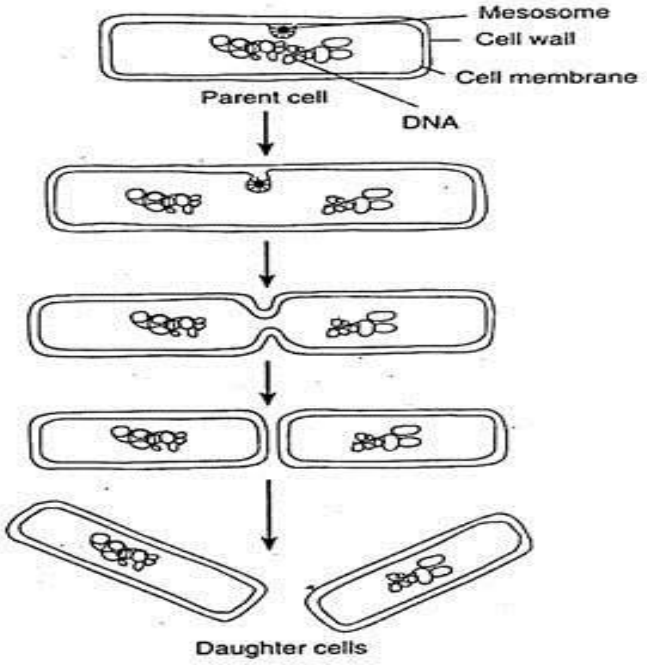


Figure 3.2: Binary fission of a bacterium

The binary fission is a rapid process and cell undergoes division at an interval of 20-30 minutes. The division becomes gradually slow after certain time due to accumulation of toxic substance and exhaustion of nutrients. Conidia formation takes place in filamentous bacteria like Streptomyces etc., by the formation of a transverse septum at the apex of the filament (Fig. 3.3A). The part of this filament which bears conidia is called conidiophore. After detachment from the mother and getting contact

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with suitable substratum, the conidium germinates and gives rise to new mycelium.

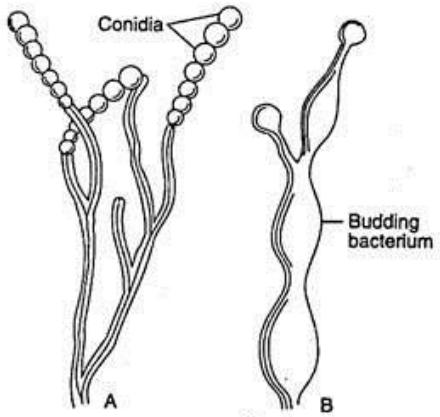

Fig. 2.21 : Asexual reproduction of bacteria : A. Conidia formation, B. Budding

Figure 3.3: Asexual reproduction of bacteria: A. Conidia formation, B. Budding

The bacterial cell develops small swelling at one side which gradually increases in size (Fig. 3.3B). Simultaneously the nucleus undergoes division, where one remains with the mother and other one with some cytoplasm goes to the swelling. This outgrowth is the bud, which gets separated from the mother by partition wall, e.g., Hyphomicrobium vulgare, Rhodomicrobium vannielia, etc. Cysts are formed by the deposition of additional layer around the mother wall. These are the resting structure and during favourable condition they again behave as the mother, e.g., many members of Azotobacter. Spores are formed during unfavourable environmental condition like desiccation and starvation. As the spores are formed within the cell, they are called endospores. Only one spore is formed in a bacterial cell. On germination, it gives rise to a bacterial cell.

Some endospore forming bacteria:

1. Gram-positive,

a) Bacilli,

(i) Obligate aerobes, e.g., Bacillus subtilis, B. anthracis.

(ii) Obligate anaerobes, e.g., Clostridium tetani, C. botulinum. (b) Cocci, e.g., Sporosarcina.

2. Gram-negative

(a) Bacillus, e.g., Coxiellaburnetii,

(b) Cocci, e.g., Escherichia coli.

3.5 BACTERIAL CULTURE AND CHARACTERISTICS OF BACTERIA

A microbiological culture, or microbial culture, is a method of multiplying microbial organisms by letting them reproduce in predetermined culture medium under controlled laboratory conditions. Microbial cultures are foundational and basic diagnostic methods used as a research tool in molecular biology. Microbial cultures are used to determine the type of organism, its abundance in the sample being tested,

or both. It is one of the primary diagnostic methods of microbiology and used as a tool to determine the cause of infectious disease by letting the agent multiply in a predetermined medium. For example, a throat culture is taken by scraping the lining of tissue in the back of the throat and blotting the sample into a medium to be able to screen for harmful microorganisms, such as Streptococcus pyogenes, the causative agent of strep throat. Furthermore, the term culture is more generally used informally to refer to "selectively growing" a specific kind of microorganism in the lab. There are several types of bacterial culture methods that are selected based on the agent being cultured and the downstream use. One method of bacterial culture is liquid culture, in which the desired bacteria are suspended in a liquid nutrient medium, such as Luria Broth, in an upright flask. This allows a scientist to grow up large amounts of bacteria for a variety of downstream applications. Liquid cultures are ideal for preparation of an antimicrobial assay in which the experimenter inoculates liquid broth with bacteria and lets it grow overnight (they may use a shaker for uniform growth).

Then they would take aliquots of the sample to test for the antimicrobial activity of a specific drug or protein (antimicrobial peptides). Microbiological cultures can be grown in petri dishes of differing sizes that have a thin layer of agar-based growth medium. Once the growth medium in the petri dish is inoculated with the desired bacteria, the plates are incubated at the optimal temperature for the growth of the selected bacteria (for example, usually at 37 degrees Celsius, or the human body temperature, for cultures from humans or animals, or lower for environmental cultures). After the desired level of growth is achieved, agar plates can be stored upside down in a refrigerator for an extended period of time to keep bacteria for future experiments. There are a variety of additives that can be added to agar before it is poured into a plate and allowed to solidify. Some types of bacteria can only grow in the presence of certain additives. This can also be used when creating engineered strains of bacteria that contain an antibiotic-resistance gene. When the selected antibiotic is added to the agar, only bacterial cells containing the gene insert conferring resistance will be able to grow. This allows the researcher to select only the colonies that were successfully transformed. Stab cultures are similar to agar plates, but are formed by solid agar in a test tube. Bacteria is introduced via an inoculation needle or a pipette tip being stabbed into the center of the agar. Bacteria grow in the punctured area. Stab cultures are most commonly used for short-term storage or shipment of cultures. Virus or phage cultures require host cells in which the virus or phage multiply. For bacteriophages, cultures are grown by infecting bacterial cells. The phage can then be isolated from the resulting plaques in a lawn of bacteria on a plate. Virus cultures are obtained from their appropriate eukaryotic host cells. For single-celled eukaryotes, such as yeast, the isolation of pure cultures uses the same techniques as for bacterial cultures. Pure cultures of multicellular organisms are often more easily isolated by simply picking out a single individual to initiate a culture. This is a useful technique for pure culture of fungi, multicellular algae, and small metazoa, for example. Developing pure culture techniques is crucial to the observation of the specimen in question.

The most common method to isolate individual cells and produce a pure culture is to prepare a streak plate. The streak plate method is a way to physically separate the microbial population, and is done by spreading the inoculate back and forth with an inoculating loop over the solid agar plate. Upon incubation, colonies will arise and single cells will have been isolated from the biomass.

3.6. ECONOMIC IMPORTANCE OF BACTERIA

Some bacteria play an important role in maintaining and others in increasing soil fertility. The fertility of soil is proportional to its nitrogen content. Nitrogen is an essential ingredient of all living protoplasm. All growing plants, therefore, require it in their metabolism. Atmosphere, no doubt, is four-fifths (80%) nitrogen, green plants generally are unable to use it. They mostly absorb it as nitrates and to some extent as ammonia from the soil.Continuous absorption of these salts results in their exhaustion in the soil. Nearly all fertilisers for the soil include a large proportion of such soluble nitrogen compounds to promote plant growth.In nature the presence of a regular supply of these salts is ensured by bacteria of certain types. These bacteria which function as Nature's farmers belong to three categories, namely, ammonifying bacteria, nitrifying bacteria and nitrogen-fixing bacteria. They are the agents of maintaining a continual circulation of nitrogen in nature between the plant world, in soil and the atmosphere. The series of changes through which the nitrogen passes due to the activities of these organisms constitute the nitrogen cycle. The saprophytic bacteria break down the proteins and other nitrogen containing remains of the plant and animal origin in the soil to amino acids by secreting enzymes.The amino acids are then converted into ammonia by a group of bacteria called the ammonifying bacteria. The liberated ammonia may combine with carbon dioxide and water in the soil to form ammonium carbonate. A few plants such as the common cereals can make use of ammonium compounds as a source of nitrogen. The majority of plants, however, cannot absorb ammonium compounds as a source of nitrogen.Ammonia is very soluble. It moves in the soil rapidly and is acted upon by microorganisms of the category of chemosynthetic autotrophs in the soil. They are the nitrifying bacteria such as Nitrosomonas and Nitmbacter. They form nitrates from ammonium compounds. Nitrosomonas oxidizes ammonium carbonate to nitrous acid liberating energy. The nitrous acid then combines with bases in the soil forming potassium nitrite. Nitrobacter oxidizes nitrites to nitrates gain liberating energy.Neither the ammonifying nor the nitrifying bacteria add to the total quantity of combined nitrogen in the soil. The ammonifying bacteria convert amino acids into ammonia. The process is called ammonification. The nitrifying bacteria convert nitrogen from the unavailable form of ammonium salts to the available nitrates. This process converting unavailable ammonium salts into available nitrates is called nitrification. A considerable amount of nitrogen is lost by denitrification and through drainage. The loss must be made good by equal gains if the soil fertility is to be maintained.The electric discharges in the atmosphere bring about the formation of traces of nitrogen compounds that are washed to the soil by rainwater. The largest additions, however, come from a biological fixation

process through the activity of two types of nitrogen-fixing bacteria.Some of them live free in the soil and others in the root nodules of leguminous plants. They are able to make use of the atmospheric nitrogen and change it into nitrogenous compounds.The nitrogen-fixing bacteria are thus unique in tapping a source of nitrogen not available to most other plants. This process of nitrogen transformation is called nitrogen fixation. They take gaseous nitrogen from the air present between the soil particles. The nitrogen combines with other elements forming organic nitrogenous compounds. These compounds are assimilated by the bacteria. In due course these bacteria die and their dead bodies containing nitrogenous compounds are decomposed by another type of bacteria called the bacteria of decomposition. During decomposition ammonia is produced. The nitrifying bacteria convert this ammonia first into nitrites and finally into nitrates. Nitrates constitute the form of nitrogen needed by the green plants. It lives in the roots of such plants as Pea Bean, Medicago and others. All these belong to the Pea family (Leguminoseae). Besides the legumes, the nodules are found on the roots of Alnus glutinosa, Casuarina, species of Coriaria and a few others. The symbiont in non- leguminous plants is a member of Plasmodiophorales. The presence of bacteria in the roots causes the formation of little nodules. These nodules or the tubercles are the homes of millions of these bacteria. They have the ability to take up free nitrogen of the air and convert it into nitrogen compounds. A part of the fixed nitrogen passes into the tissues of legume plant and a part diffuses into the surrounding soil. These bacteria enter into a mutually beneficial partnership with the host plant. They give the host the nitrogen compounds and receive in return carbohydrates manufactured by the host plant. This association is an excellent example of symbiosis. The legumes are very rich in nitrogen because of this association.Some of our best protein plant foods come from the legume family of plants. The legume plants can flourish on land that has been depleted of its nitrogen by other plants. They are sometimes grown and ploughed under by the farmer when a foot or so in height. The decaying bodies of these legume plants enrich the soil. They furnish a rich supply of nitrogen to the future crop. This is called green manuring. The soil on which repeated crops of cereals are grown becomes impoverished.It can be enriched again by growing on it a crop of some plants of Pea family. This practice of alternating cereals with leguminous crop is known as rotation of crops. The leguminous plants contain more of nitrogen than they get from the soil salts.The additional quantity is obtained from the air by Rhizobium. Neither Rhizobium nor the legume root alone can fix nitrogen. Rhizobium lives in the soil where it has the form of coccus. The legume roots secrete substances which attract bacteria on to their surface. The bacteria, in turn, secrete a growth hormone which causes the root hairs to curl.The cocci enter the curled root hairs. They grow in the root hair in the form of a continuous thread-like mass which finally reaches the root cortex. In this way many of the cortical cells become filled with a dense mass of these bacteroids.Their presence in the cortical cell serves as a stimulus causing abnormal growth. The cortical cells around the infection divide and redivide and grow to form a nodule. A nodule comprises a central mass of cells full of bacteroids.Around this zone of infection are a few cell layers thick of bacterial free cortical zone.

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At the apex of the nodule are the meristematic region and a vascular strand at its base. Within the host cells the cocci feed on the carbohydrates and other foods and undergo a change in their form they become V, T or Y shaped. The core of the nodule is red owing to the formation of red respiratory pigment haemoglobin.

Role of Bacteria in Industries

Man has utilised the activities of bacteria for various industrial processes. The butter and cheese industries entirely depend upon the activities of the lactic acid bacteria. The souring and curding of milk by lactic acid bacteria is another common example of application in everyday life. It takes place in two steps. In the first step the lactose sugar of milk is fermented into glucose by enzyme lactose secreted by the lactic acid bacteria. In the second step there is transformation of glucose into lactic acid. The latter sours the milk and coagulates the milk proteins (casein) forming curds and whey. Oxidation of alcohol into vinegar (acetic acid) is brought about by the acetic acid bacteria. The curing of tea, tobacco and manufacture of indigo are other examples of useful chemical activities of bacteria which have been controlled for the benefit of mankind. The process of tanning hides in leather making and preparing sponges also involve the use of bacteria. The production of linen is impossible without bacterial activity. The tough fibres, which are left behind, are separated. These fibres are spun and woven into linen cloth, ropes, etc. The preparation of coffee and cocoa is also dependent upon bacterial action. The cocoa beans are white in colour and quite bitter in taste. The bacteria digest the bitter coverings of seeds and give the characteristic colour, flavour and aroma. Many saprophytic bacteria in their metabolic activities excrete waste products of great commercial importance. It is useful in tanning industries. Vitamin B is the product of fermentation of sugars and starch by Clostridium acetobutilicum. The vitamins are used in medicinal preparations. Butyl alcohol, acetone and ethyl alcohol are produced in one fermentation operation when a certain bacterium is allowed to act on cooked com starch. These products are important commercial solvents.It is an important ingredient of explosives and is also used in the manufacture of photographic films.

Role of Bacteria in Medicine

1. Source of Antibiotics:

The milder antibiotics of bacterial origin are tyrothricin, subtilin, polymyxin B, and bacitracin. Bacillius subtilis is the source of subtilin. Bacitracin is obtained from a stain very much like B. subtilis. The actionomycetes which are filamentous, bacteria-like organisms produce more powerful antibiotics such as streptomycin, aureomycin and terramycin. Preparation of Serums and Vaccines: These are substances which are used to develop immunity to various diseases in man. Serums are used in advance as a therapeutic measure. They are also used when a person actually suffers from a disease. Diphtheria, lockjaw, pneumonia, etc. are the diseases in which the serums are effective. Vaccines are commonly used to make people immune to diseases like typhoid, small-pox, cholera, scarlet fever, etc. In the preparation of serums, small doses of bacterial toxins (poisons) are injected into the blood of animals such as

horses. To combat or neutralize the bacterial poisons, the body of the animal produces antibodies. The blood of the animal is then withdrawn. Impurities such as blood corpuscles and other solid matter are removed from the blood. The clear blood liquid containing the antibodies is the serum. It is used as weapon to combat diseases caused by these bacteria. To produce vaccines dead or weakened disease producing bacteria or their diluted poisons (antigens) are directly injected into a man to cause disease in a mild form. As a reaction the host is stimulated to form antibodies. The latter may remain for years in the body of the host imparting immunity against the same type of bacteria which may later enter his body. This is an incomplete account of the beneficial activities of bacteria. Anyhow it indicated their extreme importance in everyday life. Because of these beneficial activities the bacteria are called the friends of mankind.

3.7 CHECK YOUR PROGRESS

1. What is binary fission?
2.What are the major and minor nutrients of bacterial cells?

3.8 LET US SUM UP

Two separate processes. Bacteria reproduce by a process called binary fission. During binary fission, the DNA is replicated and the cell separates. Each daughter cell receives a complete genome. This process represents asexual reproduction, as no exchange or reassortment of genetic information takes place. During sexual reproduction, genes are reshuffled to increase genetic diversity. Gene transfer in eukaryotes occurs when two germ line cells from parents merge to form a zygote. The zygote contains equal amounts of DNA from both parents. Gene transfer in bacteria is a one-way street in that a donor cell gives DNA to a recipient cell, but not vice versa. Gene transfer between bacteria may occur by uptake of unprotected DNA, movement of DNA in virus particles, or cell-to-cell DNA transfer.Bacterial gene transfer occurs by one of three mechanisms: transformation, transduction, and conjugation.Transformation is the uptake of naked DNA from the environment. Transduction occurs via a bacteriophage. The bacteriophage acquires host cell genetic information during packaging and transfers this material to a new host cell during infection. Conjugation is the movement of genetic information from cell-to-cell. DNA that enters a bacterial cell may survive on its own if it is a complete replicon. Otherwise, it will be degraded unless it is recombined into the host chromosome.Incoming DNA for a bacterial cell faces three possible fates. First, the DNA may enter the cell and survive as a replicon, which means it is able to replicate independent of the host cell's chromosome, and therefore must possess an origin of replication. Second, the incoming DNA may be completely degraded. Third, some of the incoming DNA may be recombined into the host chromosome, while the rest is degraded. Recombination usually only occurs between DNA molecules that share homology.

3.9 UNIT - END EXERCISES

1. How are bacteria characterized?
2. Describe in detail about economic importance of bacteria?

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3.10 ANSWERS TO CHECK YOUR PROGRESS

1. Bacteria are single-celled organisms. They lack organelles such as chloroplasts and mitochondria, and they do not have the true nucleus found in eukaryotic cells. Instead, their DNA, a double strand that is continuous and circular, is located in a nucleoid. The nucleoid is an irregularly shaped region that does not have a nuclear membrane. Bacteria also have a cell membrane and a cell wall that is often made of peptidoglycan. Together, the cell membrane and cell wall are referred to as the cell envelope. Many bacteria need a cell wall in order to survive.Reproduction occurs through binary fission, which is the splitting of a bacterial cell after it reaches a certain size. Bacteria reproduce asexually, so the two daughter cells that result from binary fission have the same DNA as the parent cell. However, some bacteria can also exchange genetic material among one another in a process known as horizontal gene transfer. This method involves two already existing bacteria; it is not a form of transmission from parent to child.

2. Bacteria are economically important as these microorganisms are used by humans for many purposes. The beneficial uses of bacteria include the production of traditional foods such as yoghurt, cheese, and vinegar. Microbes are also important in agriculture for the compost and fertilizer production. Made to rise by fermentation, with a leaven that consists of bacteria, often combined with wild yeast enzymes. The milk-souring bacterial genus Lactobacillus is used to make yoghurt and cheese. Bacteria are used, too, to form organic acids in pickles and vinegar. Involves the use of microorganisms including bacteria and fungi in the manufacturing and services industries. These include chemical manufacturing such as ethanol, acetone, organic acid, enzymes, and perfumes. Bacteria are important in the production of many dietary supplements and pharmaceuticals. For example, is used for commercial preparation of riboflavin and vitamin K. E. coli is also used to produce D-amino acids such as D-p-hydroxyphenylglycine, an important intermediate for synthesis of the antibiotic amoxicillin. The manipulation of genes also called recombinant DNA technology. In genetic engineering, pieces of DNA (genes) are introduced into a host by a variety of techniques, one of the earliest being the use of a virus vector. The foreign DNA becomes a permanent feature of the host, and is replicated and passed on to daughter cells along with the rest of its DNA. Bacterial cells are transformed and used in production of commercially important products. Examples include production of human insulin (used to treat diabetes) and human growth hormone (somatotrophin used to treat pituitary dwarfism. Bacteria such as Clostridium butyricum are used to separate fibres of jute, hemp and flax in the process of retting. The plants are immersed in water and when they swell, inoculated with bacteria which hydrolyzepectic substances of the cell walls and separate the fibres. Alternatively, the plants are spread out on the ground, where they become wetted by dew and ret naturally. These separated fibres are used to make ropes, sacks etc. Bacteria living in the gut of cattle, horses and other herbivores, for example Ruminococcus spp., help digest cellulose by secreting the enzyme cellulose. This is how herbivores are able get the energy they need from grass and other plants.Also, Escherichia coli, part of the intestinal microbiota of humans and other animals,

converts consumed food into vitamin K2. This is absorbed in the colon and, in animal models, is sufficient to meet their daily requirement of the vitamin. Bacteria can also be used in the place of pesticides in biological pest control. This commonly uses Bacillus thuringiensis (also called BT), a Gram-positive, soil-dwelling bacterium. This bacterium is used as a Lepidopteran-specific insecticide under trade names such as Dipel and Thuricide. Because of their specificity, these pesticides are regarded as environmentally friendly, with little effect on humans, wildlife, pollinators, or other beneficial insects. Bacteria can be used to remove pollutants from contaminated water, soil and subsurface material. During the Mega Borg Oil Spill, for example, 100 pounds of bacteria were sprayed over an acre of the oil slick to break down the hydrocarbons present into more benign by-products.

3.11 SUGGESTED READINGS

1. Ananthanarayan and Paniker's Textbook of Microbiology Tenth edition with booklet Paperback – Jun 2017 by Reba Kanungo (Author)

BLOCK 2: VIRUS AND MYCOPLASMAS

NOTES

UNIT IV VIROLOGY

4.1 INTRODUCTION

A virus is a biological agent that reproduces inside the cells of living hosts. When infected by a virus, a host cell is forced to produce thousands of identical copies of the original virus at an extraordinary rate. Unlike most living things, viruses do not have cells that divide; new viruses are assembled in the infected host cell. But unlike still simpler infectious agents, viruses contain genes, which gives them the ability to mutate and evolve. Over 5,000 species of viruses have been discovered. The origins of viruses are unclear: some may have evolved from plasmids—pieces of DNA that can move between cells—while others may have evolved from bacteria. A virus consists of two or three parts: genes, made from either DNA or RNA, long molecules that carry genetic information; a protein coat that protects the genes; and in some viruses, an envelope of fat that surrounds the protein coat and is used, in combination with specific receptors, to enter a new host cell. Viruses vary in shape from the simple helical and icosahedral to more complex structures. Viruses range in size from 20 to 300 nanometres; it would take 33,000 to 500,000 of them, side by side, to stretch to 1 centimetre (0.39 in). Viruses spread in many ways. Just as many viruses are very specific as to which host species or tissue they attack, each species of virus relies on a particular method for propagation. Plant viruses are often spread from plant to plant by insects and other organisms, known as vectors. Some viruses of animals, including humans, are spread by exposure to infected bodily fluids. Viruses such as influenza are spread through the air by droplets of moisture when people cough or sneeze. Viruses such as norovirus are transmitted by the faecal–oral route, which involves the contamination of hands, food and water. Rotavirus is often spread by direct contact with infected children. The human immunodeficiency virus, HIV, is transmitted by bodily fluids transferred during sex. Others, such as the Dengue virus, are spread by blood-sucking insects. Viral infections can cause disease in humans,

animals and even plants. However, they are usually eliminated by the immune system, conferring lifetime immunity to the host for that virus. Antibiotics have no effect on viruses, but antiviral drugs have been developed to treat life-threatening infections. Vaccines that produce lifelong immunity can prevent some viral infections.

4.2 OBJECTIVES

In addition to fulfilling the learning objectives provided by individual lecturers, the student should be able to do the following.

- Describe the structure and replication strategies of the individual viruses discussed, including the processes of entry into cells, control of gene transcription and where relevant translation and gene product stability, control of and mechanism(s) of genome replication, virion assembly and egress from the cell.
- Define the process of virus latency and describe in molecular terms control of the process and activation of viral genomes during reactivation.
- Describe the growth behavior differences between normal cells and cells transformed by oncogenic DNA and RNA viruses.
- Describe the processes of senescence and apoptosis and discuss the impact of oncogenic viruses (and specific viral gene products or activities) on these processes.
- Define the term tumor suppressor genes, describe how the corresponding gene products are involved in normal cell growth control, how tumor suppressor gene products intersect growth control/survival pathways, and how tumor viruses interact with these products and their intersecting pathways.
- Describe the processes involved in the anti-tumor effects of "anti-tumor" viruses.
- Integrate experimental strategies learned in the context of individual viral systems into the design of experiments involving other systems.
- Interpret data from experiments that utilize methodologies described and draw appropriate conclusions from the data.

4.3 VIROLOGY – GENERAL FEATURES

Essentially, virology is the branch of microbiology that deals with the study of viruses (as well as various virus-like particles), their characteristics, classification, as well as the relationship with their respective hosts. Other organisms in microbiology, viruses are very unique with different characteristics (with regards to multiplication, structure, etc) that set them apart. Given that viruses are of medical and veterinary significance, virology has increasingly become one of the most important sub-disciplines of microbiology that has allowed researchers to not only discover treatments and cures for the diseases they cause, but also use them for pharmaceutical purposes. Some of the general characteristics of a virus include:

- Can only reproduce (through synthesis and assembly) in living cells
- Contain DNA/RNA or both in some cases
- Are not capable of sexual or asexual modes of reproduction

- Are not cells - They are a cellular particles that lack normal cell organelles and cytoplasm
- Very small compared to other single-celled organisms.

Unlike other true single-cells organisms, viruses are referred to as "particles" in most books because they are not considered to be "living" cells. They are parasites that fully depend on living cells for replication.

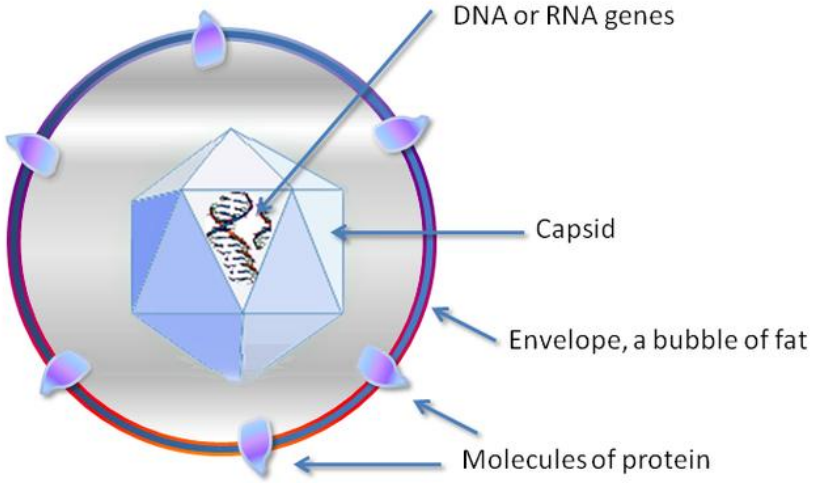

Figure 4.1: Schematic representation of Virus

4.4 VIRUS CLASSIFICATION

According to a system that was proposed in the 70s by the International Committee on the Taxonomy of Viruses (ICTV), the classification (naming) was provided:

- Phylum: Viricota
- Class: Viricetes
- Order: Virales
- Family: Viridae
- Subfamily: Virinae
- Genus: Virus
- Subgenus: Virus
- Apart from this system of classification, virology also classifies viruses based on the following characteristics:

Nature of Nucleic Acid

For the most part, cells of living organisms contain DNA in their nucleus that carries the genetic material. For viruses, however, they either carry DNA or RNA with genes that are responsible for encoding specific proteins. The DNA and RNA between various types of viruses are also different which allows specific viruses to be identified. Whereas Poxviruses and Herpes viruses contain an envelope, double-stranded DNA, the double-stranded DNA of Adenoviruses and Polyomaviruses do not have an envelope while Parvoviruses contain unenveloped, single-stranded DNA. These differences are also observed in RNA viruses. For instance, such viruses as Retroviruses and Togaviruses contain enveloped RNA while Picomaviruses lack this outer envelope. As well, the RNA of such viruses as Reoviruses is contained in a double capsid. In order to direct the synthesis of proteins, viral RNA first encodes enzymes that replicate the

RNA to DNA. The new DNA molecule is then directly responsible for the synthesis of viral proteins (Fig. 4.1). Some of the other structures of the genetic material may take the following forms:

- Linear - E.g. smallpox and rabies viruses
- Circular - E.g. Papillomaviruses
- Non-segmented - E.g. Parainfluenza viruses
- Segmented - E.g. Influenza viruses

4.5 CHARACTERISTICS – ULTRA STRUCTURE OF VIRUS

Different types of viruses also have different shape/morphology. Currently, several shapes of the virus shell have been identified, which has in turn been used to classify different types of viruses (Fig. 4.2). These include:

Helical symmetry - Viruses with this morphology contain a layer of capsomer that is stacked around the nucleic acid forming a helical shape. Assembly of the protein subunits forms an elongated helical structure that is either flexible or tough in nature. Examples of these viruses include the Sendal virus and the tobacco mosaic virus.

Icosahedral Symmetry - Typically, viruses classified under this morphology have a polyhedron structure consisting of about 20 faces/sides of equilateral triangles as well as 12 verticles/corners. Here, lines that run through the opposite vertices define the general appearance of the shell. For instance, those that run through the centers of the faces of opposite triangles result in threefold rotational symmetry axes. Here, then, icosahedra symmetry may range from fivefold to twofold rotational symmetry. Adenovirus, rhinovirus, and poliovirus are good examples of viruses that fall in this category.

Prolate - This is a type of icosahedra that is elongated. As a result, they may appear more cylindrical in shape given that the elongation is along one axis. This morphology has been associated with a majority of viruses known as bacteriophages (virus that infects and replicate in bacteria). Good examples of bacteriophages include the M13 bacteriophage and *Escherichia* Virus T4.

Complex - The complex structure is a combination of the helical and icosahedral symmetry. Therefore, viruses with capsids referred to as complex cannot be fully classified as helical or icosahedral. In some cases, these viruses may contain additional structures such as a complex cell wall. This allows for the virus to be easily identified based on any extra features. Using such extra structures as the helical tail, the virus can attach on to a cell (e.g. bacteria) before inserting their DNA. The Poxvirus that causes smallpox in human beings is an example of a virus with a complex shell.

Envelope - Compared to the other shells, the shell of viruses, referred to as an envelope, are covered by a lipid bilayer membrane. In most cases, this covering is formed as the virus exits the host cell. Some of the viruses that have the lipid bilayer envelope include the HIV and Influenza virus.

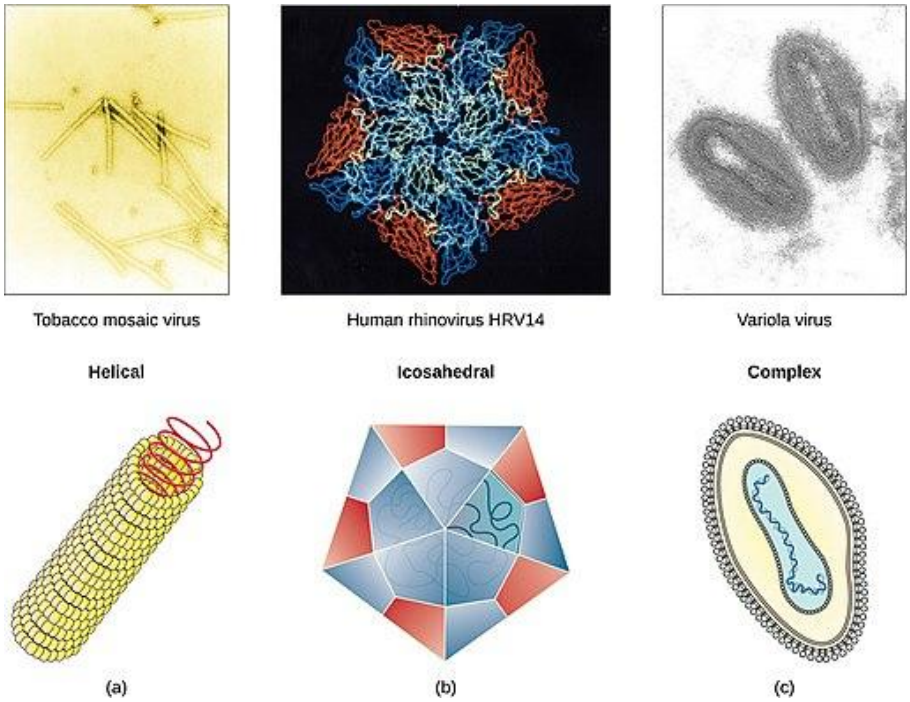

Figure 4.2: Helical, icosahedral and complex structures of viruses

4.6 CHECK YOUR PROGRESS

1. What are some common viruses?
2. How do you get a virus?

4.7 LET US SUM UP

A virus is a small parasite that cannot reproduce by it. Once it infects a susceptible cell, however, a virus can direct the cell machinery to produce more viruses. Most viruses have either RNA or DNA as their genetic material. The nucleic acid may be single- or double-stranded. The entire infectious virus particle, called a virion, consists of the nucleic acid and an outer shell of protein. The simplest viruses contain only enough RNA or DNA to encode four proteins. The most complex can encode 100 – 200 proteins. A wealth of subsequent research with bacterial viruses and animal viruses has provided detailed understanding of viral structure, and virus-infected cells have proved extremely useful as model systems for the study of basic aspects of cell biology. In many cases, DNA viruses utilize cellular enzymes for synthesis of their DNA genomes and mRNAs; all viruses utilize normal cellular ribosomes, tRNAs, and translation factors for synthesis of their proteins. Most viruses comman-deer the cellular machinery for macromolecular synthesis during the late phase of infection, directing it to synthesize large amounts of a small number of viral mRNAs and proteins instead of the thousands of normal cellular macromolecules. For instance, animal cells infected by influenza or vesicular stomatitis virus synthesize only one or two types of glycoproteins, which are encoded by viral genes, whereas uninfected cells produce hundreds of glycoproteins. Such virus-infected cells have been used extensively in studies on synthesis of cell-surface glycoproteins. Similarly, much

information about the mechanism of DNA replication has come from studies with bacterial cells and animal cells infected with simple DNA viruses, since these viruses depend almost entirely on cellular proteins to replicate their DNA. Viruses also often express proteins that modify host-cell processes so as to maximize viral replication. For example, the roles of certain cellular factors in initiation of protein synthesis were revealed because viral proteins interrupt their action. Finally, when certain genes carried by cancer-causing viruses integrate into chromosomes of a normal animal cell, the normal cell can be converted to a cancer cell. Since many viruses can infect a large number of different cell types, genetically modified viruses often are used to carry foreign DNA into a cell. This approach provides the basis for a growing list of experimental gene therapy treatments. Because of the extensive use of viruses in cell biology research and their potential as therapeutic agents, we describe the basic aspects of viral structure and function in this section.

4.8 UNIT – END EXERCISES

1. Write in detail about ultra structure of virus?
2. Describe in detail about classification of virus?

4.9 ANSWERS TO CHECK YOUR PROGRESS

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- Linear - E.g. smallpox and rabies viruses
- Circular - E.g. *Papilloma viruses*
- Non-segmented - E.g. Parainfluenza viruses
- Segmented - E.g. Influenza viruses

4.10 SUGGESTED READINGS

1. Essentials of Medical Microbiology Paperback – 2018 by Apurba S. Sastry (Author), Sandhya Bhat (Author)
2. Medical Microbiology 1st Edition, Kindle Edition by F. H. Kayser (Author), K. A. Bienz (Author), J. Eckert (Author)

UNIT- V VIRUS

NOTES

5.1 INTRODUCTION

A virus is a small infectious agent that replicates only inside the living cells of an organism. Viruses can infect all types of life forms, from animals and plants to microorganisms, including bacteria and archaea. Since Dmitri Ivanovsky's 1892 article describing a non-bacterial pathogen infecting tobacco plants, and the discovery of the tobacco mosaic virus by MartinusBeijerinck in 1898, about 5,000 virus species have been described in detail, although there are millions of types. Viruses are found in almost every ecosystem on Earth and are the most numerous type of biological entity. The study of viruses is known as virology, a sub-speciality of microbiology. While not inside an infected cell or in the process of infecting a cell, viruses exist in the form of independent particles, or virions, consisting of: (i) the genetic material, long molecules of DNA or RNA that encode the structure of the proteins by which the virus acts; (ii) a protein coat, the capsid, which surrounds and protects the genetic material; and in some cases (iii) an outside envelope of lipids. The shapes of these virus particles range from simple helical and icosahedral forms for some species to more complex structures for others. Most virus species have virions too small to be seen with an optical microscope, about one hundredth the size of most bacteria. The origins of viruses in the evolutionary history of life are unclear: some may have evolved from plasmids—pieces of DNA that can move between cells—while others may have evolved from bacteria. In evolution, viruses are an important means of horizontal gene transfer, which increases genetic diversity in a way analogous to sexual reproduction. Viruses are considered by some to be a life form, because they carry genetic material, reproduce, and evolve through natural selection, but lack key characteristics (such as cell structure) that are generally considered necessary to count as life. Because they possess some but not all such qualities, viruses have been described as "organisms at the edge of life", and as replicators. One transmission

pathway is through disease-bearing organisms known as vectors: for example, viruses are often transmitted from plant to plant by insects that feed on plant sap, such as aphids; and viruses in animals can be carried by blood-sucking insects. Influenza viruses are spread by coughing and sneezing. Norovirus and rotavirus, common causes of viral gastroenteritis, are transmitted by the faecal–oral route, passed by contact and entering the body in food or water. HIV is one of several viruses transmitted through sexual contact and by exposure to infected blood. The variety of host cells that a virus can infect is called its "host range". This can be narrow, meaning a virus is capable of infecting few species, or broad, meaning it is capable of infecting many. Viral infections in animals provoke an immune response that usually eliminates the infecting virus. Immune responses can also be produced by vaccines, which confer an artificially acquired immunity to the specific viral infection. Some viruses, including those that cause AIDS and viral hepatitis, evade these immune responses and result in chronic infections. Several antiviral drugs have been developed.

5.2 OBJECTIVES

- Explain what a virus is, in terms of its size and molecular components. Distinguish between a capsid and a viral envelope?
- Describe similarities and differences between the tobacco mosaic virus, an adenovirus, the influenza virus, and the T4 bacteriophage.
- Explain the concepts of host range and host specificity. Describe the host ranges and specificities of any two viruses.
- Explain, in general, two ways in which viruses recognize their hosts.
- Describe the life cycle of a virus, including in your description how the viral genome is replicated and how the host cell's biochemical machinery is co-opted to synthesize viral proteins.
- List the different types of nucleic acids that are found in viruses. How does replication and transcription differ from the processes we learned about in higher eukaryotes?
- Compare and contrast the lytic and lysogenic cycles of phage l
- Describe the infection cycle of HIV, including in your description the role of reverse transcriptase and provirus formation. How could you explain the fact that some people infected with HIV do not display the symptoms of AIDS?

5.3 ISOLATION OF VIRUS

Unlike bacteria, many of which can be grown on an artificial nutrient medium, viruses require a living host cell for replication. Infected host cells (eukaryotic or prokaryotic) can be cultured and grown, and then the growth medium can be harvested as a source of virus. Virions in the liquid medium can be separated from the host cells by either centrifugation or filtration. Filters can physically remove anything present in the solution that is larger than the virions; the viruses can then be collected in the filtrate (Fig. 5.1).

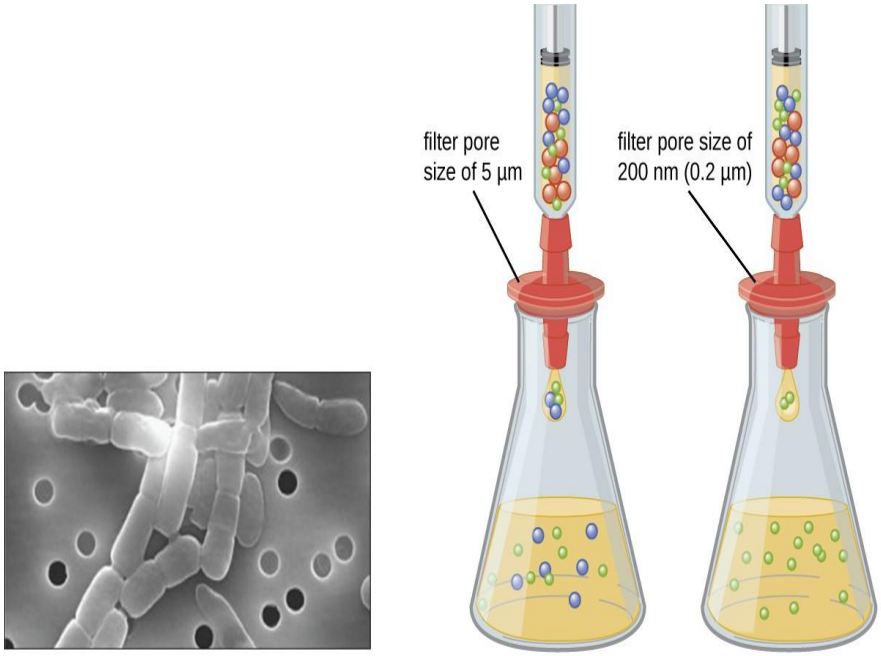

Figure 5.1: Membrane filters can be used to remove cells or viruses from a solution. (a) This scanning electron micrograph shows rod-shaped bacterial cells captured on the surface of a membrane filter. Note differences in the comparative size of the membrane pores and bacteria. Viruses will pass through this filter. (b) The size of the pores in the filter determines what is captured on the surface of the filter (animal [red] and bacteria [blue]) and removed from liquid passing through. Note the viruses (green) pass through the finer filter. (credit a: modification of work by U.S. Department of Energy)

Viruses can be grown **in vivo** (within a whole living organism, plant, or animal) or **in vitro** (outside a living organism in cells in an artificial environment, such as a test tube, cell culture flask, or agar plate). **Bacteriophages** can be grown in the presence of a dense layer of bacteria (also called a **bacterial lawn**) grown in a 0.7 % soft agar in a Petri dish or flat (horizontal) The agar concentration is decreased from the 1.5% usually used in culturing bacteria. The soft 0.7% agar allows the bacteriophages to easily diffuse through the medium. For lytic bacteriophages, lysing of the bacterial hosts can then be readily observed when a clear zone called a **plaque** is .As the phage kills the bacteria, many plaques are observed among the cloudy bacterial lawn.

Animal viruses require cells within a host animal or tissue-culture cells derived from an animal. Animal virus cultivation is important for 1) identification and diagnosis of pathogenic viruses in clinical specimens, 2) production of vaccines, and 3) basic research studies. In vivo host sources can be a developing embryo in an embryonated bird's egg (e.g., chicken, turkey) or a whole animal. For example, most of the influenza vaccine manufactured for annual flu vaccination programs is cultured in hens' eggs. he embryo or host animal serves as an incubator for viral replication (see Figure 3). Location within the embryo or host animal is important. Many viruses have a tissue tropism, and must therefore be introduced into a specific site for

growth. Within an embryo, target sites include the amniotic cavity, the chorioallantoic membrane, or the yolk sac. Viral infection may damage tissue membranes, producing lesions called pox; disrupt embryonic development; or cause the death of the embryo. For in vitro studies, various types of cells can be used to support the growth of viruses. A primary cell culture is freshly prepared from animal organs or tissues. Cells are extracted from tissues by mechanical scraping or mincing to release cells or by an enzymatic method using trypsin or collagenase to break up tissue and release single cells into suspension. Because of anchorage-dependence requirements, primary cell cultures require a liquid culture medium in a Petri dish or tissue-culture flask so cells have a solid surface such as glass or plastic for attachment and growth. Primary cultures usually have a limited life span. When cells in a primary culture undergo mitosis and a sufficient density of cells is produced, cells come in contact with other cells. When this cell-to-cell-contact occurs, mitosis is triggered to stop. This is called contact inhibition and it prevents the density of the cells from becoming too high. To prevent contact inhibition, cells from the primary cell culture must be transferred to another vessel with fresh growth medium. This is called a secondary cell culture. Periodically, cell density must be reduced by pouring off some cells and adding fresh medium to provide space and nutrients to maintain cell growth. In contrast to primary cell cultures, continuous cell lines, usually derived from transformed cells or tumors, are often able to be subcultured many times or even grown indefinitely (in which case they are called immortal). Continuous cell lines may not exhibit anchorage dependency (they will grow in suspension) and may have lost their contact inhibition. As a result, continuous cell lines can grow in piles or lumps resembling small tumor growths. An example of an immortal cell line is the **HeLa cell line**, which was originally cultivated from tumor cells obtained from Henrietta **Lacks**, a patient who died of cervical cancer in 1951. HeLa cells were the first continuous tissue-culture cell line and were used to establish tissue culture as an important technology for research in cell biology, virology, and medicine. Prior to the discovery of HeLa cells, scientists were not able to establish tissue cultures with any reliability or stability. More than six decades later, this cell line is still alive and being used for medical research. See "The Immortal Cell Line of Henrietta Lacks" below to read more about this important cell line and the controversial means by which it was obtained.

5.4 PURIFICATION

Effective virus purification capitalizes on the differences between the physical properties of the adenovirus relative to the components of the mixture from which it is being isolated. The exact composition of the milieu varies with the cell culture process and, to a lesser extent, every batch. In general, the large-scale purification of adenovirus requires the isolation of the virus from infected cell lysate taken from a bioreactor. This mixture consists of a formulated medium sometimes containing bovine serum, and less frequently antifoaming agents, or anticlumping agents (pluronics). Significant amounts of additives, however, present difficult

challenges for any recovery procedure. Efficient large-scale production requires high cell densities which in turn require high gas exchange rates. This can cause severe foaming and necessitate the addition of agents to control it. Other additives such as anticlumping agents and lipids adapt the media for large-scale cell culture. Cell lysis, necessary to release the adenovirus from the host cell, results in the additional release of DNA, protein, lipids, carbohydrates, and other cellular components. Culture conditions, media components, cell-derived contaminants, and additives may have a significant impact on downstream processing. Adenoviruses are produced by infection of cell lines in culture with a viral seed stock. The particular cell line used requires a highly developed cell culture method to achieve maximum yield. Flat stock culture, although useful for small-scale work, is generally not sufficient for larger scale applications. Some of the cell lines used in flat stock culture has resisted attempts to adapt them to the suspension and serum-free conditions preferred for large-scale processes. A compromise is struck by the culture of attachment-dependent cells using microcarriers in a bioreactor. These microcarrier-based processes introduce yet another component that must be separated from the adenovirus. Similarly, if serum is utilized, it will be necessary to consider the effective removal of its components. For downstream purification, higher titersfavor better recovery and cleaner preparations because recovery and purification are enrichment processes. Even with maximum productivity, however, adenoviral particles represent a small fraction of molecular entities produced by the end of the culture process. Therefore, factors affecting the end titer can also affect the process.The majority of adenoviral vectors for gene therapy are serotype 5 and have been rendered deficient for replication in most cells. With the exception of replication-competent adenoviruses, most vectors have been crippled to eliminate their replication in normal human cells. In general, when compared to wild-type virus, deletions or mutations in the early genes tend to attenuate viral replication in all cell lines. Attenuation for replication is typically achieved by large deletions in the immediate early region E1. These vectors require specialized packaging cell lines for efficient production. Cell lines such as HEK 2939 or PER.C610 have been transformed with adenoviral DNA and provide sufficient E1 function in trans to enable replication.In addition to E1 deletions, many vectors possess deletions for much of the E3 region. The deleted E3 genes are considered nonessential for viral replication and these deletions allow for larger transgene packaging capacity. Other deletions have been made to reduce the frequency of recombination during culture. Some vectors may have additional early gene deletions (e.g., in E4) as well as a deletion of protein IX encoding sequences.11 Elimination of certain essential genes from the virus requires that the cell line be able to complement these protein functions in trans to package the virus.

A significant unwanted by-product of adenoviral replication is DNA. Wild-type human adenoviruses are able to replicate in a variety of both quiescent and proliferating human cells due to the function of adenoviral immediate early genes. E1a proteins can be observed within an hour after infection, as cellular transcription factors are sufficient to transcribe the Ela genes. El expression initiates the adenoviral life cycle by

altering the cell cycle machinery to induce cellular DNA replication even in quiescent cells. Viral and cellular proteins activate subsequent viral transcription. New copies of viral DNA are synthesized and viral production proceeds in a replication cascade. By the end of viral DNA replication, a large amount of DNA is present in the infected cell.The purpose of a gene therapy vector is to convey a therapeutic effect by the delivery and expression of therapeutic genes. Many of these transgenes have a significant effect on the cells and adenoviral life cycle. Some genes, such as the retinoblastoma protein and the cyclin-dependent kinase inhibitor p21, directly affect the levels of activated E2F. E2F is a cellular transcription factor that is necessary for the transactivation of the adenoviral E2 promoter and thus the expression of viral DNA replication proteins. Other transgene products, such as pro-apoptotic proteins, can overcome the adenoviral block to apoptosis leading to early cell death and can interfere with adenoviral particle assembly. Secreted pleiotropic transgene products, such as growth factors, can trigger undesired effects in the packaging cell and severely attenuate production. Before adenoviral DNA can be coated with viral core proteins, the DNA is available for transcription. In this way, some adenoviral genes, especially those encoding capsid components, are not expressed until DNA replication occurs. During this phase, the expression of transgene product is enhanced by the replication cycle itself by expanding the copy number of the transgene with each copy becoming available for transcription. Strong exogenous promoters may also sequester transcription factors and the cellular protein synthesis machinery can become clogged with transgene expression leading to attenuated adenoviral protein production. If the sequence of events leading to particle maturation is disturbed, even by an imbalance in protein production, large quantities of viral proteins, incomplete particle assemblies, transgene products, abnormal cellular structures, and in some cases extreme amounts of extracellular proteins can be added to the milieu. These complications can significantly impede purification and add to the analytical requirements.

5.5 CHEMICAL NATURE OF VIRUS

On the whole viruses are much smaller than bacteria. Most animal and plant viruses are invisible under the light microscope. Some of smaller viruses are only 200Å in diameter. Viruses cannot multiply outside a living cell. No virus has been cultivated in a cell-free medium. Viruses do not have an independent metabolism. They are metabolically inactive outside the host cell because they do not possess enzyme systems and protein synthesis machinery. Thus viruses are obligatory intracellular parasites. Viruses have a very simple structure. They consist of a nucleic acid core surrounded by a protein coat. In this respect they differ from typical cells which are made up of proteins, carbohydrates, lipids and nucleic acids. Myxoviruses have a membranous envelope consisting of proteins, carbohydrate and lipid outside the usual protein coat, but this envelope is derived from the host cell. Viruses do not have any cytoplasm, and thus cytoplasmic organelles like mitochondria, Golgi complexes, ribosomes, lysosomes etc. are absent. They do not have any limiting cell membrane.

Viruses usually have only one nucleic acid, either DNA or RNA. Typical cells have both DNA and RNA. Rous Sarcoma virus (RSV), producing certain cancer, is the only virus having both DNA and RNA. Many of the smaller viruses can be crystallized, and thus behave like chemicals. Viruses do not have the power of growth and division. The genetic material of virus reproduces only in a host cell.Thus viruses do not show all the characteristics of typical living organisms. They, however, possess two fundamental characteristics of living systems. Firstly, they contain nucleic acid as their genetic material. The nucleic acid contains all the instructions for the structure and the function of the virus. Secondly, they can reproduce themselves, even if only by using the host cells' synthesis machinery.

5.6 VIRAL REPLICATION

Viral replication is the formation of biological viruses during the infection process in the target host cells. Viruses must first get into the cell before viral replication can occur. Through the generation of abundant copies of its genome and packaging these copies, the virus continues infecting new hosts. Replication between viruses is greatly varied and depends on the type of genes involved in them. Most DNA viruses assemble in the nucleus while most RNA viruses develop solely in cytoplasm. Viruses multiply only in living cells. The host cell must provide the energy and synthetic machinery and the low molecular-weight precursors for the synthesis of viral proteins and nucleic acids.The virus replication occurs in seven stages, namely;

1. Adsorption
2. Entry
3. Uncoating
4. Transcription / mRNA production
5. Synthesis of virus components
6. Virion assembly
7. Release (Liberation Stage)

Adsorption: The virus attaches to the cell membrane of the host cell. It then injects its DNA or RNA into the host to initiate infection. In animal cells these viruses get into the cell through the process of endocytosis which works through fusing of the virus and fusing of the viral envelope with the cell membrane of the animal cell and in plant cell it enters through the process of pinocytosis which works on pinching of the viruses.

Entry: **The cell membrane of the host cell invaginates the virus particle, enclosing it in a pinocytotic vacoule. This protects the cell from antibodies like in the case of the HIV virus.**

Uncoating: **Cell enzymes (from lysosomes) strip off the virus protein coat. This releases or renders accessible the virus nucleic acid or genome.**Transcription / mRNA production: **For some RNA viruses, the infecting RNA produces messenger RNA (mRNA). This is translation of the genome into protein produces. For others with negative stranded RNA and DNA, viruses are produced by transcription then translation. The mRNA is used to instruct the host cell to make virus**

components. The virus takes advantage of the existing cell structures to replicate it

Synthesis of virus components:

The following components are manufactured by the virus through the host's existing organelles:

- Viral protein synthesis: virus mRNA is translated on cell ribosomes into two types of virus protein.
- Structural: the proteins which make up the virus particle are manufactured and assembled.
- Non – structural: not found in particle, mainly enzymes for virus genome replication.
- Viral nucleic acid synthesis (genome replication) new virus genome is synthesized; templates are either the parental genome or with single stranded nucleic acid genomes, newly formed complementary strands. By a virus called polymerate or replicate in some DNA viruses by a cell enzyme. This is done in rapidly dividing cells.

Virion Assembly

A virion is simply an active or intact virus particle. In this stage, newly synthesized genome (nucleic acid), and proteins are assembled to form new virus particles. This may take place in the cell's nucleus, cytoplasm, or at plasma membrane for most developed viruses. For some RNA viruses, the infecting RNA produces messenger RNA (mRNA). This is translation of the genome into protein produces. For others with negative stranded RNA and DNA, viruses are produced by transcription then translation. The mRNA is used to instruct the host cell to make virus components. The virus takes advantage of the existing cell structures to replicate itself.

Release (Liberation Stage)

The viruses, now being mature are released by either sudden rupture of the cell, or gradual extrusion(budding) of enveloped viruses through the cell membrane.The new viruses may invade or attack other cells, or remain dormant in the cell. In the case of bacterial viruses, the release of progeny virions takes place by lysis of the infected bacterium. However, in the case of animal viruses, release usually occurs without cell lysis.

5.7 TRANSMISSION OF VIRUS

The movement of virus from one host to other host is called transmission. The transmis¬sion is dependent upon the external agents.

The transmission of plant viruses takes place by the following means:

(i) By vegetative propagating organs like tuber (potato spindle tuber virus), bulbs (Cattle disease virus), etc.

(ii) Through collateral hosts like lady’s finger and Croton bonplandianum (Yellow vein mosaic virus).

(iii) Through soil (Tobacco, Potato, and Wheat mosaic viruses).

(iv) Through seeds (Bean mosaic virus, Barley strip mosaic virus, Curly top virus of sugar beet, Ring spot virus of soyabean).

(v) Through pollen grains (Stone fruit ring spot virus, Bean mosaic virus).

(vi) Through fungi (Tobacco necrosis virus transmitted by Olpidium brassicae, the root pathogen; Synchytrium brassicae transmits the potato virus).
(vii) Through rubbing and friction between closely growing healthy and infected plants (Potato virus x, Tobacco mosaic virus, etc.).
(viii) Through insects (Yellow mosaic of mung by white fly, Bemisia; Maize mosaic virus by an aphid, Myzus; Maize dwarf mosaic virus by an aphid, Brachycaudus, etc.).

5.8 VIRIONS

The illustration at left depicts a virion – the infectious particle that is designed for transmission of the nucleic acid genome among hosts or host cells. A virion is not the same as a virus. I define virus as a distinct biological entity with five different characteristics. Others believe that the virus is actually the infected host cell. The idea that virus and virion are distinct was first proposed by Bandea in 1983. He suggested that a virus is an organism without a cohesive morphological structure, with subsystems that are not in structural continuity: Viruses are presented as organisms which pass in their ontogenetic cycle through two distinctive phenotypic phases: (1) the vegetative phase and (2) the phase of viral particle or nucleic acid. In the vegetative phase, considered herein to be the ontogenetically mature phase of viruses, their component molecules are dispersed within the host cell. In this phase the virus shows the major physiological properties of other organisms: metabolism, growth, and reproduction. According to Bandea's hypothesis, the infected cell is the virus, while the virus particles are 'spores' or reproductive forms. His theory was largely ignored until the discovery of the giant mimivirus, which replicates its DNA genome and produces new virions in the cytoplasm within complex viral 'factories'. Claverie suggested that the viral factory corresponds to the organism, whereas the virion is used to spread from cell to cell. He wrote that "to confuse the virion with the virus would be the same as to confuse a sperm cell with a human being". If we accept that the virus is the infected cell, then it becomes clear that most virologists have confused the virion and the virus. This is probably a consequence of the fact that modern virology is rooted in the study of bacteriophages that began in the 1940s. These viruses do not induce cellular factories, and disappear (the eclipse phase) early after cell entry. Contemporary examples of such confusion include the production by structural virologists of virus crystals, and the observation that viruses are the most abundant entities in the seas. In both cases it is the virion that is being studied. But virologists are not the only ones at fault – the media writes about the AIDS virus while showing an illustration of the virion. One can conclude that infected eukaryotic cells in which viral factories have taken control of the cellular machinery became viruses themselves, the viral factory being in that case the equivalent of the nucleus. By adopting this viewpoint, one should finally consider viruses as cellular organisms. They are of course a particular form of cellular organism, since they do not encode their own ribosomes and cell membranes, but borrow those from the cells in which they live.This argument leads to the assumption that viruses are living, according to the classical definition of

living organisms as cellular organisms. Raoult and Forterre have therefore proposed that the living world should be divided into two major groups of organisms, those that encode ribosomes (archaea, bacteria and eukarya), and capsid-encoding organisms (the viruses).

5.9 CHECK YOUR PROGRESS

1. Define virion?
2. What are the chemical natures of virus?

5.10 LET US SUM UP

Understanding the molecular events accompanying virus replication is essential for the proper understanding and control of all virus diseases. The virus replication cycle generates new viral genomes and proteins in sufficient quantities to ensure propagation of the viral genome; this requires that the extracellular viral genome is protected from enzymatic degradation and can be introduced into further target cells for further rounds of replication. The initial recognition between virus and host is more complex than originally supposed and may involve more than one cellular receptor. A critical first intracellular step is the generation of viral mRNA by one of a limited number of strategies first described by David Baltimore. Lacking ribosomes, viruses have no means of producing protein and are reliant on the host cell for protein synthesis. Viral proteins are often modified by host cell glycosylation during or after virus assembly. Temporal regulation of intracellular events is critical in all but the very simplest of viruses, and some form of suppression of the host innate immune response is common to nearly all human viruses. Infected cells often produce non-infectious particles with incomplete genomes, and these defective interfering particles may play a role in pathogenesis. Understanding these processes will open up a range of targets for the development of novel therapies.

5.11 UNIT - END EXERCISES

1. What are the 6 steps of viral replication?
2. What is isolation and purification?

5.12 ANSWERS TO CHECK YOUR PROGRESS

1. Viral replication is the formation of biological viruses during the infection process in the target host cells. Viruses must first get into the cell before viral replication can occur. Through the generation of abundant copies of its genome and packaging these copies, the virus continues infecting new hosts. Replication between viruses is greatly varied and depends on the type of genes involved in them. Most DNA viruses assemble in the nucleus while most RNA viruses develop solely in cytoplasm. Viruses multiply only in living cells. The host cell must provide the energy and synthetic machinery and the low molecular-weight

precursors for the synthesis of viral proteins and nucleic acids. The virus replication occurs in seven stages, namely;

1. Adsorption
2. Entry
3. Uncoating
4. Transcription / mRNA production
5. Synthesis of virus components
6. Virion assembly
7. Release (Liberation Stage)

Adsorption: The virus attaches to the cell membrane of the host cell. It then injects its DNA or RNA into the host to initiate infection. In animal cells these viruses get into the cell through the process of endocytosis which works through fusing of the virus and fusing of the viral envelope with the cell membrane of the animal cell and in plant cell it enters through the process of pinocytosis which works on pinching of the viruses.

Entry: The cell membrane of the host cell invaginates the virus particle, enclosing it in a pinocytotic vacoule. This protects the cell from antibodies like in the case of the HIV virus.

Uncoating: Cell enzymes (from lysosomes) strip off the virus protein coat. This releases or renders accessible the virus nucleic acid or genome.

Transcription / mRNA production: For some RNA viruses, the infecting RNA produces messenger RNA (mRNA). This is translation of the genome into protein produces. For others with negative stranded RNA and DNA, viruses are produced by transcription then translation. The mRNA is used to instruct the host cell to make virus components. The virus takes advantage of the existing cell structures to replicate it

Synthesis of virus components:

The following components are manufactured by the virus through the host's existing organelles:

- Viral protein synthesis: virus mRNA is translated on cell ribosomes into two types of virus protein.
- Structural: the proteins which make up the virus particle are manufactured and assembled.
- Non – structural: not found in particle, mainly enzymes for virus genome replication.
- Viral nucleic acid synthesis (genome replication) new virus genome is synthesized; templates are either the parental genome or with single stranded nucleic acid genomes, newly formed complementary strands. By a virus called polymerate or replicate in some DNA viruses by a cell enzyme. This is done in rapidly dividing cells.

Virion Assembly

A virion is simply an active or intact virus particle. In this stage, newly synthesized genome (nucleic acid), and proteins are assembled to form new virus particles. This may take place in the cell's nucleus, cytoplasm, or at plasma membrane for most developed viruses. For some RNA viruses, the infecting RNA produces messenger RNA (mRNA). This is translation of the genome into protein produces. For others with negative stranded RNA and DNA, viruses are produced by transcription then translation. The mRNA is used to instruct the host cell to make virus

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Release (Liberation Stage)

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2. Virus isolation from blood or CSF is impractical and requires the use of specialised lymphocyte culture lines. Available serological assays include IIF (indirect immunofluorescence), capture EIA, neutralization and immunoblot assays. Shortcomings of serology include the inability to distinguish between primary infection and reactivation as well as cross-reactions between HHV-6 and HHV-7. Molecular techniques, especially quantitative polymerase chain reaction (PCR) from blood, and CSF, can distinguish between latent infection and active primary infection or reactivation, and are fast becoming the mainstay of the diagnostic approach. Purification is the process of separating the virus particles from host constituents and other chemicals present in sap. Purified viral preparations help in λ Study of physico-chemical properties of the λ virus Virus morphology (Shape λ & size)

5.13 SUGGESTED READINGS

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UNIT- VI PRIONS

6.1 INTRODUCTION

Prions are infectious agents that long defied some of our basic ideas of biology. They appear to behave like other infectious organisms, yet they lack any of the most fundamental features of organisms. In particular, they lack any genetic material (DNA or RNA). Over time, work on prions has suggested that the "infectious agent" is actually a misfolded protein -- which causes a normal cellular protein to change its shape to the misfolded form.Prion diseases and prions are so unusual and so fascinating that they have been the subject of two Nobel prizes in Physiology or Medicine. In 1976 Carleton Gajdusek shared the Nobel Prize for his work showing that the human disease kuru was similar to the well known sheep disease scrapie. In 1997 Stanley Prusiner, at UCSF, was the sole recipient of the prize; Prusiner was responsible for developing the modern prion model.As diseases, prion diseases are quite rare and difficult to transmit. But they are also quite scary, because they are progressive neurodegenerative diseases, with no cure or treatment. They also have the mystique of being strange, due to the poor understanding of what prions are and how they work.The prion disease most in the news is BSE (bovine spongiform encephalopathy), often called mad-cow disease. It is rather likely that the BSE agent can be transmitted to humans, and cause vCJD (variant Creutzfeldt-Jakob disease). The number of known vCJD cases in humans is under 200, but there are so many unknowns, including a possible incubation period of many many years, that this mysterious disease strikes fear at least much uncertainty. As we learn more about prion diseases, a new part of the story is emerging. It is possible that a number of neurodegenerative diseases long considered quite distinct may share some underlying features. These include Alzheimer's disease, Parkinson's disease, Huntington disease, and the prion diseases. The common thread may be that all involve misfolded proteins. The reason for the misfolding and the details of the disease development vary, and there is no implication here that all of these are infectious. In fact, not all prion diseases are infectious.

6.2 OBJECTIVES

- Prions are infectious agents that consist of protein, but no DNA or RNA, and seem to produce their deadly effects by duplicating their shapes and accumulating in tissues.
- They are thought to contribute to several progressive brain disorders, including mad cow disease and Creutzfeldt-Jakob disease.

6.3 PRIONS

Prions are misfolded proteins with the ability to transmit their misfolded shape onto normal variants of the same protein. They characterize several fatal and transmissible neurodegenerative diseases in humans and many other animals. It is not known what causes the normal protein to misfold, but the abnormal three-dimensional structure is suspected of conferring infectious properties, collapsing nearby protein molecules into the same shape. The word prion derives from "proteinaceous infectious particle". The hypothesized role of a protein as an infectious agent stands in contrast to all other known infectious agents such as viruses, bacteria, fungi and parasites, all of which contain nucleic acids (DNA, RNA or both). Prion variants of the prion protein (PrP), whose specific function is uncertain, are hypothesized as the cause of transmissible spongiform encephalopathies (TSEs), including scrapie in sheep, chronic wasting disease (CWD) in deer, bovine spongiform encephalopathy (BSE) in cattle (commonly known as "mad cow disease") and Creutzfeldt–Jakobdisease (CJD) in humans. All known prion diseases in mammals affect the structure of the brain or other neural tissue; all are progressive, have no known effective treatment and are always fatal. Until 2015, all known mammalian prion diseases were considered to be caused by the prion protein (PrP), however in 2015 multiple system atrophy (MSA) was found to be transmissible and was hypothesized to be caused by a prion form of alpha-synuclein. Prions form abnormal aggregates of proteins called amyloids, which accumulate in infected tissue and are associated with tissue damage and cell death. Amyloids are also responsible for several other neurodegenerative diseases such as Alzheimer's disease and Parkinson's disease. Prion aggregates are stable, and this structural stability means that prions are resistant to denaturation by chemical and physical agents: they cannot be destroyed by ordinary disinfection or cooking. This makes disposal and containment of these particles difficult. A prion disease is a type of proteopathy, or disease of structurally abnormal proteins. In humans, prions are believed to be the cause of Creutzfeldt–Jakob disease (CJD), its variant (vCJD), Gerstmann–Sträussler–Scheinker syndrome (GSS), fatal familial insomnia (FFI) and kuru. There is also evidence suggesting prions may play a part in the process of Alzheimer’s disease, Parkinson's disease and amyotrophic lateral sclerosis (ALS), and these have been termed prion-like diseases. Several yeast proteins have also been identified as having prionogenic properties. Prion replication is subject to epimutation and natural selection just as for other forms of replication, and their structure varies slightly

between species. Recent scientific observations show the need to refine the prion hypothesis. Synthetic prions, created in the laboratory independent of any biological source, have little or no ability to cause infection with TSEs; however, when synthetic prions are administered in combination with cofactors, such as phosphatidylethanolamine and RNA molecules, this can transmit TSEs. It has also been shown that scrapie and Creutzfeldt–Jakob disease may require agent-specific nucleic acids for transmission of infection. Most recently, it was shown that mice with severe combined immunodeficiency do not develop scrapie following inoculation with brain tissue from animals infected with scrapie, suggesting that either the role of immunity in prion pathogenesis is incompletely understood or that there is some other flaw in current understanding of prion pathophysiology. The protein that prions are made of (PrP) is found throughout the body, even in healthy people and animals. However, PrP found in infectious material has a different structure and is resistant to proteases, the enzymes in the body that can normally break down proteins. The normal form of the protein is called PrP^{C}, while the infectious form is called PrP^{Sc} – the C refers to 'cellular' PrP, while the Sc refers to 'scrapie', the prototypic prion disease, occurring in sheep. While PrP^{C} is structurally well-defined, PrP^{Sc} is certainly polydisperse and defined at a relatively poor level. PrP can be induced to fold into other more-or-less well-defined isoforms in vitro, and their relationship to the form(s) that are pathogenic in vivo is not yet clear. PrP^{C} is a normal protein found on the membranes of cells. It has 209 amino acids (in humans), one disulfide bond, a molecular mass of 35–36 kDa and a mainly alpha-helical structure. Several topological forms exist; one cell surface form anchored via glycolipid and two transmembrane forms. The normal protein is not sedimentable; meaning that it cannot be separated by centrifuging techniques. Its function is a complex issue that continues to be investigated. PrP^{C} binds copper (II) ions with high affinity. The significance of this finding is not clear, but it is presumed to relate to PrP structure or function. PrP^{C} is readily digested by proteinase K and can be liberated from the cell surface in vitro by the enzyme phosphoinositide phospholipase C (PI-PLC), which cleaves the glycophosphatidylinositol (GPI) glycolipid anchor. PrP has been reported to play important roles in cell-cell adhesion and intracellular signaling in vivo, and may therefore be involved in cell-cell communication in the brain. Protease-resistant PrP^{Sc}-like protein (PrP^{res}) is the name given to any isoform of PrP^{c} which is structurally altered and converted into a misfolded proteinase K-resistant form in vitro. To model conversion of PrP^{C} to PrP^{Sc} in vitro, rapidly converted PrP^{C} into a PrPres by a procedure involving cyclic amplification of protein misfolding. The term "PrP^{res}" has been made to distinguish between PrP^{Sc}, which is isolated from infectious tissue and associated with the transmissible spongiform encephalopathy agent. For example, unlike PrP^{Sc}, PrP^{res} may not necessarily be infectious. The infectious isoform of PrP, known as PrP^{Sc}, or simply the prion, is able to convert normal PrP^{C} proteins into the infectious isoform by changing their conformation, or shape; this, in turn, alters the way the proteins interconnect. PrP^{Sc} always causes prion disease. Although the exact 3D structure of PrP^{Sc} is not known, it has a higher proportion of β-sheet structure in place of the

normal α-helix structure. Aggregations of these abnormal isoforms form highly structured amyloid fibers, which accumulate to form plaques. It is unclear as to whether these aggregates are the cause of cell damage or are simply a side-effect of the underlying disease process. The end of each fiber acts as a template onto which free protein molecules may attach, allowing the fiber to grow. Under most circumstances, only PrP molecules with an identical amino acid sequence to the infectious PrP^{Sc} are incorporated into the growing fiber. However, rare cross-species transmission is also possible. The physiological function of the prion protein remains poorly understood. While data from in vitro experiments suggest many dissimilar roles, studies on PrP knockout mice have provided only limited information because these animals exhibit only minor abnormalities. In research done in mice, it was found that the cleavage of PrP proteins in peripheral nerves causes the activation of myelin repair in Schwann cells and that the lack of PrP proteins caused demyelination in those cells. MAVS, RIP1, and RIP3 are prion-like proteins found in other parts of the body. They also polymerise into filamentous amyloid fibers which initiate regulated cell death in the case of a viral infection to prevent the spread of virions to other, surrounding cells. A review of evidence in 2005 suggested that PrP may have a normal function in maintenance of long-term memory. As well, a 2004 study found that mice lacking genes for normal cellular PrP protein show altered hippocampal long-term potentiation. A recent study that might explain why this is found that neuronal protein CPEB has a similar genetic sequence to yeast prion proteins. The prion-like formation of CPEB is essential for maintaining long-term synaptic changes associated with long term memory formation. A 2006 article from the Whitehead Institute for Biomedical Research indicates that PrP expression on stem cells is necessary for an organism's self-renewal of bone marrow. The study showed that all long-term hematopoietic stem cells express PrP on their cell membrane and that hematopoietic tissues with PrP-null stem cells exhibit increased sensitivity to cell depletion. There is some evidence that PrP may play a role in innate immunity, as the expression of PRNP, the PrP gene, is upregulated in many viral infections and PrP has antiviral properties against many viruses, including HIV. The first hypothesis that tried to explain how prions replicate in a protein-only manner was the heterodimer model. This model assumed that a single PrPSc molecule binds to a single PrP^{C} molecule and catalyzes its conversion into PrP^{Sc}. The two PrP^{Sc} molecules then come apart and can go on to convert more PrP^{C}. However, a model of prion replication must explain both how prions propagate, and why their spontaneous appearance is so rare. Manfred Eigen showed that the heterodimer model requires PrP^{Sc} to be an extraordinarily effective catalyst, increasing the rate of the conversion reaction by a factor of around 1015. This problem does not arise if PrP^{Sc} exists only in aggregated forms such as amyloid, where cooperativity may act as a barrier to spontaneous conversion. What is more, despite considerable effort, infectious monomeric PrP^{Sc} has never been isolated. An alternative model assumes that PrP^{Sc} exists only as fibrils, and that fibril ends bind PrP^{C} and convert it into PrP^{Sc}. If this were all, then the quantity of prions would increase linearly, forming ever longer fibrils. But exponential growth of both PrP^{Sc}

and of the quantity of infectious particles is observed during prion disease. This can be explained by taking into account fibril breakage. A mathematical solution for the exponential growth rate resulting from the combination of fibril growth and fibril breakage has been found. The exponential growth rate depends largely on the square root of the PrP^C concentration. The incubation period is determined by the exponential growth rate, and in vivo data on prion diseases in transgenic mice match this prediction. The same square root dependence is also seen in vitro in experiments with a variety of different amyloid proteins. The mechanism of prion replication has implications for designing drugs. Since the incubation period of prion diseases is so long, an effective drug does not need to eliminate all prions, but simply needs to slow down the rate of exponential growth. Models predict that the most effective way to achieve this, using a drug with the lowest possible dose, is to find a drug that binds to fibril ends and blocks them from growing any further. Until 2015 all known mammalian prion diseases were considered to be caused by the prion protein, PrP; in 2015 multiple system atrophy was found to be transmissible and was hypothesized to be caused by a new prion, the misfolded form of a protein called alpha-synuclein. The endogenous, properly folded form of the prion protein is denoted PrP^C (for Common or Cellular), whereas the disease-linked, misfolded form is denoted PrP^{Sc} (for Scrapie), after one of the diseases first linked to prions and neurodegeneration. The precise structure of the prion is not known, though they can be formed by combining PrP^C, polyadenylic acid, and lipids in a protein misfolding cyclic amplification (PMCA) reaction. Proteins showing prion-type behavior are also found in some fungi, which has been useful in helping to understand mammalian prions. Fungal prions do not appear to cause disease in their hosts.

Prions cause neurodegenerative disease by aggregating extracellularly within the central nervous system to form plaques known as amyloid, which disrupt the normal tissue structure. This disruption is characterized by "holes" in the tissue with resultant spongy architecture due to the vacuole formation in the neurons. Other histological changes include astrogliosis and the absence of an inflammatory reaction. While the incubation period for prion diseases is relatively long (5 to 20 years), once symptoms appear the disease progresses rapidly, leading to brain damage and death. Neurodegenerative symptoms can include convulsions, dementia, ataxia (balance and coordination dysfunction), and behavioural or personality changes. All known prion diseases are untreatable and fatal. However, a vaccine developed in mice may provide insight into providing a vaccine to resist prion infections in humans. Additionally, in 2006 scientists announced that they had genetically engineered cattle lacking a necessary gene for prion production – thus theoretically making them immune to BSE, building on research indicating that mice lacking normally occurring prion protein are resistant to infection by scrapie prion protein. In 2013, a study revealed that 1 in 2,000 people in the United Kingdom might harbour the infectious prion protein that causes vCJD.Many different mammalian species can be affected by prion diseases, as the prion protein (PrP) is very similar in all mammals. Due to small differences in PrP between different species it is unusual for a prion

disease to transmit from one species to another. The human prion disease variant Creutzfeldt–Jakob disease, however, is thought to be caused by a prion that typically infects cattle, causing bovine spongiform encephalopathy and is transmitted through infected meat. It has been recognized that prion diseases can arise in three different ways: acquired, familial, or sporadic. It is often assumed that the diseased form directly interacts with the normal form to make it rearrange its structure. One idea, the "Protein X" hypothesis, is that an as-yet unidentified cellular protein (Protein X) enables the conversion of PrP^{C} to PrP^{Sc} by bringing a molecule of each of the two together into a complex. Current research suggests that the primary method of infection in animals is through ingestion. It is thought that prions may be deposited in the environment through the remains of dead animals and via urine, saliva, and other body fluids. They may then linger in the soil by binding to clay and other minerals. A University of California research team, led by Nobel Prize winner Stanley Prusiner, has provided evidence for the theory that infection can occur from prions in manure. And, since manure is present in many areas surrounding water reservoirs, as well as used on many crop fields, it raises the possibility of widespread transmission. It was reported in January 2011 that researchers had discovered prions spreading through airborne transmission on aerosol particles, in an animal testing experiment focusing on scrapie infection in laboratory mice. Preliminary evidence supporting the notion that prions can be transmitted through use of urine-derived human menopausal gonadotropin, administered for the treatment of infertility, was published in 2011.

6.4 PYTOPLASMA

Phytoplasmas are obligate bacterial parasites of plant phloem tissue and of the insect vectors that are involved in their plant-to-plant transmission. Phytoplasmas were discovered in 1967 by Japanese scientists who termed them mycoplasma-like organisms. Since their discovery, phytoplasmas have resisted all attempts at in vitro culture in any cell-free medium; routine cultivation in an artificial medium thus remains a major challenge. Although phytoplasmas have recently been reported to be grown in a specific artificial medium, experimental repetition has yet to be reported. Phytoplasmas are characterized by the lack of a cell wall, a pleiomorphic or filamentous shape, a diameter normally less than 1 μm, and a very small genome. Phytoplasmas are pathogens of agriculturally important plants, including coconut, sugarcane, and sandalwood, in which they cause a wide variety of symptoms ranging from mild yellowing to death. Phytoplasmas are most prevalent in tropical and subtropical regions. They are transmitted from plant to plant by vectors (normally sap-sucking insects such as leafhoppers) in which they both survive and replicate. Phytoplasmas are Mollicutes, which are bound by a triple-layered membrane, rather than a cell wall. The phytoplasma cell membranes studied to date usually contain a single immunodominant protein of unknown function that constitutes most of the protein in the membrane. A typical phytoplasma is pleiomorphic or filamentous in shape and is less than 1 μm in diameter. Like other prokaryotes, phytoplasmic DNA is

distributed throughout the cytoplasm, instead of being concentrated in a nucleus. Phytoplasmas can infect and cause various symptoms in more than 700 plant species. One characteristic symptom is abnormal floral organ development including phyllody, (i.e., the production of leaf-like structures in place of flowers) and virescence (i.e., the development of green flowers attributable to a loss of pigment by petal cells). Phytoplasma-harboring flowering plants may nevertheless be sterile. The expression of genes involved in maintaining the apical meristem or in the development of floral organs is altered in the morphologically affected floral organs of phytoplasma-infected plants. Aphytoplasma infection often triggers leaf yellowing, probably due to the presence of phytoplasma cells in phloem, which can affect phloem function and carbohydrate transport, inhibit chlorophyll biosynthesis, and trigger chlorophyll breakdown. These symptoms may be attributable to stress caused by the infection rather than a specific pathogenetic process. Many phytoplasma-infected plants develop a bushy or "witch's broom" appearance due to changes in their normal growth patterns. Most plants exhibit apical dominance but infection can trigger the proliferation of axillary (side) shoots and a reduction in internode size. Such symptoms are actually useful in the commercial production of poinsettias. Infection triggers more axillary shoot production; the poinsettia plants thus produce more than a single flower. Phytoplasmas are spread principally by insects of the families Cicadellidae (leafhoppers), Fulgoridae (planthoppers), and Psyllidae (jumping plant lice), which feed on the phloem of infected plants, ingesting phytoplasmas and transmitting them to the next plant on which they feed. Thus, the host range of phytoplasmas is strongly dependent upon that of the insect vector. Phytoplasmas contain a major antigenic protein constituting most of the cell surface protein. This protein associates with insect microfilament complexes and is believed to control insect-phytoplasma interactions. Phytoplasmas can overwinter in insect vectors or perennial plants. Phytoplasmas can have varying effects on their insect hosts; examples of both reduced and increased fitness have been noted. Phytoplasmas enter the insect body through the stylet, pass through the intestine, and then move to the hemolymphand colonize the salivary glands: the entire process can take up to 3 weeks.Once established in an insect host, phytoplasmas are found in most major organs. The time between ingestion by the insect and attainment of an infectious titer in the salivary glands is termed the latency period. Phytoplasmas can also be spread via dodders (Cuscuta) or by vegetative propagation such as the grafting of infected plant tissue onto a healthy plant. Phytoplasmas move within phloem from a source to a sink, and can pass through sieve tube element. However, as phytoplasmas spread more slowly than solutes, and for other reasons, passive translocation within plants is thought to be unimportant. Before the molecular era, the diagnosis of phytoplasma-caused diseases was difficult because the organisms could not be cultured. Thus, classical diagnostic techniques, including symptom observation were used. Ultrathin sections of phloem tissue from plants with suspected phytoplasma-infections were also studied. The empirical use of antibiotics such as tetracycline was additionally employed. Molecular diagnostic techniques for phytoplasma detection began to emerge in the 1980s and included enzyme-linked

immunosorbent assay (ELISA)-based methods. In the early 1990s, polymerase chain reaction (PCR)-based techniques were developed: these are far more sensitive than ELISAs, and restriction fragment length polymorphism (RFLP) analysis allowed the accurate identification of various phytoplasma strains and species. More recent techniques allow infection levels to be assessed. Both quantitative PCR and bioimaging can effectively quantify phytoplasma titers within plant. In addition, loop-mediated isothermal amplification (a sensitive, simple, and rapid diagnostic method) is now available as a commercial kit allowing all known phytoplasma species to be detected in about 1 h, including the DNA extraction step. Phytoplasmas are normally controlled by the breeding and planting of disease-resistant crop varieties (perhaps the most economically viable option) and by the control of insect vectors. Tissue culture can be used to produce healthy clones of phytoplasma-infected plants. Cryotherapy (i.e., the freezing of plant samples in liquid nitrogen) prior to tissue culture increases the probability of producing healthy plants in this manner. Plantibodies targeting phytoplasmas have also been developed. Tetracyclines are bacteriostatic to phytoplasmas. However, disease symptoms reappear in the absence of continuous antibiotic application. Thus, tetracycline is not a viable agricultural control agent, but it is used to protect ornamental coconut trees. The genomes of four phytoplasmas have been sequenced: "onion yellows", "aster yellows witches' broom" (*Candidatus* [Ca] p*hytoplasmaasteris*), *Ca. phytoplasmaaustraliense*, and *Ca. Phytoplasma mali*. Phytoplasmas have very small genomes, with extremely small amount of G and C nucleotides (sometimes as little as 23%, which is thought to be the lower threshold for a viable genome).In fact, the Bermuda grass white-leaf phytoplasma has a genome size of only 530 kb, one of the smallest known genomes of all living organisms. The larger phytoplasma genomes are around 1350 kb in size. The small genome size of phytoplasma is attributable to reductive evolution from *Bacillus/Clostridium* ancestors. Phytoplasmas have lost ≥75% of their original genes, and can thus no longer survive outside of insects or plant phloem. Some phytoplasmas contain extrachromosomal DNA such as plasmids. Despite their small genomes, many predicted phytoplasma genes are present in multiple copies. Phytoplasmas lack many genes encoding standard metabolic functions and have no functioning homologous recombination pathway, but they do have a sec transport pathway. Many phytoplasmas contain two rRNA operons. Unlike other Mollicutes, the triplet code of UGA is used as a stop codon in phytoplasmas. Phytoplasma genomes contain large numbers of transposons and insertion sequences and also contain a unique family of repetitive extragenic palindromes termed PhREPS for which no role is known. However, it is theorized that the stem-loop structures in PhREPS play a role in transcription termination or genome stability. Phytoplasmas belong to the monotypic order *Acholeplasmatales*. In 1992, the Subcommittee on the Taxonomy of Mollicutes proposed the use of "Phytoplasma" rather than "mycoplasma-like organisms" "for reference to the phytopathogenicmollicutes". In 2004, the generic name phytoplasma was adopted and is currently of *Candidatus* (Ca.) status (used for bacteria that cannot be cultured). Phytoplasma taxonomy is complicated because the organisms cannot be cultured;

methods normally used to classify prokaryotes are thus not available. Phytoplasma taxonomic groups are based on differences in fragment sizes produced by restriction digests of 16S ribosomal RNA gene sequences (RFLPs) or by comparisons of DNA sequences from 16s/23s spacer regions. The actual number of taxonomic groups remains unclear; recent work on computer-simulated restriction digests of the 16Sr gene suggested up to 28 groups, whereas others have proposed fewer groups, but more subgroups. Each group includes at least one *Ca. phytoplasma* species, characterized by distinctive biological, phytopathological, and genetic properties.

6.5 MYCOPLASMA

Mycoplasma is a mollicute genus of bacteria that lack a cell wall around their cell membranes. This characteristic makes them naturally resistant to antibiotics that target cell wall synthesis (like the beta-lactam antibiotics). They can be parasitic or saprotrophic. Several species are pathogenic in humans, including *M. pneumoniae*, which is an important cause of "walking" pneumonia and other respiratory disorders, and *M. genitalium*, which is believed to be involved in pelvic inflammatory diseases. *Mycoplasma* species are the smallest bacterial cells yet discovered, can survive without oxygen, and come in various shapes. For example, *M. genitalium* is flask-shaped (about 300 x 600 nm), while *M. pneumoniae* is more elongated (about 100 x 1000 nm). Hundreds of *Mycoplasma* species infect animals. The term *Mycoplasma*, was first used by Albert Bernhard Frank in 1889 to describe an altered state of plant cell cytoplasm resulting from infiltration by fungus-like microorganisms. Julian Nowak later proposed the genus name *Mycoplasma* for certain filamentous microorganisms imagined to have both cellular and a cellular stages in their lifecycles, which could explain how they were visible with a microscope, but passed through filters impermeable to other bacteria. Later, the name for *Mycoplasma* was pleuropneumonia-like organisms (PPLO), broadly referring to organisms similar in colonial morphology and filterability to the causative agent (a *Mycoplasma*) of contagious bovine pleuropneumonia. Other species of *Mycoplasma* other than those listed below have been recovered from humans, but are assumed to have been contracted from animals. These use humans as the primary host:

- *M. amphoriforme*
- *M. buccale*
- *M. faucium*
- *M. fermentans*
- *M. genitalium*
- *M. hominis*

Mycoplasma species have been isolated from women with bacterial vaginosis. *M. genitalium* is found in women with pelvic inflammatory disease. In addition, infection is associated with increased risk of cervicitis, preterm birth and spontaneous abortion, and infertility. *Mycoplasma genitalium* has developed resistance to some antibiotics. *Mycoplasmae* are

associated with fetal respiratory distress syndrome, bronchopulmonary dysplasia, and intraventricular hemorrhage in preterm infants. Over 100 species have been included in the genus *Mycoplasma*. Microbes of the class Mollicutes, to which *Mycoplasma* belongs, are parasites or commensals of humans, animals, and plants. The genus *Mycoplasma* uses vertebrate and arthropod hosts. Dietary nitrogen availability has been shown to alter codon bias and genome evolution in *Mycoplasma* and *Phytoplasma*. Mycoplasmal bacteria are also known as mollicutes. They are the simplest and the smallest free-living prokaryotes. Mycoplasmal bacteria haven been found in the pleural cavities of cattle suffering from pleuropneumonia. These organisms are often called MLO (mycoplasma-like organisms) or PPLO (pleuropneumonia-like organisms).

Important characteristics of mycoplasmal bacteria

1. Cell wall is absent and plasma membrane forms the outer boundary of the cell.
2. Due to the absence of cell wall these organisms can change their shape and are pleomorphic.
3. Lack of nucleus and other membrane-bound organelles.
4. Genetic material is a single DNA duplex and is naked.
5. Ribosomes are 70S type. Possess a replicating disc at one end which assist replication process and also the separation of the genetic materials.
6. Heterotrophic nutrition. Some live as saprophytes but the majority are parasites of plants and animals. The parasitic nature is due to the inability of mycoplasmal bacteria to synthesise the required growth factor.

Cell morphology

Due to the lack of a rigid cell wall, Mycoplasmataceae can contort into a broad range of shapes, from round to oblong. They therefore cannot be classified as rods, cocci or spirochetes. Mycoplasma species are often found in research laboratories as contaminants in cell culture. Mycoplasmal cell culture contamination occurs due to contamination from individuals or contaminated cell culture medium ingredients. *Mycoplasma* cells are physically small – less than 1 µm, so are difficult to detect with a conventional microscope.

Mycoplasmae may induce cellular changes, including chromosome aberrations, changes in metabolism and cell growth. Severe Mycoplasma infections may destroy a cell line. Detection techniques include DNA probe, enzyme immunoassays, PCR, plating on sensitive agar and staining with a DNA stain including DAPI or Hoechst. An estimated 11 to 15% of U.S. laboratory cell cultures are contaminated with *Mycoplasma*. A Corning study showed that half of U.S. scientists did not test for *Mycoplasma* contamination in their cell cultures. The study also stated that, in former Czechoslovakia, 100% of cell cultures that were not routinely tested were contaminated while only 2% of those routinely tested were contaminated (study page 6). Since the U.S. contamination rate was based on a study of companies that routinely checked for *Mycoplasma*, the actual contamination rate may be higher. European contamination rates are higher and that of other countries are higher still (up to 80% of Japanese cell cultures). About 1% of published Gene Expression Omnibus data may

have been compromised. Several antibiotic-containing formulations of antimycoplasmal reagents have been developed over the years. A chemically synthesized genome of a mycoplasmal cell based entirely on synthetic DNA which can self-replicate has been referred to as *Mycoplasma laboratorium*. The P1 antigen is the primary virulence factor of *Mycoplasma*. P1 is a membrane associated protein that allows adhesion to epithelial cells. The P1 receptor is also expressed on erythrocytes which can lead to autoantibody agglutination from mycobacteria infection. Several *Mycoplasma* species can cause disease, including *M. pneumoniae*, which is an important cause of atypical pneumonia (formerly known as "walking pneumonia"), and *M. genitalium*, which has been associated with pelvic inflammatory diseases. *Mycoplasma* infections in humans are associated with skin eruptions in 17% of cases. *Mycoplasma* and *Ureaplasma* species are not part of the normal vaginal flora. Some *Mycoplasma* species are spread through sexual contact. Some mycoplasmae have a negative effect on fertility. *M. hominis* causes male sterility/Genitals inflammation in humans. Low birth-weight, preterm infants are susceptible to *Mycoplasma* infections. Several species of *Mycoplasma* are frequently detected in different types of cancer cells. These species are:

- *M. fermentans*
- *M. genitalium*
- *M. hominis*
- *M. hyorhinis*
- *M. penetrans*
- *U. urealyticum*

The presence of *Mycoplasma* was first reported in samples of cancer tissue in the 1960s.Since then, several studies tried to find and prove the connection between *Mycoplasma* and cancer, as well as how the bacterium might be involved in the formation of cancer. Several studies have shown that cells that are chronically infected with the bacteria go through a multistep transformation. The changes caused by chronic mycoplasmal infections occur gradually and are both morphological and genetic. The first visual sign of infection is when the cells gradually shift from their normal form to sickle-shaped. They also become hyperchromatic due to an increase of DNA in the nucleus of the cells. In later stages, the cells lose the need for a solid support to grow and proliferate, as well as the normal contact-dependent inhibition cells. Cells infected with *Mycoplasma* for an extended period of time show significant chromosomal abnormalities. These include the addition of chromosomes, the loss of entire chromosomes, partial loss of chromosomes, and chromosomal translocation. All of these genetic abnormalities may contribute to the process of malignant transformation. Chromosomal translocation and extra chromosomes help create abnormally high activity of certain proto-oncogenes, which caused by these genetic abnormalities and include those encoding c-myc, HRAS, and vav. The activity of proto-oncogenes is not the only cellular function that is affected; tumour suppressor genes are affected by the chromosomal changes induced by mycoplasma, as well. Partial or complete loss of chromosomes causes the

loss of important genes involved in the regulation of cell proliferation. Two genes whose activities are markedly decreased during chronic infections with mycoplasma are the Rb and the p53 tumour suppressor genes. Another possible mechanism of carcinogenesis is RAC1 activation by a small GTPase-like protein fragment of Mycoplasma.A major feature that differentiates mycoplasmas from other carcinogenic pathogens is that the mycoplasmas do not cause the cellular changes by insertion of their own genetic material into the host cell. The exact mechanism by which the bacterium causes the changes is not yet known. The malignant transformation induced by mycoplasmae is also different from that caused by other pathogens in that the process is reversible. The state of reversal is, however, only possible up to a certain point during the infection. The window of time when reversibility is possible varies greatly; it depends primarily on the *Mycoplasma* involved. In the case of *M. fermentans*, the transformation is reversible until around week 11 of infection and starts to become irreversible between weeks 11 and 18.If the bacteria are killed using antibiotics (i.e. Ciprofloxacinor, Clarithromycin) before the irreversible stage, the infected cells should return to normal. Epidemiologic, genetic, and molecular studies suggest infection and inflammation initiate certain cancers, including those of the prostate. *M. genitalium* and *M. hyorhinis* induce malignant phenotype in benign human prostate cells (BPH-1) that were not tumorigenic after 19 weeks of exposure. In a study to understand the effects of *Mycoplasma* contamination on the quality of cultured human colon cancer cells, a positive correlation was found between the number of *M. hyorhinis* cells present in the sample and the percentage of CD133-positive cells (a glycoprotein with an unknown function). Gastric cancer: Strong evidence indicates the infection of *M. hyorhinis* contributes to the development of cancer within the stomach and increases the likelihood of malignant cancer cell development. Lung cancer: Studies on lung cancer have supported the belief that more than a coincidental positive correlation exists between the appearance of *Mycoplasma* strains in patients and the infection with tumorigenesis. Prostate cancer: p37, a protein encoded for by *M. hyorhinis*, has been found to promote the invasiveness of prostate cancer cells. The protein also causes the growth, morphology, and the gene expression of the cells to change, causing them to become a more aggressive phenotype. Renal cancer: Patients with renal cell carcinoma (RCC) exhibited a significantly high amount of *Mycoplasma sp.* compared with the healthy control group. This suggests *Mycoplasma* may play a role in the development of RCC.

6.6 CHECK YOUR PROGRESS

1. What is virion?
2. Define phytoplasma?

6.7 LET US SUM UP

A virion is an entire virus particle consisting of an outer protein shell called a capsid and an inner core of nucleic acid (either ribonucleic or

deoxyribonucleic acid—RNA or DNA). The core confers infectivity, and the capsid provides specificity to the virus. The nucleic acid is densely coiled within. *Phytoplasmas* are wall-less parasitic bacteria living exclusively in plant phloem as consequence of transmission by sap-sucking insect vectors they have been associated with several hundred plant diseases. *Mycoplasma* is a mollicute genus of bacteria that lack a cell wall around their cell membranes. This characteristic makes them naturally resistant to antibiotics that target cell wall synthesis (like the beta-lactam antibiotics). They can be parasitic or saprotrophic.

6.8 UNIT – END EXERCISES

1. What are the symptoms of phytoplasma?
2. Write in detail about mycoplasma?

6.9 ANSWERS TO CHECK YOUR PROGRESS

1. One characteristic symptom is abnormal floral organ development including phyllody, (i.e., the production of leaf-like structures in place of flowers) and virescence (i.e., the development of green flowers attributable to a loss of pigment by petal cells). Phytoplasma-harboring flowering plants may nevertheless be sterile. The expression of genes involved in maintaining the apical meristem or in the development of floral organs is altered in the morphologically affected floral organs of phytoplasma-infected plants. A phytoplasma infection often triggers leaf yellowing, probably due to the presence of phytoplasma cells in phloem, which can affect phloem function and carbohydrate transport, inhibit chlorophyll biosynthesis, and trigger chlorophyll breakdown. These symptoms may be attributable to stress caused by the infection rather than a specific pathogenetic process. Many phytoplasma-infected plants develop a bushy or "witch's broom" appearance due to changes in their normal growth patterns. Most plants exhibit apical dominance but infection can trigger the proliferation of auxiliary (side) shoots and a reduction in internode size. Symptoms are actually useful in the commercial production of poinsettias. Infection triggers more auxiliary shoot production; the poinsettia plants thus produce more than a single flower.
2. Mycoplasmae may induce cellular changes, including chromosome aberrations, changes in metabolism and cell growth. Severe Mycoplasma infections may destroy a cell line. Detection techniques include DNA probe, enzyme immunoassays, PCR, plating on sensitive agar and staining with a DNA stain including DAPI or Hoechst. An estimated 11 to 15% of U.S. laboratory cell cultures are contaminated with mycoplasma. A Corning study showed that half of U.S. scientists did not test for Mycoplasma contamination in their cell cultures. The study also stated that, in former Czechoslovakia, 100% of cell cultures that were not routinely tested were contaminated while only 2% of those routinely tested were contaminated. Since the U.S. contamination rate was based on a study of companies that routinely checked for Mycoplasma, the actual contamination rate may be higher. European contamination rates are higher and that of other countries are higher still (up to 80% of Japanese cell

cultures). About 1% of published Gene Expression Omnibus data may have been compromised. Several antibiotic-containing formulations of antimycoplasmal reagents have been developed over the years. A chemically synthesized genome of a mycoplasmal cell based entirely on synthetic DNA which can self-replicate has been referred to as *Mycoplasma laboratorium*. The P1 antigen is the primary virulence factor of mycoplasma. P1 is a membrane associated protein that allows adhesion to epithelial cells. The P1 receptor is also expressed on erythrocytes which can lead to autoantibody agglutination from mycobacteria infection. Several *Mycoplasma* species can cause disease, including *M. pneumoniae*, which is an important cause of atypical pneumonia (formerly known as "walking pneumonia"), and *M. genitalium*, which has been associated with pelvic inflammatory diseases. *Mycoplasma* infections in humans are associated with skin eruptions in 17% of cases. *Mycoplasma* and *Ureaplasma* species are not part of the normal vaginal flora. Some *Mycoplasma* species are spread through sexual contact. Some mycoplasmae have a negative effect on fertility. *M. hominis* causes male sterility/Genitals inflammation in humans. Low birth-weight, preterm infants are susceptible to *Mycoplasma* infections. Several species of *Mycoplasma* are frequently detected in different types of cancer cells.

6.10 SUGGESTED READINGS

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UNIT- VII MICROBIAL PRODUCTS

7.1 INTRODUCTION

The term industrial microbiology refers to the use of microorganisms for industrial purposes. Such things as anticoagulants, antidepressants, vasodilators, herbicides, insecticides, plant hormones, enzymes, and vitamins have been isolated from microorganisms or produced in large quantities by genetically engineering the organisms with foreign genes. In commercial industrial plants, microorganisms are widely used to produce numerous organic materials that have far-reaching value and application. **Enzymes,** among the **enzymes** industrially produced by bacteria are **amylases**, which break down starches to smaller carbohydrates for commercial use. Amylases are also used in brewing, baking, and textile production. Bacteria have been used to produce **proteases**, which break down proteins and are used for tenderizing meats, preparing leathers, and making detergents and cheese. The food, petroleum, cosmetic and pharmaceutical industries use microorganisms to manufacture **polysaccharides.** For example, the bacterium *Xanthomonas campestris*, produces a polysaccharide called **xanthan**, which is used to stabilize and thicken foods and as a base for cosmetics. It is also a binding agent in many pharmaceuticals and is used in textile printing and dyeing. Another polysaccharide of microbial origin is **dextran.** The bacterium *Leuconostoc mesenteroides* produces this polysaccharide when it grows on sucrose. Dextran is used to extend blood plasma. **Nutrients,** Amino acids, nucleotides, vitamins, and organic acids are produced by the ton by microorganisms. Various types of research and health laboratories use these products, and health-food stores sell them as **nutritional supplements.** For example, the **lysine** prescribed by some doctors to treat herpes simplex infections is a product of the bacterium *Corynebacterium glutamicum*. **Vitamin B_{12}** (cyanocobalamine) and **vitamin B_2** (riboflavin) are produced by a bacterium and a mold, respectively. **Chemotherapeutic agents,** another valuable use of microorganisms in industry is in the production of **chemotherapeutic agents.** Almost two billion dollars worth of drugs are produced in the United States, mainly by the use of microorganisms. Antibiotics are

produced by fungi such as *Penicllium* and *Cephalosporium* and by species of the bacterium *Streptomyces*. Many of these drugs are natural, but several are synthetic or semisynthetic drugs that begin with the naturally occurring molecule, which is then modified.

7.2 OBJECTIVES

- The commercial possibilities of microbes are seemingly endless. However, intuition alone tells us that not all of these technological possibilities are likely to become commercial realities.
- This report is an update of an earlier BCC Research report published in 2011.
- Its goal is to survey microbial applications in a wide range of fields based on the most recent data, identify the applications that appear to have significant commercial potential in the near- to mid-term and to develop quantitative estimates of their current and/or future sales.
- The updated report's specific objectives support this broad goal.
- These objectives include identifying microbial technologies and applications that have the greatest commercial potential in the 2013 through 2018 time frame, identifying market drivers, evaluating obstacles to their successful commercialization, and projecting future sales of each application.

7.3 MICROBIAL PRODUCTS

Microbial products are products derived from various microscopic organisms. Microbial products may consist of the organisms themselves and/or the metabolites they produce.

Antibodies, enzymes, anticoagulants, antidepressants, vasodilators, herbicides, insecticides, plant hormones, and vitamins have been isolated from microorganisms or produced in large quantities by genetically engineering the organisms with foreign genes. Microorganisms are widely used to produce numerous organic materials that have far-reaching value and application.

7.4 ANTIBIOTICS

An antibiotic is a type of antimicrobial substance active against bacteria and is the most important type of antibacterial agent for fighting bacterial infections. Antibiotic medications are widely used in the treatment and prevention of such infections. They may either kill or inhibit the growth of bacteria. A limited number of antibiotics also possess antiprotozoal activity. Antibiotics are not effective against viruses such as the common cold or influenza; drugs which inhibit viruses are termed antiviral drugs or antivirals rather than antibiotics. It is broadly used to refer to any substance used against microbes, but in the usual medical usage, antibiotics (such as penicillin) are those produced naturally (by one microorganism fighting another), whereas nonantibiotic antibacterials (such as sulfonamides and antiseptics) are fully synthetic. However, both

classes have the same goal of killing or preventing the growth of microorganisms, and both are included in antimicrobial chemotherapy. "Antibacterials" include antiseptic drugs, antibacterial soaps, and chemical disinfectants, whereas antibiotics are an important class of antibacterials used more specifically in medicine and sometimes in livestock feed. Antibiotics have been used since ancient times. Many civilizations used topical application of mouldy bread, with many references to its beneficial effects arising from ancient Egypt, China, Serbia, Greece and Rome. The first person to directly document the use of moulds to treat infections was John Parkinson (1567–1650). Antibiotics revolutionized medicine in the 20th century. Alexander Fleming (1881–1955) discovered modern day penicillin in 1928. After realizing the great potential there was in penicillin, Fleming pursued the challenge of how to market it and translate it to commercial use. With help from other biochemists, penicillin was finally available for widespread use. This was significantly beneficial during wartime. Unfortunately, it didn't take long for resistance to begin. Effectiveness and easy access have also led to their overuse and some bacteria have developed resistance. This has led to widespread problems, and the World Health Organization has classified antimicrobial resistance as a "serious threat [that] is no longer a prediction for the future, it is happening right now in every region of the world and has the potential to affect anyone, of any age, in any country.

Medical uses

Antibiotics are used to treat or prevent bacterial infections, and sometimes protozoan infections. (Metronidazole is effective against a number of parasitic diseases). When an infection is suspected of being responsible for an illness but the responsible pathogen has not been identified, an empiric therapy is adopted. This involves the administration of a broad-spectrum antibiotic based on the signs and symptoms presented and are initiated pending laboratory results that can take several days. When the responsible pathogenic microorganism is already known or has been identified, definitive therapy can be started. This will usually involve the use of a narrow-spectrum antibiotic. The choice of antibiotic given will also be based on its cost. Identification is critically important as it can reduce the cost and toxicity of the antibiotic therapy and also reduce the possibility of the emergence of antimicrobial resistance. To avoid surgery, antibiotics may be given for non-complicated acute appendicitis. Antibiotics may be given as a preventive measure and this is usually limited to at-risk populations such as those with a weakened immune system (particularly in HIV cases to prevent pneumonia), those taking immunosuppressive drugs, cancer patients, and those having surgery. Their use in surgical procedures is to help prevent infection of incisions. They have an important role in dental antibiotic prophylaxis where their use may prevent bacteremia and consequent infective endocarditis. Antibiotics are also used to prevent infection in cases of neutropenia particularly cancer-related; there are many different routes of administration for antibiotic treatment. Antibiotics are usually taken by mouth. In more severe cases, particularly deep-seated systemic infections, antibiotics can be given intravenously or by injection. Where the site of infection is easily accessed, antibiotics may be given topically in the form of eye drops onto the

conjunctiva for conjunctivitis or ear drops for ear infections and acute cases of swimmer's ear. Topical use is also one of the treatment options for some skin conditions including acne and cellulitis. Advantages of topical application include achieving high and sustained concentration of antibiotic at the site of infection; reducing the potential for systemic absorption and toxicity, and total volumes of antibiotic required are reduced, thereby also reducing the risk of antibiotic misuse. Topical antibiotics applied over certain types of surgical wounds have been reported to reduce the risk of surgical site infections. However, there are certain general causes for concern with topical administration of antibiotics. Some systemic absorption of the antibiotic may occur; the quantity of antibiotic applied is difficult to accurately dose, and there is also the possibility of local hypersensitivity reactions or contact dermatitis occurring. Antibiotic consumption varies widely between countries. The 'WHO report on surveillance of antibiotic consumption' published in 2018 analysed 2015 data from 65 countries. As measured in defined daily doses per 1,000 inhabitants per day. Mongolia had the highest consumption with a rate of 64.4. Burundi had the lowest at 4.4. Amoxicillin and Amoxicillin/clavulanic acid were the most frequently consumed. Antibiotics are screened for any negative effects before their approval for clinical use, and are usually considered safe and well tolerated. However, some antibiotics have been associated with a wide extent of adverse side effects ranging from mild to very severe depending on the type of antibiotic used, the microbes targeted, and the individual patient. Side effects may reflect the pharmacological or toxicological properties of the antibiotic or may involve hypersensitivity or allergic reactions. Adverse effects range from fever and nausea to major allergic reactions, including photodermatitis and anaphylaxis. Safety profiles of newer drugs are often not as well established as for those that have a long history of use. Common side-effects include diarrhea, resulting from disruption of the species composition in the intestinal flora, resulting, for example, in overgrowth of pathogenic bacteria, such as *Clostridium difficile*. Antibacterials can also affect the vaginal flora, and may lead to overgrowth of yeast species of the genus *Candida* in the vulvo-vaginal area. Additional side effects can result from interaction with other drugs, such as the possibility of tendon damage from the administration of a quinolone antibiotic with a systemic corticosteroid. Exposure to antibiotics early in life is associated with increased body mass in humans and mouse models. Early life is a critical period for the establishment of the intestinal microbiota and for metabolic development. Mice exposed to sub therapeutic antibiotic treatment (STAT)– with either penicillin, vancomycin, or chlortetracycline had altered composition of the gut microbiota as well as its metabolic capabilities. One study has reported that mice given low-dose penicillin (1 μg/g body weight) around birth and throughout the weaning process had an increased body mass and fat mass, accelerated growth, and increased hepatic expression of genes involved in adipogenesis, compared to control mice.In addition, penicillin in combination with a high-fat diet increased fasting insulin levels in mice. However, it is unclear whether or not antibiotics cause obesity in humans. Studies have found a correlation between early exposure of antibiotics (<6

months) and increased body mass (at 10 and 20 months). Another study found that the type of antibiotic exposure was also significant with the highest risk of being overweight in those given macrolides compared to penicillin and cephalosporin. Therefore, there is correlation between antibiotic exposure in early life and obesity in humans, but whether or not there is a causal relationship remains unclear. Although there is a correlation between antibiotic use in early life and obesity, the effect of antibiotics on obesity in humans needs to be weighed against the beneficial effects of clinically indicated treatment with antibiotics in infancy.

7.5 ENZYMES

Enzyme, a substance that acts as a catalyst in living organisms, regulating the rate at which chemical reactions proceed without itself being altered in the process.

The biological processes that occur within all living organisms are chemical reactions, and most are regulated by enzymes. Without enzymes, many of these reactions would not take place at a perceptible rate. Enzymes catalyze all aspects of cell metabolism. This includes the digestion of food, in which large nutrient molecules (such as proteins, carbohydrates, and fats) are broken down into smaller molecules; the conservation and transformation of chemical energy; and the construction of cellular macromolecules from smaller precursors. Many inherited human diseases, such as albinism and phenylketonuria, result from a deficiency of a particular enzyme. Enzymes also have valuable industrial and medical applications. The fermenting of wine, leavening of bread, curdling of cheese, and brewing of beer have been practiced from earliest times, but not until the 19th century were these reactions understood to be the result of the catalytic activity of enzymes. Since then, enzymes have assumed an increasing importance in industrial processes that involve organic chemical reactions. The uses of enzymes in medicine include killing disease-causing microorganisms, promoting wound healing, and diagnosing certain diseases. All enzymes were once thought to be proteins, but since the 1980s the catalytic ability of certain nucleic acids, called ribozymes (or catalytic RNAs), has been demonstrated, refuting this axiom. Because so little is yet known about the enzymatic functioning of RNA, this discussion will focus primarily on protein enzymes. A large protein enzyme molecule is composed of one or more amino acid chains called polypeptide chains. The amino acid sequence determines the characteristic folding patterns of the protein's structure, which is essential to enzyme specificity. If the enzyme is subjected to changes, such as fluctuations in temperature or pH, the protein structure may lose its integrity (denature) and its enzymatic ability. Denaturation is sometimes, but not always, reversible.

Bound to some enzymes is an additional chemical component called a cofactor, which is a direct participant in the catalytic event and thus is required for enzymatic activity A cofactor may be either a coenzyme—an organic molecule, such as a vitamin—or an inorganic metal ion; some enzymes require both. A cofactor may be either tightly or loosely bound to the enzyme. If tightly connected, the cofactor is referred

to as a prosthetic group. An enzyme will interact with only one type of substance or group of substances, called the substrate, to catalyze a certain kind of reaction. Because of this specificity, enzymes often have been named by adding the suffix "-ase" to the substrate's name (as in urease, which catalyzes the breakdown of urea). Not all enzymes have been named in this manner, however, and to ease the confusion surrounding enzyme nomenclature, a classification system has been developed based on the type of reaction the enzyme catalyzes. There are six principal categories and their reactions:

i. Oxidoreductases, which are involved in electron transfer;
ii. Transferases, which transfer a chemical group from one substance to another;
iii. Hydrolases, which cleave the substrate by uptake of a water molecule (hydrolysis);
iv. Lyases, which form double bonds by adding or removing a chemical group;
v. Isomerases, which transfer a group within a molecule to form an isomer; and
vi. Ligases, or synthetases, which couple the formation of various chemical bonds to the breakdown of a pyrophosphate bond in adenosine triphosphate or a similar nucleotide.

In most chemical reactions, an energy barrier exists that must be overcome for the reaction to occur. This barrier prevents complex molecules such as proteins and nucleic acids from spontaneously degrading, and so is necessary for the preservation of life. When metabolic changes are required in a cell, however, certain of these complex molecules must be broken down, and this energy barrier must be surmounted. Heat could provide the additional needed energy (called activation energy), but the rise in temperature would kill the cell. The alternative is to lower the activation energy level through the use of a catalyst. This is the role that enzymes play. They react with the substrate to form an intermediate complex—a "transition state"—that requires less energy for the reaction to proceed. The unstable intermediate compound quickly breaks down to form reaction products, and the unchanged enzyme is free to react with other substrate molecules. Only a certain region of the enzyme, called the active site, binds to the substrate. The active site is a groove or pocket formed by the folding pattern of the protein. This three-dimensional structure (Fig. 7.1), together with the chemical and electrical properties of the amino acids and cofactors within the active site, permits only a particular substrate to bind to the site, thus determining the enzyme's specificity.

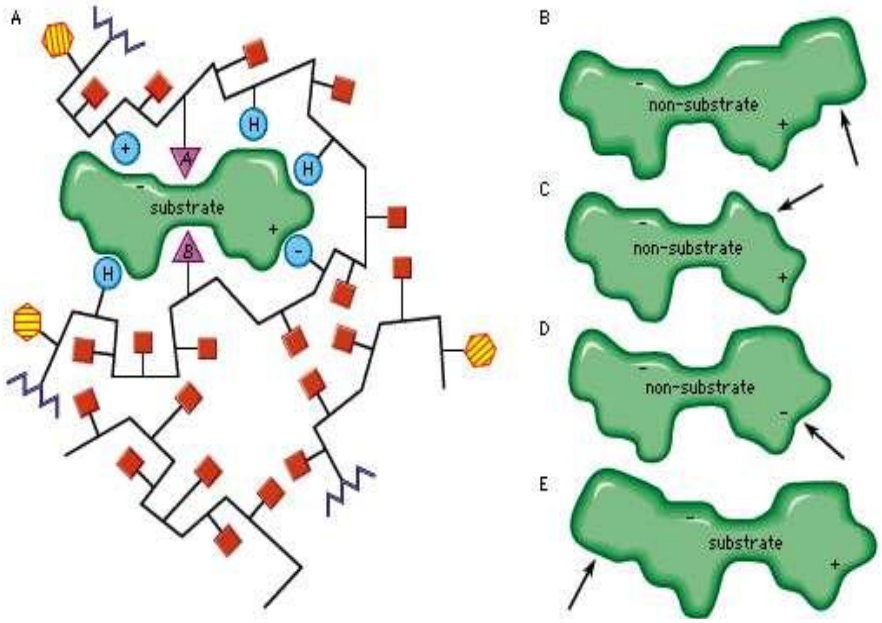

Figure 7.1 Enzyme synthesis and Activity.

Enzyme synthesis and activity also are influenced by genetic control and distribution in a cell. Some enzymes are not produced by certain cells, and others are formed only when required. Enzymes are not always found uniformly within a cell; often they are compartmentalized in the nucleus, on the cell membrane, or in subcellular structures. The rates of enzyme synthesis and activity are further influenced by hormones, neurosecretions, and other chemicals that affect the cell's internal environment.

Factors Affecting Enzyme Activity

Because enzymes are not consumed in the reactions they catalyze and can be used over and over again, only a very small quantity of an enzyme is needed to catalyze a reaction. A typical enzyme molecule can convert 1,000 substrate molecules per second. The rate of an enzymatic reaction increases with increased substrate concentration, reaching maximum velocity when all active sites of the enzyme molecules are engaged. The enzyme is then said to be saturated, the rate of the reaction being determined by the speed at which the active sites can convert substrate to product.

Enzyme activity can be inhibited in various ways. Competitive inhibition occurs when molecules very similar to the substrate molecules bind to the active site and prevent binding of the actual substrate. Penicillin, for example, is a competitive inhibitor that blocks the active site of an enzyme that many bacteria use to construct their cell walls. Noncompetitive inhibition occurs when an inhibitor binds to the enzyme at a location other than the active site. In some cases of noncompetitive inhibition, the inhibitor is thought to bind to the enzyme in such a way as to physically block the normal active site. In other instances, the binding of the inhibitor is believed to change the shape of the enzyme molecule, thereby deforming its active site and preventing it from reacting with its substrate. This latter type of noncompetitive inhibition is called allosteric inhibition; the place where the inhibitor binds to the enzyme is called the allosteric site. Frequently, an end-product of a metabolic pathway serves as

an allosteric inhibitor on an earlier enzyme of the pathway. Allosteric control can involve stimulation of enzyme action as well as inhibition. An activator molecule can be bound to an allosteric site and induce a reaction at the active site by changing its shape to fit a substrate that could not induce the change by itself. Common activators include hormones and the products of earlier enzymatic reactions. Allosteric stimulation and inhibition allow production of energy and materials by the cell when they are needed and inhibit production when the supply is adequate.

7.6 HUMAN DISEASES CAUSED BY BACTERIA

Bacterial disease, any of a variety of illnesses caused by bacteria. Until the mid-20th century, bacterial pneumonia was probably the leading cause of death among the elderly. Improved sanitation, vaccines, and antibiotics have all decreased the mortality rates from bacterial infections, though antibiotic-resistant strains have caused resurgence in some illnesses. In the early 21st century, tuberculosis, which is caused by *Mycobacterium tuberculosis*—several strains of which had developed resistance to one or more drugs widely used to treat the infection—was among the deadliest infectious diseases worldwide. Bacteria cause disease by secreting or excreting toxins (as in botulism), by producing toxins internally, which are released when the bacteria disintegrate (as in typhoid), or by inducing sensitivity to their antigenic properties (as in tuberculosis). Other serious bacterial diseases include cholera, diphtheria, bacterial meningitis, tetanus, Lyme disease, gonorrhea, and syphilis.

Pneumonia inflammation and consolidation of the lung tissue as a result of infection, inhalation of foreign particles, or irradiation. Many organisms, including viruses and fungi, can cause pneumonia, but the most common causes are bacteria, in particular species of *Streptococcus* and *Mycoplasma*. Although viral pneumonia does occur, viruses more commonly play a part in weakening the lung, thus inviting secondary pneumonia caused by bacteria. Fungal pneumonia can develop very rapidly and may be fatal, but it usually occurs in hospitalized persons who, because of impaired immunity, have reduced resistance to infection. Contaminated dusts, when inhaled by previously healthy individuals, can sometimes cause fungal lung diseases. Pneumonia can also occur as a hypersensitivity, or allergic response, to agents such as mold, humidifiers, and animal excreta or to chemical or physical injury (e.g., smoke inhalation). Streptococcal pneumonia, caused by *Streptococcus pneumoniae*, is the single most common form of pneumonia, especially in hospitalized patients. The bacteria may live in the bodies of healthy persons and cause disease only after resistance has been lowered by other illness or infection. Viral infections such as the common cold promote streptococcal pneumonia by causing excessive secretion of fluids in the respiratory tract. These fluids provide an environment in which the bacteria flourish. Patients with bacterial pneumonia typically experience a sudden onset of high fever with chills, cough, chest pain, and difficulty in breathing. As the disease progresses, coughing becomes the major symptom. Sputum discharge may contain flecks of blood. Any chest pains

result from the tenderness of the trachea (windpipe) and muscles from severe coughing. Diagnosis usually can be established by taking a culture of the organism from the patient's sputum and by chest X-ray examination. Treatment is with specific antibiotics and supportive care, and recovery generally occurs in a few weeks. In some cases, however, the illness may become very severe, and it is sometimes fatal, particularly in elderly people and young children. Death from streptococcal pneumonia is caused by inflammation and significant and extensive bleeding in the lungs that result in the eventual cessation of breathing. Streptococcal bacteria release a toxin called pneumolysin that damages the blood vessels in the lungs, causing bleeding into the air spaces. Antibiotics may exacerbate lung damage because they are designed to kill the bacteria by breaking them open, which leads to the further release of pneumolysin. Research into the development of aerosol agents that stimulate blood clotting and that can be inhaled into the lungs and possibly be used in conjunction with traditional therapies for streptococcal pneumonia is ongoing.

Mycoplasmal pneumonia, caused by *Mycoplasma pneumoniae*, an extremely small organism, usually affects children and young adults; few cases beyond the age of 50 are seen. Most outbreaks of this disease are confined to families, small neighbourhoods, or institutions, although epidemics can occur. *M. pneumoniae* grows on the mucous membrane that lines the surfaces of internal lung structures; it does not invade the deeper tissues—muscle fibres, elastic fibres, or nerves. The bacteria can produce an oxidizing agent that might be responsible for some cell damage. Usually the organism does not invade the membrane that surrounds the lungs, but it does sometimes inflame the bronchi and alveoli. Another bacterium, *Klebsiella pneumoniae*, although it has little ability to infect the lungs of healthy persons, produces a highly lethal pneumonia that occurs almost exclusively in hospitalized patients with impaired immunity. Other bacterial pneumonias include Legionnaire disease, caused by *Legionella pneumophilia*; pneumonia secondary to other illnesses caused by *Staphylococcus aureus* and *Hemophilus influenzae*; and psittacosis, an atypical infectious form.

Mycobacteria tuberculosis is the primary bacterium responsible for causing the disease commonly known as tuberculosis (TB). The disease is present worldwide and is responsible for considerable morbidity and mortality. Tuberculosis usually exists in the form of a lung infection; however, the organism may cause disease in any organ or tissue throughout the body. The tubercule bacillus responsible for the disease is usually transmitted by the infected individual through coughing or sneezing. Although a single casual contact may transmit disease, most infections result from sustained exposures. Epidemiologic Information Tuberculosis has remained endemic in developing countries; however, it has reemerged as a major threat to both developing and industrialized countries over the last decade (Porter and Adams, 1994). Around the world, there are almost nine million new cases annually with more than 7,000 deaths per day attributable to TB. Diagnosis A definitive diagnosis of tuberculosis requires the identification of the tubercule bacillus. However, most individuals have been tested for tuberculosis using common TB skin tests (e.g., purified protein derivative, PPD) where the patient develops an

infiltration (swelling) around the sight of antigen injection within 24 hours. A negative skin test, however, does not exclude the possibility of infection and patients with a past history of exposure to TB may have a positive test without an active infection. Detecting the bacillus by sputum or other culture is not always simple. Treatment and Prevention Preventing TB requires prompt identification and treatment of infected patients. Family members and close contacts of those found to be infected should be tested and also treated if they are shown to be positive, even if the infection is an asymptomatic primary one. A major risk factor for spread of TB is crowded living conditions and a depressed socioeconomic status.

Infectious diarrhea continues to be a major source of international disease and death. Although many of these infections are self limited, some are more problematic, causing disability and even death. There were a number of outbreaks of diarrhea during Operation Desert Shield. Such outbreaks can be particularly disabling during periods of deployment because of both the disability they inflict on the individual and the potential for spread to other individuals. Epidemiologic Information Hyams and colleagues collected data from U.S. troops stationed in northeastern Saudi Arabia between September and December 1990. They cultured stool from 432 individuals presenting with diarrhea, cramps, vomiting, or hematochezia. They also surveyed 2,022 soldiers in regions throughout Saudi Arabia. Researchers were able to identify a bacterial enteric pathogen in 49.5 percent of the troops with gastroenteritis. The most common bacteria were enterotoxigenic *Escherichia coli* and *Shigella sonnei*. The diarrhea is usually mild, but not always, and lasts for a few days without therapy. Sometimes the infection can persist and patients may develop a reactive arthritis that is most commonly associated with patients carrying the HLAB27 antigen. *Shigella* produces an acute infectious colitis that is commonly referred to as "bacillary dysentery." The spectrum of disease is variable from mild watery diarrhea to the fatal dysentery that is more common in less-developed regions. The incubation period is from one to two days, following which some patients develop fever, some diarrhea, and some both. Patients with dysentery experience small-volume frequent stools (several per hour) consisting of blood, mucus, and pus, with abdominal cramps and tenesmus. Most patients recover over the period of up to a week, although with severe disease, they can suffer colonic perforation that can prove fatal. Very rarely patients may experience broader persistent systemic symptoms (e.g., hemolytic uremic syndrome, arthritis, seizures). *Salmonella* are responsible for a number of diseases in humans. In addition to causing typhoid fever, infection can present as acute diarrhea or in more severe cases as septicemia, meningitis, reactive arthritis, osteomyelitis, and endocarditis. With respect to the gastroenteritis, the incubation period is generally from one to two days. Diarrhea (sometimes with the presence of blood) may be accompanied by nausea, vomiting, and abdominal cramps. Generally the illness is mild and self-limited, although immuno suppressed, elderly, and young patients are particularly at risk for more severe disease. Diagnosis generally requires isolation of the organism from stool. Common laboratory techniques exist to distinguish known bacterial pathogens that infect the gastrointestinal tract.

Treatment and Prevention:

Treatment depends on identifying the infecting organism and its antibiotic resistance pattern. In reality, most diseases are self-limited, particularly in healthy infected hosts. Once the bacterial resistance pattern is known, an appropriate antibiotic may be selected for those patients needing more aggressive therapy. For patients with severe diarrhea, fluid and electrolyte replacement may be indicated. Because these are contagious, infectious diseases, prevention centerson isolation of infected individuals until the disease resolves. Furthermore, good hygiene contributes considerably to reducing the likelihood of infection.

Neisseria meningitidis is a gram-negative bacterium that normally populates the oropharynx (upper respiratory tract) but has the potential to cause a number of diseases, most importantly meningitis (for which it is named) and bacteremia in susceptible hosts. Healthy individuals may be carriers of the infection, and sporadic epidemiologic outbreaks continue to occur in both industrialized and developing countries.

Although the diagnosis may be made by blood culture, the disease often resolves before the diagnosis is made. • Meningococcemia without meningitis—the individual shows signs of sepsis (elevated white cell count, skin rashes, malaise, weakness, headache, and hypotension) but without meningeal signs. • Meningitis with or without meningococcemia—these patients have headache, fever, and accompanying meningeal signs. Cerebrospinal fluid examination suggests infection. • Meningoencephalitic—These individuals are septic, obtunded, with meningeal signs. With active disease, the signs a patient expresses vary widely. Petechial rashes measuring from 1–2 mm may be present, particularly on the lower half of the body. These spots may coalesce to form what appear to be ecchymoses. Cardiovascular involvement is also well recognized with this infection, with accompanying arrhythmias, congestive heart failure, decreased tissue perfusion, and pulmonary edema. The most devastating findings are septic shock and diffuse intravascular coagulation. Because the organism commonly colonizes the oropharynx, the mere isolation of *N. meningitidis* is insufficient to confirm an infection. In fact, many healthy individuals harbor this organism. Therefore, diagnosis depends on isolation of the bacteria from what is otherwise a sterile body environment (e.g., blood, cerebrospinal fluid (CSF), pleural fluid, pericardial fluid). Bacterial culture is the standard for diagnosis, although gram-negative diplococci can be seen with abundant infections on initial Gram's stain. However, diagnosis is conventionally done by serologic measures through detection of antigens from body fluids (e.g., blood, joints, CSF). These tests (e.g., latex agglutination, counter immunoelectrophoresis) offer accurate rapid diagnosis. These tests also enable demonstration of the specific serogroup responsible for infection. More recently, use of the polymerase chain reaction has emerged as an additional powerful diagnostic technique for meningococcal infection Particularly with the development of antibiotic resistant strains of *N. meningitidis*, efforts have been undertaken to develop vaccines using the bacterial antigens as targets for the vaccine. In large populations, achieving a sufficient number of protected individuals creates what is known as "herd

immunity" whereby the risk of epidemics is reduced because the number of individuals harboring the infection is low, and there are no clusters of infectious individuals. In the health care setting, it is important to avoid direct contact with potentially infectious individuals, particularly those with a respiratory infection, by adhering to droplet precautions. Secondary prevention includes chemoprophylaxis in those with known exposures. Treatment for meningococcal disease has dramatically altered the course of epidemics. Penicillin administered either intravenously or intramuscularly, remains the first-line treatment.

Vibrio cholerae is a gram-negative comma-shaped bacterium that has been known for many years to cause diarrheal illness secondary to intestinal infection. The infection is frequently mild or asymptomatic, but it can be severe. Approximately 5 percent of infected persons have severe disease (cholera) characterized by profuse watery diarrhea, vomiting, and leg cramps. In these individuals, rapid loss of body fluids leads to dehydration and shock; without aggressive treatment, death can occur within hours. When cholera is suspected, definitive diagnosis can be made through microbiologic examination of the stool. The organism can be identified by trained microbiologists on examination of fresh stool, and the bacterium can be cultured using readily available media. Newer immunologic and molecular mechanisms now exist to aid in the diagnosis of this disease. A short-acting vaccine is available for individuals exposed to cholera; however, the vaccine is not usually recommended for individuals traveling to areas (e.g., Latin America) where cholera is commonly found. Treatment for cholera is supportive, with replacement of fluids and electrolytes through intravenous and oral therapy. When recognized and treated, patients recover from their infection without long-term consequences.

7.7 HUMAN DISEASES CAUSED BY VIRUS

Viruses are very small infectious agents. They're made up of a piece of genetic material, such as DNA or RNA, that's enclosed in a coat of protein. Viruses invade cells in your body and use components of those cells to help them multiply. This process often damages or destroys infected cells. A viral disease is any illness or health condition caused by a virus. Read on to learn more about some of the main types of viral diseases: Not all viral diseases are contagious. This means they aren't always spread from person to person. But many of them are. Common examples of contagious viral diseases include the flu, the common cold, HIV, and herpes. Other types of viral diseases spread through other means, such as the bite of an infected insect. Respiratory viral diseases are contagious and commonly affect the upper or lower parts of your respiratory tract. Common symptoms of a respiratory viral disease include:

- Runny or stuffy nose
- Coughing or sneezing
- Fever
- Body aches

Examples of respiratory diseases include:

- Flu

- Common cold
- Respiratory syncytial virus infection
- Adenovirus infection
- Parainfluenza virus infection
- Severe acute respiratory syndrome (SARS)

Transmission

Respiratory viruses are spread by droplets generated through coughing or sneezing. If someone with a viral illness coughs or sneezes nearby and you inhale these droplets, you may develop the disease. These viruses can also be spread through contaminated objects, such as doorknobs, tabletops, and personal items. If you touch one of these objects and then touch your nose or eyes, you could develop a disease.

Treatment

Respiratory viral diseases usually heal on their own. But over-the-counter (OTC) medications, including nasal decongestants, cough suppressants, and pain relievers, can help to reduce symptoms. In addition, Tamiflu, an antiviral drug, is sometimes prescribed if someone is in the very early stages of developing the flu.

Prevention

The best way to avoid respiratory viral diseases is to practice good personal hygiene. Wash your hands often, cover your mouth when you cough or sneeze, and limit your interactions with people who show symptoms of a respiratory condition. There's also a vaccine that can help to reduce your risk of getting the seasonal flu. Gastrointestinal viral diseases affect your digestive tract. The viruses that cause them are contagious and usually lead to a condition called gastroenteritis, also called the stomach flu. Common symptoms of gastrointestinal viral diseases include:

- Abdominal cramps
- Diarrhea
- Vomiting

Examples of gastrointestinal viral diseases include:

- Norovirus infection
- Rotavirus infection
- Some adenovirus infections
- Astrovirus infection

Transmission

Gastrointestinal viruses are shed in the stool during bowel movements. Food or water that's been contaminated by feces can spread the virus to others. You can also get the virus from sharing utensils or personal objects with someone who has a virus. There aren't any treatments for gastrointestinal viral diseases. In many cases, they resolve on their own within a day or two. In the meantime, drink plenty of fluids to replace those lost from diarrhea or vomiting.

Prevention

You can prevent gastrointestinal viral diseases by washing your hands often, especially after using the bathroom. Wiping down contaminated surfaces and not sharing personal items or eating utensils can

also help. There's also a vaccine for rotavirus that's recommended as part of a child's vaccination schedule. Exanthematous viruses cause skin rashes. Many of them cause additional symptoms as well. Many of the viruses in this category, such as the measles virus, are highly contagious.

Examples of exanthematous viral diseases include:

- Measles
- Rubella
- Chickenpox/Shingles
- Roseola
- Smallpox
- Fifth disease
- Chikungunya virus infection

Transmission

- Many exanthematous viruses are spread through respiratory droplets from the cough or sneeze of someone with the virus.
- Other exanthematous viral diseases, such as chickenpox and smallpox, can be transmitted by coming into contact with fluid in broken skin lesions.
- Shingles only occurs in people who've had chickenpox at some point. It's a reactivation of the varicella-zoster virus that's been lying dormant in your cells.
- Chikungunya virus is spread through a mosquito bite and cannot be transmitted from person to person.
- Treatment
- Treating exanthematous viral diseases focuses on managing symptoms. Fever-reducing medications, such as acetaminophen, can help with some of the more bothersome symptoms.
- Antiviral drugs, such as acyclovir, may be given for chickenpox or shingles.

Prevention

- Measles, rubella, chickenpox, shingles, and smallpox can all be prevented through vaccination. You can reduce your risk of a chikungunya virus infection by protecting yourself from mosquito bites.

The hepatic viral diseases cause inflammation of the liver, known as viral hepatitis. The most common Trusted Source types of viral hepatitis are hepatitis A, B, and C. It is worth noting that diseases caused by other viruses, such as cytomegalovirus and the yellow fever virus, can also affect the liver.

Examples of hepatic viral diseases include:

- Hepatitis A
- Hepatitis B
- Hepatitis C
- Hepatitis D

Many neurologic viruses are spread through the bite of an infected animal or bug, such as a mosquito or tick. Other viruses, such poliovirus and other enteroviruses, are quite contagious and spread through close contact with someone with the virus. Contaminated objects can also contribute to the spread of these viruses.

Treatment

There's no specific treatment for people with mild viral meningitis or encephalitis. Getting plenty of rest, staying hydrated, and taking OTC anti-inflammatories to ease pain or headaches can all help. In some cases, antiviral medication may be prescribed. Polio or severe cases of meningitis or encephalitis may require additional treatment, such as breathing assistance or IV fluids.If an animal that's suspected to have the rabies virus bites you, you'll be given a series of shots to help prevent the rabies virus from infecting you.

Prevention

There's a vaccine for both poliovirus and the mumps virus, which can cause meningitis and encephalitis. Practicing good hygiene, avoiding close contact with those who have the virus, and protecting against insect bites can all help to reduce the spread of encephalitis and meningitis. To reduce the risk of spreading rabies, keep your pets vaccinated and avoid approaching wild animals. Cutaneous viral diseases cause lesions or papules to form on the skin. In many cases, these lesions can stick around for a long time or come back after disappearing for a while.

Examples of Cutaneous viral diseases include:

- Warts, including genital warts
- Oral herpes
- Genital herpes
- Molluscumcontagiosum

Hemorrhagic viral diseases are severe conditions that involve damage to your circulatory system.

Symptoms of a hemorrhagic viral disease include:

- High fever
- Body aches
- Weakness
- Bleeding under the skin
- Bleeding from the mouth or ears
- Bleeding in internal organs

Examples of viral hemorrhagic diseases include:

- Ebola
- Lassa fever
- Dengue fever
- Yellow fever
- Marburg hemorrhagic fever
- Crimean-Congo hemorrhagic fever

Transmission

Some hemorrhagic viral diseases, such as dengue fever and yellow fever, are spread through the bite of an infected insect. Others, such as Ebola, are spread to other people through contact with the blood or other bodily fluid of someone with the virus. Lassa fever is spread through inhaling or consuming the dried feces or urine of a rodent with the virus.

Treatment

There's no specific treatment for hemorrhagic viral diseases. It's important to stay hydrated if you have a viral hemorrhagic disease. Some people may need intravenous (IV) fluids to maintain electrolyte balance.

Supportive care to maintain hydration and electrolyte balance is essential. In some cases, the antiviral drug ribavirin may be given.

Prevention

Researchers are in the process of developing vaccines for several hemorrhagic viruses. A yellow fever vaccine is currently available for people traveling to areas where yellow fever is common. If you live or work in an area where viral hemorrhagic diseases are common, you can do the following to reduce your risk: Use proper protection, such as gloves, glasses, or a face shield, when working around people who have a virus. Avoid being bitten by insects, especially mosquitos and ticks, by wearing protective clothing or using insect repellent. Protect against rodent infestation by keeping food covered, removing garbage often, and making sure windows and doors are secured properly.

Neurologic viral diseases

Some viruses can infect the brain and surrounding tissues, causing neurologic viral diseases. This can result in a range of symptoms, including:

- Confusion
- Drowsiness
- Ceizures
- Coordination problems

Examples of neurologic viral diseases include:

- Polio
- Viral meningitis
- Viral encephalitis
- Rabies

There are many viral diseases. Some, such as the common cold or the stomach flu, are minor and go away on their own within a few days. Others, however, are more serious. Unlike bacterial infections, viral diseases don’t respond to antibiotics. Instead, treatment usually focuses on managing symptoms and supporting the immune system with plenty of rest and hydration.

7.8 CHECK YOUR PROGRES

1. Define antibiotic?
2. What are the viral diseases?

7.9 LET US SUM UP

Antibiotics are drugs used to treat bacterial infections. They are ineffective against viral infections and most other infections. Antibiotics either kill microorganisms or stop them from reproducing, allowing the body's natural defenses to eliminate them. Enzymes are catalysts that, within the mild conditions of temperature, pH, and pressure of the cells, carry out chemical reactions at amazing high rate. They are characterized by a remarkable efficiency and specificity. ... Coenzymes are small nonprotein molecules that are associated to some enzymes. Bacteria are living things that have only one cell. Under a

microscope, they look like balls, rods, or spirals. They are so small that a line of 1,000 could fit across a pencil eraser. Most bacteria won't hurt you - less than 1 percent of the different types make people sick. Many are helpful. Some bacteria help to digest food, destroy disease-causing cells, and give the body needed vitamins. Bacteria are also used in making healthy foods like yogurt and cheese. But infectious bacteria can make you ill. They reproduce quickly in your body. Many give off chemicals called toxins, which can damage tissue and make you sick. Examples of bacteria that cause infections include *Streptococcus, Staphylococcus, and E. coli*. Antibiotics are the usual treatment. When you take antibiotics, follow the directions carefully. Each time you take antibiotics, you increase the chances that bacteria in your body will learn to resist them causing antibiotic resistance. Later, you could get or spread an infection that those antibiotics cannot cure. Viruses are very tiny germs. They are made of genetic material inside of a protein coating. Viruses cause familiar infectious diseases such as the common cold, flu and warts. They also cause severe illnesses such as HIV/AIDS, smallpox, and Ebola. Viruses are like hijackers. They invade living, normal cells and use those cells to multiply and produce other viruses like themselves. This can kill, damage, or change the cells and make you sick. Different viruses attack certain cells in your body such as your liver, respiratory system, or blood. When you get a virus, you may not always get sick from it. Your immune system may be able to fight it off. For most viral infections, treatments can only help with symptoms while you wait for your immune system to fight off the virus. Antibiotics do not work for viral infections. There are antiviral medicines to treat some viral infections. Vaccines can help prevent you from getting many viral diseases.

7.10 UNIT –END EXERCISES

1. Describe in detail about mycobacterium tuberculosis
2. Explain in detail about viral diseases of influenza

7.11 ANSWERS TO CHECK YOUR PROGRESS

1. *Mycobacterium tuberculosis* is a species of pathogenic bacteria in the family Mycobacteriaceae and the causative agent of tuberculosis. First discovered in 1882 by Robert Koch, M. tuberculosis has an unusual, waxy coating on its cell surface primarily due to the presence of mycolic acid. This coating makes the cells impervious to Gram staining, and as a result, *M. tuberculosis* can appear either Gram-negative or Gram-positive. Acid-fast stains such as Ziehl-Neelsen, or fluorescent stains such as auramine are used instead to identify *M. tuberculosis* with a microscope. The physiology of *M. tuberculosis* is highly aerobic and requires high levels of oxygen. Primarily a pathogen of the mammalian respiratory system, it infects the lungs. The most frequently used diagnostic methods for tuberculosis are the tuberculin skin test, acid-fast stain, culture, and polymerase chain reaction. *M. tuberculosis* is part of a complex that has at least 9 members: *M. tuberculosis sensustricto, M. africanum, M. canetti, M. bovis, M. caprae, M. microti, M. pinnipedii, M. mungi, and M. orygis*. It requires oxygen to grow, does not produce spores, and is nonmotile. *M. tuberculosis* divides every 15–20 hours. This is extremely slow compared

with other bacteria, which tend to have division times measured in minutes (*Escherichia coli* can divide roughly every 20 minutes). It is a small bacillus that can withstand weak disinfectants and can survive in a dry state for weeks. Its unusual cell wall is rich in lipids such as mycolic acid and is likely responsible for its resistance to desiccation and is a key virulence factor. Other bacteria are commonly identified with a microscope by staining them with Gram stain. However, the mycolic acid in the cell wall of *M. tuberculosis* does not absorb the stain. Instead, acid-fast stains such as Ziehl-Neelsen stain, or fluorescent stains such as auramine are used. Cells are curved rod-shaped and are often seen wrapped together, due to the presence of fatty acids in the cell wall that stick together. This appearance is referred to as cording, like strands of cord that make up a rope. *M. tuberculosis* is characterized in tissue by caseating granulomas containing Langhans giant cells, which have a "horseshoe" pattern of nuclei.

Treatment and Prevention:

Preventing TB requires prompt identification and treatment of infected patients. Family members and close contacts of those found to be infected should be tested and also treated if they are shown to be positive, even if the infection is an asymptomatic primary one. A major risk factor for spread of TB is crowded living conditions and a depressed socioeconomic status.

2. Viral disease, disease caused by viruses. Long-term immunity usually follows viral childhood diseases. The common cold recurs into adulthood because many different viruses cause its symptoms, and immunity against one does not protect against others. Some viruses mutate fast enough to reinfect people after recovery or to keep the immune system from fighting them off. Certain cancers are caused by viruses. Vaccines can prevent some viral diseases. Antiviral drugs work only against specific viruses; antibiotics are ineffective against viral diseases. Influenza, also called flu or grippe, an acute viral infection of the upper or lower respiratory tract that is marked by fever, chills, and a generalized feeling of weakness and pain in the muscles, together with varying degrees of soreness in the head and abdomen. Influenza is caused by any of several closely related viruses in the family Orthomyxoviridae (a group of RNA viruses). Influenza viruses are categorized as types A, B, C, and D. These major types generally produce similar symptoms but are completely unrelated antigenically, so that infection with one type confers no immunity against the others. The A viruses cause the great influenza epidemics, and the B viruses cause smaller localized outbreaks. The C viruses cause only mild respiratory illness in humans. Influenza D viruses are not known to infect humans and have been observed only in pigs and cattle. Influenza A viruses are classified into subtypes, and both influenza B and subtypes of influenza A are further divided into strains. Subtypes of influenza A are differentiated mainly on the basis of two surface antigens (foreign proteins)—hemagglutinin (H) and neuraminidase (N). Examples of influenza A subtypes include H1N1, H5N1, and H3N2. Strains of influenza B and strains of influenza A subtypes are further distinguished by variations in genetic sequence.

Between worldwide outbreaks, known as pandemics, influenza viruses undergo constant, rapid evolution (a process called antigenic drift), which is driven by mutations in the genes encoding antigen proteins. Periodically, the viruses undergo major evolutionary change by acquiring a new genome segment from another influenza virus (antigenic shift), effectively becoming a new subtype. Viral evolution is facilitated by animals such as pigs and birds, which serve as reservoirs of influenza viruses. When a pig is simultaneously infected with different influenza A viruses, such as human, swine, and avian strains, genetic reassortment can occur. This process gives rise to new strains of influenza A. Newly emerged influenza viruses tend to be initially highly infectious and virulent in humans because they possess novel antigens to which the human body has no prepared immune defence (i.e., existing antibodies). Once a significant proportion of a population develops immunity through the production of antibodies capable of neutralizing the new virus, the infectiousness and virulence of the virus decreases. Although outbreaks of influenza viruses are generally most fatal to young children and the elderly, the fatality rate in people between ages 20 and 40 is sometimes unexpectedly high, even though the patients receive treatment. This phenomenon is believed to be due to hyper-reaction of the immune system to new strains of influenza virus. Such reaction results from the overproduction of inflammatory substances called cytokines. The release of excessive amounts of these molecules causes severe inflammation, particularly in the epithelial cells of the lungs. Individuals whose immune systems are not fully developed (such as infants) or are weakened (such as the elderly) cannot generate such a lethal immune response. Influenza pandemics are estimated to occur on average once every 50 years. Epidemics happen much more frequently, and seasonal influenza appears annually in most parts of the world, sometimes in epidemic proportions. Influenza type A virus is the most frequent cause of seasonal influenza. When influenza A virus undergoes an antigenic shift, a pandemic affecting most of the world can occur within a matter of months. The influenza pandemic of 1918–19, the most destructive influenza outbreak in history and one of the most severe disease pandemics ever encountered, was caused by a subtype of influenza A known as H1N1.

7.12 SUGGESTED READINGS

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NOTES

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BLOCK 3: PLANT PATHOLOGY

UNIT – 8 INTRODUCTON TO PLANT PATHOLOGY

NOTES

Structure

8.1 INTRODUCTION

Phytopathology is the study of the diseases of plants and covers the entire field of biological and scientific activity concerned with the understanding of this complex phenomenon. Phytopathology is thus the study of the nature, development, and control of plant diseases.

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Disease, being a complex phenomenon, is difficult to define in a few words. We have to keep in mind what Locke said in Human understanding: though definitions will serve to explain the names of substances as they stand for our ideas, yet they leave them not without great imperfection as they stand for things. According to modern conception, dieases is an interaction among the host, parasite, and environment. A simple dictionary meaning of disease is: any departure from health, presenting marked symptoms, malady, illness, disorder. Diseased plant are distinguished by changes in their morphological structure or physiological process, which are brought about by unfavourable environment or by parasitic agencies. Several definitions of diseases in plants have been proposed. Some example are: "a series of harmful physiological processes caused by continuous irritation of the plant by a primary agnet"; "a harmful deviation from the normal functioning of physiological processes"; "a continuous impairment of metabolism". Stakman and Harrar (1957) defined plant disease as a physiological disorder or structural abnormality that is harmful to the plant or to any of its part or products that reduces the economic value.

Plant disease, an impairment of the normal state of a plant that interrupts or modifies its vital functions. All species of plants, wild and cultivated alike, are subject to disease. Although each species is susceptible to characteristic diseases, these are, in each case, relatively few in number. The occurrence and prevalence of plant diseases vary from season to season, depending on the presence of the pathogen, environmental conditions, and the crops and varieties grown. Some plant varieties are particularly subject to outbreaks of diseases while others are more resistant to them.

8.2 OBJECTIVES OF PLANT PATHOLOGY

Plant Pathology (Phytopathology) deals with the cause, etiology, resulting losses and control or management of the plant diseases. The objectives of the Plant Pathology are the study on:
(1) The living entities that cause diseases in plants;
(2) The non-living entities and the environmental conditions that cause disorders in plants;

8.3 INTRODUCTION TO PLANT PATHOLOGY

Plant pathology or phytopathology is the science, which deals with the plant diseases. It is concerned with health and productivity of growing plants. Phytopathology (Greek Phyton = plant + pathos - disease, ailments + logos = discourse, knowledge) is the branch of agricultural, botanical or biological science which deals with the cause, etiology (aetiology), resulting in losses and management methods of plant diseases.

Plant pathology can also be defined as the study of the nature, cause and prevention of plant diseases. Plant pathology is related to most of the old

and new sciences like biology, physics, chemistry, physiology, mathematics, genetics, soil science, biochemistry, biotechnology etc.

8.3.1 Plant diseases

Plant diseases are recognized by the symptoms (external or internal) produced by them or by sick appearance of the plant. The term plant disease signifies the condition of the plant due to disease or cause of the disease. Plant disease is mainly defined in terms of the damage caused to the plant or to its organ. The other definitions for the term disease are:

1. Disease is a malfunctioning process that is caused by continuous irritation, which results in some suffering producing symptoms. This definition is accepted by both American Phytopathological Society and British Mycological Society.

2. Disease is an alteration in one or more of the ordered sequential series of physiological processes culminating in a loss of coordination of energy utilization in a plant as a result of the continuous irritation from the presence or absence of some factor or agent.

3. A plant is said to be „diseased" when there is a harmful deviation from normal functioning of physiological process (Federation of British Plant Pathologists, 1973).

4. The disease can also be defined as 'any disturbance brought about by a living entity or non-living agents or environmental factors which interfere with manufacture, translocation or utilization of food, mineral nutrients and water in such a way that the affected plant changes in appearance with or without much loss in yield than that of a normal healthy plant of the same variety. In general disease is an interaction among the host, parasite and the environment.

8.4 HISTORY OF PLANT PATHOLOGY

Man became painfully 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, Atharvanaveda (1500-500 B.C.), the Artha Shashtra of Kautilya (321-186 B.C.), Sushrute 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

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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.

8.4.1 Mycology

1675 - Dutch worker Anton von Leeuwenhoek developed the first microscope.

1729 - Italian botanist P. A. Micheli proposed fungi comes from spores; father of Mycology.

1755 - French botanist Tillet published a paper on bunt or stinking smut of wheat; discovered bunt is a disease of wheat.

1807 - French scientist I. B. Prevost showed bunt of wheat is a fungus and showed evidence that a disease is caused by a microorganism.

1821 - E. M. Fries published Systema Mycologicum for naming of fungi; he was named as Linnaeus of Mycology.

1821 - Robertson of England stated that sulphur is effective against peach mildew.

1845 - Irish Potato famine due to Phytophthora infestans caused starvation of million and immigration of 1.5 million people.

1858 - J. G. Kuhn published first textbook in Plant Pathology – The Diseases of Cultivated Crops, their Causes and their Control.

1861 -Anton de Bary (Germany) worked out the life cycle of potato late blight and first to prove experimentally Phytophthora infestans is the cause of potato late blight. He proved that fungi are causes but not the results of diseases. He is the Father of Modern Plant Pathology.

1865 – Anton de Bary reported heteroecious nature of wheat stem rust.
1869 – England loses coffee production to coffee rust, forced to grow tea.

1874 -Robert Hartig published a book entitled, “Important Diseases of Forest Trees”.

1875-1912 - Brefeld discovered the methods of artificial culture of microorganisms; he also illustrated the complete life cycles of cereal smut fungi and diseases caused by them.

1877 – M. S. Woronin discovered and named the Club root of Cabbage pathogen as Plasmodiophora brassicae.

1878 – M. S. Woronin found out the life cycle of potato wart disease.

1878 -Downy mildew of grapevine was introduced into Europe from America. The disease almost ruined the wine industry.

1881 -H.M. Ward worked out the life cycle of coffee leaf rust. He is called as Father of Tropical Plant Pathology.

1882 -Robert Hartig published a textbook -Diseases of Trees. He is called as "Father of Forest Pathology".

1885 -Pierre Marie Alexis Millardet accidentally discovered the Bordeaux mixture for the control of downy mildew of grapevine.

NOTES

1885 – A. B. Frank defined and named mycorrhizal associations in plant roots. 1887 -Burgundy mixture was introduced by Mason of France.

1894 -Swedish scientist Eriksson described the phenomenon of physiologic races in cereal rust fungus, *Puccinia graminis*.

1899 – W. A. Orton selected and bred water-melon, cowpea and cotton for resistance to Fusarium wilt diseases. He is considered as a pioneer worker in the development of disease resistant varieties. 1904 – A. F. Blakeslee, American Geneticist founded heterothallism in Rhizopus

1904 – R. H. Biffen was the first to show that resistance to pathogens in plants can be inherited as a Mendelian character; pioneer in genetics of plant disease resistance.

1912 – H. Burgeff reported that within a cell of a fungus, fusion between dissimilar nuclei can occur. He called this phenomenon as heterokaryosis.

1917 -E. C. Stakman demonstrated physiologic forms in stem rust of wheat.

1918 -E.J.Butler published book on Fungi and Disease in Plants; he made exhaustive study on Indian fungi and the diseases caused by them. He is called as the Father of Modern Plant Pathology in India; He joined as the first Director of Imperial Bureau of Mycology (Commonwealth Mycological Institute, CMI) now CAB International Mycological Institute in Kew, England in 1920. He began the journal Review of Applied Mycology; with S.G. Jones he wrote, 'Plant Pathology' in 1949.

1929 -Sir Alexander Fleming isolated the antibiotic, Penicillin from the fungus, Penicillium notatum.

1932 – H. N. Hansen and R. E. Smith were the first to demonstrate the origin of physiologic races through heterokaryosis.

1934 -W. H. Tisdale and I. Williams studied the organic fungicides by discovering alkyl dithiocarbamates.

1938 – H. N. Hansen found out dual phenomenon in Fungi Imperfecti.

1942 – H. H. Flor developed gene-for-gene hypothesis in flax rust.

1943 – Great Bengal Famine due to Helminthosporium oryzae caused death of 2 million people in India.

1943 -Dimond, Heuberger and Horsfall discovered the ethylene bis dithiocarbamates.

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1945 -J. G. Horsfall explored the mechanism of fungicidal action.

1948 -B. B. Mundkur started Indian Phytopathological Society with its journal Indian Phytopathology. He has written a book 'Fungi and Plant Diseases' in 1949, which is the second, book in plant pathology in India.

1951-57 -E. A. Gaümann was one of the first to investigate the physiology of the wilts caused by Fusarium spp. He put forth the involvement of toxin (toxin theory) in wilt diseases.

1952 -N.F. Jensen suggested blending of different resistant genotypes of similar agronomic characters in fields of oats to reduce the spread of rust and losses from rust.

1953 -N. E. Borlaug and associates developed multiline cultivars for wheat.

1953 – Pontecorvo and his associates demonstrated parasexualism in fungi.

1956 -J. G. Horsfall published a book entitled "Principles of Fungicidal action"

1957 – E. C. Stakman with J. G. Harrar wrote a book Principles of Plant Pathology.

1963 - J. E. Van der Plank found out vertical and horizontal types of resistance in crop plants.

1966 -van Schmeling and Marshall Kulka were the first to find out systemic fungicides (oxathiin compounds – carboxin and oxycarboxin).

1970 -S. D. Garrett investigated the management of root diseases and he is the pioneer worker in the field of biological control.

1972 – G. Rangaswami wrote a book on Diseases of Crop Plants in India.

8.4.2 Plant Bacteriology

1683 – Anton von Leeuwenhoek first observed bacteria.

1876 -Louis Pasteur and Robert Koch -They proved that anthrax disease of cattle was caused by specific bacterium.

1876 -Robert Koch of Germany described the theory called "Koch's postulates." He established the principles of pure culture technique.

1876 -Robert Koch and Pasteur disproved the theory of spontaneous generation of diseases and propose germ theory in relation to the diseases of man and animal.

1882 -American Plant Pathologist -T. J. Burrill first time proved that fire blight of apple and pear was caused by a bacterium (now known as Erwinia amylovora).

1901-1920 E.F.Smith of U.S.A gave the final proof of the fact that bacteria could be incitants of plant diseases. He also worked on the

bacterial wilt of cucurbits and crown gall disease. He is also called as "Father of Phytobacteriology". Chilton and his coworkers demonstrated that crown gall bacterium transforms plant cell to tumour cell by introducing into them a plasmid.

1910 -C. O. Jensen related crown gall of plants to cancer of animals.

1952 -J. Lederberg coined the term plasmid

1952 – S. A. Waksman won Nobel prize for the discovery of streptomycin.

1952 – Zinder and J. Lederberg discovered transduction in bacteria

1962 – H. Stolp discovered bdellovibrios.

1972 – P. B. New and A. Kerr success in biological control of A. radiobacter strain K.

1972 – I. M. Windsor and L. M. Black observed a new kind of phloem inhabiting bacterium causing clover club leaf disease.

1974 – I. Zanen et al. demonstrated Ti plasmid in Agrobacterium tumefaciens.

1980 – D. W. Dye et al. introduced the pathovar in the taxonomy of plant pathogenic bacteria.

8.4.3 Plant Virology

1886 -Adolf Mayer described a disease of tobacco called mosaikkranheit (tobacco mosaic). Adolf Mayer demonstrated the sap transmission of Tobacco Mosaic Virus disease.

1892 -Dimitri Ivanowski demonstrated that the causal agent of tobacco mosaic could pass through bacterial filter.

1895 -E.F. Smith of U.S.A. showed the peach yellows was a contagious disease.

1898 -M.W. Beijerinck -a Dutch microbiologist and founder of virology proved that the virus inciting tobacco mosaic is not a microorganism. He believed it to be contagium vivum fluidum (infectious living fluid). He was the first to use the term virus, which is the Latin word for poison.

1929 -F. O. Holmes provided a tool by which the virus could be measured by showing that the amount of virus present in a plant sample preparation is proportional to the number of local lesions produced on appropriate host plant leaves rubbed with the contaminated sap.

1935 -W. M. Stanley proved that viruses can be made as crystals. He got Nobel Prize in 1946.

1936 -F. C. Bawden and, N.W. Pirie (Britain) found that the crystalline nature of the virus contains nucleic acid and protein.

1939 -Kausche and colleagues first time saw the TMV virus particles with the help of Electron microscope.

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1956 -Gierer and Schramm proved that the nucleic acid fraction of the virus is actually the infectious agent.

1959 -Munday succeeded in inducing TMV mutations.

1966 -Kassanis discovered the satellite viruses.

1971 -T. O. Diener discovered viroids, which only consist of nucleic acids. Smaller than viruses, caused potato spindle tuber disease (250-400 bases long of single-stranded circular molecule of infectious RNA).

8.4.4 Phytoplasma

1967 – Doi et al and Ishiie et al, the Japanese scientists found that mycoplasmalike organisms (MLO) could be responsible for the disease of the yellows type. Doi observed that MLO's are constantly present in phloem while Ishiie observed MLO's temporarily disappeared when the plants are treated with tetracycline antibodies.

8.4.5 Spiroplasma

1972-Davies et al., observed that a motile, helical wall-less microorganism associated with corn stunt diseases, which could be cultured and characterized and they named it as spiroplasma.

Check your Progress- 1
Note: a. Write your answer in the space given below
b. Compare your answer with those given at the end of the unit.
1. What is Phytopathology?
2. What is a plant disease?
……
…….

8.5 CAUSAL AGENTS RESPONSIBLE FOR PLANT DISEASES

Plant diseases are classified on the basis of type of pathogenic or non-pathogenic causes of the disease. The classification is based on the plant pathogenic organisms as follows.

8.5.1 A. Parasites: They include both biotic and mesobiotic agents. The diseases are incited by parasites under a set of suitable environment. Association of definite pathogen is essential with each disease.

8.5.1.1 i. Biotic agents: They are also called as animate causes. They are living organisms.

Biotic agents include

8.5.1.2 Prokaryotes

a. True bacteria or bacteria (Facultative parasites) e.g. Citrus canker.

b. Rickettsia-like bacteria (RLB) e.g. Citrus greening, Pierce's disease of grape

c. Mollicutes or wall-less prokaryotes

i. Mycoplasma-like organism (MLO) e.g. Sesame phyllody, egg plant little leaf.

ii. Spiroplasma e.g. Corn stunt, Citrus stubborn

8.5.1.3 Eukaryotes

a. Protists (Unicellular, coenocytic or multicellular with little or no differentiation of cells and tissues).

i. Fungi e.g. wilt of cotton

ii. Protozoa e.g. heart rot of coconut

iii. Algae e.g. red rust of mango

b. Plants - Parasitic flowering plants or phanerogamic parasites - Broomrape of tobacco.

c. Animals (Multicellular, extensive differentiation of cells and tissues) e.g. Nematodes -Root knot nematode.

ii. Mesobiotic agents: They include viruses and viroids. They are infectious agents. They can be crystallized and are considered non-living. But their multiplication in the living plants ensures that they are living. Hence they are called as mesobiotic agents. Viruses e.g. yellow mosaic of blackgram Viroids e.g. spindle tuber of potato

8.5.2 B. Non-parasites or Abiotic agents:

They are also called as non-infectious or physiological disorders. When no pathogen is found, cultured from or transmitted from a diseased plant, then the disease is said to be caused by a non-living or environmental factor. These diseases occur because of disturbances in the plant system by the improper environmental conditions in the air or soil or by mechanical influences. They are listed below.

i. Too low or too high temperature

ii. Lack or excess of soil moisture

iii. Lack or excess of light

iv. Lack of oxygen

v. Air pollution (Toxic gases in the atmosphere etc.)

vi. Mineral deficiencies or toxicities

vii. Soil acidity or alkalinity

viii. Toxicity of pesticides

ix. Improper agricultural practices.

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8.5.3 a. FUNGI

Fungi are eukaryotic, achlorophyllous organisms that may reproduce sexually and asexually and whose filamentous branched somatic structures are typically surrounded by cell walls containing chitin or cellulose.

8.5.4 b. BACTERIA

Bacteria are microscopic, unicellular prokaryotes, which lack chlorophyll. These microorganisms are with a primitive nucleus lacking a clearly defined membrane. The bacteria are smaller than fungi and measure about 0.5 to 1.0 x 2.0 to 5.0 µm in size. More than 1,600 bacterial species are known. Majority of them are saprophytes. Several species cause diseases in human beings and animals. About 200 species of bacteria cause diseases in plants. First report of plant disease by bacteria was made by T.J. Burrill of the University of Illinois. He showed that fire blight of apple and pear is caused by a bacterium, Erwinia amylovora. Bacteria have been defined by Clifton as "extremely minute, rigid essentially unicellular organisms, free of true chlorophyll and generally devoid of any photosynthetic pigments; most commonly multiplying asexually by simple transverse fission, the resulting cells being of equal or nearly equal size".

8.5.4.1 Fastidious vascular bacteria (Rickettsia-like bacteria – RLB)

Fastidious vascular bacteria are otherwise called Rickettsia - Like bacteria, Rickettsia like organisms (RLO), or fastidious prokaryotes or rickettsia -like walled bacteria. They are small bacteria with a cellular ultrastructure of typical gram- negative bacteria. They are very exacting in their nutritional requirements, refusing to grow on routine bacteriological media. They have a cell wall unlike MLO and spiroplasma. MLO is restricted to phloem tissues where as RLB are restricted mostly to xylem or phloem. A common habit for both is the insect body fluid (haemolymph). Both the groups are dependent on insect vectors for transmission. Non-tissue restricted RLB have also been observed in plant diseases. They reproduce by binary fission. Mostly insect vectors transmit them. Nematode (Xiphinema index) also helps in transmission of RLB (yellow disease of grapevine). Mechanical inoculations (as in Pierce's disease of grapevine, almond leaf scorch and alfalfa dwarf) or vegetative propagation also reproduce disease symptoms. They produce phytoalexins, which induce characteristic symptoms of the disease. They are cultured in artificial media e.g., Pierce's disease of grapevine, almond leaf scorch, phony disease of peach and plum leaf scald. Xylem restricted RLB can be more successfully cultured than limited-limited bacteria.

Penicillin is effective against RLB. Sulpha drugs also inhibit them. The RLB can be divided into three groups.

1 Xylem-limited RLB

2 Phloem-limited RLB and

3 Non-tissue restricted RLB

8.5.4.2 Phloem-limited RLB

Phloem limited bacterium was first recognized by D.Lefleche and J.M.Bowe in 1970. Twelve phloem-restricted RLB have been identified Examples of phloem limited RLB include citrus greening, clover club leaf (CCL), white clover disease, clover rugose leaf curl, potato leaflet stunt, little leaf of *Sida cordifolia* and stunting of dodder.

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a. Symptoms: Stunting, yellowing of young leaves, virescence of floral parts, premature death of the entire plant.

b. RLB: They are mostly rigid rods and Gram-negative and sensitive to penicillin. The cells measure 0.2 to 0.5 x 1.0 to 2.0 (0.3 x 1.3) μm and are bound by a double membrane or a cell wall and cytoplasmic membrane. RLB have not been cultured in vitro and Koch's postulates proved. Therefore, not much is known about their nature, taxonomy and serological relationships.

Transmission: Transmission is by leafhoppers, dodder and grafting. Citrus greening is transmitted by citrus psylla (Psyllina sp.) and by vegetative propagation. Clover clubleaf multiplies in its vector, Agalliopsis novella. The vector retains infectivity throughout its life cycle and the RLB is transovarially transmitted.

8.5.4.3 Xylem-limited RLB

Pierce's disease of grapevine, almond leaf scorch, phony disease of peach, wilt of periwinkle, Sumatra disease of cloves, elm leaf scorch, alfalfa dwarf, plum leaf scald. The RLB causing phony disease of peach is named as Xylella fastidiosa. Symptoms: Symptoms include marginal necrosis of leaves, stunting of plants, decline in vigour and reduction in yield. RLB: In general xylem-limited Gram-negative bacteria have elongated cells of 0.2 to 0.5 into 1.4 μm size (Davis et. al., 1981). The cells usually have well defined cell wall and plasma membrane. Both are triple layered in structure. The walls are ridged or ripped due to periodic infolding of the outer membrane of the wall. These width of the ridges is about 45 to 75 nm. The cell wall ultrastructure is typical of Gram negative bacterial. In culture, the cells of Pierce's disease of grapevine and almond leaf scorch are non-motile, gram negative, oxidase negative and catalase positive. They are susceptible to tetracyclines but not to penicillin. The G+C content of the DNA is about 53.1 moles per cent.

Transmission

Transmission of RLB takes place mostly through xylem feeding insects. Sap transmission and transmission through vegetative propagation have been reported. The insect vectors belong to sharp shooter leafhoppers (Cicadellidae) and spittle bug or froghoppers (Cercopidae). Pierce's disease of grapevine is spread by Homaladisca coagulata, Oncometopia undulata, Cuerna costalis, Draeculacephala portola, D.minerva, Corneocephala fulgide and Graphocephala atrapunctata. RLB of Pierce's disease of grapevine is transmitted by the vector in a noncirculative but persistent manner. There is no incubation period in the body of the vector and

infectivity is lost after moulting. This is because the RLB accumulate only in the salivary syringes where they appear to attach in a polar orientation. The transmission is accompanied by regurgitation of the bacteria into the xylem stream. The RLB are not pathogenic to the vector. There is no transovarially transfer of RLB.

8.5.4.4 Non-tissue restricted RLB:

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They are also found in parenchyma and meristematic cells of yellows of grapevine, chlorosis and Aspermy of wheat, apple proliferation, carrot proliferation and necrosis of grapevine. A yellow of grapevine is transmitted by a nematode, Xiphinema index. Not much is known about RLB of these diseases.

8.5.5 PHYTOPLASMA

Phytoplasma lack cell wall and are bounded by a unit membrane. They are pleomorphic. They lack cell wall. They have fried egg appearance of colony. They are filterable through 450 nm membrane. They have both DNA and RNA. They cannot be grown on artificial media. They produce symptoms like little leaf, phyllody, spike, yellows, stunting, witches' broom etc. They are mostly transmitted by leafhoppers. They are insensitive to penicillin and sensitive to tetracycline. e.g. phyllody of sesame, little leaf of brinjal.

8.5.6 SPIROPLASMA

Spiroplasma is helical, wall-less prokaryotes requiring cholesterol for growth and cause diseases in plants, insects and rats. They are insensitive to penicillin and sensitive to erythrocin and tetracycline. e.g. corn stunt, citrus stubborn.

8.5.7 VIRUS

Viruses are ultramicroscopic, nucleoprotein entities, which are infectious agents and obligately parasitic pathogens, which are less than 200 mμ in size. They are devoid of enzymes and depend on the host protein synthesis machinery (ribosomes). They have only one type of nucleic acid viz., RNA or DNA. Most of the plant virus is having RNA. e.g. TMV. Few viruses contain DNA. e.g. Cauliflower mosaic virus, banana bunchy top virus, maize streak virus and sugar beet curly top virus.

8.5.8 VIROIDS

Viroids are small low molecular weight ribonucleic acids that can infect plant cells replicate themselves and cause disease. They are also called as mini viruses. e.g. Potato spindle tuber, Chrysanthemum stunt, Coconut Cadang cadang. ALGAE Algae are eukaryotic, unicellular or multicellular organisms and mostly occur in aquatic environments. Many algae thrive as terrestrial or subterranean algae. The size of algae ranges from 1.0mm to many centimetres in length. They contain chlorophyll and are photosynthetic. They reproduce by asexual and sexual processes. The study of algae is called phycology or algology.

8.5.9 PROTOZOA

Protozoa (trypanosomatid flagellates) belonging to the class Mastigophora, order Kinetoplastida and family Trypanosomatidae have been known to parasitize plants. Protozoa attacking plants move by flagella. Protozoa or trypanosomatid flagellates belonging to the class Mastigophora, order: Kinetoplastida and family Trypanosomatidae have been known to parasitize plants. The Mastigophora, or flagellates, are characterized by one or more long slender flagella at some or all stages of their life cycle. The flagella are used for locomotion and food capture. They are also used as sense organs. The body of the flagellates has a definite long, oval or spherical form, which is maintained by a thin, flexible membrane cover.

In some groups it may be armoured. Flagellates reproduce by longitudinal fission. Flagellates apparently cause the phloem necrosis disease of coffee, the heart rot disease of coconut palm and the Marchitez suppressive (sudden wilt or wither) disease of oil palm, Marchitez suppressive is one of the important diseases in oil palm. Phytomonas staheli was described from sieve tubes of coconut and oil palm.

8.5.10 PHANEROGAMIC PARASITES

Phanerogamic parasites are flowering plants or seed plants, which lead a parasitic life on other living plants. They parasitize a great number of economic plants and cause considerable loss in yield. The phanerogamic parasites invade stem or root of the host plants. Some of these parasites possess chlorophyll, which manufacture carbohydrates to a limited extent and depend on the host for mineral, salts and water. These are generally called as semi or partial parasites. Some of the parasites, which do not have chlorophyll, depend entirely on the host plants for their food materials. They are called holo or total parasites. Nearly 2,500 species of phanerogamic parasites in 11 families have been recorded throughout the world. Among them Orobanchaceae, Scrophulariaceae, Loranthaceae, Convolvulaceae and Lauraceae are important.

8.6 Answer to Check Your Progress Questions

1. Phytopathology is the study of the diseases of plants and covers the entire field of biological and scientific activity concerned with the understanding of this complex phenomenon. Phytopathology is thus the study of the nature, development, and control of plant diseases.
2. Plant diseases are recognized by the symptoms (external or internal) produced by them or by sick appearance of the plant. The term plant disease signifies the condition of the plant due to disease or cause of the disease. Plant disease is mainly defined in terms of the damage caused to the plant or to its organ.

8.7 Summary

Plant pathology is a branch of botany. It deals with the diseases of plants, helps to maintain good health of plants, and also take proper steps

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to increase the productivity. Control of plant diseases is crucial to the reliable production of food, and it provides significant problems in agricultural use of land, water, fuel and other inputs. Plants in both natural and cultivated populations carry inherent disease resistance, but there are numerous examples of devastating plant disease impacts such as the Great Famine of Ireland and chestnut blight, as well as recurrent severe plant diseases like rice blast, soybean cyst nematode, and citrus canker. However, disease control is reasonably successful for most crops. Disease control is achieved by use of plants that have been bred for good resistance to many diseases, and by plant cultivation approaches such as crop rotation, use of pathogen-free seed, appropriate planting date and plant density, control of field moisture, and pesticide use. Across large regions and many crop species, it is estimated that diseases typically reduce plant yields by 10% every year in more developed settings, but yield loss to diseases often exceeds 20% in less developed settings. Continuing advances in the science of plant pathology are needed to improve disease control, and to keep up with changes in disease pressure caused by the ongoing evolution and movement of plant pathogens and by changes in agricultural practices. Plant diseases cause major economic losses for farmers worldwide. The Food and Agriculture Organization estimates indeed that pests and diseases are responsible for about 25% of crop loss. To solve this issue, new methods are needed to detect diseases and pests early, such as novel sensors that detect plant odours and spectroscopy and biophotonics that are able to diagnose plant health and metabolism.

8.8 Key Words

Plant diseases: A plant disease is any abnormal condition that alters the appearance or function of a plant. It is a physiological process that affects some or all plant functions.

Plant Pathology: Plant pathology is the scientific study of diseases in plants caused by pathogens and environmental conditions.

Causal Agent: A factor associated with the definitive onset of an illness

8.9 Self Assessment Question and Exercises

1. What is phytopathology?
2. What are the different types of plant diseases?
3. What are the causal agents for plant diseases?

UNIT – 9 TYPES OF PLANT DISEASES

Structure

9.1INTRODUCTION

In general, a plant becomes diseased when it is continuously disturbed by some causal agent that results in an abnormal physiological process that disrupts the plant's normal structure, growth, function, or other activities. This interference with one or more of a plant's essential physiological or biochemical systems elicits characteristic pathological conditions or symptoms.

Plant diseases can be broadly classified according to the nature of their primary causal agent, either infectious or noninfectious. Infectious plant diseases are caused by a pathogenic organism such as a fungus, bacterium, mycoplasma, virus, viroid, nematode, or parasitic flowering plant. An infectious agent is capable of reproducing within or on its host and spreading from one susceptible host to another. Noninfectious plant diseases are caused by unfavourable growing conditions, including extremes of temperature, disadvantageous relationships between moisture and oxygen, toxic substances in the soil or atmosphere, and an excess or deficiency of an essential mineral. Because noninfectious causal agents are not organisms capable of reproducing within a host, they are not transmissible.

In nature, plants may be affected by more than one disease-causing agent at a time. A plant that must contend with a nutrient deficiency or an imbalance between soil moisture and oxygen is often more susceptible to infection by a pathogen, and a plant infected by one pathogen is often prone to invasion by secondary pathogens. The combination of all disease-causing agents that affect a plant make up the disease complex. Knowledge of normal growth habits, varietal characteristics, and normal variability of

plants within a species—as these relate to the conditions under which the plants are growing—is required for a disease to be recognized.

9.2 OBJECTIVITIES

After going through the unit you will be able to:

1. Understand the types of plant diseases
2. Understand the methods used to study the plant disease
3. Identify the various sympotms of plant disease caused by casual organism.

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9.3 PLANT DISEASES

9.3.1 Introduction to Plant Diseases

Tens of thousands of diseases affect cultivated and wild plants. On average, each kind of crop plant can be affected by a hundred or more plant diseases. Some pathogens affect only one variety of a plant. Other pathogens affect several dozen or even hundreds of species of plants. Plant diseases are sometimes grouped according to the symptoms they cause (root rots, wilts, leaf spots, blights, rusts, smuts), to the plant organ they affect (root diseases, stem diseases, foliage diseases), or to the types of plants affected (field crop diseases, vegetable diseases, turf diseases, etc.). One useful criterion for grouping diseases is the type of pathogen that causes the disease. The advantage of such a grouping is that it indicates the cause of the disease, which immediately suggests the probable development and spread of the disease and also possible control measures. On this basis, plant diseases in this text are classified as follows:

9.3.2 Types of Plant Diseases

9.3.2.1 I. Infectious, or biotic, plant diseases

Infectious diseases are disorders caused by organisms — such as bacteria, viruses, fungi or parasites. Many organisms live in and on our bodies. They're normally harmless or even helpful. But under certain conditions, some organisms may cause disease. Some infectious diseases can be passed from person to person.

1. Diseases caused by fungi

1) Cereal rusts
2) Cereal smuts
3) Ergot of rye and wheat
4) Late blight of potato
5) Brown spot of rice
6) Southern corn leaf blight
7) Powdery mildew of grapes
8) Downy mildew of grapes
9) Downy mildew of tobacco
10) Chestnut blight
11) Dutch elm disease
12) Pine stem rusts
13) Dwarf mistletoes
14) Coffee rust Asia
15) Banana leaf spot or Sigatoka

16) Rubber leaf blight South America
17) Fusarium scab of wheat North America

2. Diseases caused by prokaryotes (bacteria and mollicutes)

1. Citrus canker
2. Fire blight of pome fruits
3. Soft rot of vegetables

3. Diseases caused by parasitic higher plants and green algae

4. Diseases caused by viruses and viroids

1. Sugar cane mosaic
2. Sugar beet yellows
3. Citrus tristeza (quick decline)
4. Swollen shoot of cacao
5. Plum pox or sharka
6. Barley yellow dwarf
7. Tomato yellow leaf curl
8. Tomato spotted wilt virus

5. Diseases caused by nematodes

1. Root knot
2. Sugar beet cyst nematode
3. Soybean cyst nematode

6. Diseases caused by protozoa

9.3.2.2 II. Noninfectious, or abiotic, plant diseases

They are not associated with any animate or viral pathogen, so they cannot be transmitted from an infected plant to a healthy one. These are due to disturbances in the plant body caused by lack of certain inherent qualities, by improper environmental conditions of soil and air & by mechanical influences. Examples: 1)Low/high temperature, 2) unfavourable oxygen levels,3) unfavourable water levels, 4) hail, 5)wind, 6)air pollution toxicity etc.

1. Diseases caused by too low or too high a temperature

2. Diseases caused by lack or excess of soil moisture

3. Diseases caused by lack or excess of light

4. Diseases caused by lack of oxygen

5. Diseases caused by air pollution

6. Diseases caused by nutrient deficiencies

7. Diseases caused by mineral toxicities

8. Diseases caused by soil acidity or alkalinity (pH)

9. Diseases caused by toxicity of pesticides

10. Diseases caused by improper cultural practices

Check your Progress- 1

Note: a. Write your answer in the space given below

b. Compare your answer with those given at the end of the unit.

1.What is the infectious disease?

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2. What is the non-infectious disease?

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9.3.3 METHODS OF STUDYING PLANT DISEASES

To diagnose a plant disease it is necessary to first determine whether the disease is caused by a pathogen or an environmental factor. In some cases, in which typical Symptoms of a disease or signs of the pathogen are present, it is fairly easy for an experienced person to determine not only whether the disease is caused by a pathogen or an environmental factor, but by which one. Frequently, comparing the symptoms with those given in books that list the known diseases and their causes for specific plant hosts or in books like those of the compendia series of the American Phytopathological Society helps narrow the number of likely causes and often helps identify the cause of the disease. In most cases, however, a detailed examination of the symptoms and an inquiry into characteristics beyond the obvious symptoms are necessary for a correct diagnosis.

Infectious Diseases

In diseases caused by pathogens (fungi, bacteria, parasitic higher plants, nematodes, viruses, mollicutes, and protozoa), a few or large numbers of these pathogens may be present on the surface of the plants (some fungi, bacteria, parasitic higher plants, and nematodes) or inside the plants (most pathogens). The presence of such pathogens on or in a plant indicates that they are probably the cause of the disease. Someone with experience can detect and identify pathogens, in some cases with the naked eye or with a magnifying lens (some fungi, all parasitic higher plants, and some nematodes). More frequently, identification can be accomplished only by microscopic examination (fungi, bacteria, and nematodes). If no such pathogens are present on the surface of a diseased plant, then one must look for additional symptoms and, especially, for pathogens inside the diseased plant. Such pathogens are usually at the margins of the affected tissues, at the vascular tissues, at the base of the plant, and on or in its roots.

Diseases Caused by Parasitic Higher Plants

The presence of a parasitic higher plant (e.g., dodder, mistletoe, witchweed, or broomrape) growing on a plant is sufficient for diagnosis of the disease.

(i) Diseases Caused by Nematodes

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If a plant parasitic nematode is present on, in, or in the rhizosphere of a plant showing certain kinds of symptoms, the nematode may be the pathogen that caused the disease or at least was involved in the production of the disease. If the nematode can be identified as belonging to a species or genus known to cause such a disease, then the diagnosis of the disease can be made with a degree of certainty.

(ii) Diseases Caused by Fungi and Bacteria

When fungal mycelia and spores, or bacteria, are present on the affected area of a diseased plant, two possibilities must be considered: (1) the fungus or bacterium may be the actual cause of the disease or (2) the fungus or bacterium may be one of the many saprophytic fungi or bacteria that can grow on dead plant tissue once the latter has been killed by some other cause, perhaps by even other fungi or bacteria.

(iii) Fungi

To determine whether a fungus found on or in a diseased plant is a pathogen or a saprophyte, one first studies under a microscope the morphology of its mycelium, fruiting structures, and spores. The fungus can then be identified and checked in an appropriate book of mycology or plant pathology to see whether it has been reported to be pathogenic, especially on the plant on which it was found. If the symptoms of the plant correspond to those listed in the book as caused by that particular fungus, then the diagnosis of the disease is, in most cases, considered complete. If no such fungus is known to cause a disease on plants, especially one with symptoms similar to the ones under study, then the fungus found should be considered a saprophyte or, possibly, a previously unreported plant pathogen, and the search for the proof of the cause of the disease must continue. In many cases, neither fruiting structures nor spores are initially present on diseased plant tissue, and therefore no identification of the fungus is possible. For some fungi, special nutrient media are available for selective isolation, identification, or promotion of sporulation. Others need to be incubated under certain temperature, aeration, or light conditions to produce spores. With most fungi, however, fruiting structures and spores are produced in the diseased tissue if the tissue is placed in a glass or plastic "moisture chamber," i.e., a container to which wet paper towels are added to increase the humidity in the air of the container.

(iv)Bacteria and Mollicutes

Diagnosis of a bacterial disease and identification of the causal bacterium is based primarily on the symptoms of the disease, the constant presence of large numbers of bacteria in the affected area, and the absence of any other

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pathogens. Bacteria are small (0.8 by 1mm), however, and although they can be seen with a compound microscope, they all resemble tiny rods and have no distinguishing morphological characteristics for identification. Care must be taken, therefore, to exclude the possibility that the observed bacteria are secondary saprophytes, i.e., bacteria that are growing in tissue killed by some other cause. Selective media are available for the selective cultivation of almost all plant pathogenic bacteria free of common saprophytes so that the genus and even some species can be identified. The easiest and surest way to prove that the observed bacterium is the pathogen is through isolation and growth of the bacterium in pure culture and, using a single colony for reinoculation of a susceptible host plant, reproducing the symptoms of the disease and comparing them with those produced by known species of bacteria. Since the late 1970s, immunodiagnostic techniques, including agglutination and precipitation, fluorescent antibody staining, and enzyme-linked immunosorbent assay, have been used to detect and identify plant pathogenic bacteria. Such techniques are quite sensitive, fairly specific, rapid, and easy to perform, and it is expected that soon standardized, reliable antisera will be available for serodiagnostic assays of plant pathogenic bacteria. Since 1980, newer techniques have been used involving an automated analysis of fatty acid profiles of the bacteria or of the substances utilized by the bacteria for food (Biolog). Additional identification tests include comparison of the number of DNA pieces released by certain restriction enzymes, or degrees (percentages) of hybridization of the DNA of an unknown bacterium with the DNA of a known one. Some of the molecular techniques are now used for the identification of fastidious vascular bacteria. Diseases caused by mollicutes appear as stunting of plants, yellowing or reddening of leaves, proliferation of shoots and roots, production of abnormal flowers, and eventual decline and death of the plant. Mollicutes are small, polymorphic, wall-less bacteria that live in young phloem cells of their hosts; they are generally visible only under an electron microscope and, except for the genus Spiroplasma, cannot be cultured on nutrient media. The diagnosis of such diseases, therefore, is based on symptomatology, graft transmissibility, transmission by certain insect vectors, electron microscopy, sensitivity to tetracycline antibiotics but not to penicillin, sensitivity to moderately high (32–35.8°C) temperatures, and, in a few cases in which specific antisera have been prepared, on serodiagnostic tests.

Diseases Caused by Viruses and Viroids

Many viruses (and viroids) cause distinctive symptoms in their hosts, and so the disease and the virus (or viroid) can be identified quickly by the symptoms. In the many other cases in which this is not possible, however, the diseases are diagnosed and the viruses are identified primarily as follows: (1) through virus transmission tests to specific host plants by sap inoculation or by grafting, and sometimes by certain insect, nematode, fungus, or mite vectors; (2) for viruses for which specific antisera are available, by using serodiagnostic tests, primarily enzyme-linked immunosorbent assays (ELISA), gel diffusion tests, microprecipitin tests, and fluorescent antibody staining; (3) by electron microscopy techniques

such as negative staining of virus particles in leaf dip or purified preparations, or immune-specific electron microscopy (a combination of serodiagnosis and electron microscopy); (4) by microscopic examination of infected cells for specific crystalline or amorphous inclusions, which usually are diagnostic of the group to which the virus belongs; (5) through electrophoretic tests, useful primarily for detection and diagnosis of viroids and of nucleic acids of viruses; and (6) via hybridization of commercially available radioactive DNA complementary to a certain virus DNA or RNA, or viroid RNA, with the DNA or RNA present in plant sap and attached to a membrane filter (immunoblot).

Diseases Caused by More Than One Pathogen

Quite frequently a plant may be attacked by two or more pathogens of the same or different kinds and may develop one or more types of disease symptoms. It is important to recognize the presence of the additional pathogen(s). Once this is ascertained, the diagnosis of the disease(s) and the identification of the pathogen(s) proceed as described earlier for each kind of pathogen.

Noninfectious Diseases

If no pathogen can be found, cultured, or transmitted from a diseased plant, then it must be assumed that the disease is caused by an abiotic environmental factor. The number of environmental factors that can cause disease in plants is almost unlimited, but most of them affect plants by interfering with normal physiological processes. Such interference may be a result of an excess of a toxic substance in the soil or in the air, a lack of an essential substance (water, oxygen, or mineral nutrients), or a result of an extreme in the conditions supporting plant life (temperature, humidity, oxygen, CO2, or light). Some of these effects may be the result of normal conditions (e.g., low temperatures) occurring at the wrong time or of abnormal conditions brought about naturally (flooding or drought) or by the activities of people and their machines (air pollutants, soilcompaction, and weed killers). The specific environmental factor that has caused a disease might be determined by observing a change in the environment, e.g., a flood or an unseasonable frost. Some environmental factors cause specific symptoms on plants that help determine the cause of the malady, but most of them cause nonspecific symptoms that, unless the history of the environmental conditions is known, make it difficult to diagnose the cause accurately.

Identification of a Previously Unknown Disease:

Koch’s Rules (Postulates)

When a pathogen is found on a diseased plant, the pathogen is identified by reference to special manuals; if the pathogen is known to cause such a disease and the diagnostician is confident that no other causal agents are involved, and then the diagnosis of the disease may be considered completed. If, however, the pathogen found seems to be the cause of the disease but no previous reports exist to support this, then the steps

described on under Koch's postulates are taken to verify the hypothesis that the isolated pathogen is the cause of the disease

Check your Progress- 2

Note: a. Write your answer in the space given below

b. Compare your answer with those given at the end of the unit.

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3.How do you identify plant diseases?

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9.3.4KOCH'S POSTULATES

Robert Koch (1843–1910) was a medical doctor and a bacteriologist. He was the first to show, in 1876, that anthrax, a disease of sheep and other animals, including humans, was caused by a bacterium that he called *Bacillus anthracis*. He subsequently discovered, in 1882, that tuberculosis and, in 1883, that cholera are each caused by a different bacterium, which led to the general conclusion that each disease is caused by a specific microbe. These experiments confirmed for the first time the germ theory of disease proposed earlier by Louis Pasteur. Before Koch's experiments, and while Koch himself was carrying out the work on the diseases mentioned earlier, there was confusion and uncertainty about the occurrence and the cause of each disease. Much of the time when bacteria or fungi were isolated from diseased or dead human, animal, or plant tissues, the isolated bacteria or fungi were subsequently shown to be saprophytes, i.e., they coexisted with the microorganism that caused the disease but could not by themselves cause the disease for which they were being considered. Based on his experiences, in 1887, Koch set out the four steps or criteria that must be satisfied before a microorganism isolated from a diseased human, animal, or plant can be considered as the cause of the disease. These four steps, rules, or criteria are known as "Koch's postulates."

1. The suspected causal agent (bacterium or other microorganism) must be present in every diseased organism (e.g., a plant) examined.
2. The suspected causal agent (bacterium, etc.) must be isolated from the diseased host organism (plant) and grown in pure culture.
3. When a pure culture of the suspected causal agent is inoculated into a healthy susceptible host
(Plant), the host must reproduce the specific disease.
4. The same causal agent must be recovered again from the experimentally inoculated and infected host, i.e., the recovered agent must have the same characteristics as the organism in step 2.

Koch's rules are possible to implement, although not always easy to carry out, with such pathogens as fungi, bacteria, parasitic higher plants, nematodes, most viruses and viroids, and the spiroplasmas. These organisms can be isolated and cultured, or can be purified, and they can then be introduced into the plant to see if they cause the disease. With the other pathogens, however, such as some viruses, phytoplasmas, fastidious

phloem-inhabiting bacteria, protozoa, and even some plant pathogenic fungi that are obligate parasites of plants (such as the powdery mildew, downy mildew, and rust fungi), culture or purification of the pathogen is not yet possible and the pathogen often cannot be reintroduced into the plant to reproduce the disease. Thus, with these pathogens, Koch's rules cannot be carried out, and their acceptance as the actual pathogens of the diseases with which they are associated is more or less tentative. In most cases, however, the circumstantial evidence is overwhelming, and it is assumed that further improvement of techniques of isolation, culture, and inoculation of pathogens will someday prove that today's assumptions are justified. However, in the absence of the proof demanded by Koch's rules and as a result of insufficient information, all plant diseases caused by phytoplasmas (e.g., aster yellows) and fastidious vascular bacteria (e.g., Pierce's disease of grape) were for years thought to be caused by viruses. Despite the difficulties of carrying out Koch's postulates with some causal agents, they have been and continue to be applied, sometimes with certain modifications, in all cases of disease. They have had and continue to have a tremendous effect in deciding and in convincing others that a particular microorganism is the cause of a specific disease. By attempting to carry out Koch's postulates in all newly discovered diseases, a great deal of work with potential saprophytes has been avoided, while, at the same time, doubt and criticism are reduced to a minimum while confidence in and use of the identification increase greatly and quickly.

Check your Progress- 3

Note: a. Write your answer in the space given below

b. Compare your answer with those given at the end of the unit.

4. What are the 4 rules of Koch's postulate?

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9.3.5SYMPTOM OF PLANT DISEASES

A disease manifests itself in the form of some typical external and internal changes in the host plant.Such visible changes, abnormalities or signs which serve to recognize the disease in the lost plant are called symptoms of the disease.A diseased plant can easily be distinguished from a normal healthy one on the basis of a symptom. The symptoms provide clues to find out the nature of the disease and the casual agent operating on the host. All the visible symptoms are collectively called syndrome.The symptoms of plant diseases are of following

Anthracnose:

These are circular to angular or irregular spots occurring along the stems, petioles, leaf veins and fruits. The affected tissues are finally killed leaving behind characteristic lesions.

NOTES

Blight:
Blight is a symptom in which the diseased plant is killed suddenly. The pathogen rapidly kills the foliage, blossom and other above-ground parts of the host.

Callus:
This is the outgrowth of tissues in response to wounding.

Cankers:
These are the necrotic lesions, developing in the cortical tissues of stem, leaves or fruits, ultimately resulting in the corky growths in the affected parts.

Chlorosis:
Under this symptom, the normal green pigments of the plant are destroyed and the tissue becomes yellow.

Curling:
Sometimes, the infection of the pathogen results in the abnormal bending of stem, leaves and shoots of the host. This symptom is called 'curling'. It is actually due to the localized overgrowth of cells and tissues.

Damping-off:
In this symptom, the stem of young seedling is affected at the ground level. This results in the toppling down and ultimate death of young seedlings.

Die-back:
Twigs or branches start dying from the tip downward, providing them a burnt appearance in this symptom.

Discolouration:
The yellowing of green parts due to lack of light is called etiolation. But, when it happens due to mineral deficiency, low temperature or infection of pathogens is called chlorosis. When leaves appear transparent due to lack of any pigment is called albinism. Colour conversion to red, purple or orange is called chromosis. Sometimes due to viral or fungal infection yellow and green patches found irregularly called mosaic. The

NOTES

discolouration surrounding leaf veins is called vein banding and the discolouration along leaf veins is called vein clearing.

Dwarfing:
This is the subnormal development of most of the plant parts resulting in the reduction of the size of stem, leaves and ultimately the plant height.

Edema:
The eruption or swelling of the epidermal cells of the infected plant is called "edema' or intumescence.

Elongated Internodes:

This is the abnormal elongation of internodes of the infected plant due to hypertrophy (increase in size of the individual cells) and/or hyperplasia (increase in the number of cells due to cell division).

Etiolation:
his is the under-development of chlorophyll in plants developing in insufficient light.

Exudation (Bleeding and Gummosis):
This symptom results in the exuding of fluids from the diseased tissues. If the thick discoloured fluid flows regularly from the diseased tissue, it is called bleeding. But if a gummy substance oozes out from the diseased tissue and dries as a hard gummy mass, it is called gummosis.

Fasciculation:
If several plant organs, such as stem, leaves, flowers and fruits cluster together around a common focus, the symptom is called 'fasciculation'.

Galls and Tumours:
Hypertrophy results in the formation of some irregular-shaped galls ranging from a few mm to 2 or 3 cm. Relatively large, fleshy or hard galls are called 'tumours'.


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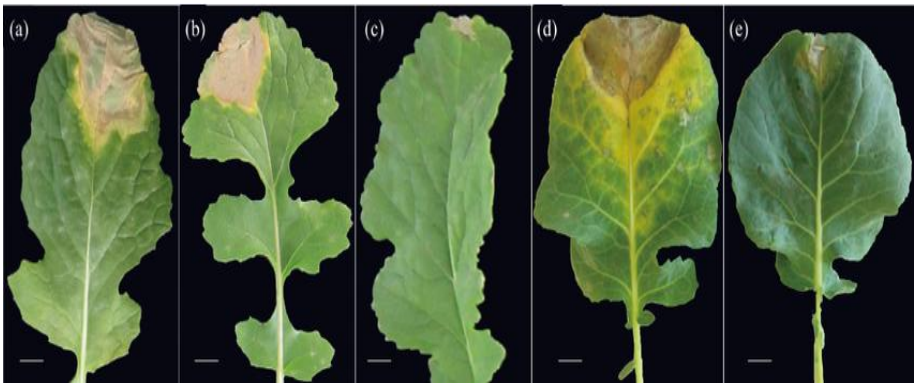
Mosaic
Uneven development of chlorophyll resulting in light green patches alternating with dark green areas is the symptom called ‘mosaic’. Mosaic is usually the symptom of viral infection.

Mummification:
When fruits become dry due to rotting and form a dark, wrinkled, hard mass, the symptom is called ‘mummification’.

Phyllody:
When the infection results in the formation of floral parts (sepals, petals, stamens, etc.) into leaf-like structures, the symptom is called ‘phyllody’.

Prolepsis:
When there is a premature development of shoots from buds, the symptom is called ‘prolepsis’.

Rot:
When the infection leads to the disintegration of the affected tissues, the symptom is called ‘rot’. Based on its nature, it may be soft rot, hard rot, dry rot, black rot, white rot, etc.



Russetting:
When the infection leads to the formation of brownish, superficial roughening of the skin of tubers, fruits, etc., the symptom is called 'russetting'.

Rust Pustules:

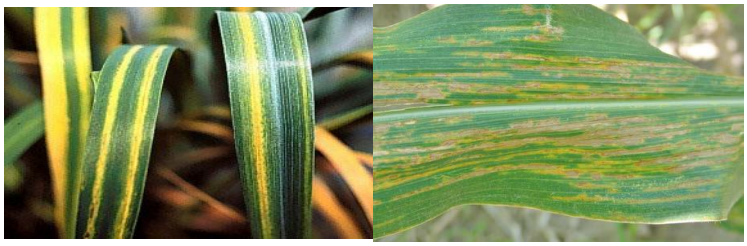
These are the small pustules of spores which may be erumpent or submerged, linear or circular, and are often surrounded by chlorotic areas. Rusty pustules may be yellow, light-brown, dark-brown or red in colour. Often formed by members of Uredinales (e.g. Puccinia), white rust is caused by Albugo while the red rust of tea is caused by an alga Cephaleurosvirescens.

Scab:
When outgrowth of epidermal and cortical cells results in the formation of ulcer-like lesions on tubers, stem, leaves and fruits, the symptom is called 'scab'.

Smuts:
These are the malformations, containing masses of spores which provide the colony a colour of deep brown or dark black to the affected parts, such as stem, leaves, inflorescence and rarely to the underground parts of the host plant.

Spots:
Spots are the necrotic symptoms of different shapes, sizes and colours. They may be isolated or may coalesce in the later stages.

Streaks and Stripes:
The streaks are the linear lesions which develop due to infection on the leaf blade, leaf sheath and stem. The enlarged streaks form stripes.



Variegation:
This is a pattern of white patches formed by the non-development of chlorophyll in certain cells of the host due to the infection of the pathogen.

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Vein-clearing:
This is viral symptom developed due to the inhibition of chlorophyll formation in the veins of the host.

Wilting:
It is withering or drooping of whole plant due to loss of turgidity. It generally caused due to excessive transpiration, injuring to root system, toxins of pathogens etc.

Witche's Broom:
The woody branches of infected tree become swollen from which upward turned shoots and small leaves arise which give broom – like appearance e.g. witches brooms of deodar plant caused by *Peridermiumcedri*.

Mildews:

These are the coloured superficial patches on the host surface due to fungal infection. When the superficial patches appear cottony or downy called downy mudews and when dusty or powdery called powdery mudew appears.

Check your Progress- 4

Note: a. Write your answer in the space given below

b. Compare your answer with those given at the end of the unit.

5. What is Anthracnose?

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9.4 ANSWER TO CHECK YOUR PROGRESS QUESTIONS

1. Infectious diseases are disorders caused by organisms — such as bacteria, viruses, fungi or parasites. Many organisms live in and on our bodies. They're normally harmless or even helpful. But under certain conditions, some organisms may cause disease. Some infectious diseases can be passed from person to person.

2. They are not associated with any animate or viral pathogen, so they cannot be transmitted from an infected plant to a healthy one.

3.A symptom of plant disease is a visible effect of disease on the plant. Symptoms may include a detectable change in color, shape or function of the plant as it responds to the pathogen. Leaf wilting is a typical symptom of verticilium wilt, caused by the fungal plant pathogens *Verticilliumalbo-atrum* and *V. dahliae*.

4. (i)The bacteria must be present in every case of the disease.

(ii) The bacteria must be isolated from the host with the disease and grown in pure culture.

(iii) The specific disease must be reproduced when a pure culture of the bacteria is inoculated into a healthy susceptible host.

(iv)The bacteria must be recoverable from the experimentally infected host.

5. These are circular to angular or irregular spots occurring along the stems, petioles, leaf veins and fruits. The affected tissues are finally killed leaving behind characteristic lesions.

9.5SUMMARY

Plant disease, an impairment of the normal state of a plant that interrupts or modifies its vital functions. All species of plants, wild and cultivated alike,

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are subject to disease. Although each species is susceptible to characteristic diseases, these are, in each case, relatively few in number. There are two types of plant disease has been noted namely Infectious diseases are disorders caused by organisms — such as bacteria, viruses, fungi or parasites and noninfectious disease which are not associated with any animate or viral pathogen, so they cannot be transmitted from an infected plant to a healthy one. There are several methods has been employed to study the plant diasea which including koch's postulates methods. The bacteria must be present in every case of the disease. There are four rules of Koch's postulate such as (i)The bacteria must be present in every case of the disease. (ii) The bacteria must be isolated from the host with the disease and grown in pure culture.(iii) The specific disease must be reproduced when a pure culture of the bacteria is inoculated into a healthy susceptible host.(iv)The bacteria must be recoverable from the experimentally infected host.

A diseased plant can easily be distinguished from a normal healthy one on the basis of a symptom. The symptoms provide clues to find out the nature of the disease and the casual agent operating on the host. All the visible symptoms are collectively called syndrome.The symptoms of plant diseases are of followingsuch as Anthracnose, Blight, Callus, Cankers, Chlorosis, Curling, Damping-off, Die-back, Discolouration, Dwarfing, Edema, Elongated Internodes, Etiolation, Exudation, Fasciculation, Galls and Tumours, Mosaic, Mummification, Phyllody, Prolepsis and Others.

9.6KEY WORDS

Chlorosis: the failure to accumulate or maintain the normal complement of chlorophyll resulting in an abnormal yellow appearance.

Canker: dead places on bark and cortex of twigs or stems; often discolored and raised or sunken.

Damping-off: destruction of seeds in the soil, or seedlings near the soil line, resulting in reduced stand, or the seedling falling over on the ground.

Mildew: a plant disease in which the pathogen is seen as a growth on the surface of the host; e.g., downy mildew, powdery mildew, caused by very different fungi, but both having the name Mildew.

Mosaic: symptom of certain viral diseases of plants characterized by intermingling patches of normal green and light green or yellowish colors.

Vein clearing: destruction of chlorophyll adjacent or in the vein tissue as a result of infection by a virus or other pathogen.

Wilt: loss of rigidity and drooping of plant parts generally caused by insufficient water in the plant.

Witches' broom: broom-like growth or massed proliferation caused by the dense clustering of branches in woody plants.

9.7SELF ASSESSMENT QUESTION AND EXERCISES

1. What are the symptoms of bacterial disease in plants?
2. What is infectious and non-infectious disease and examples?
3. Define Mildew.
4. Comments on Koch's postulates
5. Write essay on symptoms of plant disease.

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9.8FURTHER READING

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UNIT – 10 ETIOLOGY

Structure

10.1 INTRODUCTION

Etiology is the cause of a disease or the science that deals with such causes. The word etiology comes from the Greek *etio-*, which means 'causation' and *-ology*, which refers to the scientific study of something. A disease's etiology, or cause, generally falls into three main categories; intrinsic, extrinsic and idiopathic.

Intrinsic - means coming from within. Therefore, any pathological, or disease-causing, change that has occurred from inside the body has occurred as a result of intrinsic factors. Example: Inherited conditions, or conditions that are passed down to you from your parents. An example of this is hemophilia, a disorder that leads to excessive bleeding. The second category of disease etiology is extrinsic etiologies. This means the cause of the disease, or pathological change, came from outside of the body. Example: Infectious agents like bacteria, viruses, fungi, and parasites. Idiopathic means unknown cause.

10.2 OBJECTIVES

After going through the unit you will be able to:

1. Understanding the plant diseases and their etiology
2. Understanding plant disease epidemics and forcasting

10.3 ETIOLOGY

10.3.1 Introduction to Etiology

Etiology is the study of causation, or origination. The word is derived from the Greek aitiología, "giving a reason for" (aitía, "cause"; and -logía). More completely, etiology is the study of the causes, origins, or reasons behind the way that things are, or the way they function, or it can refer to the causes themselves. The word is commonly used in medicine, (where it is a branch of medicine studying causes of disease) and in philosophy, but also in physics, psychology, government, geography, spatial analysis, theology, and biology, in reference to the causes or origins of various phenomena.

In the past, when many physical phenomena were not well understood or when histories were not recorded, myths often arose to provide etiologies. Thus, an etiological myth, or origin myth, is a myth that has arisen, been told over time or written to explain the origins of various social or natural phenomena. For example, Virgil's Aeneid is a national myth written to explain and glorify the origins of the Roman Empire. In theology, many religions have creation myths explaining the origins of the world or its relationship to believers.

10.3.2 PLANT DISEASE EPIDEMICS

The epidemiology of diseases is the study of the outbreak of disease, its course, its intensity causes and effects, and the various factors governing it. When a disease affects single or isolated individuals within a population, its occurrence is termed sporadic. On the other hand, when the disease shows a concentration either in time or space, it is known as an epidemic or epiphytotic. When as epidemic is periodic and appears after certain intervals of time, it is termed cyclic. On the other hand, when an epidemic extends over most of a continent and causes mass mortality, it is termed as pandemic.

10.3.2.1 The form of epidemics

The form of an epidemics may be defined quantitatively, i.e., the incidence of the disease, and qualitatively, i.e., the severity of the disease. The form of epidemics has two extreme types- explosive epidemics and tardy epidemics. The graph of the former type shows a steep rise, a short acute peak, and a steep decline, whereas that of latter shows slow progress.

10.3.2.2 Annual fluctuations of epidemics:

In temperate climates, most epiphytotics in which the pathogen lacks a perennating mycelium shows an annual cycle or periodicity. Annual rhythms become more complicated in those infectious disease which attack several organs, and therefore develop in several ways. Thus, in downy mildew of vine (*Plasmopara viticola*), the foliage and first buds are attacked where as in late blight of potato (*Phytophthora infestans*), the foliage and the tubers are attacked.

10.3.2.3 Secular fluctuations of epidemics:

The secular or long-term periodicity of progressive epidemics differs from that of endemic epidemics. Progressive epidemics (epiphytotics) usually develop bilaterally. During first year it increase destructively, but later it settles down to a constant value, e.g., powdery mildew of vine (*Uncinula necator*) in France.

10.3.2.4 Factors responsible for the establishment of an epidemic:

There are several important factors under which an infection gives rise to an epidemic. Such conditions or factors must occur simultaneously in both the pathogen and the environment. There may be three categories of these factors:

10.3.2.4.1 (A) Nature of the host

(i) Accumulation of susceptible individuals; (ii) heightened disease proneness of the host; (iii) the presence of appropriate alternate hosts.

10.3.2.4.2 (B) Nature of the pathogen

(iv) the presence of an aggressive pathogen; (v) high reproductive capacity of pathogen; (vi) efficient dispersal of pathogen and (vii) unexacting growth requirements.

10.3.2.4.3 (C) Environment

(viii) optimal weather conditions for the development of the pathogen.

When all the above mentioned eight factors are realized in a particular locality at one and same time, the epidemic is resulted. These

eight factors which are responsible for the establishment of an epidemic are being discussed here separately.

10.3.2.4.4 (i) **Accumulation of susceptible individuals:**

With increasing distance from the source of infection the probability of infection reduces sufficiently, and therefore, an epidemic would rarely breakout in host plants which occurs as isolated individuals. The accumulation of susceptible plants in widespread localities is an important factor favoring epidemics. The destructive epidemic of 'Tikka disease of Groundnut' in Maharashtra during 1912-13, and the blast of rice during 1948-49 in the parts of same state, were mainly because of cultivation of local varieties.

10.3.2.4.5 (ii) **Heightened disease proneness of host:**

The development of an epidemic is promoted by heightened disease proneness of host which may be due either to innate or to environmental factors. For example, the susceptibility of potato plants to late blight (*Phytophthora infestans*) increases with the age of the plant and with the growing season. In case of potato blight, the resistance decreases during the vegetative period, whereas in the case of leaf curl of peaches, it increases during the vegetative period.

The environmental factors which heighten disease proneness can be arranged in three groups: epidemiologically, the susceptibility of host increased by (a) general lowering of host's vitality, (b) the external temperature and (c) nutrition.

10.3.2.4.6 (iii) **Presence of appropriate alternate hosts:**

The presence of appropriate alternate host is a condition for an epidemic only in those diseases with heterogeneous disease-cycles, e.g., black rust of wheat, blister rust of pine (*Cronartium ribicola*) and certain viral diseases such as leaf roll of potato.

10.3.2.4.7 (iv) Presence of an aggressive pathogen:

The infection of plant makes an essential condition for an epidemic. In the absence of pathogen, no infectious disease or epidemic can break out even when all other conditions are fulfilled. But only pathogen does not make a condition for an epidemic. An epidemic occurs when all the factors coincide in time and place.

Sometimes, the flora of a given region may not contain a particular pathogen, but this may be introduced. Secondly, the distribution of a cultivated plant may be extended artificially by world trade and thus its native parasites may be left behind. In other cases, a cultivated plant temporarily evades its parasites because resistant varieties are developed.

Occasionally, a cultivated plant in a new area is succumbed to a new disease because of the attack of an indigenous parasite. The large scale cultivation of agricultural plants all over the world forms a reservoir of disease from a wide variety of hosts.

Sometimes, a pathogen increases its parasitic faculties and becomes the beginning of a new epidemic. A new race, i.e., a new pathogen, may arise from a parasitic strain through change of habit, hybridization or mutation. This change may be temporary or permanent and may increase the epidemic.

10.3.2.4.8 (v) **High reproductive capacity of the pathogen:**

The pathogens of high reproductive capacity can give rise to an epidemic. The reproductive capacity of a pathogen may be determined by certain factors. They are follows:

10.3.2.5 Fecundity of pathogen: only fungi of high reproductive capacity of pathogen can give rise to epidemics. For example, *Corticium vagum* which rarely sporulates, cannot give rise to progressive epidemics. On the other hand, pathogens of the type of powdery and downy mildews, rusts of wheat, potato blight, rice blast, etc., are most suitable for an epidemic.

10.3.2.6 Numerical threshold of infection:

For example, if one smut spore of Ustilago tritici is carried to a distance of half kilometre by the wind where it falls on a wheat stigma, and germinates there, it is quite unimportant epidemiologically, since it cannot give rise to infection directly. On the other hand, even single spore infection with uredospores of *Puccinia graminis* on cereal leaves can be effective.

10.3.2.7 Rhythm of successive generations:

The length of the life-cycle of pathogen, also known as rhythm of successive generations, is an important factor for the outbreak of an epidemic. The rate of succession of generations in any pathogen is dertmined by the time required for the production of fructifications, and the formation and germination of spores. The time required for the formation of spores should be small for the outbreak of any epidemic. For example, powdery mildews, potato blight, downy mildews and cereal rusts are quite efficient for the outbreak of an epidemic, because the spores of successive generations are formed in very short period of time, i.e., about a week or so.

Similarly for an epidemic, the speed of formation of propagative spores, and the speed of germination of propagative spores, with least resting period should be high.

10.3.2.8 (vi) **Ready dissemination of pathogen**:

The efficient dispersal of the propagative units of the pathogen also play an important role in the outbreak of an epidemic. In the case of fungi, the transmission is effected mostly by wind, whereas in bacteria and virus by means of insects. In fungi, the conidia, oidia, uredospores, etc., must be physically capable of being transported by the wind and biologically able to survive it. In such cases, only those spores are capable of causing an epidemic which are formed on the external surface of the host. The length pf life and resistance of propagative spores to environmental factors are low in comparison with resting spores. Under natural conditions, the oidia of the powdery mildews of wheat and barely lose their power of infection only after 48 hours. However, they can retain their power to infect in laboratory conditions with high humidity (95%) and low temperature (0^{o} C for 7 days in the case of wheat mildew and 51 days in the case of barely mildew.

10.3.2.9 (vii) **Unexacting growth requirements of pathogen**:

Epidemiogenic pathogens are like those weeds which never die out. The pathogens can produce epidemics because they are not selective and can survive in any type of ecological conditions.

10.3.2.10 (viii) **Optimal weather condition (Meteoropathology)**: meteoropathology is the study of the relation between weather condition and epidemics. Thus, the environment in meteorological conditions plays an important role and determines the course, and severity of epidemics. The fluctuations in weather conditions are mainly responsible for an epidemic.

In most of cases, the epidemiological conditions for infection and reproduction are alike, being favoured by high atmospheric humidity, dew, intermittent rains, mist, etc. The mycelium inside the leaf ceases to grow at temperature below 10-12^{o} C and above 30^{o}C. The optimum air temperature for the spread of epidemics lies between 22-24^{o}C.

The development and abstraction of conidia are also favored by high air temperature and humidity in downy mildews, whereas in powdery mildews both the number of spores and their germiability are greater if they are developed in bright sunlight than in rainy, dull weather.

10.3.2.11 (ix) **Cause of the progressive severity of epidemics**:

The persistence and momentum of annual and secular epidemic increase both in quality and quantity of the disease. This depends on two main causes. The plant epidemics usually start from local infection, and in subsequent links of the chain, the number of spores or germs increase in geometrical progression. As the inoculum increases, the resistance is broken and disease becomes general. On the other hands, as the amount of spore materials grows, it becomes easier for the pathogen to start infections, and ultimately the incidence and the severity of the disease increase together to a maximum. In addition to the increase in number of pathogen, increase in virulence of individual parasitic strains, and increase in virulence of parasitic populations are also needed for progressive epidemic.

10.3.2.12 Conditions for decline of epidemic

After reaching the peak, every epidemic subsides. In the case of annual epidemics, the climatic factors are chiefly responsible for its decline. For instance, in the late summer a few cool nights are sufficient to check the further increment of the pathogen.

To arrest the secular epidemics, three important factors are responsible. They are as follows:

10.3.2.13 Saturation of host population by disease: when an epidemic occurs it eliminates susceptible individuals and only the resistant individuals of the population survive, which may breed further.

10.3.2.14 Reduction in disease proneness of host plants: the disease proneness of a host is reduced when it becomes immune. For instance, the annual epidemic of black rust of wheat is favored by the rise of temperature in summer, and it should decline again as the maximum summer temperatures are passed, but by that time the crop is being harvested.

10.3.2.15 Decreased in vitality of the pathogen: by and by the aggressiveness of the pathogen is begin reduced. As the plants stock becomes saturated by disease, the susceptible individuals are eliminated, and the pathogen tends to parasitize the more resistant plants of the same species. But in most of the cases, the pathogen loses its vitality and ultimately dies out.

As the adverse conditions of the pathogen increase, not only the vitality of the spores, but also their number tends to diminish. Because of the regression of spore formation and dissemination, ultimately a lower limiting value is reached, and the epidemic declines. Thus, when an epiphytotic changes in character and subsides, both host and parasite are responsible for the event.

Check your Progress- 2

Note: 1. What is etiology?

2. What are the plant disease epidemics?

3. What is plant disease forcasting?

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10.3.3 FORECASTING OF PLANT DISEASES

Forecasting of plant diseases is predicting the occurrence of disease in an epi-phytotic form in a particular area. Plant diseases vary in incidence from season to season due to differences in the nature and amount of inoculum, environmental condi-tions, numbers and activity of vectors, and other factors which affect the development and spread of pathogens.

Reliable forecasting of the likely incidence of plant diseases can save a great deal of money provided it can be done sufficiently early to organize effective control measures. This will also avoid wasting time and money on unnecessary control measures. Early forecasting gives crop growers sufficient time to rearrange their crop schedules and to avoid susceptible crop in a season when disease is likely to be severe.

The aim of disease forecasting should also be to arrange control measures before the inoculum is likely to infect the crop. It is thus very important that forecasts should be correct so that an effective disease warning system is reliable to the crop growers.

10.3.3.1 Forecasting Methods:

A rational method of disease forecasting method should be based on:

(i) Factors (microclimatic) which influence the initial appearance and subsequent spread of inoculum

(ii) Thorough knowledge of the life cycle of the pathogen

(iii) The ways in which the pathogen perennates

(iv) Rough estimate of the quantities of inoculum expected to be disseminated through propagating stock, soil, air, vectors, etc.

(v) Mechanism of host infection

(vi) Knowledge of the sus-ceptibility of the host plant at different stages of growth

(vii) Meteorological data (macroclimatic conditions) of the area.

10.3.3.2 Methods of disease forecasting may be:

(i) empirical, in which correlation bet-ween the results of disease surveys and the corresponding weather conditions in a particular area has to be related to the biology of host plant and pathogen.

(ii) fundamental, in which the effects of different weather conditions (moisture, tem-perature, etc.) on plant and pathogen, separately and together, are investigated in the laboratory and conclusions tested under field conditions. Besides these, many methods are based on a survey of the viable inoculum available at the beginning of the crop season.

These methods provide an early warning but have the disadvantage that many other factors may subsequently modify the course of expected outbreak, e.g., unsuitable weather may prevent disease development despite the presence of ample inoculum.

Again other forecasting systems are based on weather conditions during the crop season, but these assume the presence of sufficient inoculum and will be irrelevant if this is not available. Forecasting systems should therefore take account of inoculum sources as well as the weather conditions which affect the deve-lopment and dissemination of inoculum.

Hence forecasting methods should be basi-cally based on: (i) Weather conditions during the intercrop period, particularly as affects survival of inoculum; (ii) Weather conditions during the crop season; (iii) The amount of disease in the young crop; and (iv) The nature of propagating organs of the pathogen in the air, soil, or planting material.

Weather conditions during the intercrop period are usually related to survival of the pathogen or its vectors bet-ween crops. Intercrop weather which reduces pathogen or its vectors is likely to mini-mize inoculum sources for the following crop. Weather conditions during the crop season are important in the development and spread of inoculum.

The effects of weather conditions on plant diseases are complex, influencing not only the events of the infection cycle, but also the resistance of the plant, its ability to throw off or survive attacks, and the numbers and activity of any vectors of the pathogen.

Hence the results of laboratory experiments on the pathogens are not always a completely reliable indication as to what happens under more varied conditions of the field.

Detailed observations over a number of years may be necessary before forecasting systems based on weather conditions can be prepared. Some diseases originate from inoculum blown in from distant sources and information on the incidence of the disease in such areas, if known can be useful in forecasting the date and severity of the disease in the area expected to receive inoculum. This may be supplemented by spore trapping to determine when the inoculum will be arriving in large quantities. Such study should be supported by the knowledge of the circulation of the air currents which is responsible for the transport of inoculum.

The distribution and concentra-tion of soil borne pathogens can be estimated by suitable laboratory experiments and the data collected may be utilized for both forecasting and controlling of diseases. Some pathogens are partly or exclusively seed-borne and the degree of seed-borne infection can be estimated in the laboratory by germinating seeds under conditions suitable for disease development. According to virulence of infection or contamina-tion the propagating materials may be rejected or treated to destroy contaminating pathogen to render them safe for sowing. In such a case both forecasting and control are combined.

10.3.4 PLANT DISEASE BASICS: THE DISEASE TRIANGLE

Plant diseases can be analysed conveniently using the concept called the 'Disease Triangle'. This places the three factors which must interact to cause plant disease at the three corners of a triangle. Those three factors are:

- **Susceptible host**,
- **Disease causing organism** (the pathogen)
- **Favourable environment** for disease.

The **host** is the plant itself; some can fall victim to many diseases, others only suffer particular ones. So all plants have a range of susceptibilities to a range of diseases. The **pathogen** is the disease. Diseases of plants are most often caused by fungi but there ***are*** some plant pathogenic bacteria and viruses.

Without the right host in the right conditions, pathogens cannot cause any harm. Some pathogens are specific to only one or a few host plants, others have broad abilities to attack almost everything. The **favourable environment** essentially means the weather conditions needed for a pathogen to thrive (this is an important point; it's 'a favourable environment for disease' and if the pathogen is present and disease results, it's obviously an **unfavourable** environment for the plant).

Disease results only if all of these three things occur simultaneously; if one or more of the factors is not present, then disease does not occur. The disease triangle was probably first recognised at the beginning of the 20th century and it has become one of the paradigms of plant pathology. It holds a position in plant pathology rather similar to that held by Ohm's Law (which relates current, resistance, and voltage) in electrical and electronic engineering.

It is a paradigm because occurrence of a disease caused by a biological agent **absolutely requires** the interaction of a susceptible host with a virulent pathogen under environmental conditions favourable for disease development. The mechanisms that contribute to pathogenesis can all be thought of as modifying the disease triangle by reducing or eliminating one of the corners of the triangle. Examples (from among many) include:

- the lack of defences in the host,
- efficient spore dispersal by the pathogen,
- weather conditions favouring spore production, etc.

Methods of disease control (again from among many) include:

- breeding for resistance in the host,
- applying pesticide to hinder the pathogen,
- irrigating to relieve water stress.

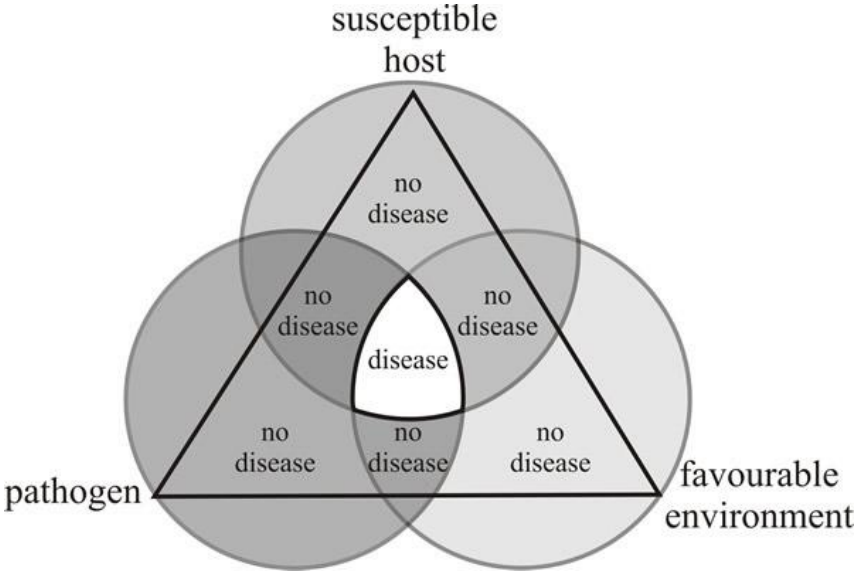


Figure. The disease triangle illustrating the phenomenon of plant disease as the interior space of a triangle with the three essential factors (susceptible host, favourable environment for disease, and pathogen) at the vertices.

It is usually stated that this triangular relationship is unique to plant pathology because the immobility of plants prevents them escaping from inhospitable environments, plants have little thermal storage capacity and are therefore subject to temperature stress much more than animals (even poikilothermic animals can 'bask in the sun' or retire to the shade as appropriate), and the immune system of vertebrates arms them with sophisticated mechanisms to recognise and neutralise pathogens. Also, the predominance of fungi in causing plant diseases is held to reinforce the uniqueness of the plant disease triangle because fungi are also highly dependent on environmental conditions.

However, this triangular relationship is only unique to plants if you ignore the fact that members of kingdom Fungi also suffer disease, and the severity of that disease also depends on the three essential factors: a susceptible host in an environment favourable for disease challenged by a virulent pathogen.

Some plant pathologists have suggested elaborating the disease triangle by adding additional parameters, such as human activities, disease vectors, and time. Humans contribute to the disease triangle because human activity in agriculture is pervasive and, if you think about it, impacts on all three factors so far discussed, so can profoundly affect the occurrence and severity of plant diseases. This means that humans are already represented implicitly in the basic triangle configuration and this is the main counterargument against including human activity as a new vertex in a 'disease rectangle'.

Animal and other vectors are not essential to all plant diseases even though they play a critical role in many. Vectors are therefore only worth including in those special cases, where the triangular relationship can be modified by placing the vector on the disease triangle side that connects the host and pathogen vertices; this arrangement emphasises the dependence of the pathogen on its vector.

Time is an essential dimension and has been added to the disease triangle by several authors, primarily to convey the idea that disease onset and intensity are affected by the duration that the three prime factors are aligned. Some duration of favourable alignment is necessary for disease to occur; but the length of time depends on your level of analysis. Physiological events in the host that define infection can take place in minutes or hours; disease symptoms in the field can take days or weeks to appear. Showing time as a dimension on the triangle (perhaps converting it into a pyramid) could be a more realistic adaptation of the diagram.

10.3.5 DISEASE CYCLES

In order for a disease to develop, a pathogen must be present and successfully invade plant host tissues and cells. The chain of events involved in disease development includes inoculation, penetration, infection, incubation, reproduction, and survival (Figure).

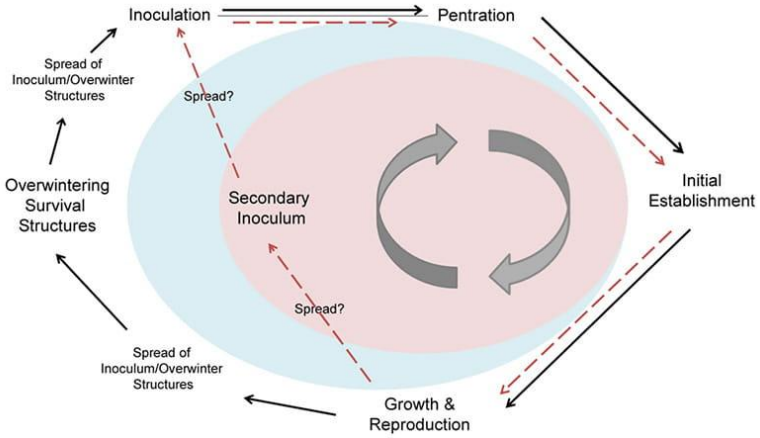


Figure. The monocyclic pathogen follows the black arrows to complete its cycle. Polycyclic pathogens follow the red arrows for the majority of the season and the black arrows at the end of the season.

10.3.5.1 Stages of Development

Inoculation:

This describes the introduction of the plant pathogen to the host. Different pathogen groups employ different inoculation methods and are equipped with various specialized mechanisms that aid in the inoculation process. For example, some fungal pathogens release spores into the air and the spores are then spread with the aid of air currents.

Penetration:

Wound sites and natural plant openings, such as stomata and hydathodes, facilitate the entrance of some plant pathogens; others have evolved unique mechanisms for direct penetration. Fungi and nematodes are able to actively penetrate host tissues and cells if environmental conditions, such as moisture and temperature, are favorable for the penetration process.

Infection:

This occurs when the pathogen invades the plant tissue and establishes a parasitic relationship between itself and the plant. Viruses, bacteria, and phytoplasmas are not able to actively penetrate or enter plant

host tissues. Therefore they must rely on other methods to infect plant tissues and cells. Associations with insect vectors have been established by these pathogens to aid inoculation and dispersal.

Incubation

Once inside the plant, pathogens may undergo an incubation period and remain latent for a period of time before initiating disease.

10.3.5.2 Reproduction

Plant pathogens can reproduce sexually and asexually. It is dependent on the pathogen.

10.3.5.3 Survival

Plant pathogens have evolved so they can survive prolonged periods of unfavorable weather conditions. For example, brown spot is a fungal pathogen that produce spores that are dark in coloration which reduces the amount of UV light penetrating and preventing cell death. In addition, Soybean cyst nematode lay their eggs within a cuticle casing. The cuticle casing is very hard and prevents other microbes and chemicals to penetrate killing the eggs prior to hatching.

If any step is disturbed in the cycle, the disease will be less severe or fail to develop. Knowing and understanding the disease cycle for a particular disease is very helpful in managing the disease. There are two types of disease cycles, monocyclic and polycyclic.

10.3.5.3.1 Monocyclic Plant Disease:

When pathogens are able to complete only one or part of disease cycle in one year, they are called monocyclic pathogens and the disease as monocyclic dis-ease. In monocyclic disease, pathogen develops primary inoculum which is the only inoculum available for the entire season. The secondary inoculum and secondary infection are totally absent in such diseases, e.g., smuts (loose smut of wheat, c.o. *Ustilago segetum*), root rot (root rot of turmeric, c.o. *Pythium aphanidermatum*) and vascular wilt (wilt of pigeon pea, c.o. *Fusarium udum*), etc.

10.3.5.3.2 Polycyclic Plant Disease:

When pathogens are able to complete more than one generation (2-30) in a growing season, they are called poly-cyclic pathogens and the disease as polycyclic disease. The amount of inoculum in each cycle is multiplied manyfold, e.g., leaf spot (brown spot disease of rice, c.o. *Helminthosporium oryzae*), blight (late blight of potato, c.o. *Phytophthora infestans*), powdery mildews (powdery mildew disease of cucurbits, c.o. *Erysiphe cichoracearum*), etc.

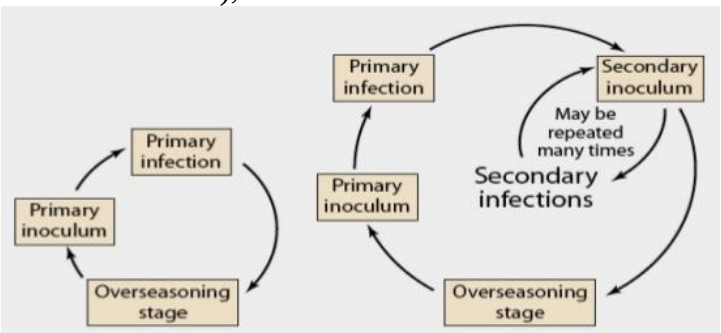


Figure. Diagrams of (left) monocyclic and (right) polycyclic plant diseases. Monocyclic disease lack secondary inoculum and secondary infection during the same year in polycyclic plant diseases.

10.4 Answer to Check Your Progress Questions

1**.** Etiology is the cause of a disease or the science that deals with such causes. The word etiology comes from the Greek *etio-*, which means 'causation' and *-ology*, which refers to the scientific study of something.
2. Plant disease epidemiology is the study of disease in plant populations. Much like diseases of humans and other animals, plant diseases occur due to pathogens such as bacteria, viruses, fungi, oomycetes, nematodes, phytoplasmas, protozoa, and parasitic plants
3. Forecasting of plant diseases is predicting the occurrence of disease in an epi-phytotic form in a particular area. Plant diseases vary in incidence from season to season due to differences in the nature and amount of inoculum, environmental condi-tions, numbers and activity of vectors, and other factors which affect the development and spread of pathogens.

10.5 Summary

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. For example, 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.

In 1963 J. E. van der Plank published "Plant Diseases: Epidemics and Control", a seminal work that created a theoretical framework for the study of the epidemiology of plant diseases.This book provides a theoretical framework based on experiments in many different host pathogen systems and moved the study of plant disease epidemiology forward rapidly, especially for fungal foliar pathogens. Using this framework we can now model and determine thresholds for epidemics that take place in a homogeneous environment such as a mono-cultural crop field.

10.6 Key Words

Etiology: Etiology is the cause of a disease or the science that deals with such causes.
Pathogenicity: Pathogenicity is the potential disease-causing capacity of pathogens
Epidemic: An epidemic is the rapid spread of infectious disease to a large number of people in a given population within a short period of time, usually two weeks or less.

Disease Triangle: The disease triangle is a conceptual model that shows the interactions between the environment, the host and an infectious (or abiotic) agent. This model can be used to predict epidemiological outcomes in plant health and public health, both in local and global communities.
Disease cycle: In order for a disease to develop, a pathogen must be present and successfully invade plant host tissues and cells. The chain of events involved in disease development includes inoculation, penetration, infection, incubation, reproduction, and survival.

10.7 Self Assessment Question and Exercises

1. What is plant pathology?
2. Why is Plant Pathology Important?
3. What are the factors responsible for the establishment of an Epidemic?
4. Types of plant forcasting methods?

UNIT – 11 HOST-PATHOGEN INTERACTION

Structure

11.1 INTRODUCTION

Plant-pathogen interaction is a multifaceted process, mediated by the pathogen-and plant derived molecules which mainly include proteins, sugars and lipopolysacchatides. Secretd molecules, derived from the pathogen, are the key factors which determine their pathogenicity and allow their successful colonization inside the host. On the other hand, plant derived molecules are involved in the recognition of these pathogens in order to elicit the defense response. The recognition may lead to activation of the host defence mechanisms that are able to prevent the infection. A successful pathogen needs to suppress the host's immune responses during the infection. In this unit, author have discussed about the host-pathogen interaction particulary infection process can be broadly divided into

following three phases: adhesion, penetration, colonisation and defense mechanisms in plants including induced physical or structural or histological barriers and induced biochemical changes.

11.2 OBJECTIVITIES

After going through the unit you will be able to:

1. Understanding the strategies plants have evolved to cope with pathogens and
2. Understanding plant defence mechanism against pathogens.

11.3 HOST-PATHOGEN INTERACTIONS

11.3.1 Introduction to Host-pathogen Interactions

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.

11.3.2 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:

(1) Adhesion
(2) Penetration
(3) 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.

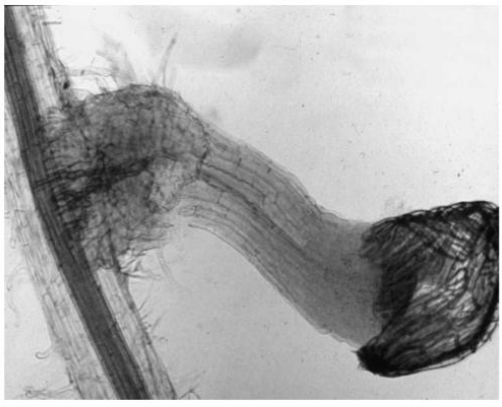
11.3.3 Adhesion

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 pathogens from being displaced by not being blown or rinsed by water and/or wind from a potentially suitable environment.

1. It is required for host penetration via mechanical pressure,
2. It is required for thigmo differentiation.
3. It is required for thigmo tropism.
4. It facilitates interaction between pathogen and host.
5. It increases the surface area of contact with its host.
6. 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 specialized penetration structures, such as appressoria, while others utilize 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 specialized penetration structure, the appressorium (Fig.1). Sugars, amino acids and minerals secreted by plants at the leaf surface can nonspecifically trigger spore germination or provide nutrition for the pathogen. Some pathogenic spores will not germinate in the absence of these substances. 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.



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

11.3.4 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) 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.

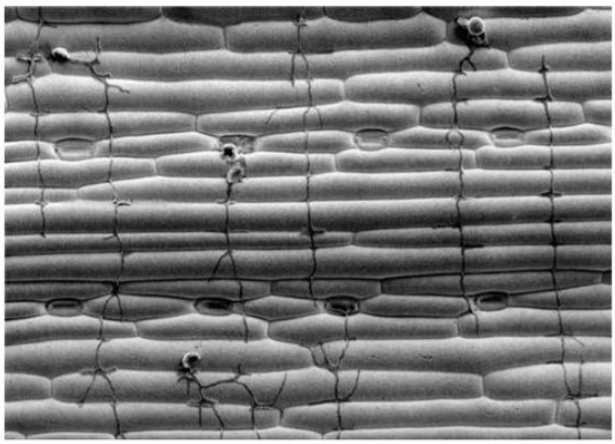


Fig: Germ tubes of P. graminis f. sp. tritcii orienting themselves at the grooves on the host surface corresponding to the epidermal cell junctions.

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.

11.3.4.1 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.

Pathogen Entry through Natural Opening

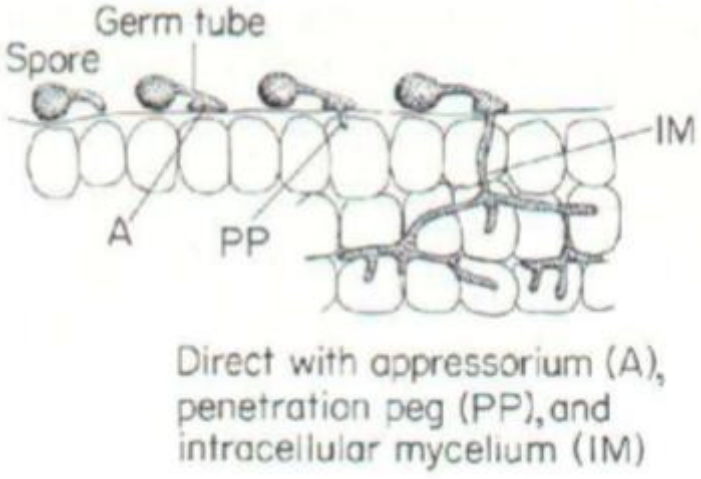
11.3.4.2 Mechanical Forces

11.3.4.2.1 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.



Steps of Pathogen Spore Germination on host Tissue

11.3.4.3 Chemical Forces

11.3.4.3.1 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.

11.3.4.3.1.1 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.

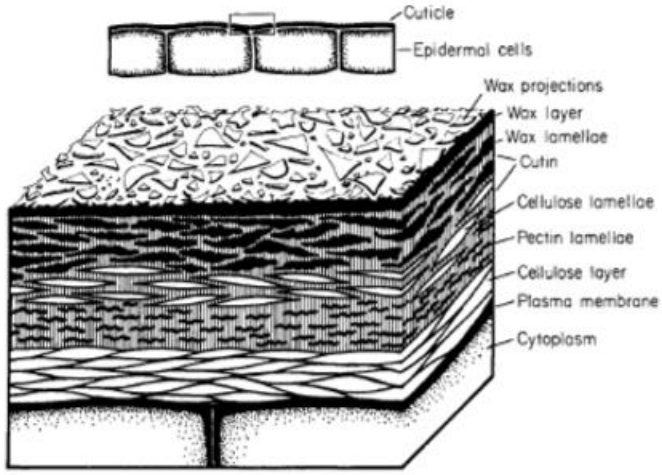


Fig. Schematic Representation of Structure and Composition of Cuticle of Leaf Epidermal Cells

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 crosslinkages 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.

11.3.4.3.1.2 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 –

1. are extremely poisonous
2. are very effective in low concentrations
3. can potentially injure host cells
4. can very seriously affect membrane permeability
5. have the ability to deactivate or inhibit plant enzymes
6. 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.

11.3.4.3.1.3 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 disease 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.

11.3.4.3.1.4 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.

11.3.5 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. Airborne 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.

Check your Progress- 1

Note: a. Write your answer in the space given below

b. Compare your answer with those given at the end of the unit.

1. How can pathogens enter a plant?

.……

…….

11.4 DEFENSE MECHANISMS IN PLANTS

11.4.1 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.

11.4.2 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). 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

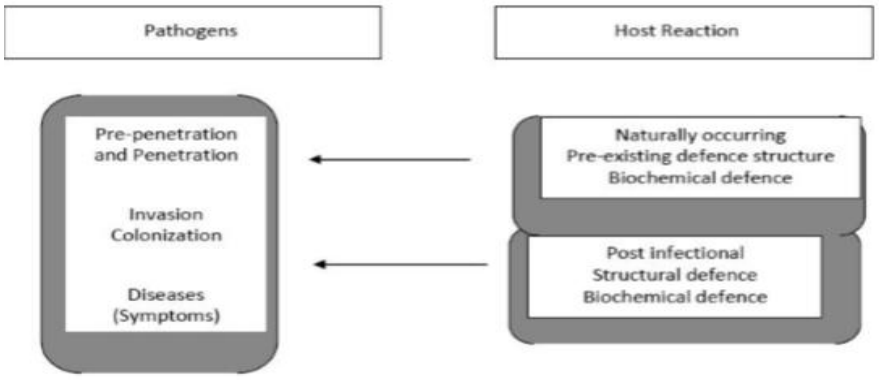


Fig: Defense Mechanisms: Pre-infective or Post infective

11.4.3 Pre existing or Pre-Infective Defense Mechanism

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.

a) 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.

b) 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.

c) 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.

d) 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.

11.4.4 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.

a) 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.

b) 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.

c) 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.

d) 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.

e) 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.

11.4.5 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-

1. Induced Physical or Structural Defense
2. 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.

11.4.5.1 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.

a) 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.

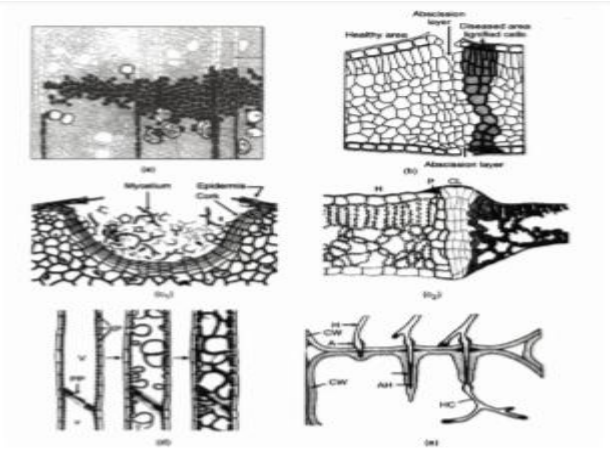
b) Suberization: In several plants, as soon as the infection occurs in the cells, the infected cells become surrounded by suberized cells. This isolates

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.

c) 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. Shot holes in leaves of fruit trees are a common feature.

d) 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. 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.

e) 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, resulting in the death or starvation of infective propagules.



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

11.4.5.2 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:

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

ii. The substance can be isolated from the host showing protection against the disease.

iii. Introduction of isolated substance to the appropriate susceptible host confers protection.

iv. The nature of protection so induced resembles that of the natural agents of a resistant plant.

a) 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.

b) 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).

c) 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.

d) **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, phenoloxidizing 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.

e) 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.

f) 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.

g) 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 hostpathogen 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.

Check your Progress- 2

Note: a. Write your answer in the space given below

b. Compare your answer with those given at the end of the unit.

3. What is Tyloses?

……

……

11.5 ANSWER TO CHECK YOUR PROGRESS QUESTIONS

1. Bacteria can be sucked into a plant through natural plant openings such as stomata, hydathodes or lenticels. They can enter through abrasions or wounds on leaves, stems or roots or through placement by specific feeding insects.

2. 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. 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

11.6 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, the physical and chemical characteristics of these are first discussed. 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.

Plant have different defence mechanism to overcome the pathogen invasion. In plants some structures are already present to defend the attack while in others, the structures to defend the host develops after the infection. In this way, structural defense can be characterised as (A) Preexisting defense structures and (B) Defense structures developed after the attack of the pathogen. Although structural defense mechanisms do prevent the attack of the pathogen, the defense mechanism also includes the chemical substances produced in the plant cells before or after the infection. It has now been established that biochemical defense mechanisms play more important role than the structural defense mechanisms. This has been supplemented by the fact that many pathogens entering non host plants naturally or artificially inoculated fail to cause infections in absence of any structural barriers. Induced biochemical such as phenolic compounds, phytoalexins, new protein (Phenyl Ammonia Lyase (PAL), enzymes have important role on prevent or kill the pathogens in plants.

11.8 SELF ASSESSMENT QUESTION AND EXERCISES

1. Define penetration.
2. What is defence mechanism?
3. Describe induced structural and biochemical defence mechanism in plants.
4. Describe pre-existing biochemical defence mechnnism in plants.
5. Write eassy on penetration of pathogen into the host.

11.7 KEY WORDS

Penetration: the initial invasion of a host by a pathogen.

Colonization: The growth of a pathogen, particularly a fungus, in the host after infection is called colonization.

Pectin: a methylated polymer of galacturonic acid found in the middle lamella and the primary cell wall of plants.

Pectinase: An enzyme that breaks doen pectin.

Pathogenesis: is the chain of events that lead to development of disease in the host or sequence of progress in disease development from the initial contact between the pathogen and its host to the completion of the syndrome.

Tylosis: An overgrowth of the protoplast of a parenchyma cell into an adjacent xylem vessel or tracheid.

Elicitor: a physical, chemical or biological stimulus that triggers defence responses in plants.

Inducible or Induced: A substance, usually an enzyme, whose production has been or may be stimulated by another compound, often a substance or a structurally related compound called an inducer.

11.9 FURTHER READING

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UNIT – 12 CONTROL MEASURES

Structure

12.1 INTRODUCTION

Since the beginning of agriculture, generations of farmers have been evolving practices for combating the various plagues suffered by our crops. Following our discovery of the causes of plant diseases in the early nineteenth century, our growing understanding of the interactions of pathogen and host has enabled us to develop a wide array of measures for

the control of specific plant diseases. Successful disease control requires thorough knowledge of the causal agent and the disease cycle, host-pathogen interactions in relation to environmental factors, and cost. Disease control starts with the best variety, seed, or planting stock available and continues throughout the life of the plant. For harvested crops, disease control extends through transport, storage, and marketing. Most control measures are directed against inoculum of the pathogen and involve the principles of exclusion and avoidance, eradication, protection, host resistance and selection, and therapy. Relatively few diseases are controlled by a single method; the majority require several approaches. These often need to be integrated into a broad program of biological, cultural, and chemical methods to control as many different plant diseases. In this unit, author have briefly discussed about the various control measure such as cultural practices, chemical control and biological control measure and integrated plant disease management.

14.2 OBJECTIVITIES

After going through the unit you will be able to:

The goal of plant disease management using various measures is to reduce the economic and aesthetic damage caused by plant diseases.

1. Understand the various approach in cultural practices methods to control plant diseases.
2. Understand the various chemical employed in chemical control measures to curtail plant diseases.
3. Understand the antagonist's behaviors of organism which helps to use biocontrol agent for plant disease.
4. Understand the importance of integrated plant disease management on plant disease.

12.3 CULTURAL PRACTICES

12.3.1 Introduction

Cultural practices which include manipulation and/or adjustment of crop production techniques have been as old as possibly agriculture itself. In early stage of agriculture development, the growers through their experiences and observations had known that repeated cultivation of a particular crop species or variety on a piece of land often resulted in crop sickness. As a matter of fact, in the present day agriculture, cultural practices are being considered as essential backup methods for management of plant diseases. Adequate adjustment in crop production techniques can modify the environment in such a manner that it becomes unfavorable for the pathogen and pathogenesis. Based on this the disease control affected by cultural practices are preventive, these practices aim at reducing the activity and density of inoculum.

12.3.2 Cultural methods

Procedures for disease control through cultural practices are discussed under following heads

12.3.2.1 Eradication

Eradication is the elimination of pathogen after it has become established in the area where host is growing. The following are the important methods followed to prevent the spread of the disease:

i. Eradication of alternate hosts,

ii. Eradication of collateral and self-sown overwintering hosts

iii. Eradication of the affected plants or trees,

iv. Eradication of pathogens from infected plant parts by surgery and

v. Eradication of culled out plant materials, debris, etc., through different cultural practices

12.3.2.1.1 Eradication of alternate hosts

Removal of alternate hosts helps to prevent and check the spread of the disease caused by heteroecious rust pathogens in the primary hosts. Barberry bush is the alternate host for black stem rust pathogen Puccinia graminis tritici on wheat where the pathogen survives in the offseason. The eradication of barberry had two benefits i.e., it elimination of early spring primary inoculum and prevention of the formation of new physiologic races of the pathogens.

12.3.2.1.2 Eradication of collateral and self-sown overwintering hosts

There are many weed hosts or wild species of cultivated plants act as collateral hosts or volunteer plants of an economic crop which act as reservoirs of pathogens of annual crop. Reservoir hosts help the pathogen to continue the infection chain. The primary inoculum is produced on and dispersed from these hosts to the cultivated crop hosts. If these wild or uneconomic host plants of the pathogen are destroyed, the sources of primary inoculum are eliminated and chances of initiation of the disease in the crop hosts are reduced. Destruction of these hosts breaks the life cycle of the pathogen and the infection chain. Reservoir hosts or indigenous plant species which are not actually involved with the life cycle of the pathogen but provide additional sites for its persistence and multiplication. In some cases such plant species act as symptomless carriers, especially for viruses and root pathogens. Regional elimination of such hosts requires careful attention to roadside areas and other non-agricultural land also. Self sown crops / volunteer plants help the pathogen to overwinter / oversummer in the absence of economic hosts. In Sudan it was enforced through legislation to pull out the cotton plants to prevent regrowth which facilitate the carryover of the cotton leaf curl virus. Wheat streak mosaic virus has been effectively controlled by eliminating the volunteer wheat plants that served as reservoirs for the virus.

12.3.2.1.3 Eradication of affected plants or trees

In some threatening plant diseases, it is essential to eradicate the host and the pathogen from an area. Citrus, canker (Xanthomonas axonopodis pv. citri) is an example of success of an eradication programme. This disease was first noticed in Florida citrus trees in 1913. An eradication campaign was started in 1915. All the citrus nurseries and orchards were inspected and the infected trees were cut and burnt. The eradication programme continued till 1927 and no citrus canker was present in that area. Peach yellows and peach rosette were also controlled by removal and destruction of diseased trees.

12.3.2.1.4 Eradication of pathogens from infected plant parts by surgery

Eradication of affected plant parts (tree surgery) are also practiced in certain cases which reduces the source of primary inoculum. Lesions caused by fire blight bacterium (*Erwinia amylovora*) on pear and apple trees are removed during winter months. This not only prevents further

spread in the affected trees but also reduces the amount of inoculum that can spread to other branches and trees. Tree surgery is also practiced in coconut trees affected by stem bleeding disease (*Ceratocystis paradoxa*), citrus gummosis (*Phytophthora citrophthora*), *Dendrophthoe* spp. on citrus, bud rot of palms (*Phytophthora palmivora*) and koleroga of arecanut (*P.arecae*).

12.3.2.2 Crop rotation

Crop rotation is essentially a preventive measure and has its effect mainly on the succeeding crop. Crop rotation is the oldest and cheapest method adopted in agriculture for eradication of certain types of pathogens from infested soil. Continuous cropping or monoculturing provides opportunity for perpetuation of pathogenic organisms in the soil when the same crop is raised year after year in the same field. The soil-borne pathogens of that crop easily perennate in the soil and increase in their population. After sometime, the soil becomes so heavily infested that it becomes unfit for cultivation of the particular crop. Virus diseases of crop plants and their vectors are found to increase after every crop if a crop is cultivated continuously in a field. On the other hand, when immune, resistant or non-host crops are grown for a definite duration after a susceptible crop in the field it is expected that in the absence of nutrition, the pathogen will be starved off and the population of such pathogens consequently decreases.

Few examples has been listed. Crop rotation with sugarcane or paddy is effective in the control of 'Panama wilt' of banana (*Fusarium oxysporum* f.sp. *cubense*) and crop rotation with paddy or green manures is effective in the control of red rot of sugarcane (*Colletotrichum falcatum*). Rotation of cereal crops like pearlmillet, finger millet or fox-tail millet is recommended for the control of Macrophomina root rot of pulse crops.

12.3.2.3 Fallowing

Fallowing starves the pathogen and helps in reduction of the inoculum by elimination of the host. Diseases like Macrophomina root rot on different crop plants is controlled by following this method. Flood fallowing is to a depth of 0.6 to 1.5 m for 4 to 6 months markedly reduced the Panama wilt pathogen *Fusarium oxysporum* f.sp.*cubense* inoculum in banana. Soil inoculum of *Phytophthora parasitica* var. *nicotianae*, the causal organism of black shank of tobacco was destroyed by flooding the field for 3 to 4 months and by raising swamp rice in a 2 year rotation with tobacco-rice crop in Java. Flooding the soil strewn with debris infected by *Xanthomonas axonopodis* pv. *malvacearum* for 4 days reduced the inoculum level and thus the incidence of disease was only 2.1% as against 69.5% in unflooded fields. Wet fallowing makes the pathogenic propagule in or on the soil to germinate, spent them, is become susceptible attack of saprophytes. Example, Sclerotium rolfsii and Verticillium dahliae. The sclerotia or microsclerotia of these fungi are activated in the absence of root exudates of this host. They germinate quickly when there is alternate wetting and drying of the soil. When the population of Pythium myriotylum is not high wet fallowing is successful in reducing the population. Wet fallowing reduces saprophytic survival of Alternaria solani on crop debris.

12.3.2.4 Application of organic manures

Addition of organic manures like farm yard manure or green manures or oil cakes to the soil increases the antagonistic microorganisms in the soil. Buildup of antagonistic microorganisms reduces the population of soil borne plant pathogens and the diseases caused by them. Application of farm yard manure at the rate of 12.5 tonnes/ha reduced the incidence of Macrophomina root rot of cotton. Application of 5 kg of neem cake/tree three times in a year reduces the basal stem rot (*Ganoderma lucidum*) of coconut. In the control of sesame root rot (*Macrophomina phaseolina*) application of neem cake at the rate of 150 kg/ha is recommended. Application of neem cake at the rate of 2 tonnes/ha in two split doses and covering with mud reduced foot rot disease in betelvine garden.

12.3.2.5 Soil amendment

It has been proved that the organic amendments rich in carbon and deficient in nitrogen control the take-all disease (Ophiobolus graminis) of wheat. There is considerable liberation of CO2 by soil saprophytes which suppresses the pathogenic activity of this fungus .In the process of survival also, low nitrogen content in the soil reduces the longevity of the fungus. Phytophthora root rot of avocado is controlled by amending the soils with alfalfa meal- a material of low C/N ratio.

12.3.2.6 Summer ploughing

Deep ploughing during summer periods buries the inocula of fungi of soil borne nature. Fungal propagules, sclerotia and different types of spores conidia on plant refuses die when exposed to sunlight due to the higher temperature prevailing during the summer. Further infected self-sown plants, volunteer host's plants, weed hosts, regrowths from the plant roots, alternate hosts and alternative hosts are also destroyed. Here, the spread of the disease is avoided. Groundnut blight (*Corticium rolfsii*) is controlled by ploughing the soil to a depth of 20 cm.

12.3.2.7 Crop growing seasons

Rice blast becomes serious when the rice crop is raised from August to September in Tamil Nadu. Ragi blast becomes serious when sowing is made between June and August. Similarly yellow mosaic of blackgram/green gram and phyllody of sesame are serious during kharif season in South India. Incidence of powdery mildews of different crops is found to be high during rabi when compared to kharif and summer seasons. In bhendi, yellow vein mosaic incidence is very high during summer. The seasons with high incidence of diseases should be avoided in the epidemic areas.

12.3.2.8 Adjustment of sowing time

In many diseases the incidence is more severe when the susceptible stage of the plant growth and favourable conditions for the pathogens coincides. While choosing the time of sowing it should be taken into consideration that susceptible stage of the crop growth and soil conditions and other environments favourable for maximum activity of the pathogen does not fall at the same time. Properly adjusting the sowing dates can give good dividends. Late planted wheat crops contract less infection than

wheat planted on normal dates. Early and late sown crops have been found to be free from Oodhubathi disease of rice.

Avoiding cool and cloudy days for planting will help to reduce red rot of sugarcane. Late sowing of winter wheat and barley is considered to be the most effective measures in reducing take all disease of wheat. Rapeseed sown in mid to late August is more liable to attack by leaf spot (*Alternaria brassicae*) than late-sown crops. Pea and gram planted soon after rains when soil temperature and moisture are at a high level, show high incidence of root rot and blight. As the soil temperature falls and moisture becomes less (Nov-Dec) these diseases are also reduced. In areas where these diseases are serious, late sowing helps in saving the crop. Stem rust of wheat damages the late sown crop more than the early sown crop. Because, time of onset of disease and ear formation coincides. Sowing from January to April or October to December is advocated to escape from the attack of neck blast of finger millet. Peas and chickpea sown in October usually suffer heavily from root rot and wilt (a complex of *Fusarium, Rhizoctonia* and *Sclerotium*). When these crops are sown late, the diseases are not so severe or almost absent. The groundnut rosette is transmitted by *Aphis craccivora*.

12.3.2.9 Adjustment of harvesting time

Harvesting of groundnut should not coincide with the rainy days and it helps to avoid infection by *Aspergillus flavus*. Freedom of onions and roses grown in rainless seasons from downy mildew diseases and freedom of beans, chilli and cucurbits from bacterial diseases in such seasons are the best examples for sowing of crops at correct season to avoid disease outbreaks. In the case of deciduous fruit trees and grapevines, the season of sprouting, flowering and fruit set can be advanced or delayed by pruning practices or by treatments to break dormancy. Advantage can sometimes be taken of this fact to avoid coincidence of all or one of these phases of host growth with weather periods particularly favourable to specific pathogens that attack trees in the phases.

12.3.2.10 Growing of seed crops

Coffee can be grown in the western Hemisphere usually free from coffee rust which causes heavy losses in Eastern Hemisphere. In the case of virus diseases this will be more useful. By growing seed materials in isolated places where the population of vectors is very low and the condition is uncongenial for the vectors. Virus free potato tubers to be used as seeds are grown in cool and windy places in many parts of the world. Under tropical and subtropical countries, such conditions prevail in the hills at high altitudes. Obtaining seed from disease-free localities has been very successfully resorted to the elimination of many seed –borne diseases. In the U.S.A. seed-potatoes are invariably grown in northern snow-clad sections, where viruses are practically absent and then exported to various other sectors in the south. Similar practice has been in vogue in India, where seed-potatoes are annually imported in southern states from Simla hills for control of virus diseases and bacterial ring. In the U.S.A, the seed growing areas have been shifted to arid pacific regions for crops like cabbage, turnip, beans and peas for obtaining disease-free seed and indirectly controlling such diseases like black leg and black rot of cabbage

and turnip and anthracnose of beans and peas. Similar practice is obtained in parts of Bombay, where the foot rot of ginger (*Pythium myriotylum*) prevalent in the southern parts, is controlled through the importation of seed-rhizomes from disease-free arid regions of the north, where the disease is practically non-existent on account of the dry climate, lighter soils and moderate rainfall.

12.3.2.11 Selection of seeds and seed materials

Seeds and seed materials carry many fungi, bacteria, viruses and phytoplasmas and may introduce these pathogens into the field, i.e., seeds and seed materials form the primary source of infection. Seed and seed materials like cuttings, tubers, grafts, setts etc., should be well matured, disease free, uninjured and have a high germinating capacity. The absence of an initial inoculum in seeds is definitely helpful in delaying or suppressing the incidence of the disease. It is a preventive method.

The diseases like foot rot, brown spot, short smut of sorghum, loose smut of wheat, bacterial blight of rice, bacterial blight of cotton, leaf crinkle of blackgram etc., are transmitted through seeds. Virus diseases and black ring of potatoes, foot rot of ginger, foot rot of betelvine, Panama disease of banana, red rot of sugarcane cassava mosaic, bunchy top and virus diseases of fruit trees are transmitted through tubers, setts, rhizomes, corns, grafts and budwoods. 'Tuber indexing' is a special method to obtain disease free seed materials in potato. It is commonly practiced by nurseries and seed merchants selling potato seed tubers. Use of seeds in the place of rhizome/sucker is recommended in the control of 'katte' disease of cardamom.

12.3.2.12 Leveling of the field and provision of drainage facilities

Water stagnation in different patches of field favours the fungi like Pythium, Phytophthora, *Rhizoctonia solani*, etc., for which proper leveling of the field before sowing or planting is very essential. Further improving the drainage is necessary in the control of sheath blight of rice. Provision of drainage channels in orchard crops like citrus, jack, mango etc., in the garden is necessary before planting. In the control of damping-off diseases of vegetable and other crops, raising seedling in the raised beds method is followed. Foot rot of ginger (*Pythium myriotylum*) is also controlled by following the raised bed system of nursery.

12.3.2.13 Burning of stubbles and crop residues

Burning of plant wastes, crop residues, stubbles, etc., in the areas selected for raising nurseries for vegetable crops, tobacco, chillies and forest trees etc. heats the soil and kills the inoculum of the pathogens present in the top layer of the soil. When nurseries are raised in these areas incidence of damping off disease is highly reduced. This practice is also followed in pits made for planting coconut, banana, fruit trees etc., Burning of wheat plant every second or third year is suggested for eradication of pathogen in the field when *Cephalosporium gramineum* infects wheat. Otherwise, debris in the field helps the perpetuation of the pathogen and the disease. Burning of rice crop residues avoid carryover of sheath blight (*Rhizoctonia solani*); stem rot (*Sclerotium oryzae*) of rice and bacterial blight of cotton.

12.3.2.14 Depth of sowing

Depth of sowing greatly influences seed transmission of smuts. Shallow planting in wet soils protects wheat plants from *Urocystis tritici* (flag smut) of wheat. Deep planting may cause delay in the emergence of seedlings, which may be vulnerable to pre-emergence damping off. Early emergence results in early lignification of tissues which become resistant to attack of soilborne pathogens.

12.3.2.15 Spacing

Closer spacing invariably alters the microclimate underneath the canopy of the crop which may provide favourable environment for development of diseases. Boll rot in cotton is quite common in crowded crop. Defoliation of plants or skip cropping gives better control against the boll rot disease. In certain virus diseases like groundnut rosette the incidence is observed to be less when wider spacing is adopted. Closer spacing favours many air borne diseases because of high humidity in the crop canopy. Early and late blight of groundnut and blister blight of tea are more in dense canopy. Early spread of black rot of cabbage takes place in closer spacing. Crowded stands may reduce some systemic diseases. Cotton wilt caused by *Verticillium alboatrum* will be less in closely planted crop if the fungal inoculum is less in the soil. Similarly closer spacing of rice reduces rice tungro virus infection particularly when vector population is less. Avoiding shade and providing wider spacing reduces the incidence of powdery mildew of tobacco. Late blight of potato and downy mildew of grapevine spread fast in closer spaced crops. In the case of bud necrosis of groundnut caused by tomato spotted wilt virus, seeds are sown adopting closer spacing of 15x15cm to compensate the rogued out plants with regard to plant population and yield. These are examples where dense sowing helps in disease reduction. The virus of tomato leaf curl, transmitted by *Bemisia tabaci*, is less severe in a crowded planting than in spaced planting.

12.3.2.16 Method of sowing/planting

In places where water accumulation is a problem to the crop growth sowing of seeds on the sides or ridges is found effective in reducing the incidence of *Sclerotium rolfsii* on groundnut and vegetable crops and *Sclerotinia sclerotiorum* and *Rhizoctonia solani* on vegetable crops and Phytophthora blight of pigeonpea. High ridging prevents infection of potato tubers, by zoospores from leaf lesions in late blight diseases. Ridging is disadvantageous in water deficit areas where it encourages pathogens like *Macrophomina phaseolina*.

12.3.2.17 Avoiding injury

Injury of plant parts should be avoided in order to check the entry of pathogens. Clipping of tips of tall rice seedlings favours the entry of bacterial blight pathogen and incidence of the disease. Hence clipping should be avoided at the time of transplanting of rice. While harvesting the pods in groundnut, fruits in tree crops and vegetable crops injuries to the fruits pave the way for the pathogen and causing pod/fruit rot. It also reduces the storage life of fruits and vegetables. Hence much care should be given to avoid wounds during the harvest time.

12.3.2.18 Altering the soil pH

In certain soil borne diseases adjustment of soil reaction helps in the reduction of inoculum level of the pathogens. The altered pH of the environment forms a barrier against the pathogen. A very low pH less than 5.2 is unfavourable to common scab bacterium on potato (Streptomyces scabies). Thus, use of acid forming fertilizers (like sulphur) and avoiding lime and calcium ammonium nitrate application are effective in controlling the common scab disease. On the other hand the club root pathogen of cabbage (*Plasmodiophora brassicae*) cannot live and infect when the soil pH is 7.0 or more. Hence liming which increases the soil pH gives satisfactory control of club root disease. In Punjab, root rot of tobacco (*Macrophomina phaseolina*) has been overcome by application of 2.5 to 5.0 tons of lime /ha to the soil.

12.3.2.19 Mixed cropping

Mixed cropping materially helps in checking certain diseases. Blight of pulse crop (*Phyllosticta phaseolina*) has been successfully overcome by growing pulses as a mixed crop with cereals like sorghum and pearlmillet.

12.3.2.20 Intercropping

Intercropping is also a device in the control of some soil borne diseases. Intercrops should be properly chosen so that they should not have any common pathogen for e.g., *Macrophomina phaseolina* has got wide host range and hence common host should not be grown as intercrops. Intercropping with moth bean (*Phaseolus aconitifolius*) in a cotton field reduced the root rot (*M.phaseolina*) incidence. Due to reduction in the number of host plants there is sufficient spacing between them and chances of contact between foliage of roots of diseased and healthy plant are greatly reduced. Therefore, root pathogens are unable to spread from diseased to healthy roots and spread of foliar pathogens is also reduced to a great extent.

Intercropping of sorghum in pigeonpea field reduced the wilt (*F. udum*) incidence. The roots of non-host plants may act as a barrier obstructing the movement of pathogens in soil. They may release toxic substances from their roots which may suppress the growth of the pathogens attacking the main crop. Hydrocyanic acid (HCN) in root exudates of sorghum is toxic to *F. udum,* the pigeonpea wilt fungus.

12.3.2.21 Barrier cropping

Taller crops can be grown to protect a crop of lesser height from virus vectors. The insects may land at the taller crops (barrier crops) and the dwarf crop may escape from virus diseases by those insects. Barrier cropping with 3 rows of maize or sorghum or pearlmillet around the main crop namely blackgram or greengram is effective in reducing the vector population and incidence of yellow mosaic. Another best example is growing of 3 rows of kale or barley as barrier crops in cauliflower seed beds and undersown beet steckling against cauliflower mosaic and beet yellows diseases respectively. The incoming aphids are thought to land on the barley or kale and probe briefly, causing them to lose the non-persistently transmitted virus they are carrying. Maize or sunflower are the other barrier crops considered for these crops.

12.3.2.22 Decoy crop and trap crop

Decoy crops (hostile crops) are non-host crops sown with the purpose of making soilborne pathogens waste their infection potential. This is effected by activating dormant propagules of fungi, seeds of parasitic plants, etc. in absence of the host.

Trap crops are host crops of the pathogen, sown to attract pathogens but destined to be harvested or destroyed before they complete their life cycle. Fodder sorghum can be raised as a trap crop to reduce downy mildew of sorghum.

12.3.2.23 Trenching

Trenching between rows of trees in orchards has been effectively utilized in arresting the growth and spread of the pathogen in the soil to the neighbouring trees. *Ganoderma lucidum* root rot infected citrus trees should be isolated by digging a trench of 30 cm wide and 60 cm to 90 cm deep around the tree at a distance of 2.5 to 3.0 m from the base to prevent the contact of diseased roots with healthy roots. Thereby the spread of the pathogen to neighboring tree is prevented. Similar method is also followed in the control of basal stem rot (*Ganoderma lucidum*) of coconut in India.

12.3.2.24 Isolation distances

The distance between seed production and commercial plots has been worked out for reducing seed borne loose smut of barley and wheat. Barley and wheat crops should be isolated by at least 50 m from any source of loose smut infection for production of certified seeds in the U.K. the number of viruliferous insects reaching a healthy crop from a diseased one decreases with distance between them so that cultivation of susceptible crops at a distance from each other delays or reduces the severity of virus diseases. Incidence of lettuce and cucumber mosaic viruses is about 3% if the new lettuce crop is sown 0.8 km away from an old lettuce field much greater incidence of mosaic in sugarbeet fields occurs within 90 metres of a seed crop than in the fields at a greater distance. Beet mosaic and beet yellows are markedly reduced by isolating beet fields by 19 to 24 km and 24 to 32 km mites respectively from a large source of infected beets.

12.3.2.25 Yellow sticky traps

Sticky, yellow polythene sheets erected vertically on the windward side of red pepper fields have been sown to reduce the incidence of potato virus Y (PVY) and cucumber mosaic virus (CMV) in the crop. The aphids are attracted to the yellow colour and are caught on the sticky polythene. The control obtained was so successful that the method has become a standard control procedure in red pepper crops in Israel. Similar traps have also been used to protect 'seed' potato crops, against potato leaf roll virus. Yellow sticky traps are in use to attract and kill the whitefly vectors which spread yellow mosaic of blackgram and greengram and bhendi yellow vein mosaic.

12.3.2.26 Mulching

Mulching or covering of top soil with organic residues often helps in reducing plant diseases. Mulches of non-host origin should be used in the field. These mulches are known to release inhibitory substances in the underlying soil and also promote development of parasites and predators of nematodes. Reflective surfaces (mulches) laid on the soil around the crop

plant, have been found to be highly effective in controlling aphid vectors. Aluminium strips or grey or white plastic sheets are used as mulch and it has successfully protected red peppers against CMV and PVY in Israel and summer squash against watermelon mosaic virus in the Imperial valley of California. Straw mulches have been successfully used to control the white fly – transmitted tomato yellow leaf curl virus in tomato crops in Israel. It is believed that the colour of the straw attracts the whiteflies and they are subsequently killed by the reflective heat. The disadvantage with straw mulches is that they eventually lose their yellow colour, but prolonged control may be obtained if straw is replaced by yellow polythene sheets.

12.3.2.27 Irrigation water management

Irrigation to the crop in the field is to wet the soil to the extent that roots easily get water and nutrients. If excess water is added to soil, it may directly affect activity of pathogens and/or it may affect disease incidence through the effect on the host. By irrigating the field, soil temperature is brought down, stress is removed and the disease is suppressed. When excess irrigation is made the juvenile stage of plants is lengthened making it susceptible to attack of fungi like Pythium. Supply of frequent but low quantity of irrigation water is, therefore, recommended for reducing chances of damping off in nurseries. Under conditions of excess water, respiration of roots is inhibited and many soluble salts accumulate in toxic amounts around the roots and base of the stem. This increases disease proneness of the roots. Irrigation increases guttation. Guttation drops on leaves serve as media for multiplication and penetration of many pathogens, such as *Helminthosporium* spp. on cereals and *Xanthomonas campestris* on Brassica spp.

Pathogens directly taking advantage of excess water are those that need wet soil for (i) activation of their resting structures and (ii) for movement of these propagules. Thus, in presence of excess free water bacterial cells and zoospores of Pythiaceous fungi are dispersed easily. Therefore, at the plant stage when these pathogens can attack the crop irrigation should be avoided.

Sclerotia, smut spores, chlamydospores, oospores and mycelium found in the soil are carried from one field to another through irrigation and drainage water. Stem rot, sheath blight and bacterial blight diseases of rice, damping off of vegetables and Macrophomina root rots of many crops spread mainly through irrigation and drainage water. Hence care should be taken not to irrigate a healthy field using drainage/irrigation water from a diseased field.

12.3.2.28 Field and plant sanitation

Field and plant sanitation is an important method of disease control through cultural practices. The inoculum present on field plants in the field may multiply on the plant or in the soil and in due course of time may be sufficient to nullify or reduce the effect of control practices. Many pathogens overwinter or oversummer on plant debris during the off-seasons and become active when the crop is again grown in the field. Hence plants bearing pathogens or plant debris introducing inoculum into the soil should be removed as early as possible. In most of the soil borne diseases like wilt and root rot, it has been reported that as long as the dead roots and other roots and other affected parts are present in the soil, the

fungus continue its growth. When such diseased plant materials are removed, there is quick decline in the population of pathogens in the soil.

In this manner Fusarium wilt of cotton, pigeonpea and banana, Verticillium wilt of cotton, root rot of beans, downy mildew of pearlmillet, sorghum, maize and peas, foot rot of betelvine, bacterial blight of cotton, white rust of crucifers, black spot of rose, powdery mildew of pea and cereals are reduced. In certain areas the linseed rust fungus (Melampsora lini), the rice blast and brown spot fungi and the fungus causing early blight of potato also perennate through dormant stages in diseased crop debris. Destruction of crop debris by burning immediately after harvest reduces the amount of inocula which survive through debris.

Avoidance of the transfer of inoculum from one field to another by man, machine or water is one of the ground rules of cultural control. Where soil-borne diseases are concerned, anything that carries soil is suspect, this includes wheels, boots and water flowing either from adjacent fields, or through drainage ditches from distant fields. As regards sap-borne viruses, attention must be paid to disinfection of wheels and of the hands of labourers, as they pass from one field to another. Where such virus can also be carried on clothing. The work should be planned so that the labourers do not go from older to younger fields on the same day.

12.3.2.29 Roguing

Roguing consists of completely removing or uprooting the diseased plants to prevent further spread of the disease. This method is widely adopted in the control of virus diseases spread by insects (cassava mosaic, yellow mosaic of blackgram and greengram, citrus tristeza, katte disease of cardamom, bunchy top of banana) and basal stem rot of coconut, green ear of pearlmillet and broomrape (Orobanche) in tobacco. The whip smut of sugarcane (*Ustilago scitaminea*) in the canal areas of Bombay in Co.475 variety has been greatly checked by roguing carried out over wide areas and long period. In Jamaica, a country-wide campaign of destroying infected plants has succeeded in the control of Panama wilt of banana. Root rot and wilt attached plants after their death should be as and when noticed in the field uprooted and burnt to check the inoculum build up in the soil.

12.3.2.30 Management of plant nutrients

The plant nutrients in general when applied in excess may increase or reduce the resistance in plants to diseases. Increased application of nitrogenous fertilizers increases the incidence of many diseases. Crops fed with heavy doses of nitrogenous was fertilizers grow robust with foliage and succulent tissue but become highly susceptible to the attack of diseases like rust powdery mildew, blast, tobacco mosaic and some bacterial diseases. Some diseases are favored by ammoniacal form of nitrogen while others are favoured by nitrate form of nitrogen. In general wilts (*Fusarium* sp.) and root rots (*Rhizoctonia* spp.) are favoured by ammoniacal nitrogen while Verticillium wilts and root rots due to Pythium spp. are favoured by nitrate nitrogen. Repeated application of phosphatic fertilizers delays the onset and lessens the severity of take-all disease of barley (*Gaeiimannomyces graminis*). Potassium application reduces the disease incidence in many crop diseases probably by increasing phenolics synthesis in plants. Application of potash induces resistance in groundnut

against root rot caused by *Macrophomina phaseolina*. Calcium application suppresses the lesions due to the *R.solani* on bean roots. It is due to formation of calcium pectate, which is less available to action by polygalacturanase (PG) enzyme than is pectic acid. Manganese reduces late blight of potato, ferric chloride controls rice brown spot and silicon application reduced rice blast.

12.3.2.31 Time of harvesting

Time of harvesting affects the cleanliness of the seeds. Delayed harvesting of grain crops in temperate climatic conditions enables the pathogen more time to contaminate the seeds. The best example is grain mould of sorghum where contamination by species of *Fusarium*, *Curvularia*, *Alternaria*, *Aspergillus*, Phoma is seen. Potato tubers harvested when the tops are green get easily contaminated by the late blight pathogen present on the leaves. Removal of tops and making them to dry before digging the tubers kills the sporangia and avoids contamination of tubers harvested later.

Check your Progress- 1

Note: a. Write your answer in the space given below

b. Compare your answer with those given at the end of the unit.

1. How do you prevent viral disease in crops through cultural practices?

……

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12.4 CHEMICAL CONTROL

12.4.1 Introduction and Concept

The word fungicide has originated from two Latin words: viz. fungus and caedo (to kill). So literally, a fungicide would be any agency (physical or chemical) which kills a fungus. However, the word is restricted to chemicals. Hence, the word fungicide should mean a chemical capable of killing fungi. However, there are a number of compounds which do not kill the fungus. They simply inhibit growth or spore germination temporarily. If the fungus is freed from such substances, it would revive. Such a chemical is called a "FUNGISTAT" and the phenomenon as "FUNGISTASIS". Some other chemicals, like certain phenantherene derivatives and Bordeaux mixture may inhibit spore production without affecting vegetative growth. These are called "ANTISPORULANTS ". There are other groups of chemicals which exhibit very poor or no antifungal activity *in – vitro* condition but provide protection to the plants either by inhibiting the penetration of host surface by the fungi or by inducing the host defense system. The former type of chemicals are termed as "ANTIPENETRANTS "and later as "ANTIPATHIC AGENTS ". Even though fungistats, antisporulants, antipenentrants, and antipathic agents do not "kill" fungi, they are included under the broad term fungicide because by common usage, the word fungicide has been defined as chemical substance which has ability to prevent damage caused by fungi t plants and their products. Fungicide which is effective only if applied prior to fungal infection is called "PROTECTANTS ". On the other hand, fungicide which is capable of eradicating a fungus after it has caused infection, and thereby " curing " the plant, is called "THERAPEUTANT ".

12.4.2 FORMULATIONS

Formulations is an art which is mainly concerned with methods of presenting the active ingredient, in the most effective form, that is, with regard to storage , application , and ultimate biological activity. Actually the amount of fungicide that is biologically active/effective at the plant surface is so minute that for economic use the chemical must be diluted, either with a solid dust, powder, etc.) or liquid (concentrates, emulsions, suspensions, etc.) before application. Water provides a cheap and effective dilution medium and, with few exceptions is used as the carrier for agricultural sprays. As majority of the fungicides are of very low water solubility they must be formulated to make them compatible with water, hence surface active agents are required to prepare water-dispersible powders, stock emulsions, or emulsifiable concentrates. In addition, surface active agents may be needed to improve the suspending, spreading, and wetting properties of the sprays. Other supplements that may be incorporated with sprays include stickers to improve the weather resistance of the deposit, and materials to improve storage stability, deposition on plant surface and penetration of plant surface and/or fungal cells. The type of formulation chosen for a fungicide is determined by a variety of factors which cost and biological efficiency are the most important.

12.4.3 Applications of Fungicides for Diseases Management

12.4.3.1 Seed treatment

Seeds, tubers, bulbs and other propagating materials are often treated with fungicides for eradication of the pathogen propagules present on and/or in them as well as for preventing their rot in the soil after planting. The chemicals can be applied on the seed as dusts, or as thick water suspension (slurry) mixed with the seed. The seeds can also be soaked in water or solvent solution of the chemical and then allowed to dry. On the basis of their mode of action of the seed treatment chemicals can be of three types:

a) Those which kill the pathogen present on the seed and do not remain active for long after the seed has been planted (seed disinfectants).

b) Those which act as eradicants and destroy the pathogen established in seed tissues. A number of systemic chemicals are being used for this purpose (seed disinfectants), and

c) When seed are treated with protective fungicides, the pathogens present on the seeds are destroyed and the compounds remain on seed surface for sufficiently long times (seed protectants). In treating seed and other propagative organs, it should be ensured that enough chemical has sticked to the seeds. It should also be ensured that their viability is not lowered or destroyed.

12.4.3.2 Soil Treatment

The purpose of soil treatment with chemicals is to reduce or eradicate the inoculums density of the pathogens present in *soil* or *soil invaders* or as *soil inhabitants.* The chemical soil treatment can be done in any of the following ways.

1. Soil Drenching

Fungicidal suspension or solution is applied to soil surface in quantities enough to wet 10-15 cm depth of the soil before or after planting.

2. Broadcasting

Non-volatile fungicides (dusts, powders, granules, etc.) are mixed with soil or fertilizers and spread on the soil surface. Light ploughing or harrowing is then done to mix the chemical in sufficient depth.

3. Furrow Application

In this method fungicides are applied either as dusts or with water to the furrow at the planting time. This method is possible in crops planted in furrows/rows such as potato and sugarcane. This method is good for control of diseases that occur at the base of the plants. This method requires much less quantity of the chemicals per hectare than the broadcast method.

4. Fumigation

This method is usually used to control plant parasitic nematodes. The chemicals used are generally volatile and on coming in contact with soil moisture release gases which diffuse in the soil and kill the nematodes. Special equipments (guninjectors) are required for application since diffusion is restricted to certain distance. The chemicals are injected into soil at regular distance (12-15 inch) all over the field. The depth of application is kept at 6-9 inches.

12.4.3.3 SPRAYING

This is the most commonly adopted method. Spraying of fungicides is done on leaves, stems, flowers and fruits. Wettable powders are most commonly used for preparing sprays. The most common diluent or carrier is water. The dispersion of the spray is usually achieved by its passage under pressure through nozzles of sprayers. Spraying is of two types, namely, high volume and low volume. When sprays involve large quantities of liquid per unit area, they are termed high volume. Six hundred litres and above per hectare would be considered to be of the high volume category. With low volume sprays, it is usually possible to cover one hectare with about 100 litres or less.

12.4.3.4 DUSTING

Dusts are applied to leaves, fruits, and stems of plants as an alternative to spraying. Dry powders are used for covering host surface. Dusting is practicable only in the calmest weather and high effectively is obtained if the dust is applied when the plant is wet with dew or rain.

12.4.3.5 PASTER OR PAINTS

In orchards, the wounds created during pruning and training of trees often serve as entry points of the pathogens. To protect these wounds fungicidal pastes or paints are used as protective layer. These pastes are prepared with fungicides in a suitable carrier such as raw linseed oil, lanolin, glycerine, etc. the residual effect of the pastes lasts long enough to permit natural healing of the cut surfaces.

12.4.4 SYSTEMICITY

A fungicide when applied on the plant surface either may remain on the surface (non-systemic) or absorbed by the plant. The latter may remain

there in treated plant parts (locosystemic); may move in the direction of evapotranspiration stream (apoplastic) or may move with photosynthates to "sink" (symplastic) or in both directions (ambimobile).

The symplast is the living part of the plant which is enclosed by membranes, i.e. protoplasts, and plasmodesmata, including the phloem sieve cells. Long distance transport in phloem is symplastic. The apoplast is the nonliving part of the plant, i.e., cell walls and cuticle, including xylem vessels and tracheids. Long distance movement in the transpiration stream is apoplastic.

12.4.4.1 Apoplastic Translocation

Apoplastic translocation, within the plant, is usually directed from roots to transpiring areas, especially leaves. Fungicides are absorbed by the roots, mainly in root hair zone, along with water. Root absorption is usually a passive phenomenon. Radial movement through the cortex zone occurs either symplastically or apoplastically. Symplastically moving substances cross the plasmalemma and are transported via protoplast and plasmodesmata of cortex cells and then through the epidermis cells to the vessels of the xylem.

Fungicides with typical apoplastic translocation display following properties.

1. Upward movement within the plant following plant seed, root, or stem application. 2. Movement into various plant organs is dependent on their transpiration rate. 3. Accumulation at tips and margins of leaves. In monocotyledons, where venation is convergent and palmate parallel type, metalaxyl is drawn much strongly through veins resulting in its accumulation at tips and margins. 4. In dicotyledons, because of reticulate venation, driving forces towards the periphery get drastically reduced. Thus, allowing metalaxyl to enter cytoplasm. In such cases, the fungicide is either uniformly distributed in entire lamina or accumulation at margins is quite delayed. However, majority of the fungicides with typical apoplastic translocation are accumulated at tips and margins of leaves irrespective of plants species involved.

12.4.4.2 Symplastic Translocation

Symplastic movement takes place within the living parts of the cell. The movement is in the sieve tubes of the phloem tissues. This is an active movement and requires the expenditure of metabolic energy both for uptake inside the cells as well as for movement in the sieve tubes. Fully expanded photosynthesizing leaves serve as a source and roots, flowers, young growing leaves, and fruits serve as a sink. A fungicide with symplastic movement, after entry into the phloem, follows the same source to sink pathway as followed by the phloem assimilates.

12.4.5 Classification of Fungicides

Ever since the introduction of systemic fungicides, systemically has been the most popular criterion for the broad classification of the fungicides. They were classified as 1. NON-SYNTHETIC, and 2. SYSTEMIC. However, in this book selectively has been preferred over systemically and based on that fungicides are broadly classified as 1.Non-selective, and 2.Selective.

12.4.5.1 Non selective fungicides

1. Sulfur fungicides

Several inorganic and organic sulfur fungicides are in use for control of plant diseases. All sulfur fungicides are nonsystemic, and except lime sulfur, are phytotoxic (excluding "sulfur shy" varieties) and compatible with most of the pesticides. As far as their mode of action is concerned, elemental sulfur interferes with energy production by intercepting electron on the substrate side of cytochrome C in the mitochondrial electron transport system. The dialkydithiocarbamates are known to inhibit a multitude of enzymes; therefore, fungitoxicity probably involves concurrent inhibition of enzymes at several sites. The pyruvate dehydrogenase reaction is particularly highly sensitive to dialkydithiocarbamates.

2. Copper Fungicides

Ever since the discovery of Bordeaux mixture in 1885, copper fungicides predominated the field of fungicidal plant disease control for more than 50 years until synthetic organic fungicides invaded the market. Even today some of the copper compounds are used widely in many countries. Copper is a multisite biochemical inhibitor (probably interact with-SH groups of enzymes) with little biological specificity.

3. Mercury Fungicides

Mercury is a general biocide. Several organomercurial compounds were introduced. Most of them are now withdrawn due to high mammalian toxicity. Only two organic and three inorganic mercury compounds are in use and that too in very limited cases. Mercury also exhibits multisital action due to its interaction with the –SH group of the suscetible enzymes.

4. Phthalamide and Quinone Fungicides

One of the quinone derivatives, chloranil was introduced as a fungicide in 1940. Now it has been replaced by dichlone, another quinone derivative introduced in 1943. Quinone fungicides are multisital in their mode of action. The two mechanisms which are thought to be most likely for dichlone and chloranil are 1. Binding of the quinone nucleus to –SH and –NH2 groups in fungal cell, and 2. Interference with electron transport system (Nene and Thapliyal, 1996). Among the Phthalamide compounds, captan was introduced first and is still being used widely. Basically these fungicides are protective in nature but limited systemicity has been reported for captan and captafol. Phthalamides are highly reactive against thiol (–SH) groups of proteins (enzymes) and low molecular weight metabolites (cystein, glutathione, etc).

12.4.5.2 Selective Fungicides

1. Benzimidazoles and related fungicides

Benzimidazoles and thiophanates represent a group of highly effective broad spectrum systemic fungicides. Most *Ascomycetes*, some of the *Basidiomycetes* and *Deuteromycetes*, and none of the *phycomycetes* are sensitive to these fungicides. Their mild cytokinin like effects on some plants tend to retain chlorophyll and in some cases increase yield and delay maturity. Benzimidazoles bind with the Beta- tubulin subunit of microtubules of sensitive fungi and thereby inhibit formation of spindle and subsequently chromosomal separation during nuclear division.

2. Carboxins and related compounds

Among systemic fungicides carboxin and oxycarboxin were first to be discovered and introduced for plant disease control. These fungicides are

readily absorbed by the seeds, roots, and leaves and translocated apoplastically. They are very effective mainly against Basidiomycetes, smuts, bunts and rusts of cereals, and soil fungus R. solani. Both have low animal toxicity and are quickly degraded. Their mode action is very specific. They interact with succinate ubiquinone reductase complex resulting in inhibition of oxidation of succinate via electron transport chain.

3. Sterol Biosynthesis Inhibiting Fungicides

This group includes numerous fungicides from chemically heterogenous classes. However, all these compounds inhibit ergosterol biosynthesis in fungi and therefore, are classified as "ergosterol biosynthesis inhibitors " (EBIs) or sterol biosynthesis inhibitors(SBIs). They are effective against a wide range of fungi except Oomycetes which do not need sterol for growth. Several fungicides belonging to morpholines, piperidine, piperzine, pyridine, and azole are known sterol biosynthesis inhibitors.

4. Anti-oomycetes fungicides

The past decade has witnessed the introduction of five classes of fungicides controlling diseases caused by Oomycetes: 1. the carbamates (e.g.prothiocarb, propamocarb), 2. the isoxazoles (e.g.hymexazol), 3. Cyanoacetamide oximes (e.g.cymoxanil), 4. Ethyl phosphonates (e.g.fosetyl-AL), and 5. Phenylamides. The last class Phenylamides, cover three groups of compounds: 1 acylalanines (e.g. furalaxyl, metalaxyl, benelaxyl), 2. Acylamino butyrolactones (e.g. ofurace), and 3. Acylamino-oxazo lidinones (e.g. cyprofuram, oxadixyl). All these fungicides show comperatively high water solubility.

5. Antipenetrants

There are several compounds which show no or poor fungitoxicity under in-vitro conditions but prevent infection by interfering with the penetration process.

6. Host Defense Inducing compounds

Various chemicals which have been found to trigger host resistance mechanism and by doing so provide protection against diseases on prior or sometimes simultaneous application with the pathogen. They include both chemical constituents of plants or microbes (biomolecules or metabolites) and synthetic compounds which are foreign to the living system (xenobiotics).

7. Miscellaneous selective fungicides

There are miscellaneous fungicides belonging to diverse chemical groups which show at least some degree of selectivity in their antifungal spectrum. Chinomethionate, dichlofluanid, and chlorothalonil, which interact with NH2, and / or -SH group and hence, their mode of action is multisital.

Check your Progress- 2

Note: a. Write your answer in the space given below

b. Compare your answer with those given at the end of the unit.

2. What is chemical control in plant diseases?

……

…….

3. What is the chemical used to kill oomycetes fungi?

.……

……

12.5 BIOLOGICAL CONTROL

12.5.1 Introduction

Biological control is the reduction of inoculum density or disease producing activities of a pathogen or parasite in its active or dormant state, by one or more organism accomplished naturally or through manipulation of the environment, host or antagonist, or by mass introduction of one or more antagonists.

12.5.2 Theories and Mechanisms

Management of associated microbiota is a major form of biological control Cook (1977) has discussed five element in the theoretical base.

12.5.2.1 Reduction of Inoculum Density

Inoculum density can be reduced by destroying propagules or by preventing their formation. Crop rotation adds chemically different plant residues to soil and therefore, helps in complexities of soil microbiota. It starves the pathogen due to absence of host and weakens to the extent that it is more rapidly destroyed by the microflora. Dormant sclerotia are killed off by *Trichoderma spp., Fasarium roseum, Coniothyrium minitans*, and other fungi and bacteria. Microbial activities helping in decay of organic residues and release of several chemicals, stimulate germination of resting structure like sclerotia and reduce their resistance so that they are easily colonized by antagonists and destroyed. The germtube that comes out is lysed without forming fresh structures. Other treatments that predispose resting structure to microbial decay are wetting and drying, flooding, and sublethal fumigation. Destruction of resting structure occurs in soil by direct activities of bacteria and actinomycetes. There is evidence that perforations appear in the walls of resting propagules due to activities of some microbes. These microbes subsequently enter through these perforations and destroy them.

12.5.2.2 Displacing the pathogen from host Residues

This applies to those pathogens that depend for survival on occupancy of the host remains during host free period. The pathogens use the restudies both as shelter and as a food base. This gives them the advantage of pioneer colonization. The system of residue possession by root pathogens is either PASSIVE or ACTIVE or both. *Pythium spp*. that attack succulent roots exemplify passive possession. They invade thoroughly, digest extensively, store the surplus food in their resting bodies (oospores) and then abandon the fragile exhausted host remains to other saprophytes. In active possession the organism invade the residue, usually as parasite while the tissues are still alive and active, become established in some tissues in which they persist and are metabolically active within the

dead host remains. Utilization of the substrate is slow and the pathogen persistently defends the substrate against saprophytes. Normally the active possessor does not retreat into a dormant structure. *Cephalosporium gramineum* is a typical example. *G graminis* and *F.graminearum* have some characteristics of active possessors. It is for these pathogens that efforts are to be made for their displacement or for nullifying their pioneer colonization. *F. oxysporum*, *F. solani*, and *F. culmorum* come under the category of combination possessors, that is, they are both active as well as passive possessors. They also need displacement for control through cultural practices.

12.5.2.3 Suppression of Germination and growth of the pathogen

This form of biological control has two aspects; 1. Reduction or prevention of germination (soil fungistasis) and 2. Slowing down of growth of germlings due to starvation, antibiotics, bacteriocins, mycoviruses, etc.

12.5.2.4 Protection of Infection court

This approach aims at encouraging the soil microbiota in or on the infection court which slow or prevent infection by the particular pathogen. Such protection mainly includes conditions where a week pathogen or non-pathogenic organism take possession of the sites of infection on the host. The mechanisms by which these precolonizers may protect the infection court include 1. Prior use of essential nutrients or oxygen needed by the pathogen, 2. Modification of rhizosphere pH, redox potential, and other abiotic factors that place the pathogen at a competitive disadvantage, 3. Production of antibiotics, 4. Hyperparasitism or exploitation of the pathogen, and 5. Modification of the host resistance.

12.5.2.5 Stimulation of host's resistance Response

This include cross protection provided by a weak strain of the same pathogen or by another pathogen. A tomato variety resistant to F. oxysporum f. sp. lycopersici, if inoculated with that pathogen, becomes resistant to *Verticillium dahlia*. Mint is resistant to *V. dahilae* if inoculated first with *V. nigrescens*.

12.5.3 Mechanism and process of decline

12.5.3.1 Antagonism - Antagonism is one main sub-division of microbial associations in soil. It implies that in any association of two or more species, at least one of the interacting species is harmed due to the activities of one or more of the rest. The mechanism of antagonism operate through:

12.5.3.2 Antibiosis - it is define as the condition in which one or more metabolites excreted by an organism have harmful effects on one or more other organism. In such antagonistic relationship species A produces a chemical substance that is harm or inimical to species B without species A deriving any direct benefit. However, the species A may have an indirect benefit in having a better competitive ability, thereby getting an advantage over species B for substrate colonization.

12.5.3.3 Competition – it is the indirect rivalry between two species for some feature of the environment that is in short supply. Broadly speaking competition could involve all kinds of interplay between organisms in which one is favored at the expense of the other. But in strict sense, if we

keep antibiosis or even exploitation restricted to their specific mode of action and results, competition has been defined as a more or less active demand in excess of the immediate supply of material or condition on the part of two or more organism.

12.5.3.4 Exploitation - when species A inflicts harm by the direct use of species B for its own benefit, it is exploitation (parasitism and predation). The two terms parasitism and predation have basically same effects, i.e., destruction of the host or prey. From a plant pathologist's view point, predation is a form of parasitism. However, the mode of operation makes the two terms somewhat distinct. In parasitism, some sort of etiological relationship between the parasite and the host is established and the host is not rapidly eliminated. A predator physically eliminates its prey by direct feeding on it without establishing any etiological relationship.

12.5.4 Mycoparasitism

Mycoparasitism is an act where one fungus parasitizes the other one. Barnett and Binder (1973) classified mycoparasitism into two main groups: i.e. necrotrophic and biotrophic, on the basis of nutritional relationship of parasite with the host. The necrotrophic (destructive) parasite makes contact with its host, excretes toxic substance which kills the host cells and utilizes the nutrients that are released. The mycoparasitism is of common occurrence and examples can be found among all groups of fungi from chytrids to the higher basidiomycetes. The mycoparasitism includes different kinds of interaction, viz. coiling of hyphae, penetration, production of haustoria and lysis of hyphae.

12.5.5 Biological control by Introduced Antagonists

From numerous experiments that have been conducted all over the world, a common fact emerges: reduction in the activity of a pathogen is often correlated with an increased in the population of antagonists as assessed in the soil. The use of selected antagonists is a direct method based on the theory that these antagonists when introduced in the soil, can act directly on the behavior of the pathogen. However, with few exceptions, the biological control of plant pathogen by augmenting soils with antagonists has remained restricted to research studies. Nevertheless continued scientific efforts have resulted in identification of several antagonists which have given successful biocontrol in the experimental plots and field when augmented in soil (Table).

Table: Example of biological control by Introduced Antagonists.

HOST DISEASE	PATHOGEN	ANTAGONISTS
Carnation disease	*F. oxysporum f. sp. dianthi, F. roseum, R. solani*	*Bacillus subtilis, Pseudomonas fluorescens, Thrichoderma spp.*
Chickpea root rot/wilt complex	*F. oxysporum f. sp. cicero, , R. solani, S. rolfsii*	*Thrichoderma harzianum, G. virens*
Cotton root rot	*R. solani,*	*Thrichoderma harzianum*

Cotton wilt	*F. oxysporum f. sp. vasinfectum*	*Thrichoderma harzianum, G. virens, Pseudomonas fluorescens*
Crown gall of wood trees	*Agrobacterium tumifaciens*	*Agrobacterium radiobacter*
Potato scab	*Streptomyces scabies*	*Bacillus subtilis*
Take all of cereals	*G. graminis var tritici*	*Pseudomonas fluorescens*

12.5.6 Biological seed treatment – A feasible delivery system

A more economical, effective and relatively nonpolluting delivering system of the antagonists is biological seed treatment because only small amount of materials is applied per unit area and comes in immediate contact with the target site. There are two general classes of seed treatment: those that alleviate stress associated with soil environment, and those that directly improve or increase plant growth. Several strategies can be employed for enhancing efficacy of biological seed treatments, as:
1. Permitting the bioprotectant to colonize the seed surface before planting.
2. Applying the bioprotectant in a thin layer that permits it to proliferate for a few hours after planting in the absence of competitive microflora, and 3. inclusion of adjuvants in seed treatment, which helps to control pH to a level favourable to the biocontrol agent but unfavourable to competitive microflora, add base selectively to the bioprotectant, use selective toxicants which do not restrict the growth of antagonists, and after the timing i.e. the period when antagonists is active in relation to the pathogen.

Table: Biological seed Treatment

Host	Pathogen	Antagonist
Bean	*Pythium spp.* *S. sclerotiorum* *B. cinerea*	*Trichoderma spp.* *T. koningii* *G. catenulatum*
Cauliflower	*P. aphanidermatum*	*T. harzianum*
Maize	*R. solani*	*T. harzianum*
Pea	*R. solani, P.ultimum*	*T. harzianum* *T. hamatum*

12.5.7 Organic Amendments- modification of soil environment-biological control

Modification of soil environment is one of the methods to achieve biological control. Amendment of soil with decomposable organic matter is recognized as an effective method of changing soil and rhizosphere environment. Such changes adversely affect the pathogens and empower plants to resist infection through better vigour and/ or altered root physiology. Such methods bring multiple pathogen suppression, lasting effects, less cost, no hazards, and improvement of soil fertility and nutrients uptake by plants. Crop residue decomposition encourages microbial activity both quantitatively and qualitatively. The enhanced microbial activity has two effects; it increases the variety of complex

organic compounds in soil and promotes the population of antagonists in soil. Together, these two factors increase the biological buffering capacity of soil. This capacity of the soil helps in disease prevention particularly in area where the pathogen is not yet established.

Amendments act against diseases through 1. Direct effect on the active pathogen on the root or in the rhizosphere, 2. Direct effect on the pathogen during its survival in the absence of the host, and 3. Indirect effect on the pathogenesis through the host. In the latter are included all those consequences of amendment which results from uptake of organic compounds by the plant roots, changed host physiology and possible development of resistance. Therefore, the amendments can reduce inoculum density (ID), inoculum capacity (IC), host pronensess and can also increase host resistance; the net result being reduction in disease severity.

Check your Progress- 3

Note: a. Write your answer in the space given below

b. Compare your answer with those given at the end of the unit.

4. What is Biological Control of plant diseases?

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12.6 INTEGRATED PLANT DISEASE MANAGEMENT

12.6.1 Meaning of Integrated Plant Disease Management

In a disease pyramid, three principal components are considered. These are host, pathogen, and the environment. All the three components act together during disease develop-ment. Thus, to protect a plant from disease(s) one has to tackle all the three parameters at the same time.

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

12.6.2 Procedure of Integrated Plant Disease Management

The procedure of management is different in respect of the principal component:

12.6.2.1 Management of Host

It includes: (a) Procedure to improve plant vigour. (b) Induction of disease resistance, and (c) Breeding for disease resistance.

12.6.2.2 Management of Pathogen

It includes: (a) Eradication or reduction of inoculum. (b) Application of chemicals on plant sur-face, to discourage the pathogen. (c) Prevention through legislation (i.e., keeping the pathogen away from the host) by quarantine, etc.

12.6.2.3 Management of Environment

It includes: (a) Crop management. (b) Soil management, and (c) Water management.

The main goals of an integrated plant disease control program are: 1. To eliminate or reduce the amount of initial inoculum. 2. To reduce the

effectiveness of initial inoculum. 3. To increase the resistance of the host. 4. To delay the onset of disease, and 5. To slow down the secondary cycle.

12.6.3 Programmes of Integrated Plant Disease Management

The programme of Integrated Plant Disease Control can be taken against a particular com-mon disease, such as late blight disease of pota-to or against all diseases affecting a particular crop such, as Potato, Apple, Citrus etc.

12.6.3.1 Integrated Management in an Annual Crop

1. Stock tuber should be free from any disease i.e., a healthy tuber is needed.
2. Field should be cleaned from previous years plant debris and tubers, if any, where patho-gen may perpetuate for the next season.
3. Crop rotation must be done with legume or any other crop plant, except Solanaceous members.
4. Plant should be protected from disease through foliar spray at regular intervals.
5. Make the soil loose by ploughing and expose it to sunlight for few days in summer months and/or fumigate the soil with chemicals.

12.6.3.2 Integrated Management in a Perennial Crop

1. Nursery-stock should be fumigated (to era-dicate the nematode, if present).
2. The stock should be fumigated (to eradicate the nematode, if present).
3. The growing field should be fumigated before planting to eradicate any pathogen like Armillaria, Phytophthora, if present.
4. Proper drainage in the field should be main-tained.
5. Stock should not grow in old field near the old plants that may carry different pathogens like canker, etc.
6. The field should be properly irrigated, and supplemented by fertilisers.
7. Plants should be sprayed regularly to protect them from different diseases and insect pests.
8. Usually the fruits become susceptible to several fruit rotting and fruit spotting fungi that attack at different stages till harvest or even in storage. So the fruits should be sprayed at 10-14 days' interval. Sometimes insects can damage at any stage, thereby the fungi infect the fruit through wound. Thus, the insecticides should be applied at intervals.

So the integrated control programme includes several control methods like legislation (quarantine, inspection etc.); cultural practices (sanitation, crop rotation etc.); chemical controls (spraying of plants, fumigation of soil, surface sterilisation of planting materials etc.). All the required procedures for a particular crop should be followed to overcome the disease(s) and/or insect pests.

Check your Progress- 4

Note: a. Write your answer in the space given below

b. Compare your answer with those given at the end of the unit.

5. What is Integrated disease management?

……
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12.7 ANSWER TO CHECK YOUR PROGRESS QUESTIONS

1. Roguing consists of completely removing or uprooting the diseased plants to prevent further spread of the disease. This method is widely adopted in the control of virus diseases spread by insects (cassava mosaic, yellow mosaic of blackgram and greengram, citrus tristeza, katte disease of cardamom, bunchy top of banana) and basal stem rot of coconut, green ear of pearlmillet and broomrape (Orobanche) in tobacco.
2. A variety of chemicals are available that have been designed to control plant diseases by inhibiting the growth of or by killing the disease cauing pathogens. Chemical used to control bacteria (bactericides), fungi (fungicides), and nematodes (nematicides) may be applied to seeds, foliage, flowers, fruit or soil.
3. There are few fungicides used to controlling diseases caused by Oomycetes: a. the carbamates (e.g.prothiocarb, propamocarb), b. the isoxazoles (e.g.hymexazol), c. Cyanoacetamide oximes (e.g.cymoxanil), d. Ethyl phosphonates (e.g.fosetyl-AL), and e. Phenylamides.
4. Biological control is the control of disease by the application of biological agents to a host animal or plant that prevents the development of diseases by a pathogens.
5. Integrated disease management (IDM) strategies advocate managing plant disease with minimal use of synthetic pesticides. However, IDM strategies are based on understanding the pathogen eitology, diversity and evolution.

12.8 SUMMARY

Measures taken to prevent incidence of a disease, reduce the amount of inoculum that initiates the spreads of disease and finally minimises the loss caused by the disease have traditionally being called as control measures. Various different measures is carried out to eliminate or manage the crop disease via cultural practies, biological control and chemical control measures. Cultural practices Manipulation of cultural practices to the disadvantage of the pest. It usually influence the development of disease in plants by affecting the environment. Cultural practices that leads to disease control have little effect on the climate of a region. Points to be selected for cultural practices is land selection, Site selection, Field Sanitation, Soils and Nutrition, Rogueing, Healthy planting materials, Time of sowing, Planting spacing, Fertilizer management, Water Management, Crop Rotation, Multiple cropping/Mixed Cropping, Trap Cropping, Soil amendment, Weed management, Selection of tolerant cultivar, Tillage operation. Biological control involves the use of microbial antagonists such as bacteria or fungi to suppress plant disease pathogens. Biocontrol have several importance and advantages over other control methods. Their mode of actions include antibiosis, competition, and parasitism and induced systemic resistance. There are however some limitations to the general use of biological control agents such variability in effectiveness, low spectrum action, short shelf life of products etc. A biological control agent *Trichoderma* spp. *T. koningii and G. catenulatum* can be applied as seed treatment.

Various chemical has been used to kill plant pathogen. The fungicides has been used widely to control plant pathogen. The fungicides can be

classified in to selective fungicides and non selective fungicides. Benzimidazoles and thiophanates represent a group of highly effective broad spectrum systemic fungicides. The past decade has witnessed the introduction of five classes of fungicides controlling diseases caused by Oomycetes: Some of the selective fungicides can be used to kill oomycetes. Anti- oomycetes are lisited namely 1. the carbamates 2. The isoxazoles, 3. Cyanoacetamide oximes, 4. Ethyl phosphonates, and 5. Phenylamides. The last class Phenylamides, cover three groups of compounds: 1. acylalanines, 2. Acylamino butyrolactones, and 3. Acylamino-oxazo lidinones. Non selective fungicides such as sulfur fungicides, copper fungicides, Mercury Fungicides, and Phthalamide and Quinone Fungicides has been used to kill plant pathogens.

Integrated plant disease management, which combines biological, cultural, physical and chemical control strategies in a holistic way rather than using a single component strategy proved to be more effective and sustainable.

12.9 KEY WORDS

Antagonism: Injury, killing or inhibition of the growth of one species of microorganism to another microorganism.

Antagonists: an organism having the capacity or ability to inhibit the growth or interfere with the activity of another microorganism.

Eradication: the control of plant diseases by eliminating the pathogen after it is established or by eliminating all of the plants that carry the pathogen.

Roguing: the removal of (usually diseased) plants from a growing crop.

Crop rotation: the successive planting of different crop species; often used to improve soil fertility or to reduce disease and pest problems.

Sanitation: the destruction or removal of infected and infested plants or plant parts; decontamination of tools, equipment, containers, work space, hands, etc.

Integrated Pest Management (IPM): Combining different management practices (cultural, chemical, physical, and biological) to reduce the amount of disease to a tolerable level (threshold) in a manner that is economical, efficient, and environmentally-safe.

Antibiosis: The inhibition or destruction of one organism by a metabolite produced by another organism.

12.10 SELF ASSESSMENT QUESTION AND EXERCISES

1. Define biological control.
2. What is crop Rotation?
3. Write short notes on fungicide.
4. What is Integrated Pest Management?
5. Wite essay on Chemical control.
6. Discuss in detail about the cultural practies method to control diseases.

UNIT – 13 PLANT DISEASES

Structure

13.1 INTRODUCTION

Plant disease, an impairment of the normal state of a plant that interrupts or modifies its vital functions. All species of plants, wild and cultivated alike, are subject to disease. Although each species is susceptible to characteristic diseases, these are, in each case, relative few in number. The occurrence and prevalence of plant diseases vary season to season, depending on the presence of the pathogen, environment conditions, and the crops and varieties grown. Some plant varieties are particularly subject to outbreaks of disease while others are more resistant to them. Infectious plant disease are caused by bacteria, fungi or viruses and can range in severity from mild leaf or fruit damage to death. In this unit, author has discussed about various crop disease such as tobacco mosaic virus, bunchy top of banana, blast and sheath blight of paddy, citrus canker, red rot of sugarcane and downey mildew of grapes. Unit deals with study about the disease, symptoms, causal organism, disease cycle and control or management crop disease.

13.2 OBJECTIVITIES

After going through the unit you will be able to:

1. Identify the name of the disease in various crop plants.
2. Understand the various symptoms of crop diseases.
3. Identify the causal organism of diseases in crop plants
4. Understand the disease cycle in infected crop plants.
5. Understand the suitable control measures or management of various crop diseases.

13.3 TOBACCO MOSAIC VIRUS

13.3.1 Introduction to Tobacco Mosaic Virus

This is the best known of all virus diseases. TMV is a simple rod-shaped helical virus (Fig. 13.20) consisting of centrally located single-stranded RNA (5.6%) enveloped by a protein coat (94.4%). The rod is considered to be 3,000 Å in length and about 180 Å in diameter. The tobacco mosaic virus affects all dicotyledonous plants of which most important are tobacco and tomato. But it does not affect any monocotyledonous plants.

Although Adolph Mayer in 1886 first pointed out the mosaic pattern on leaves of affected tobacco plants, it was not until 1898 the first scientific proof of the exis-tence of a virus was given by Beijerinck. Earlier than this, in 1892 Iwanowski demon-strated that tobacco mosaic virus would pass through a bacteria-proof filter. He was able to demonstrate that a diseased tobacco plant juice was able to induce mosaic disease in healthy tobacco plants. But Iwanowski could not find out the true signi-ficance of this.

Holmes in 1929 described the primary infection lesions of tobacco mosaic virus and in 1935 Stanley first isolated crystals of tobacco mosaic

virus and indicated their paracrystalline nature. Again Takahashi and Rawlins in 1933 de-monstrated the physical phenomenon of tobacco mosaic virus. Whereas, C. A. Knight showed that the tobacco mosaic virus is made up of sixteen amino acids.

The tobacco mosaic virus affects photosynthetic tissue of the host leading to distortion, blistering and necrosis. It also causes dwarfing of affected plants. It is one of the most damaging viruses of plants, causes enormous loss of tobacco crop by re-ducing yield and quality.

13.3.2 Symptoms of Tobacco Mosaic Virus

The symptom is systemic mosaic type. The primary symptom on young leaves is faint circular chlorotic lesions appear with gradual vein clearing.

This is followed by the development of characteristic systemic mosaic. With the maturity of the leaves, abnormally dark-green spots appear which develop into irregular crumpled blister-like areas while the rest of the tissue becoming more or less chlorotic (Fig. 392). Various degrees of leaf malformation like enations follow and some leaves exhibit only a mild diffuse mottle.

The development of symptoms is governed by many variable factors of which the most important is the difference in virulence of the virus strains. For example, one strain of tobacco mosaic virus may cause yellow mottling on the leaves, a second may cause necrosis only, whilst a third induces a gross malformation. Another variable factor is the variety of plant affected. In flowers, petals show mosaic symptoms. Severe strains cause streaking of stem. The disease is seldom fatal to the host.

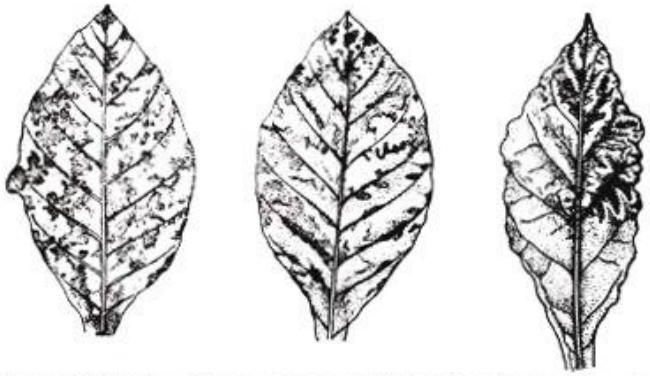

Fig. 392. Tobacco Mosaic Virus. Disease symptoms on tobacco leaves induced by ordinary or field strain.

13.3.3 Causal Organism of Tobacco Mosaic Virus

The typical tobacco mosaic virus is Tobacco mosaic virus 1, Marmor tabaci Holmes. The virus remains active in extracted host plant juice even up to 25 years. It is a very resistant virus, can stand desiccation for 25 years or more. It occurs in very high concentration in plant and its dilution end point is 10-6. The thermal inactivation point of the virus is 90°C.

The virus particles are rod-shaped (Fig. 393) measuring 280µ in length by 15µ, in width. The X-ray studies reveal that the virus particle consists of a number of protein subunits set in helical array with 49 subunits to one turn of the helix and 2,130 subunits in one rod. The ribonucleic acid thread inter-twines more or less centrally between the protein subunits.

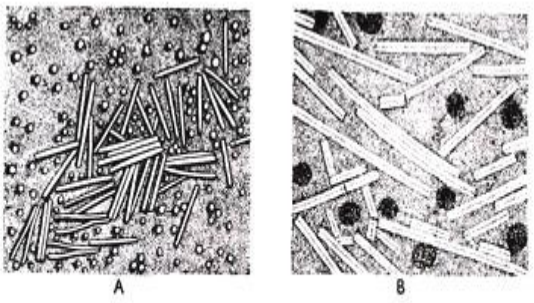

Fig. 393. Tobacco Mosaic Virus. A—B. Virus particles. A. Shadowed with palladium-gold. B. Stained with phosphotungstic acid.

The cells of tobacco plants infected with tobacco mosaic virus are characterized by the presence of certain cell inclusions. They are: (i) two types of intracellular inclusions, and (ii) intra-nuclear inclusion. The intracellular inclusions are: (a) X- bodies (Fig. 340C), and (b) striate material of crystalline plates (Fig. 340C).

The X-bodies are amorphous, protoplasmic more or less vacuolate inclusions. Whereas striate material of crystalline plates gives protein reaction. These crystals resemble the purified virus-protein crystals. The intra-nuclear fibrous and crystalline inclusions are produced by a yellow-mottling strain of tobacco mosaic virus.

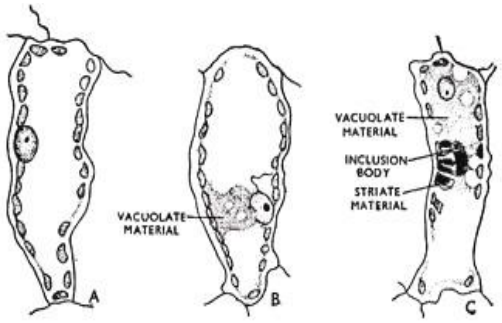

Fig. 340. Intracellular inclusions. A. Virus-free palisade cell of potato. B. Mild mosaic virus-infected palisade cell of potato showing vacuolate material close to the nucleus. C. Common tobacco mosaic virus-infected palisade cell of potato showing inclusion body (X-body).

13.3.4 Disease Cycle of Tobacco Mosaic Virus

The virus perennates in infected tobacco plant debris, tobacco refuse from warehouses, cigarettes, cigars, pipe and chewing tobacco and in perennating hosts which form the source of primary inoculum. This is one of the most infectious of the plant viruses. The virus is disseminated from plant to plant by mechanical transmission, by handling tobacco plants during transplanting; through other field operations; and contact by man and cultivation implements. The virus enters in the host tissue, it multiplies very rapidly producing disease symptoms.

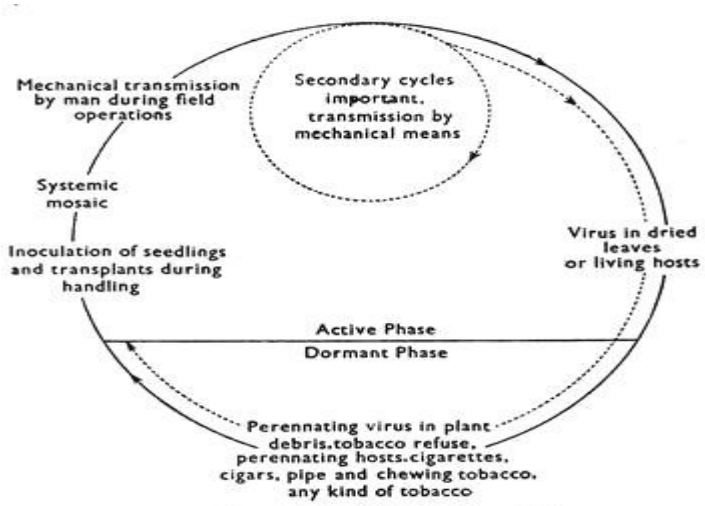

Fig. 394. Disease cycle of Tobacco Mosaic Virus.

Disease cycle of Tobacco Mosaic Virus is presented in Figure 394.

13.3.5 Control of Tobacco Mosaic Virus:

Following are some of the suggested control measures:

(i) Seed beds should be located at a great distance from the tobacco warehouses.

(ii) Seed beds should be free from any tobacco refuse.

(iii) Seed bed soil should be sterilized by steam.

(iv) Care should be taken to avoid contamination through hands and cultivation implements.

(v) Since pipe tobacco, cigarettes and chewing tobacco are all sources of primary inoculum, smoking or chewing of any kind of tobacco should be avoided.

(vi) Susceptible hosts, weed or otherwise in which virus may harbour, should be destroyed.

(vii) Previous year's plant debris should be destroyed by burning.

(viii) Diseased plants should be removed and burnt to stop further spread of the disease.

(ix) Growing resistant varieties produces good results.

Check your Progress- 1

Note: a. Write your answer in the space given below

b. Compare your answer with those given at the end of the unit.

1. What is tobacco mosaic virus?

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2. What are the symptoms of the tobacco mosaic virus?

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13.4 BUNCHY TOP OF BANANA

13.4.1 Introduction to Bunchy Top of Banana

Banana bunchy top disease is caused by Single strand DNA Virus (BBTV) and one of the most serious diseases of banana. It occurs in most banana-growing countries of the world except in Central and South America. BBTV is widespread in Southeast Asia, the Philippines, Taiwan, most of the South Pacific islands, and parts of India and Africa. In Hawaii,

BBTV was first observed in 1989 and is now widely established on Oahu. In 1995 it was discovered in the Kona area of the island of Hawaii, and in 1997 it was found on the island of Kauai. It causes severe losses because infected plants produce no fruit. New leaves of infected plants develop dark green streaks on their petioles and veins while the margins become chlorotic. The leaves at the top of the plant are narrower, upright, and closer together, making the top of the plant appear bunchy.

13.4.2 Symptoms of Bunchy Top of Banana

Bunchy top is named after one of the most characteristic symptoms of an advanced infection, when the leaves become progressively dwarfed, upright and bunched at the top of the plant, with wavy and chlorotic margins that tend to turn necrotic. The initial symptoms of banana bunchy top virus consist of dark green streaks in the veins of lower portions of the leaf midrib and the leaf stem (petiole). The streaks also occur, but are less prominent, in the veins of the leaf blade (lamina). This symptom is sometimes referred to as "Morse code streaking" because the streaks are irregular and resemble a series of "dots" and "dashes."Rubbing away the waxy white coating that covers the petiole or midrib makes it easier to see the streaking. Also, dark green, hook-like extensions of the leaf lamina veins can be seen in the narrow, light-green zone between the midrib and the lamina. The short hooks point down along the midrib toward the petiole. They can best be seen by back-lighting the leaf against the sky. Severely infected banana plants usually will not fruit, but if fruit is produced, the banana hands and fingers are likely to be distorted and twisted. The true stem (suckers) that develop after a "mother" plant has been infected with BBTV are usually severely stunted, with leaves that do not expand normally and remain bunched at the top of the pseudostem. These leaves are stiff and erect, are shorter and narrower than normal leaves, and have chlorotic edges. Suckers with these symptoms will not produce fruit.

13.4.3 Causal Organism of Bunchy Top of Banana

BBTV is the sole member of the genus *Babuvirus* in the family Nanoviridae. The genome of BBTV is made up of at least six circular, single-stranded DNA components, each about 1 kilo-base pair in length. Replication takes place by rolling circle replication, a unidirectional nucleic acid replication that can result in rapid synthesis of single-strands of DNA. It is known that Banana aphid (*Pentalonia nigronervosa*) transmits the virus from infected to healthy plants by feeding. Aphids feed on the plant phloem tissues by injecting their thin, flexible style into the epidermis of the plant tissue until it reaches the phloem of the leaves. Then the aphid injects saliva and sucks the cell contents. This ingestion of viral components is done inadvertently by the aphid. Vector transmission of the BBTV is circulative and non-propagative, meaning that transmission of the virus occurs from and to the phloem tissues and the virus does not replicate within the aphid's mid-gut. Acquisition of the virus by the banana aphid requires about 18 hours of feeding and then the aphid can retain the virus for approximately two weeks.

An early symptom of BBTV infection is "Morse code

The "hooking" symptom of BBTV, where the veins of the leaf blade extend into the normally clear area along the

13.4.4 Disease Cycle of Bunchy Top of Banana

Banana bunchy top virus is spread by the banana aphid, which acquires the virus after at least four (but usually about 18) hours of feeding on an infected plant (Figures 4 and 5). The aphid can retain the virus through its adult life, for a period of 15–20 days. During this time, the aphid can transmit the virus to a healthy banana plant by feeding on it, possibly for as little as 15 minutes but more typically for about two hours. Disease symptoms usually appear about a month after infection.

13.4.5 Control of Bunchy Top of Banana

The most important factors in controlling banana bunchy top virus are killing the aphid vector (disease carrier) and rouging (removing and destroying) infected banana plants. By killing the aphids on the banana plant, dispersal of virus-carrying aphids to nearby, healthy banana plants is avoided. Since the only host of BBTV is banana, rouging infected trees reduces spread of the virus by reducing the opportunity for aphids to acquire the virus or for people to obtain and transport infected suckers or planting material.

Check your Progress- 2

Note: a. Write your answer in the space given below

b. Compare your answer with those given at the end of the unit.

3. How do you prevent banana bunchy top virus?

……

……

13.5 BLAST DISEASE OF PADDY

13.5.1 Introduction to Blast Disease of Paddy

Blast disease or rotten neck is one of the most serve disease affecting paddy. It is a fungal disease prevalent all over the word. It is a major problem in rice production in countries like Japan, India, Taiwan and the USA. Blast disease is more serve in areas with high humidity and rainfall. Losses due to the disease may be up to 90% of the total corp. In India, the blast disease is more common in southeren parts, particularly coastal areas.

13.5.2 Symptoms of Blast Disease of Paddy

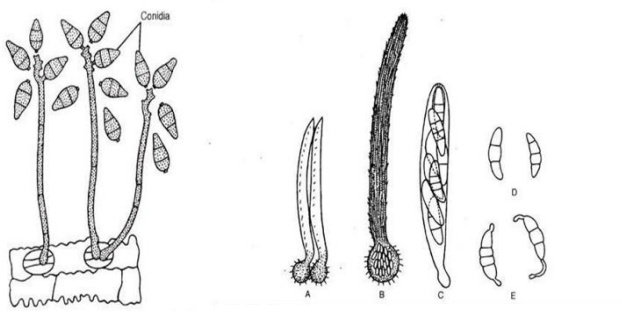
The blast is a foliage disease. The symptoms also occur in other plant parts. Leaf blade, leaf sheath, petiole, rachis, stem etc. are affected by the disease. Brownish lesion and spots are formed on leaf blade, leaf sheath culms and panicles. The spots are spindle-shaped with grey or white central part and brownish or reddish borers. The spots enlarge as the disease progress. Brown to black spots or rings is formed on the rachis of the mature inflorescence. Small brown or black spots on ear heads. Shriveled culms in server cases. The culms were covered with fluffy mycelium of the pathogen. The occurrence of bluish patches on the neck or stem. If the infection occurred before the grain formation, panicles droops and no grain formation. If the infection occurred after grain formation, the grains become small, whitish and chaffy. In advanced stages of the disease, necrotic rotting of neck and falling of the ears occurs. Plants become stunted and untimely leads to the complete death of the plant.

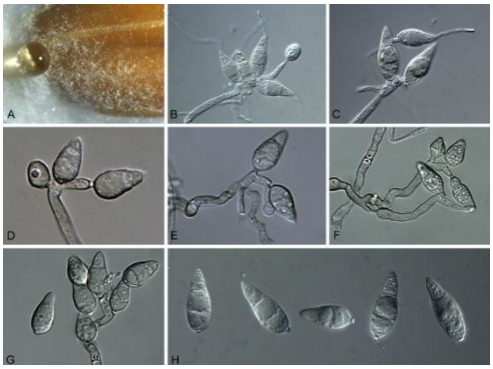
Blast disease of paddy: Symptom- Initial Stage

Spindle-shaped spots with grey/white central part and brownish borders

13.5.3 Causal Organism of Blast Disease of Paddy

The causal organism i.e., pathogen of the blast disease of rice is *Pyricularia grisea* (Cooke) Sacc. named earlier as *Trichothecium griseum*, *Pyricularia oryzae* Cav. or Dactylaria oryzae. The perfect stage of the patho-gen is *Magnaporthe grisea* (Herbert) Yaegashi and Uddagawa, an ascomycetous fungus. The mycelium is septate, uni- or multi-nucleate, branched and hyaline to olivaceous in colour. The conidiophores develop singly or in fascicles through the stomata. They are simple or rarely branched, 2-4 septate, with swollen base and show truncated denticles along its length (in mature stage). Conidia are pyriform (pear-shaped) to obclavate in shape with rounded base and narrowed tip, 2-septate, rarely 1-3 septate; hyaline or pale olive, 14-40 µm x 6-1 3 µm, with a distinctly protruding basal hylum. A typical lesion on leaf can develop 4,000 to 6,000 conidia/night for more than two weeks. During germination, appresoria are deve-loped at the tip of germ tube, either on host surface, on glass slide or on cellophane paper. They are globose, oblong or ovoid and measure 5-15 µm in diameter.The fungus often produces chlamydospores of about 5-12 µm in diameter.Pathogen can develop perithecia in culture. It is named as Magnaporthe grisea, and also called as Ceratosphaeria grisea and Phragmoporthe grisea. Perithecia are dark brown to black, glabrous, elongated, with spher-ical to subspherical base, mostly 100-180 µm in diameter with a long neck 500-1,200 µm in length.The neck is cylindrical of more than half of its length i.e., up to 1,100 µm. Asci (Fig. 5.11C) are cylindrical to clavate, hyaline, uni- tunicate (ascus has single wall), with short stipe measuring 60-80 µm x 10-12 µm. It contains 8 ascospores. The asci are intermingled with paraphyses. Ascospores are hyaline, biseriate, fusiform, curved with rounded ends, 1-4 septate (generally 3 septate), slightly con-stricted at the septa measuring 18-23 µm x 5-7 µm. Germination is bipolar.





Pyricularia oryzae: Mycelium with Conidia

13.5.4 Disease Cycle of Blast Disease of Paddy

The pathogen can perpetuate in grain both externally and internally, on straw piles and also on host other than rice. The inocula, i.e., the conidia, developed from all or any of the above sources can infect the leaf of the rice plant. The infected regions develop crops of conidia; those are disseminated and infect the different regions of the same plant and /or the other rice plants. Towards the end of the season, the pathogen may perennate as coni-dia on seed and also on straw piles. Next season, perennating conidia may serve as a source of primary inoculum. Likewise, in some areas they develop perithecia containing asci and ascospores. The perithecia remain with the plant debris or straw piles. Next season, the ascospores may cause infection.

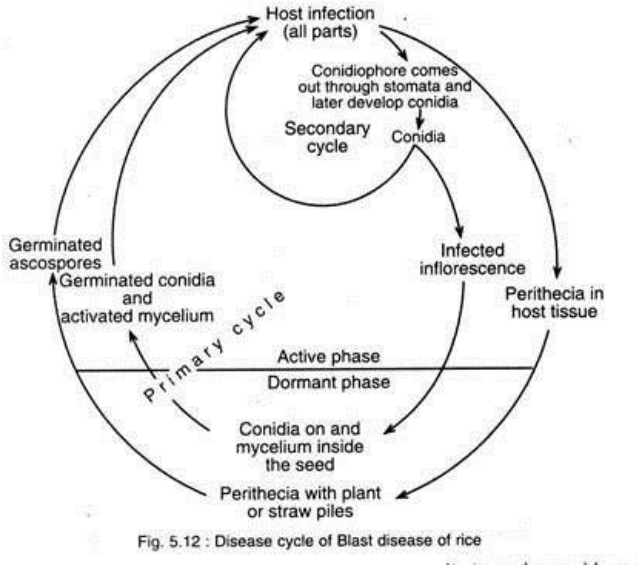


Fig. 5.12 : Disease cycle of Blast disease of rice

Disease cycle of Blast Disease of paddy

13.5.5 Control of Blast Disease of Paddy

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

A. Cultural Methods:

The following practices are used to reduce the disease incidence:

1. Early Planting:

Early planting shows less prevalence of disease than late planting.

2. Sanitation:

Sanitation i.e., cleaning of the plant debris from the field reduces the source of primary inoculum, thereby reduces the disease incidence.

3. Destruction of Collateral Hosts:

Destruction of collateral hosts like *Panicum repens, Leersia hexandra* etc., reduces the disease incidence.

B. Chemical Methods:

Chemical of different groups are used in different ways to control the disease.

These are:

1. Seed Treatment:

The pathogen is seed borne, thereby seed treatment is essen-tial to reduce the disease.

The seeds are treated with different chemicals to reduce the disease incidence:

i. Seed treatment with Agrosan GN (organomercurial) is effective to eliminate the seed borne inoculum present externally.

ii. Copper sulphate (20 ppm) mixed with Aureofungin (20 ppm) is effec-tive to control seed borne inoculum.

iii. Kapoor and Singh (1982) reported that seed treatment with Benomyl (1 : 400 w/w) gives protection to the seedling by inhibiting spore germi-nation and appresorium formation of the pathogen.

iv. Fungorene (8g / kg seed) is found to be highly effective in controlling the seed borne inoculum.

2. Foliar Spray:

Cu-fungicides are not effective during severe appearance of the disease. It causes phototoxicity, thereby reduces yield. Copper fungicide mixed with mercury compound and Phenyle mercuric acetate is highly effective as foliar spray.

C. Disease Tolerant Varieties:

The following cultivars are recommended as disease tole-rant varieties: A-67, A-90 and A-200 (in Maharastra); T-141 and T-603 (in Orissa); Co-4 (in Tamil Nadu); Kamala and Kukulu (in Bihar); Jaya and Pankaj (in Madhya Pradesh), etc.

Check your Progress- 3

Note: a. Write your answer in the space given below

b. Compare your answer with those given at the end of the unit.

4. What is Rice blast?
5. What do I look for?
6. How does it spread?

……
……..

13.6 SHEATH BLIGHT OF PADDY

13.6.1 Introduction to Sheath Blight of Paddy

Sheath blight is a fungal disease caused by *Rhizoctonia solani*. Infected leaves senesce or dry out and die more rapidly, young tillers can also be destroyed. As a result, the leaf area of the canopy can significantly be reduced by the disease. This reduction in leaf area, along with the diseased-induced senescence of leaves and young infected tillers are the primary causes of yield reduction. Sheath blight occurs in areas with high temperature (28−32°C), high levels of nitrogen fertilizer, and relative humidity of crop canopy from 85−100%. Plants are more vulnerable to sheath blight during the rainy season. High seeding rate or close plant

spacing, dense canopy, disease in the soil, sclerotia or infection bodies floating on the water, and growing of high yielding improved varieties also favor disease development.

13.6.2 Symptoms of Sheath Blight of Paddy

Symptoms are usually observed from tillering to milk stage in a rice crop and include the following:

1. Oval or ellipsoidal greenish gray lesions, usually 1-3 cm long, on the leaf sheath, initially just above the soil or water level in the case of conventionally flooded rice.

2. Under favorable conditions, these initial lesions multiply and expand to the upper part of the sheaths, the leaves, and then spread to neighboring tillers belonging to different hills (transplanted rice) or plants (direct-seeded rice).

3. Lesions on the leaves usually have irregular lesions, often with gray-white centers and brown margins as they grow older.

4. In subtropical environments, the disease is mostly initiated by sclerotia (up to two million of which can be produced per square meter in a diseased crop).

In tropical ecosystems, however, the role of sclerotia in initiating the disease is uncertain. In any case, the pathogen does not produce aerially dispersed spores; its dispersal is essentially dependent on hyphae running on plant tissues and producing new infections

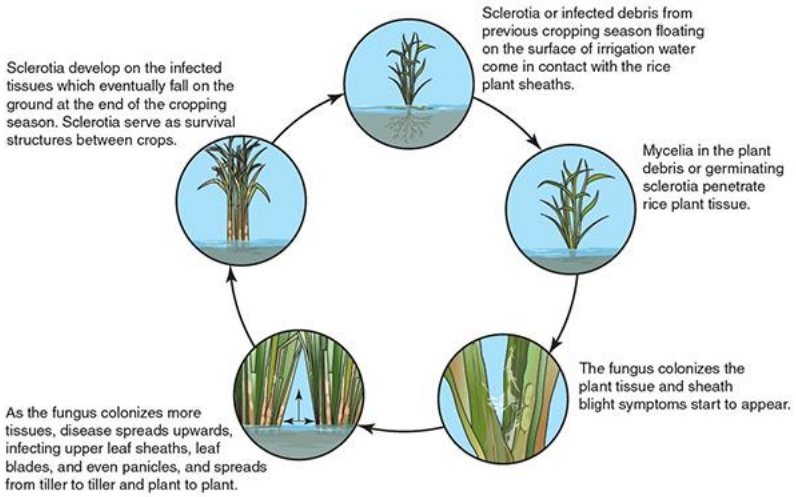
Sheath blight has symptoms similar to stem rot and stem borer infestation. To confirm the cause of disease, check for irregular lesions usually found on the leaf sheaths (initially water-soaked to greenish gray and later becomes grayish white with brown margin). Also check for presence of sclerotia.

Symptoms of Sheath blight of Paddy

13.6.4 Disease Cycle of Sheath Blight of Paddy

Sclerotia and mycelia in infected plant debris are two primary sources of inoculum. During the cropping season or at harvest, sclerotia fall on the ground and serve as survival structures from one cropping season to other. They survive for long periods in the soil, with up to 2 years in temperate rice production areas, and frequently accumulate in the field over time. Field water movement and irrigation support the dispersal of sclerotia and infected plant debris. Initial infections start with a

sclerotium or a piece of infected debris floating on the water surface and coming in contact with the sheath. The fungus gets attracted to the chemical stimuli released by the rice host. Germinating sclerotia or mycelia in debris penetrate the plant tissue either by means of natural openings or by specialized infection structures called appressoria or infection cushions. The fungus also produces extracellular enzymes that degrade plant cell walls to facilitate colonization. Once the fungus penetrates and colonizes the plant tissue, symptoms are initiated. The fungus grows upwards on the plant, penetrates, and infects upper leaf sheaths, leaf blades, and panicles (Figure 8). The fungus spreads in the field by growing its runner hyphae from tiller to tiller, from leaf to leaf, and from plant to plant, resulting in a circular pattern of damage. The infection spreads most quickly when susceptible varieties are grown under favorable conditions such as warm temperature (28 to 32°C), high humidity (95% or above), and dense stands with a heavily developed canopy. The disease frequently starts during the late tillering to joint elongation stages of plant growth and becomes more aggressive as the rice plant shifts to the panicle differentiation (reproductive) stage.



13.6.5 Control of Sheath Blight of Paddy

There is currently no resistant rice variety available for cultivation. The main management options available to minimize sheath blight include:

1. Use a reasonable level of fertilizer adapted to the cropping season.
2. Use reasoned density of crop establishment (direct seeding or transplanting).
3. Carefully control of weeds, especially on the levees.
4. Drain rice fields relatively early in the cropping season to reduce sheath blight epidemics.
5. Use fungicide to treat seeds.
6. Improve canopy architecture by reducing seeding rate or providing wider plant spacing.

Check your Progress- 4

Note: a. Write your answer in the space given below

b. Compare your answer with those given at the end of the unit

7. Name the causal agent for blast and sheath blight of paddy disease.

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……

8. How does it spread?

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……..

13.7 CITRUS CANKER DISEASE

13.7.1 Introduction to Citrus Canker Disease

Citrus Canker is a bacterial disease of worldwide distribution occurring wherever citrus is grown. It is a serious menace to our most valued citrus orchards causing objectionable blemishes on the fruit. The disease causes serious damage in India, China, Japan and Java. The pathogen incites severe canker disease in a number of citrus species on stems, leaves and fruits. The disease attacks most of the species/varieties of citrus. The most susceptible species are the acid lime plants, the sweet orange and the grape fruit.

13.7.2 Symptoms of Citrus Canker Disease

Crast-like disease lesions or scabby spots and small cankers (open wounds or dead tissue surrounded by living tissue) appear on all over ground parts of the plant such as leaves, young branches and fruits. The trees are, however, not commonly killed. The lesions on the foliage, at first, appear on the lower surface as small round raised spots. These are translucent and of yellowish brown colour. Later the spots turn white or greyish and finally rupture. The older lesions are corky and brown, sometimes purplish.

The necrotic brownish canker regions are surrounded by a yellowish brown to green raised margin and distinct watery yellow halo region. The yellow halo region is free from the pathogen. The cankerous lesions contain the pathogen in millions. Mairie suggested that the halo regions are formed due to the response of the host tissue to a diffusible metabolite of the pathogen. Padmanabhan et al. (1975) reported accumulation of malic acid in the halo region due to increased respiration in this region. The lesions on the twigs are usually irregular in form. The lesions on the fruit are similar to those on the leaves but lack the yellow halo.

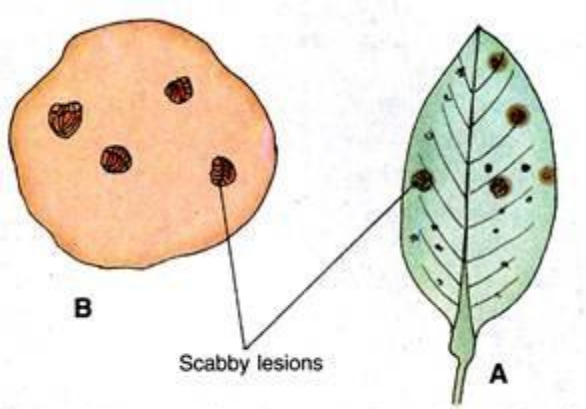

Fig. 22.28. Symptoms of Citrus Canker on leaves (A) and fruits (B).

13.7.3 Causal Organism of Citrus Canker Disease

The causal organism is the bacterial pathogen *Xanthomonas citri*, now called *X. campestris* pv. citri (Hasse) Dowson. It consists of a short, motile rod (1.5-2.0 x 0.5- 0.75 µ) furnished with a single polar flagellum (monotrichous). It lacks endospore formation. It is a gram negative, aerobic form surrounded by a mucilaginous capsule. It forms chains. The climate factors which favour the disease are the mild temperature and wet weather. The most suitable range of temperature appears to be 20ºC to 30ºC.

13.7.4 Disease Cycle of Citrus Canker Disease

Infection takes place through the stomata and wounds. The disease is not soil borne. The pathogen perennates in the old lesions on the twigs still attached to the host plant. From there it is carried by driving rains and by insects to new localities. Man functions as the chief agent of dissemination by planting infected nursery stock in new localities.

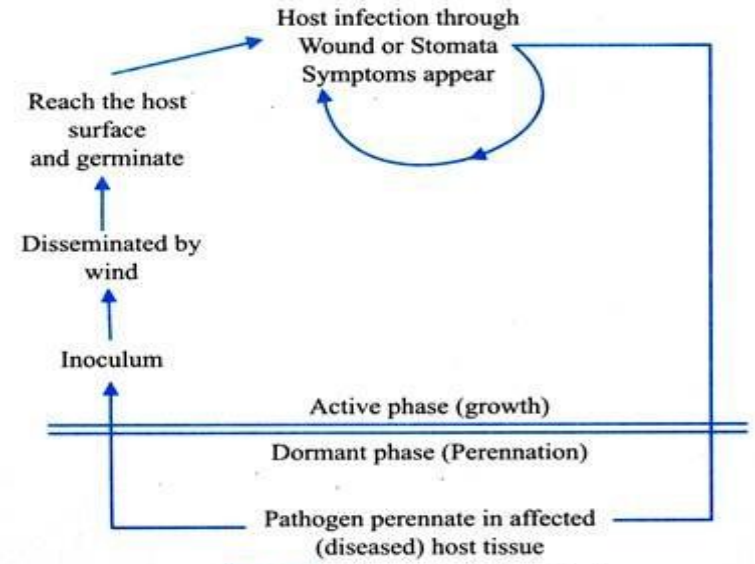

Fig. 22.29. Disease cycle of Citrus Canker.

13.7.5 Control of Citrus Canker Disease

To combat the disease in order to prevent economic loss or to reduce to a low level following measures can be suggested:

1. Eradication:

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

2. Pruning:

The infected trees may be cured by removing the diseased foliage and branches with pruning scissors and then spraying the trees with one percent Bordeaux mixture at regular intervals.

3. The use of disease free nursery stock for planting is the best method of controlling the disease.

4. The fallen infected leaves and twigs should be collected and burnt.

5. Spraying: Spraying with Bordeaux mixture and lime sulphur is a useful measure to protect the fruit. It should be done during the first three months after the beginning of fruit formation. Spraying should commence before the onset of rains and repeated during the rainy season.

6. Citrus nurseries should be raised in places away from the regions of heavy and protracted rainfall. There should be no "khatti" hedge around the nurseries.

7. Rangaswamy (1957) reported that the use of antibiotic sprays is useful in controlling the disease. Streptomycin sulphate and Phonomycin have been found to be effective. Vaheeddudin (1959) found that spraying with neem-cake is effective in controlling citrus canker.

Check your Progress- 5

Note: a. Write your answer in the space given below

b. Compare your answer with those given at the end of the unit

9. What is citrus canker disease?

10. How do you prevent citrus canker disease?

13.8 RED ROT OF SUGARCANE

13.8.1 Introduction to Red Rot of Sugarcane

This is one of the most severe of the known diseases of sugarcane. It was first described from Java by Went in 1893. It is widely distributed throughout the sugar-cane-growing countries of the world, and in fact it is extremely doubtful if there are any sugarcane-growing areas where it does not exist, although it may be much more destructive in some places than others.

The disease was very widespread and virulent in North Behar and Eastern part of the United Provinces during 1939 and 1942. It was so destructive that it almost whipped out the sugarcane plantations in those areas.

13.8.2 Symptoms of Red Rot of Sugarcane

The first external evidences of disease are the drooping, withering, and finally yellowing of the upper leaves. This is followed by a similar wilting of the entire crown, and finally the entire plant shows indications of

disease and dies. When not severe, the eyes frequently die and blacken and the dead areas extend out from the nodes.

Infection in the stem being internal, the presence of the disease is not visible externally. Upon splitting a diseased cane during the early stages of the disease, it will be found that the fibro-vascular bundles near the base are reddish in colour. The host tissue reacts vigorously to the presence of the fungus and some kind of reaction or change sets in the host cells in advance of the hyphal invasion.

The protoplasm changes colour and a gummy dark-red material oozes out of the cells filling the in-tercellular spaces. The soluble pigment present in this ooze, is absorbed by the cell wall producing the characteristic red rot appearance.

However, the presence of a red colour in the fibrovascular bundles is not necessarily an indication of this disease, since the colour may be due to any one of many other causes. As the disease advances the red colour spreads to the surrounding tissues extending through many internodes and irregular discoloured blotches are formed, which may be reddish or yellowish or white with red margins (Fig. 374A).

These white areas with red margins are a positive proof of the disease. When the stem is completely rotted inside, the natural bright colour of the rind disappears and turns dull as it shrivels. Black specks appear on shrivelled rind. The stem shrinks at the nodes (Fig. 374C). Split cane gives sour smell and shows red tissue with white cross-bands.

About this time the upper leaves of the stem turn pale and gradually droop down. These leaves then wither at the tips and along the margins. Ultimately the entire plant withers and droops down. In areas where the disease appears in a severe epidemic form, the en-tire crop withers and droops resulting in a complete loss of crop.

Though the fungus attacks all parts of the host above ground, stems and midribs of leaves are more susceptible to fungal attack. Infection in the leaves is visible along the midribs as dark-reddish zones having tendency to elongate rapidly turning blood-red enclosed by dark margins (Fig. 374B). When the infection becomes old, the central blood-red colour changes to straw colour.

The hyphae after ramifying in the infected host tissue collect beneath the epidermis and form a stroma of densely packed cells and ultimately an acervulus is developed resulting in the rupture of host epidermis. The acervulus bears long septate setae along with short conidiophores on which falcate (sickle-shaped) conidia are borne (Fig. 374D to F).

After growing for a period within the host tissue, the hyphae produce a large number of chlamydos-pores in the pith parenchyma. The chlamydospores persist in the soil for a long time.

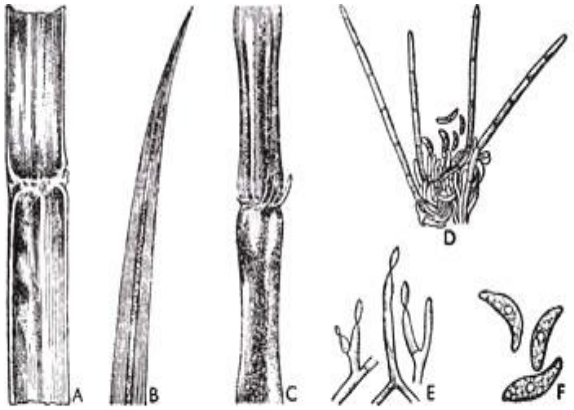

Fig. 374. Red Rot of sugarcane. A—C. Disease symptoms. A and C. On stem. B. On leaf. D. An acervulus. E. Conidiophores producing conidia. F. Conidia.

An examination of the diseased tissues with a microscope will reveal more or less mycelial threads of the fungus, or if the diseased canes are split and put in a moist chamber the fungus will develop readily and be easily recognized.

13.8.3 Causal Organism of Red Rot of Sugarcane

Red rot of sugarcane disease is caused by *Colletotrichum falcatum* Went, the perfect stage of which is *Glomerella tucumanensis* (Speg.) Arx and Muller. There has been considerable difference in opinion as to the nature of the fungus that causes this disease. Some insisted that this fungus is more strictly saprophytic than parasitic, and that it cannot attack healthy canes.

Others said that it cannot attack mature canes except through wounds, but that it can attack young plants. However, the young canes are usually protected by the leaf sheaths. In some places the fungus has been reported to grow on the dead canes only and the disease is not known.

The mycelium of the fungus grows both inter- and intracellularly in the parenchymatous cells of the host tissue. The hyphae are colourless, slender, freely branched and septate. Acervuli appear just above or below the nodes along the de-pressions or ridges.

They are black velvety bodies, develop in clusters. Acervuli are cuspidate with irregularly arranged setae (Fig. 374D). Aseptate conidiophores 20µ long and 8µ wide, on which one-celled falcate conidia are borne. Conidia are 16 to 48µ long and 4 to 8µ broad. They bear large oil globule in the centre. Chlamydospores are terminal or intercalary.

The perfect stage was reported from India under cultural condition in 1952 and under natural conditions on sugarcane leaves in 1953. It comprises of perithecia which are globose superficial with bottom embedded in the host tissue. Asci are numerous, clavate and paraphysate bearing 8 ascospores which are aseptate, hyaline and el-liptical.

13.8.4 Disease Cycle of Red Rot of Sugarcane

The sources of primary inoculum are the old fragmented stalks and leaves and other rubbish on which the fungus grows saprophytically; and unknowingly planted diseased stock during cultivation. Ratoon crops also serve as a source of primary inoculum. Opinions differ whether the fungus is strictly saprophytic or parasitic.

The conidia that are produced in the acervuli developed along the midribs of the diseased leaves during primary infection, form the secondary inoculum. They are disseminated by wind, rain splashes, irrigation water and also by insects. The conidia germinate readily by germ tube which on coming in contact with any hard surface, e.g., soil particles or plant parts, forms appressorium from-which infection hypha is produced.

The pathogen may gain entrance through the nodes at the leaf scars, through any kind of wound, through root primordia and seed-cuttings. The diseased canes are frequently found to be injured by insects, especially borers, and no doubt these wounds facilitate the entrance of the fungus, which in turn does much more damage than the insects.

Red rot is not a root disease, though roots are often infected by the fungus. High humidity due to water-logging, weak growth of host plant for want of proper cultural operations, continuous cultivation of the same variety of sugarcane in a particular locality, and cultivation of susceptible cane variety in the neighbouring areas are some of the aspects that help disease incidence and often to epiphytotic.

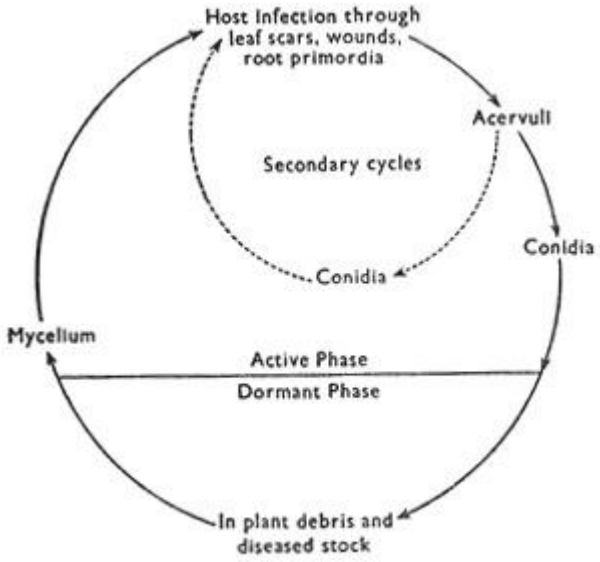

Fig. 375. Disease cycle of Red Rot of sugarcane.

Disease cycle of Red Rot of sugarcane is presented in Figure 375.

13.8.5 Control of Red Rot of Sugarcane

Red rot of sugarcane is hard to control because the stalk from which seeds are prepared has been largely affected from the time of planting, and fungicides cannot reach the infected tissues inside a diseased seed sett. Therefore careful selec-tion of red rot-free seed setts is recommended for planting. Seed should always be taken from disease-free nurseries examined regularly by the cane protection staff. Before planting, each seed sett should be carefully examined and those setts which show reddening should be discarded.

The spread of the red rot can be prevented during the growing season by timely rogueing and burning of the affected clumps with utilization of the green leaves for cattle fodder. In no case ratoons of sugarcane should be kept in the red rot affected fields. Attention should always be given to sanitation by digging out stubbles of di-seased canes

and burning them with other trash in the field. Where facilities are available for hot water treatment of seeds, they can be uti-lized for controlling red rot of seed (treat in water at 50°C., for two hours). Treating seed with fungicides like Arasan (0.25 per cent.) is often effective. The use of sugarcane varieties resistant to red rot is also recommended. Some of the resistant varieties are: Co. 975, 1148, 1158, 1336 and 6611; Co. S 561, 574; B.O. 3, 10, 47. The possibilities of an epidemic is very much minimized with the practice of long crop rotations (2 to 3 years) where planting is done in plots. One of the best ways to reduce the incidence of the disease is to raise healthy stock for planting in plots especially fertilized, cultivated, and kept disease-free by constant care.

Check your Progress- 6

Note: a. Write your answer in the space given below

b. Compare your answer with those given at the end of the unit.

11. Name the causal agent of the red rot disease of sugarcane.

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13.9 DOWNEY MILDEW OF GRAPES

13.9.1 Introduction to Downey Mildew of Grapes

This is one of the best known of the many diseases of plants of economic impor-tance. It has an interesting historical background associated with the accidental discovery of Bordeaux mixture as a fungicide by the French plant pathologist, Millardet who perfected the Bordeaux mixture as a spray for this disease.

The best information available indicates that the downy mildew of grape is endemic in North America, where it was for the first time reported by Schweinitz in 1837. This disease was introduced in France in 1874 and subsequently in other parts of Europe by 1917, where it became a serious disease because of frequent epiphytotics. It is also well established in North and South Africa, Australia and New Zealand.

The disease is confined largely to species and varieties of grapes (Vitis) although it has been reported also on the five-leaved ivy (*Parthertocissus quinquefolia* Planch.), and on English ivy (*P. tricuspidata* Planch.).

13.9.2 Symptoms of Downey Mildew of Grapes

The disease attacks all green parts of the plant leaf blades, petioles, tendrils, green shoots, and fruits at different stages of development. Early symptoms of the disease on the leaves consist of round light-green spots of an oily appearance on the upper surface, which enlarge even 1 /2 cm or more in diameter. On the corresponding under side white downy mildew consisting of the tufts of sporangiophores soon appear, bearing sporangia in great numbers (Fig. 361B).

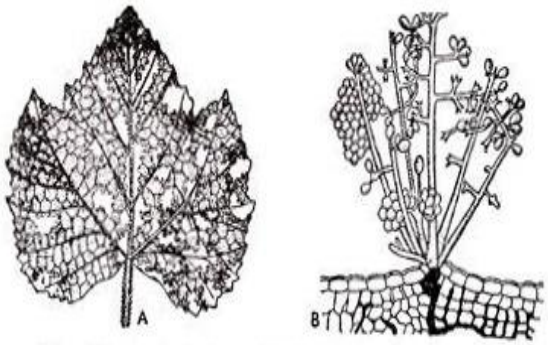


Fig. 361. Downy mildew of grape A. A diseased leaf. B. Section of a diseased leaf with sporangiophores emerging from a stoma.

In moist weather it persists; in extremely dry weather it may disappear. Later the spots become yellow, or variegated with tints of yellow and yellowish-brown forming patches of irregular shape, especially between the larger veins of the leaf (Fig. 361 A). Old spots turn brown owing to the killing of the leaf tissue.

The leaf spots may be few in number or as numerous as to, coalesce and involve nearly the entire leaf area. At this time the leaves are in a morbid condition, and the fungus within enters upon the sexual phase, producing oogonia, anteridia, and finally oospores which survive the winter in the fallen leaves.

On the stems, the lesions are brown and sunken, and along with the death of the affected parts, portions of the wine become brittle and break off easily. In extreme cases of infection the whole shoot may be dwarfed, the leaves remaining very small and densely covered with the mildew.

Flowers may be completely blighted by early attacks of the disease. Fruits may be attacked when young or when approaching maturity. The young fruits show brow-nish spots and later become covered with downy mildew, their growth is checked. The young fruits then darken and finally dry up. They may also assume a reddish- brown colour and failing to ripen, develop a soft root. On full-grown fruits, brownish patches appear and the fruits harden ultimately becoming mummified. The disease may often strip plants of their leaves and tendrils, flowers may fail to set, fruit may be destroyed in the early stages of growth, causing severe loss.

Symptoms of Downy Mildew of Grape *Plasmopara viticola*

13.9.3 Causal Organism of Downey Mildew of Grapes

Plasmopara viticola (Berk, and Curt.) Berl. the characteristic aseptate, intercellular mycelium produces knob-like haustoria to absorb

food from the host cells. Fasciculate- sporangiophores arise from the hyphae in the inter-cellular spaces just beneath the lower epidermis and emerge through the stomata.

They are branched monopodially along the main trunk and dichotomously at the extremities. The branches in turn give off other branches more or less at right angles so that the system of branches shows many small branches with arms projecting out in cross-like fashion.

The tips of the branches are provided with short finger-like pro-jections on which the sporangia are borne. The sporangia are hyaline, ovate, and attached to the sporangiophore branches at the small end, in which the wall is thin-nest. The profuse growth of sporangiophores and sporangia on the surface of the host accounts for the downy appearance which characterizes the downy mildew.

The sporangia germinate by secondary zoospores which after an active period shed their flagella and each one produces a. germ tube from which aseptate mycelium is deve-loped. The oospores are produced from the mycelium embedded within the leaf tissue. The oospore consists of a thick endospore wall surrounded by a thinner rough expo-sure wall. On germination, an oospore sends up a short un-branched stalk, at the apex of which a single sporangium is produced. The sporangium so formed germinates by zoospores exactly in the same manner as the sporangia borne on the sporangiophores.

13.9.4 Disease Cycle of Downey Mildew of Grapes

In general, the sporangia are not adopted for long survival but serve to spread the disease in localities where the leaves may remain on the vines all the year round. In such cases infection by sporangia may be continuous from season to season. On the other hand, the oospores are capable of survival in the soil for at least a year.

Oospores which remain in the fallen leaves, vine debris, or shriveled fruits on the ground are probably responsible for the primary infections which break out in the spring. Moreover the fungus may, in some localities, survive in the form of mycelium perennating in the winter buds.

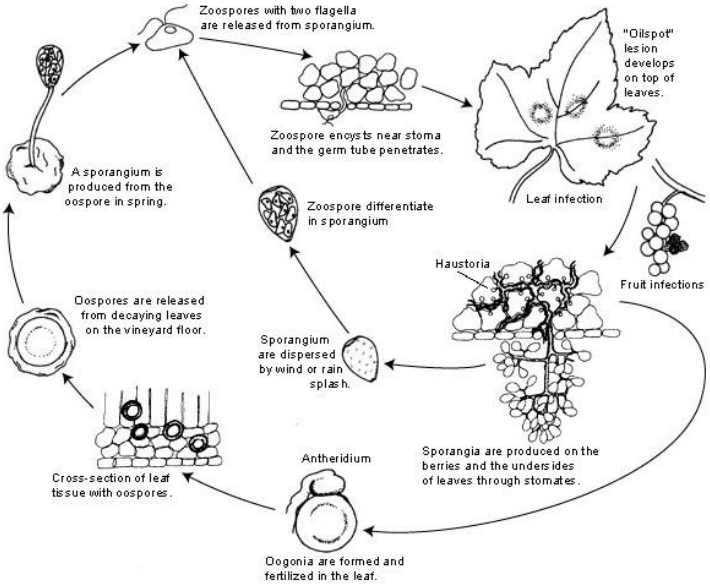
The oospores germinate at a temperature range of 13°C to 33 °C. Primary in-fections are believed to occur when the zoospores formed during oospore germination, are conveyed by splashing raindrops from the soils on to the lowermost leaves of the vines.

It may so happen that the sporangia produced during oospore germination may themselves be carried on the leaves near the ground and then zoospores are produced there. In any case, the earliest symptoms usually appear on the leaves near the ground.

Host penetration of the pathogen takes place through the stomata. The mycelium invades the intercellular spaces, ramifies in the host tissue deriving its nutrients in a large measure through haustoria which penetrate the cell lumen. In almost all cases, infection of the leaves takes place through the lower surface.

The optimum temperature for the development of the disease is 18°C to 24°C. Prolonged warm wet weather is conducive to an epidemic of the disease. Dry weather, however, checks the growth of the fungus. After the mycelium has established itself in the host tissue, sporangiophores are

sent to the host surface through the stomata (Fig. 361B). Sporangia produced on the sporangiophores serve for rapid dissemination of the disease. They also serve as the source of secondary inocula of the disease. Through the secondary inocula, secondary infections are induced resulting in secondary cycles .The sporangia are transported from host to host and to different parts of the host by both wind and rain. Disease cycle of Downy mildew of grape is very similar to that of Downy mil-dew of pea, except that the sporangia germinate by secondary zoospores.



Disease Cycle of Downy Mildew of Grape

13.9.5 Control of Downey Mildew of Grapes

The chief methods of control of the disease are as follows:

(i) Sanitation:

Destruction of old leaves is recommended to eradicate the source of inoculum since the causal organism of the disease usually hibernates as oospores in the fallen leaves.

(ii) Spraying of Bordeaux mixture:

Bordeaux mixture, 5-5-50 strength is usually recommended as a protective spray to the susceptible parts; but even up to 8-8-50 is often recommended for epiphytotic conditions. Sporangia or zoospores absorb copper until a toxic limit is reached, the more epiphytotic the disease, the stronger must be the spray to be effective. But emphasis is laid on the value of timely and early spraying, and of the thinning of the foliage during the growing season in order to gain better access for sprays, and the removal of the shoot tips in order to increase resistance in the remaining parts.

The first application of spray should be made soon after the buds open, a second before the flowers open, a third after the fall of the petals, and a final application should be given 14 days later. The time of application, however, varies with prevailing environment and the relative suscep-tibility of the host concerned.

(iii) Spraying of Other Fungicides:

The spread of the disease can be effec-tively controlled by spraying the vines with 0.3 per cent. Blitox-50, Dithane Z-78, Ferbam or Captan

first when the shoots are 6 to 8 inches high, again when they are about to flower, and finally when the fruits are just about to change their colour. Depending on the local weather conditions and the nature of virulence of the disease number of spray applications may be increased to 6 to 8 times.

(iv) Improved Cultivation Practices:

Certain improved methods of culti-vation are helpful to control the disease incidence. Vineyards should be sufficiently open to allow the foliage to dry quickly after rains. This method not only ensures free access of air but facilitates spraying as well. The risk of infection from resting oospores is reduced. All green shoots or suckers that develop at the base of the stocks should be removed, as they are very susceptible to primary infections.

(v) Use of Resistant Varieties:

The use of resistant varieties is of course a very useful practice. But even with resistant vines, spraying with Bordeaux mixture is advisable.

Check your Progress- 7

Note: a. Write your answer in the space given below

b. Compare your answer with those given at the end of the unit.

12. Describe the Life cycle of Downy Mildew of Grape

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13.10 ANSWER TO CHECK YOUR PROGRESS QUESTIONS

1. Tobacco mosaic virus is a single-stranded RNA virus that commonly infects Solanaceous plants, which is a plant family that includes many species such as tomatoes and tobacco.
2. Symptoms vary with the species of plant infected and environment conditions. Symptoms associated with TMV infections: stunting, mosaic pattern of light and dark green (or yellow and green) on the leaves, malformation of leaves, yellow streaking of leaves, yellow spotting on leaves and distinct yellowing only of vein.
3. Control of banana bunchy top is achieved by killing the banana aphids then destroying all infected materials. First, the aphids should be killed on the infected banana materials, and then all the plant material should be destroyed to prevent the spread of the virus.
4. It is one of the most feared diseases of rice, and southeastern Australia is the only rice-growing area in the world free of this disease. It can devastate rice crop yield, cause loss of markets and would require fungicide sprays to control.
5. Rice blast (Magnaporthe grisea) is a fungal disease of rice that causes lesions on the following parts of the rice plant: leaf, leaf collar, culm, culm nodes, panicle neck node and panicle. The fungus can infect rice plants at any growth stage.
6. Rice blast spreads by windborne spores, by water and on infected plant material.

7. Pathogen of the blast disease of rice is *Pyricularia grisea* (Cooke) Sacc., named earlier as *Trichothecium griseum, Pyricularia oryzae* Cav. or Dactylaria oryzae. The perfect stage of the pathogen is *Magnaporthe grisea* (Herbert) Yaegashi and Uddagawa, an ascomycetous fungus. Sheath blight is a fungal disease caused by *Rhizoctonia solani*.
8. The pathogen can survive as sclerotia or mycelium in dry soil for about 20 months but for 5-8 months in moist soil. Sclerotia spread through irrigation water.
9. Citrus canker is a serious bacterial disease of commercial varieties of citrus, and relatives, caused by the bacterium *Xanthomonas citri subsp.citri.* the disease affects the leaves, twigs and fruits causing the leaves to drop and fruit to fall to the ground before it ripen.
10. Eradication: The disease is controlled by the eradication of diseased trees. This is accomplished by removing the trees with advanced infection and burning them.
    Pruning: The infected trees may be cured by removing the diseased foliage and branches with pruning scissors and then spraying the trees with one percent Bordeaux mixture at regular intervals.
11. The causal agent of the red rot disease of sugarcane is the form species *Colletotrichum falcatum* Went, of form class fungi Imperfecti.
12. Spore are released in spring and spread to the leaves and berries by slashing rain and wind. The fungus has two types of spores, both germinating into swimming spores. These spores swim to the stomata's of the plants and initiate infection. Water is essential for the spores to swim and infect, so outbreaks of the disease coincide with period of wet weather.

13.11 SUMMARY

Plant disease, an impairment of the normal state of a plant that interrupts or modifies its vital functions. Infectious plant disease are caused by bacteria, fungi or viruses and can range in severity from mild leaf or fruit damage to death. In this unit, author has discussed about various crop disease such as tobacco mosaic virus, bunchy top of banana, blast and sheath blight of paddy, citrus canker, red rot of sugarcane and downey mildew of grapes. Unit deals with study about the disease, symptoms, causal organism, disease cycle and control or management crop disease.

Tobacco mosaic virus (TMV) is a single-stranded RNA virus that commonly infects Solanaceous plants, which is a plant family that includes many species such as tomatoes and tobacco. Symptoms associated with TMV infections: stunting, mosaic pattern of light and dark green (or yellow and green) on the leaves, malformation of leaves, yellow streaking of leaves, yellow spotting on leaves and distinct yellowing only of vein. The virus is disseminated from plant to plant by mechanical transmission, by handling tobacco plants during transplanting; through other field operations; and contact by man and cultivation implements.

Red rot disease of sugarcane is the form species *Colletotrichum falcatum* Went, of form class fungi Imperfecti. Infected leaves displayed yellowing and drying. If the diseased stalk split open, reddened internal tissues with intermingled white spots may been seen. The pathogen may

gain entrance through the nodes at the leaf scars, through any kind of wound, through root primordia and seed-cuttings. The diseased canes are frequently found to be injured by insects, especially borers, and no doubt these wounds facilitate the entrance of the fungus, which in turn does much more damage than the insects.

Citrus canker is a serious bacterial disease of commercial varieties of citrus, and relatives, caused by the bacterium *Xanthomonas citri subsp.citri*. the disease affects the leaves, twigs and fruits causing the leaves to drop and fruit to fall to the ground before it ripen. Infection takes place through the stomata and wounds. The disease is not soil borne. The pathogen perennates in the old lesions on the twigs still attached to the host plant. From there it is carried by driving rains and by insects to new localities. Man functions as the chief agent of dissemination by planting infected nursery stock in new localities. The disease is controlled by the eradication of diseased trees. This is accomplished by removing the trees with advanced infection and burning them.

Downy mildew is an extremely serious fungal disease of grapes that can result in severe crop loss. It is caused by the fungus *Plasmopara viticola*. The pathogen attacks all green parts of the vine, especially the leaves. Lesions on leaves are angular, yellowish, sometimes oily, and located between the veins. As the disease progress, a white cottony growth can be observed on the lower leaf surface. Spore are released in spring and spread to the leaves and berries by slashing rain and wind. The fungus has two types of spores, both germinating into swimming spores. These spores swim to the stomata's of the plants and initiate infection. Water is essential for the spores to swim and infect, so outbreaks of the disease coincide with period of wet weather. There are various control measures to control downy mildew disease such as maintain plant vigor, sanitation, pruning, and applying fungicides etc.

Banana bunchy top disease is caused by Single strand DNA Virus (BBTV) and one of the most serious diseases of banana. It stunts the plants and reduces yield severely. The disease is caused by banana bunchy top virus, which is transmitted by banana aphids *Pentalonia nigronervosa* Coquerel. Aphid acquires the virus after at least four (but usually about 18) hours of feeding on an infected plant. The aphid can retain the virus through its adult life, for a period of 15–20 days. During this time, the aphid can transmit the virus to a healthy banana plant by feeding on it, possibly for as little as 15 minutes but more typically for about two hours. The most important methods to control BBTV involve, killing the aphid vector and roguing (removing and destroying) infected banana plants. By killing the aphids on the banana plant, dispersal of virus-carrying aphids to nearby healthy banana plants is avoided. Since the only host of BBTV is banana, roguing infected trees reduce spread of virus.

Blast disease or rotten neck is one of the most serve disease affecting paddy. The causal organism i.e., pathogen of the blast disease of rice is *Pyricularia grisea* (Cooke) Sacc., named earlier as *Trichothecium griseum*, *Pyricularia oryzae* Cav. or Dactylaria oryzae. The perfect stage of the patho-gen is *Magnaporthe grisea* (Herbert) Yaegashi and Uddagawa, an ascomycetous fungus. The blast is a foliage disease. The symptoms also occur in other plant parts. The spots are spindle-shaped

with grey or white central part and brownish or reddish borers. The pathogen can perpetuate in grain both externally and internally, on straw piles and also on host other than rice. The inocula, i.e., the conidia, developed from all or any of the above sources can infect the leaf of the rice plant. The infected regions develop crops of conidia; those are disseminated and infect the different regions of the same plant and /or the other rice plants. Towards the end of the season, the pathogen may perennate as conidia on seed and also on straw piles. Next season, perennating conidia may serve as a source of primary inoculum. Likewise, in some areas they develop perithecia containing asci and ascospores. The perithecia remain with the plant debris or straw piles. Next season, the ascospores may cause infection.

Sheath blight is a fungal disease caused by *Rhizoctonia solani*. Symptoms are usually observed like oval or ellipsoidal greenish gray lesions, usually 1-3 cm long, on the leaf sheath, initially just above the soil or water level in the case of conventionally flooded rice. Lesions on the leaves usually have irregular lesions, often with gray-white centers and brown margins as they grow older. Initial infections start with a sclerotium or a piece of infected debris floating on the water surface and coming in contact with the sheath. The fungus gets attracted to the chemical stimuli released by the rice host. Germinating sclerotia or mycelia in debris penetrate the plant tissue either by means of natural openings or by specialized infection structures called appressoria or infection cushions. The fungus also produces extracellular enzymes that degrade plant cell walls to facilitate colonization. Once the fungus penetrates and colonizes the plant tissue, symptoms are initiated.

13.12 KEY WORDS

Downy mildew: A plant disease in which the sporangiophores and spores of a fungus appear as a downy growth on the lower surface of levaes and stem, fruit, etc., caused by fungi in the family Peronosporaceae.

Symptom: The extrenal and internal reactions or alternations of plant as a result of a disease.

Tobacco mosaic virus: a single-standeded RNA virus (species Tobacco mosaic virus of the genus Tobamovirus) that causes mosaic disease in plants such as tobacco and tomato.

Bunchy Top: is a viral disease caused by the Banana bunchy top virus (BBTV). The disease, often called BBTD for banana bunchy top disease, gets its name from the bunchy appearance of infected plants.

Rot: the softening, discoloration, and often disintegration of succulent plant tissue as a result of fungal or bacterial infection.

Blast and sheath blight: Rice blast disease caused by the fungus *Magnaporthe oryzae* and rice sheath blight disease caused by the fungus *Rhizoctonia solani* are two major hurdles for stable rice production worldwide.

13.13 SELF ASSESSMENT QUESTION AND EXERCISES

1. Describe Tobacco Mosaic Disease
2. Define bunchy top of Banana
3. Give the name of causal organism of Citrus Canker Disease.

4. Give any two symptom of Red Rot of Sugarcane.
5. Given an account of downy mildew of grapes.
6. Wite essay on blast and sheath blight of paddy with reference to causal organism, symptoms, and disease cycle and control measures.

13.14 FURTHER READING

1. Banana Bunchy Top Virus by Cooperative Extension Service, Plant Disease Dec. 1997, PD-12. (https://www2.ctahr.hawaii.edu/oc/freepubs/pdf/PD-12.pdf).
2. http://www.promusa.org/Bunchy+top accessed on 11 September 2019.
3. Plant Pathology, 5th Edition, George N. Agrios, Elsevier Academic Press.

UNIT – 14 PLANT DISEASES

Structure

14.1 INTRODUCTION

Plant disease, an impairment of the normal state of a plant that interrupts or modifies its vital functions. All species of plants, wild and cultivated alike, are subject to disease. Although each species is susceptible to characteristic diseases, these are, in each case, relative few in number. The occurrence and prevalence of plant diseases vary season to season, depending on the presence of the pathogen, environment conditions, and the crops and varieties grown. Some plant varieties are particularly subject

to outbreaks of disease while others are more resistant to them. Infectious plant disease are caused by bacteria, fungi or viruses and can range in severity from mild leaf or fruit damage to death. In this unit, author has discussed about various crop disease such as late blight of potato, leaf spot disease of groundnut, anthracnose of mango, wilt of cotton, and rust of wheat. Unit deals with study about the disease, symptoms, causal organism, disease cycle and control or management crop disease.

14.2 OBJECTIVITIES

After going through the unit you will be able to:

1. Identify the name of the disease in various crop plants.
2. Understand the various symptoms of crop diseases.
3. Identify the causal organism of diseases in crop plants
4. Understand the disease cycle in infected crop plants.
5. Understand the suitable control measures or management of various crop diseases.

14.3 LATE BLIGHT OF POTATO

14.3.1 Introduction to Late Blight of Potato

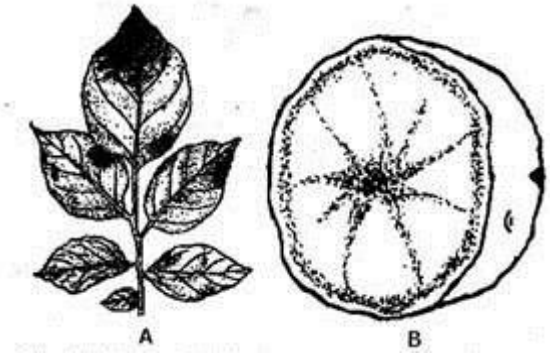
The disease causes blight symptom which appears towards middle to late in the season, that's why the name is given. Potato is a native of the North Andes in South America. Initially, late blight of potato was available in that area as an endemic disease. Gradually, by 1840-1847, it spread throughout USA and Europe. The disease became well established in Ireland, Europe and England by 1842. Later, in 1845, it spread as epiphytic disease throughout the Europe and brought famine in Ireland, causing the death of one million (1,000,000) people and more than one million migrated to other countries. In India, the Late blight was introduced in the Nilgiri hilts between 1870-1880 from England. Now-a-days, the disease is frequently available in the potato growing areas of India.

The loss from this disease is less in West Bengal and Bihar (5-10%), which gradually increases in Punjab (20-25%), U.P. (15-50%) and Haryana (40-50%) and causes a monetary loss of Rs. 4 crores per year. The loss is gradually reduced by the introduction and popularisation in culti-vation of Kufri Chandramukhi, and some other varieties.

14.3.2 Symptoms of Late Blight of Potato

Symptoms of Late Blight of Potato are available on foliage and inside the Tuber: 1. On Foliage: Symptoms appear as hydrotic areas with indefinite margins at the apex and edges of the leaflets (Fig). The infected regions gradually turn brown to black due to necrosis. In early stage of disease development, chlorotic border develops around the necrotic regions. During moist weather, the disease progresses very rapidly, causes much more decay of the infected regions and produces a characteristic bad odour. The sporangiophore and sporangia are visible as matty growth on the abaxial surface of the leaflet. The entire plant may be damaged completely within a few days. During dry weather, the disease progresses very slowly and the affected regions curl and shrivel. The diseased areas become hard and easily break with small disturbance.

2. Inside the Tuber: The pathogen does not go down through the stem to the tuber. The Tuber Infection takes place in different ways: (a) During Growing Season: Hydrotic areas develop on the tuber surface and the regions become necrotic. If condition favours, the entire potato turns brown and becomes damaged completely before harvest. (b) During Harvest: During harvesting, the tuber with delicate skin may get rup-tured and the tuber may find contact with the infected leaf and cause infec-tion. Later, the symptom is visible after cutting the tuber as a wheel marked by small brown dots.

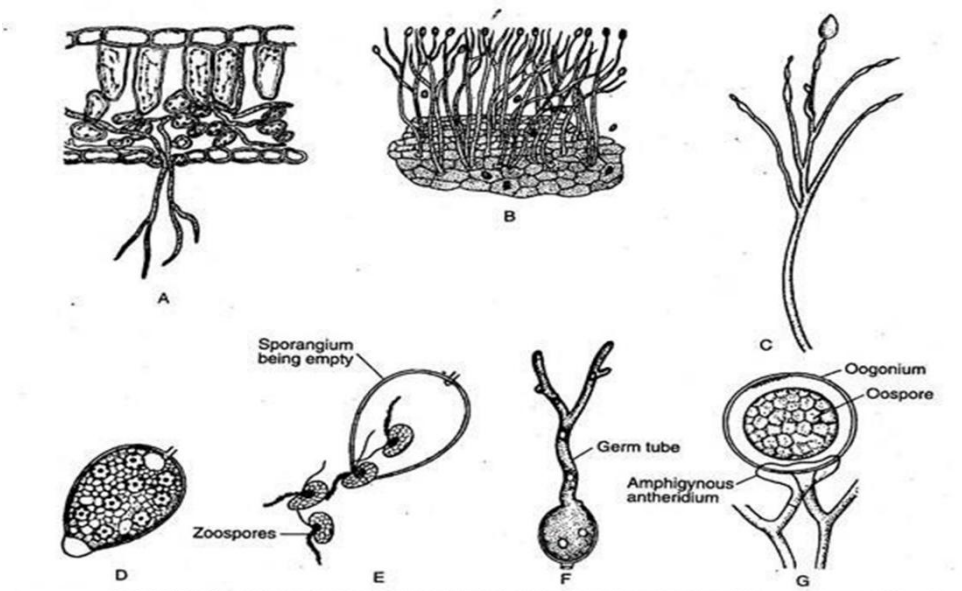


14.3.3 Causal Organism of Late Blight of Potato

The causal organism, *Phytophthora infestans*, belongs to the subdivision Mastigomycotina under the Division Eumycota. The plant body is a coenocytic mycelium which grows both intra- and intercellularly inside the host tissue. The sporangiophore develops either singly or in groups on the young seedling and also from the lower side of the leaf of adult plant.

It is branched and develop spo-rangia at the apices. Initially, one sporangium develops at the apex and, at maturity, the sporangiophore elongates from one side and shifts the sporangium towards the opposite side — another sporangium develops at the apex.

The sporangia are lemon-shaped and colourless with a small stalk below and thin papilla above (Fig. 5.19D). During favourable condition sporangium produces a number of biflagellate secondary zoospores (Fig. 5.19E). After swimming for some time in dewdrop or thin film of water, the zoospores encyst and then ger-minate by producing germ tube.



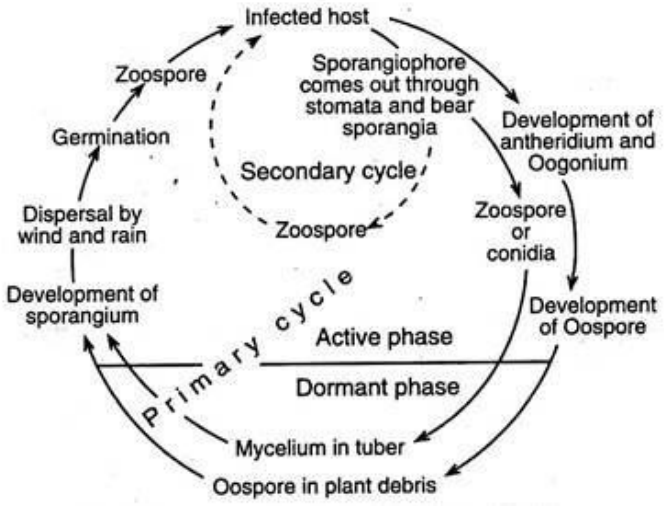
Late blight of potato : A. Section of diseased leaf showing developing sporangiophore coming out through stomata. B. Huge number of sporangiophores coming out through stomata from a diseased tissue. C. Sporangium on branched sporangiophore. D. Single sporangium. E. Germination of sporangium by producing zoospores. F. Direct germination of sporangium. G. Single Oospore

The germ tube penetrates directly through stomata or develop appresorium at its tip (on contact with cell wall), which then penetrates the host wall both by mechanical pressure as well as by enzy-matic action. During unfavourable condition the sporangium behaves as conidium, which germi-nates directly by producing germ tube. Optimum temperature for the growth of mycelium is 16-18°C, for sporulation is 21 °C, for germination of sporangia by zoospore is 12°C, while at 21 °C it germinates directly by germ tube. Relative humidity of 100% favours the abundant production of sporangium, while below 90% RH inhibits its production. The fungus is heterothallic. During sexual reproduction oospores are produced inside the aerial parts by the union between antheridium and Oogonium.

14.3.4 Disease Cycle of Late Blight of Potato

In the dormant phase, the fungus could perennate in the tuber as mycelium and in plant debris as oospore. During favourable condition, the perennating mycelium becomes active and develops active mycelium. The active mycelium develops branched sporangiophore bearing sporangia on it. On the other hand, the oospore germinates by producing germ tube bearing sporangia at their tip. In both the cases, the developed sporangia disperse by wind or rain, germinate on host surface by producing zoospores and cause infection to the host tissue. The infected host again develops sporangia on sporangiophore. The sporangia develop zoospore which cause further infection. This process repeats several times in the growing season - this is the secondary cycle. During favourable condition, the zoospores may come down into the soil and may cause infection to the tubers. The tuber infection may also take place on contact with infected foliage during harvest. The perennating mycelium inside the tuber remains active, if the infected tubers are kept in the storage for seed potato. In the next season, after sowing the seed, the perennating mycelium becomes active and causes further infection. Towards the end of the season, inside the aerial parts, both the sex organs — antheridium (male) and oogonium (female) — develop and undergo sexual reproduction of amphigynous

type. The product of sexual reproduction is oospore, which has hard protective covering. The oospore remains dormant inside the host tissue during unfavourable season. During favourable condition in the next season they germinate and produce next crop of zoospores.



Disease Cycle of Late Blight of Potato

14.3.5 Control of Late Blight of Potato

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

A. Cultural Methods: The methods useful in controlling the late blight of potato are: 1. Seed tubers should not be collected from the disease field. 2. Tubers should be harvested after the maturity of the tubers (when skin of the tubers comes tough). 3. In infected field, the aerial part of the plants should be dried completely before harvest of tubers. 4. Infected plant should be cut out and collectively buried deep in the soil. 5. The previous year's plant debris should be cleaned from the field and it must be burnt outside the field. 6. The volunteer potato plants that may harbour the infection mainly in hills should be eradicated during appearance. 7. Planting of alternate row of susceptible and resistant variety will reduce the intensity of the disease. 8. Cultivation of tuber on high ridges and covering the tuber with soil will prevent the fungus to grow out from the infected mother tuber, thus reduce the spread of the disease. 9. The nitrogen fertiliser should be applied at a reduced rate. 10. During harvesting, the potato plants should be placed on one side and tubers on other side, thereby contact between tuber and infected plant can be avoided.

B. Physical Methods: Following methods are used to control the disease:

1. The perenating mycelium in the tuber can be killed by exposure to 30°C for 65 hours or to 40°C for 4 hours. 2. The perenating mycelium can be killed by dipping the tuber in water at 45°C for 4-5 hours or at 40°C for 24 hours.

C. Chemical Methods: 1. Dutt (1962), while working with 16 different fungicides in hilly region, found that Bordeaux mixture (5:5: 50) is the most effective giving better result than. Burgundy mixture. 2. Fungicides like Brestan (1 kg/ha), Dithane M-45 (2 kg/ha) and Difolitan 80WP (2.5 kg/ha) are used as foliar spray and found highly effective in con-trolling

late blight disease of potato in plains of India. 3. According to Khanna and Sharma (1981), Metalaxyl (1 kg/ha), a systematic fungi-cide, is found highly effective in con-trolling the disease, thus increasing the yield about 3 times than the control.

D. Biological Methods (Biocontrol): 1. Jindal et al., (1988) reported the role of *Epicoccum purpurascens*, *Trichoderma koningii* and Stachybotrys atra in controlling the disease. 2. Roy et al. (1991) reported the role of *Penicillium aurantiogriseum* and *Myrothecium verrucaria* in late blight disease management. 3. Arora (1999) reported the role of *Penicillium viridecatum*, *Chaetium brasilense* and *Trichoderma viride* in controlling the late blight disease. E. Disease tolerant varieties. It is better to use tolerant varieties. In India, Central Potato Breeding Station (Simla) has developed some tolerant varieties like Kufri Jyoti, Kufri Naveen, Kufri Badshah, Kufri Jeevan, Kufri Neela etc. for commercial cultivation. Cardinal and Diament (Dutch cultivrs) and Kufri Chandramukhi and Kufri Chipsona-1 (Indian cultivars) are the varieties showing good performance in alluvial zone of West Bengal.

Check your Progress- 1

Note: a. Write your answer in the space given below

b. Compare your answer with those given at the end of the unit.

1. What is Late Blight of Potato?

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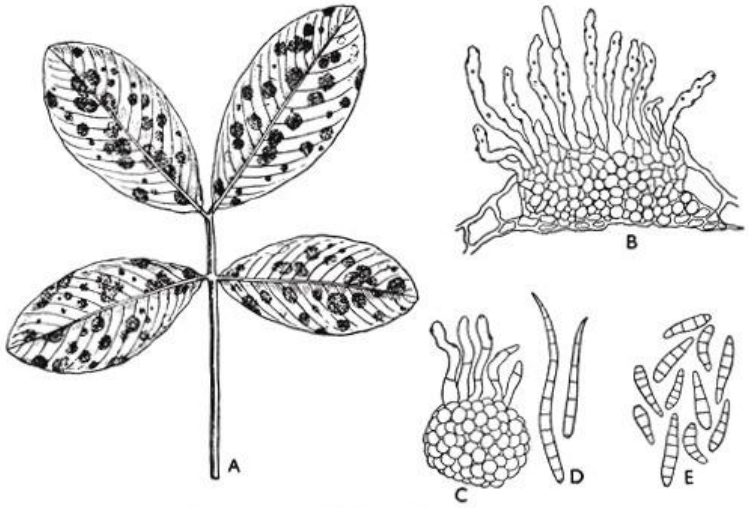
14.4 LEAF SPOT DISEASE OF GROUNDNUT

14.4.1 Introduction to Leaf spot disease of Groundnut

The disease, caused by Cercospora, is also known as Cercospora leaf spot. It is available in all the groundnut growing areas of India and caused about 20-50% reduction in yield. The size as well as the quality of the nuts are generally affected.

14.4.2 Symptoms of Leaf spot disease of Groundnut

All the aerial parts of the plants attacked by the pathogen shows characteristic symptoms. Initially, the lower leaflets get infected showing dark spots and later on, each spot becomes surrounded by yellowish halo. Large number of spots, almost circular in outline, develops on the leaf. With severity and maturation, the spots become dark brown to almost black, parti-cularly on the upper surface of the leaflets.



Stems and petioles are also with spots but less in number. With severity of disease, the spots become coalesced and defoliation of the leaf takes place. Due to defoliation, the size and quality of the nuts become greatly reduced. Infection at younger stage causes defoliation and nuts fail to develop in them. Due to this disease, the ulti-mate effect is the greater loss in yield.

14.4.3 Causal Organism of Leaf spot disease of Groundnut

Cercospora arachidicola Hori (perfect stage *Mycosphaerella arachidicola* W. A. Jenkins) and *Cercosporidium personatum* (Berk and Curt) Deighton, earlier known as Cerco-spora personata (Berk and Curt) Ell and Eve (perfect stage Mycosphaerella berkeleyii W. A. Jenkins.).

1. *Cercospora arachidicola* (Early Leaf Spot): The mycelial plant body grows both exter-nally and internally, without any haustorium. The mycelia by aggregation form stroma. The conidiophores are normally amphigenous, present only on the upper surface in younger spots. The conidiophores are geniculate, septate or aseptate, those develop hyaline or slightly olivaceous coloured, 4-13 septate, obclavate and often curved conidia. Conidia mea-sure 38-108 µm x 2-5 µm.

The perfect stage of the pathogen is Mycosphaerella arachidicola. The ascostroma is perithecial in nature, grows scattered, common-ly along the lesion margin, partly embedded in the host tissue, ovate to nearly globose in shape, black, ostiolate and measure 47-84 µm x 44 -74 µm. Perithecia contain asci without paraphyses.

Asci are cylindrical club-shaped with short stipes, bitunicate, 8-spored and measure 27-37.8 µm x 7-8.4 µm. Ascospores are 2-celled, slightly curved, upper cell somewhat larger, hyaline and measure 7-15 µm x 3-4 µm.

2. *Cercosporidium Personatum* (Late Leaf Spot):

The mycelial plant body of the pathogen grows intercellularly and draws nutrition from the neighbouring cells by haustoria. With aggregation, the mycelium forms dense stroma from where long septate to non- septate, geniculate, hypophyllous conidio-phores develop.

Conidiophores emerge in tufts by rupturing host epidermis. Conidia develop on conidiophores. Conidia are obclavate or cylindrical, septate, pale- brown and measure 30-50 µ x 5-6 µ.

The perfect stage of the pathogen is Mycosphaerella berkeleyii. The ascostroma is perithecial in nature, grows scattered, common-ly along the lesion margin, partly embedded in the host tissue, broadly ovate to globose in shape, black, ostiolate and measured 84-140 µm x 70-112 µm. Perithecia contain asci without paraphyses. Asci are cylindrical club-shaped with short stipes, bitunicate, 8-spored and measured 30-40 µm x 4-6 µm. Ascospores are 2-celled, slightly curved, upper cell somewhat larger, constricted at septum, hyaline and mea-sure 11-19.5 µm x 3-3.8 µm.

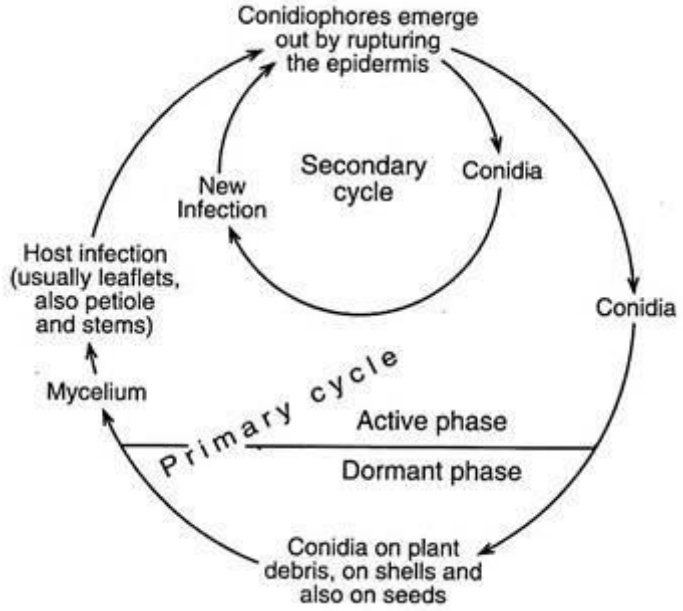
14.4.4 Disease Cycle of Leaf spot disease of Groundnut

The pathogen can perennate through conidia on diseased plant debris that remain in the soil, on shell of fruits and also on seeds. Perennating pathogen in all the above three places acts as a source of primary inoculum.

It favours high temperature ranges between 26°C to 31°C and high humi-dity. Continuous low temperature along with dew also favours infection. Infection takes place through stomata or by piercing the host epider-mal cell.

Infection commonly takes place through upper epidermis and after entering the host tissue, the pathogen ramifies and by aggregation of mycelium, stroma develops.

With further development, the stroma creates a pressure and the conidiophores come out through ruptured epidermis. Conidia are developed on conidiophores. The conidia after detachment, disseminate through wind to the different regions of the same plant or to the different plants and cause further infection and thus help to spread the disease. This cycle repeats several times in the growing season.



Disease Cycle of Leaf spot disease of Groundnut

14.4.5 Control of Leaf spot disease of Groundnut

Following procedures are adapted to control or reduce the disease incidence:

A. Cultural Methods: 1. Sanitation: Cleaning and destruction by burning the previous year's plant debris, reduce the source of primary inoculum, thereby the disease incidence becomes reduced. 2. Crop Rotation: Crop

rotation for 2-4 years is a very good procedure to reduce the rate of infection. 3. Early Sowing: It is a very useful proce-dure, where the plants mature early, thus the damage through disease becomes very less.

B. Chemical Methods: 1. Seed Treatment: As the disease is seed- borne, seed treatment gives good result to control the disease. In this procedure, the seeds are properly separated from the shell and treated with 0.5% CuSO4 solution for 30 minutes before sowing. Dry dressing with Agrosan GN is also very much effective to reduce the source of primary inoculum.

2. Foliar Spray: To reduce the source of secondary inoculum, foliar spray of chemicals is commonly used. Different Chemicals used as Foliar Spray are: a. Bordeaux mixture (4: 4: 50 or 5: 5: 50). It is used along with linseed oil (as sticker) at an interval of 15 days, which is much effective to reduce the disease to a significant level. b. Other chemicals. Chemicals like Dithane Z-78 (0.2%), Dithane M- 45, Cosan and CuSO4 (15 and 25 lb/ acre), Fycol 8E etc., are very much effective but requires 5-6 spray. c. Systemic fungicides. Bavistin, Benlate, Cercopin etc. are the sys-temic fungicides found to be very effective in controlling the disease. But, the chemicals are costly and it is advised to calculate the cost benefit ratio before application.

Check your Progress- 2

Note: a. Write your answer in the space given below

b. Compare your answer with those given at the end of the unit.

2. Which fungus is responsible for the Leaf spot disease of Groundnut?

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14.5 ANTHRACNOSE OF MANGO

14.5.1 Introduction to Anthracnose of Mango

Worldwide, mango anthracnose is the most important and destructive disease of mango. Mango is in the plant family Anacardiaceae. Mango grows throughout the tropics and subtropics and is regarded as one of the world's most important fruit crops. Mango is a perennial, branching, evergreen tree approximately 30–40 feet tall. Its fruit is a large, fleshy drupe containing a laterally compressed stone housing the seed. Mango cultivars vary considerably in fruit size, color, shape, flavor, texture, and taste.

14.5.2 Symptoms of Anthracnose of Mango

On mango, anthracnose symptoms occur on leaves, twigs, petioles, flower clusters (panicles), and fruits. On leaves, lesions start as small, angular, brown to black spots that can enlarge to form extensive dead areas. The lesions may drop out of leaves during dry weather. The first symptoms on panicles are small black or dark-brown spots, which can enlarge, coalesce, and kill the flowers before fruits are produced, greatly reducing yield. Petioles, twigs, and stems are also susceptible and develop the typical black, expanding lesions found on fruits, leaves and flowers. Ripe fruits affected by anthracnose develop sunken, prominent, dark brown to black decay spots before or after picking. Fruits may drop from trees prematurely. The fruit spots can and usually do coalesce and can eventually penetrate deep into the fruit, resulting in extensive fruit rotting.

Most green fruit infections remain latent and largely invisible until ripening. Thus fruits that appear healthy at harvest can develop significant anthracnose symptoms rapidly upon ripening. A second symptom type on fruits consists of a "tear stain" symptom, in which are linear necrotic regions on the fruit that may or may not be associated with superficial cracking of the epidermis, lending an "alligator skin" effect and even causing fruits to develop wide, deep cracks in the epidermis that extend into the pulp. Lesions on stems and fruits may produce conspicuous, pinkish-orange spore masses under wet conditions. Wet, humid, warm weather conditions favor anthracnose infections in the field. Warm, humid temperatures favor postharvest anthracnose development.

14.5.3 Causal Organism of Anthracnose of Mango

The ubiquitous fungus *Colletotrichum gloeosporioides* Penz and Sacc. is the anamorph stage (asexual stage of the pathogenic fungus). *C. gloeosporioides* is responsible for many diseases, also referred to as "anthracnose," on many tropical fruits including banana, avocado, papaya, coffee, passion fruit, and others.

14.5.4 Disease Cycle of Anthracnose of Mango

Dissemination: spores (conidia) of the pathogen are dispersed passively by splashing rain or irrigation water.Inoculation: spores land on infection sites (panicles, leaves, branch terminals). Infection and pathogen development: on immature fruits and young tissues, spores germinate and penetrate through the cuticle and epidermis to ramify through the tissues. On mature fruits, infections penetrate the cuticle, but remain quiescent until ripening of the climactic fruits begins. Symptom and disease development: black, sunken, rapidly expanding lesions develop on affected organs. Pathogen reproduction: sticky masses of conidia are produced in fruiting bodies (acervuli) on symptomatic tissue, especially during moist (rainy, humid) conditions. Many cycles of disease can occur as the fungus continues to multiply during the season. Pathogen survival: the pathogen survives between seasons on infected and defoliated branch terminals and mature leaves.

14.5.5 Control of Anthracnose of Mango

Integrated disease management practices

Management of mango anthracnose consists of five approaches:

• Site selection • cultivar selection • cultural practices in the field (sanitation, plant spacing, intercropping, etc) • fungicide sprays in the field • postharvest treatments (physical, chemical).

A range of foliar fungicides are registered for control of mango anthracnose, including products containing clarified neem oil, mono- and di-potassium salts of phosphorous acid, chlorothalonil, basic cupric sulfate, copper hydroxide, wet table sulfur, harpin protein, and copper salts of fatty and rosin acids. These products vary in their mode of action and efficacy. Consult the CTAHR Cooperative Extension Service for current product names and specific recommendations.

Check your Progress- 3

Note: a. Write your answer in the space given below

b. Compare your answer with those given at the end of the unit.

3. What is anthracnose of mango?

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14.6 WILT OF COTTON

14.6.1 Introduction to Wilt of Cotton

Wilt is one of major disease of cotton, found wherever this crop is grown. It is believed to have originated in Mexico or Central America. In India the disease was reported from Maharashtra, Tamil Nadu, Karnataka and other states. The fungus also infects the other hosts like brinjal, chilli, tobacco and bhendi. Fusarium wilt of cotton, caused by the fungus, *Fusarium oxysporum f. sp. vasinfectum* Race 4 (FOV4), was confirmed in numerous fields in El Paso and Hudspeth counties in Texas in 2017. Undoubtedly, it was present in those fields for several years, but how the disease entered the area remains unknown. FOV4 was first identified in the United States in a single county of the San Joaquin Valley (SJV) of California in 2003 and has since become widespread across the SJV. *Fusarium* causes vascular wilts of vegetables and flowers, herbaceous perennial ornamentals, plantation crops, and the mimosa tree (silk tree). Most of the wilt causing *Fusarium* fungi belongs to the species *Fusarium oxysporum*. Different host plants are attacked by special forms or races of the fungus. The fungus that attacks tomato is designated *F. oxysporum* f. sp. *lycopersici*; cucurbits, *F. oxysporum* f. sp. *conglutinans*; banana, *F. oxysporum* f.sp. *cubense*; cotton, *F. oxysporum* f. sp. *vasinfectum*; carnation, *F. oxysporum* f. sp. *dianthii*; and so on. The disease was more severe forms in heavy soil with soil temperature 20-30 ^{0}C during the crop season.

14.6.2 Symptoms of Wilt of Cotton

- Earliest -seedling is the yellowing and browning of the cotyledons.
- Leaves lose their turgidity first turn yellow and then brown and finally drop off.
- The tap root stunted and laterals are less abundant.
- Browning and blackening of vascular tissues.
- Discolorations of leaves starts from the margins and spread towards midribs.
- Wilting may be complete or partial.

14.6.3 Causal Organism of Wilt of Cotton

Fusarium oxysporum f.sp. *Vasinfevtum*

The fungus is present both inter and intra cellularly in the host tissue. The mycelium plugs the xylem vessels partially or completely. The macro conidia are 1-5 septate, hyaline, thin walled, linear to falcate, the tapering the micro conidia are hyaline, thin elliptical to spherical, single or two celled. Survival of the fungus in soils not planted to cotton for over 10 years.

14.6.4 Disease Cycle of Wilt of Cotton

Fusarium oxysporum f.sp. *Vasinfevtum* can survive in soil, in plant debris, and in infected seed beneath the seed coat, even after acid delinted and treated with fungicide seed treatments. The fungus produces multiple types of spores and some types can survive in soil for many years. Spread within a field occurs when infested soil is moved by implements, vehicles or personnel, or when water carries infested soil or plant debris in irrigation or storm water to other fields. Local spread by soil movement can be reduced with practices of segregating or thoroughly cleaning equipment by power washing with detergents.

14.6.5 Control of Wilt of Cotton

Management of Fusarium wilt in cotton is difficult. Chemical management is limited and expensive, and crop rotation is not effective because of the ability of *F. oxysporum f. sp. vasinfectum* to survive in soil for long periods. Since wilt is often associated with the root-knot nematode, resistance, chemical, and rotation practices directed at management of the nematode have sometimes proven effective in reducing the incidence and severity of Fusarium wilt. Seed treatments that eliminate *F. oxysporum f. sp*. vasinfectum from seed have been reported from China, India, and Australia. These treatments will likely be ineffective in fields already infested with the pathogen, but may be important in limiting the spread of the pathogen on seed to new areas. Crop rotation is often recommended to reduce the incidence of Fusarium wilt, but the ability of the fungus to survive in the soil for long periods in the absence of cotton limits the effectiveness of rotations.

Check your Progress- 4

Note: a. Write your answer in the space given below

b. Compare your answer with those given at the end of the unit.

4. What is the causal organism of cotton wilt?

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14.7 RUST OF WHEAT

14.7.1 Introduction to Rust of Wheat

Stem rust of wheat occurs worldwide and affects wheat wherever it is grown. Similar rusts affect other cultivated cereals and probably most wild grass genera and species. The stem rust fungus attacks all the aboveground parts of the wheat plant.

14.7.2 Symptoms of Rust of Wheat

Plants do not usually show obvious disease symptoms until 7 to 15 days after infection when the oval pustules (uredinia) of powdery, brick-red uredinio spores break through the epidermis. Microscopically, these red spores are covered with fine spines. The pustules may be abundant and produced on both leaf surfaces and stems of grass hosts. Later in the season, pustules (telia) of black teliospores begin to appear in infected grass species. Microscopically, teliospores are two celled and thick walled.

The symptoms on wheat appear as elliptical blisters or pustules, known as uredia that develop parallel with the long axis of the stem, leaf, or leaf sheath. Blisters may also appear on the neck and glumes of the wheat spike. The epidermis covering the pustules is later ruptured irregularly and pushed back, revealing a powdery mass of brick red-colored uredospores. The uredia vary in size from 1 to 3 millimeters wide by 10 millimeters long. Later in the season, as the plant approaches maturity, the pustules turn black as the fungus produces teliospores instead of uredospores and uredia are transformed into black telia. Sometimes telia may develop independently of uredia. Uredia and telia may exist on wheat plants in such great numbers that large parts of the plant appear to be covered with the ruptured areas, which are filled with the rust-red uredospores, the black teliospores, or both.

14.7.3 Causal Organism of Rust of Wheat

The causal agent of this disease is *Puccinia graminis tritici* Erikss. and Henn. (*P. graminis* Pers.,). It is a heteroecious parasite which completes its disease cycle in two hosts namely wheat and barberry (Berberis) or Mahonia.

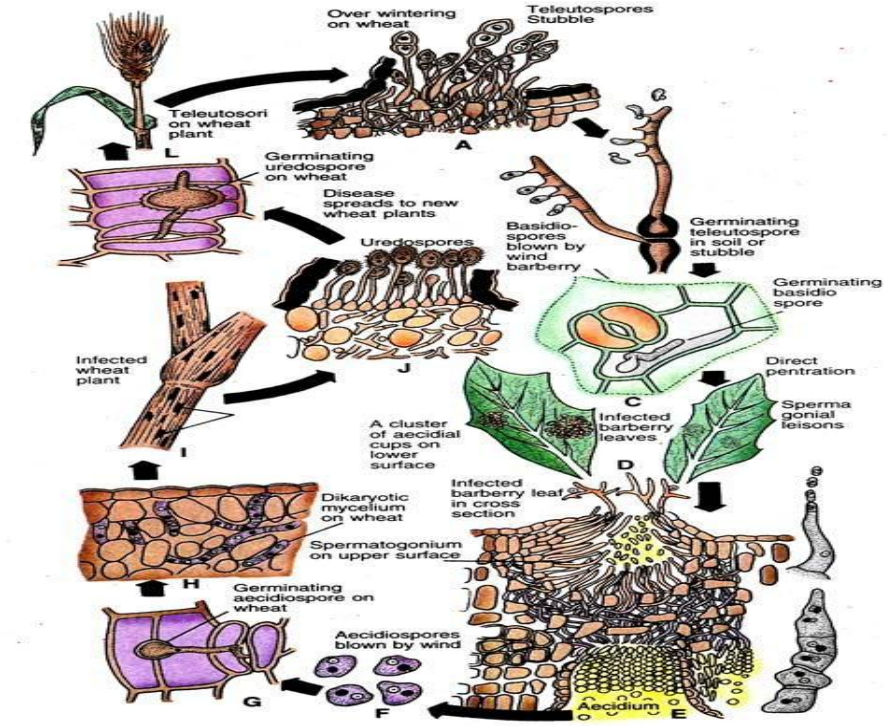
14.7.4 Disease Cycle of Rust of Wheat

Eriksson on the basis of his observations in cereal rusts, especially *Puccinia glumarum* proposed the "mycoplasm theory". He held that on the onset of winter, the fungal hyphae degenerated in the host plant. The fungus cytoplasm mingled with that of the host protoplasm in the cell. As soon as the winter was over, the mycoplasm (fungal cytoplasm) migrated into the intercellular spaces of the host. There the mycoplasm reforms the hyphae and haustoria. The mycoplasm theory was vehemently opposed. It was not widely accepted because of opposition and thus fell by the wayside. The commonly held view is that normally teleutospores are the overwintering structures. But in the plains in India uredospores constitute the primary inoculum. They are carried from the distant high altitude hills by wind. After the usual resting period the teleutospores germinate in situ (on wheat stem and stubbles in the field). Each cell produces a short promycelium (epibasidium) into which the synkaryon migrates. Each synkaryon undergoes meiosis in the promycelium or the epibasidium. Segregation of the sexual strains takes place. Walls are laid between the haploid nuclei so that each promycelium or epibasidium becomes septate and four-celled. Each cell of the basidium produces a single, uninucleate haploid basidiospores at the end of a sterigma. Of the four basidiospores thus produced two are of plus strain and two minus strain. The basidiospores are disseminated by air currents. While floating in the air they may chance to fall on young barberry leaves. If temperature and moisture conditions are favourable the basidiospores germinate. Each basidiospore develops a germ tube or a primary hypha. It penetrates the cuticle directly and brings about infection of the new host. Within the host tissue the primary hypha branches freely to form a monokaryotic or haplomycelium. The hyphae constituting it ramify in the intercellular spaces between the mesophyll cells. The cells are uninucleate. The nuclei in the mycelium are either of plus strain or minus strain depending upon the nature of the germinating spore. The mycelium feeds and grows vigorously. Eventually it enters the reproductive phase and forms thick

mats of hyphae here and there beneath the upper and lower epidermis. The hyphal mats beneath the upper eipdermis function as primordia of spermogonia. Those beneath the lower epidermis function as primordia of aecidia or aecia. In about a week's time the primordia beneath the upper epidermis produce small flask-shaped fruiting bodies called the spermagonia. They are embedded in the tissue in orange yellow spots on the upper surface of the leaves of barberry bush. Each spermagonium opens to the outside through a small aperture called an ostiole which projects above the surface of the leaf. The spermagonium contains three types of hyphal threads: 1. Periphyses: These are slender sterile, hyphae guarding the ostiole and projecting through it. 2. Spermatiophores: These are numerous, fine, elongated hyphae arising from the interior of the swollen portion of the spermagonium. They abstrict small, hyaline spermatia at their tips in succession. The abstricted spermatia lie free in the cavity of the spermagonium. 3. Receptive or Flexuous Hyphae: These are the fine, hair-like hyphal threads seen interspersed between the periphyses. They extend out through the ostiole and project much beyond the periphyses. The contents of the spermagonium are entirely plus or minus according as the spermagonium has developed from a plus or a minus mycelium. The spermatia emerge in a viscous sugary liquid through an ostiole to the leaf surface along with the flexuous hyphae. Sexual union or spermatisation, as it is called, takes place between spermatia of one strain and flexuous hyphae of the other strain. The intervening walls between the spermatium and the flexuous hypha dissolve at the point of contact. The spermatium nucleus passes into the receptive or flexuous hypha through the pore. The spermatium nucleus now passes down the receptive hypha through the septal pores and reaches the basal cell which becomes binucleate or dikaryotic. The dikaryotic cell develops into a secondary or a dikaryotic mycelium. The transference of spermatia from leaf to leaf is the work of insects. They are attracted by the nectar and visit one spermagonium after another. Meanwhile the hyphal mats beneath the lower epidermis develop into spherical masses of cells. These are known as the protoaecidia. By this time the secondary mycelium formed from the dikaryotic cells at the base of the receptive hypha reaches the young aecidium. Its cells mingle with the haploid tissue of young aecidium. As a result a palisade-like layer of binucleate cells is formed at the base of aecidium. These binucleate basal cells produce binucleate aecidiospores in terminal chains. The wall of the aecidium splits open. The aecidium now assumes a cup-shaped form. The lower epidermis also ruptures and the aecidiospores are now exposed. They are unable to reinfect barberry. The aecidiospores are binucleate and are carried by air currents to the wheat host. Here the aecidiospores germinate each by putting out a germ tube or an infection hypha which enters the host tissue through a stoma. The tip of the infection hypha swells to form an appressorium which covers the mouth of a stoma. The contents of the aecidiospore migrate into the appressorium. A narrow, peg like infection hypha emerges from the appressorium, passes through the stomatal opening and enters the substomatal cavity to form a vesicle. From the substomatal vesicle arise hyphae which proceed intercellularly into the parenchymatous tissue of the host leaf to form the intercellular mycelium. The hyphae send small round

or branched haustoria into the host cells. About ten days after the infection of the grain host (wheat) rusty red and powdery masses of uredospores appear on the stem and the leaves in oblong to circular sori. They appear in the months of February to March. The uredospores are shortly stalked, oblong, echinulate structures with four equatorially arranged germ pores on the outer wall. These spores are binucleate. Being exposed they are easily carried by the wind to other wheat plants. Here, within few hours each uredospore germinates in the surface moisture provided by rain or dew. It develops a germ tube which enters the host tissue through a stoma. Within a week's time provided the weather conditions are favourable, the new dikaryotic mycelium produces a new crop of uredospores. They are disseminated in the same way. In this way the disease is spread rapidly and widely during the growing period. The uredospores are the only spores in the life cycle which can reinfect the host plant on which they are produced.

When the grain is almost ripe, black teleutospores begin to appear in the uredosori. Soon after the teleutospores develop in new and independent dark brown or black teleutosori or teliosori. The teleutospores are two-celled, thick-walled structures. They are the resting spores. The part of the life cycle which is passed on the grain host or the wheat plant represents the dikaryophase. During this phase each cell of the mycelium, each uredospore and each cell of teleutospore has a pair of nuclei called the dikaryon. One of these nuclei is of plus strain and the other of minus strain. Towards maturity plus and minus strain nuclei in each cell of the teleutospore fuse to form a synkaryon or the fusion nucleus. The mature teleutospores thus represent the reduced diplophase. *Puccinia graminis* tritici is a polymorphic species as it produces a succession of different types of spores. However, investigations carried out by late Dr. K.C. Mehta (1923-40) in India revealed that there is no local source of infection in the plains. The uredospores perish in the high summer temperature of the plains. There are no barberry bushes in the plains and thus the teleutospores which are unable to survive the high summer temperatures following the harvest remain ineffective. Hence, the sexual stage comprising the spermagonium and aecidia is cut out from the life cycle. The actual disease cycle is completed with uredospores alone. The teleutospores are produced but they are non-functional. This eliminates the chances of perennation of the rust on the alternate barberry host in the hills. This has been confirmed by the fact that the races of P. graminis found on the barberry bushes on the hills are different from those occurring on the cereals in the plains. In spite of the above-mentioned facts, there is annual recurrence of the rusts in the plains in India. It is due to the fact that the uredospores remain viable on the hills. The summer temperature at an altitude of 5,000 ft. and above is quite congenial for their survival. There at different altitudes, the uredospores over summer on the out of season wheat plants, stubbles, and grass hosts in the uredial stage. These serve as primary inoculum for the wheat crops when sown. The infection thus starts from the hills. The uredospores are wafted down from the higher altitudes to the foot hills. From there the infection is carried to the crops in the plains.



Disease Cycle of Rust of Wheat

14.7.5 Control of Rust of Wheat

The most effective, and the only practical, means of control of wheat stem rust is through the use of wheat varieties resistant to infection by the pathogen. A tremendous amount of work has been and is being done on the development of wheat varieties resistant to existing races of the fungus. The best varieties of wheat that combine rust resistance and desirable agronomic characteristics are recommended annually by the agricultural experiment stations and change periodically in order to evade the existing rust races. Much effort is now directed toward the development of varieties with general or partial resistance and toward the development of multiline cultivars. Several fungicides can effectively control the stem rust of wheat. In most cases, however, 4 to 10 applications per season are required for complete control of the rust; because of the low income return per acre of wheat, such a control program is not economically practical. Two applications of some fungicides, coordinated with forecasts of weather conditions favouring rust epidemics, may reduce damage from stem rust by as much as 75%. These chemicals, which have both protective and eradicative properties, and therefore even two sprays, one at trace to 5% rust prevalence and the second 10 to 14 days later, can give an economically rewarding control of rust. Certain systemic fungicides also control stem rust when applied as one or two sprays 1 to 3 weeks apart during the early stages of disease development. Seed treatments with some systemic chemicals inhibit early but not late season infections. Damage by the stem rust fungus is usually lower in fields in which heavy fertilization with nitrate forms of nitrogen and dense seeding have been avoided.

Check your Progress- 5

Note: a. Write your answer in the space given below

b. Compare your answer with those given at the end of the unit.

5. What are the symptoms of rust of wheat?

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14.8 ANSWER TO CHECK YOUR PROGRESS QUESTIONS

1. Late blight of potatoes and tomatoes, the disease that was responsible for the Irish potato famine in the mid-nineteenth century, is caused by the fungus-like oomycete pathogen Phytophthora infestans. It can infect and destroy the leaves, stems, fruits, and tubers of potato and tomato plants.

2. Leaf spot is a common disease in ground nut caused by *Cercospora arachidicola* and *Cercospora personata*.

3. Mango anthracnose is caused by the fungus Colletotrichum gloeosporioides var minor (also known by the name of its perfect stage Glomerella cingulata var minor). Spore production by this fungus is favoured by wet or humid weather. The dispersal of these spores is particularly favoured by rain and wind.

4. Vascular cotton wilt (Fusarium oxysporum f.sp. vasinfectum) Symptoms of Fusarium wilt can appear at any stage of crop development. At high inoculum density or when infection initiates from the seed, plants may be killed at the seedling stage.

5. Symptoms of wheat leaf rust is reddish-brown, pustules develop on leaves and sheaths. Pustules break through the leaf epidermis, and spores are easily dislodged by rain, wind, or contact.

14.9 SUMMARY

Plant disease, an impairment of the normal state of a plant that interrupts or modifies its vital functions. Infectious plant disease are caused by bacteria, fungi or viruses and can range in severity from mild leaf or fruit damage to death. In this unit, author has discussed about various crop disease such as late blight of potato, leaf spot disease of groundnut, anthracnose of mango, wilt of cotton, and rust of wheat. Unit deals with study about the disease, symptoms, causal organism, disease cycle and control or management crop disease.

Late blight of potato is caused by *Phytophthora infestans*, belongs to the subdivision Mastigomycotina under the Division Eumycota. Symptoms of Late Blight of Potato are available on foliage and inside the Tuber. Disease Cycle of Late Blight of Potato: In the dormant phase, the fungus could perennate in the tuber as mycelium and in plant debris as oospore. During favourable condition, the perennating mycelium becomes active and develops active mycelium. The active mycelium develops branched sporangiophore bearing sporangia on it. On the other hand, the oospore germinates by producing germ tube bearing sporangia at their tip. In both the cases, the developed sporangia disperse by wind or rain, germinate on host surface by producing zoospores and cause infection to the host tissue. The infected host again develops sporangia on sporangiophore. The sporangia develop zoospore which cause further

infection. This process repeats several times in the growing season - this is the secondary cycle. During favourable condition, the zoospores may come down into the soil and may cause infection to the tubers. The tuber infection may also take place on contact with infected foliage during harvest. The perennating mycelium inside the tuber remains active, if the infected tubers are kept in the storage for seed potato. The disease can be controlled or reduced by the following methods such as Cultural Methods, physical, chemical and biological methods.

Leaf spot disease of Groundnut is caused by *Cercospora arachidicola* and *Cercosporidium personatum*. All the aerial parts of the plants attacked by the pathogen shows characteristic symptoms. Initially, the lower leaflets get infected showing dark spots and later on, each spot becomes surrounded by yellowish halo. With severity and maturation, the spots become dark brown to almost black, parti-cularly on the upper surface of the leaflets. With severity of disease, the spots become coalesced and defoliation of the leaf takes place. The pathogen can perennate through conidia on diseased plant debris that remain in the soil, on shell of fruits and also on seeds. Infection takes place through stomata or by piercing the host epidermal cell. Infection commonly takes place through upper epidermis and after entering the host tissue, the pathogen ramifies and by aggregation of mycelium, stroma develops. With further development, the stroma creates a pressure and the conidiophores come out through ruptured epidermis. Conidia are developed on conidiophores. The conidia after detachment, disseminate through wind to the different regions of the same plant or to the different plants and cause further infection and thus help to spread the disease. This disease can be control or reduce the disease incidence through tow methods 1. Cultural methods (sanitation, crop rotation, Early Sowing) and 2. Chemical Methods (Seed Treatment and Foliar Spray- a. Bordeaux mixture b. Other chemicals. c. Systemic fungicides.

Anthracnose of Mango disease is caused by *Colletotrichum gloeosporioides*. On mango, anthracnose symptoms occur on leaves, twigs, petioles, flower clusters (panicles), and fruits. Disease cycle: Dissemination: spores (conidia) of the pathogen are dispersed passively by splashing rain or irrigation water. Inoculation: spores land on infection sites (panicles, leaves, branch terminals). Symptom and disease development: black, sunken, rapidly expanding lesions develop on affected organs. Pathogen reproduction: sticky masses of conidia are produced in fruiting bodies (acervuli) on symptomatic tissue, especially during moist (rainy, humid) conditions. Many cycles of disease can occur as the fungus continues to multiply during the season. Pathogen survival: the pathogen survives between seasons on infected and defoliated branch terminals and mature leaves. Management of mango anthracnose consists of five approaches: Site selection, cultivar selection, cultural practices in the field (sanitation, plant spacing, intercropping, etc), fungicide sprays in the field and post harvest treatments (physical and chemical methods).

Wilt of Cotton disease is caused by *Fusarium oxysporum* f.sp. *Vasinfevtum.* Symptoms of Wilt of Cotton is earliest seedling is the yellowing and browning of the cotyledons. Leaves lose their turgidity first turn yellow and then brown and finally drop off. The tap root

stunted and laterals are less abundant. Browning and blackening of vascular tissues. Discolorations of leaves starts from the margins and spread towards midribs. Wilting may be complete or partial. *Fusarium oxysporum* f.sp. *Vasinfevtum* can survive in soil, in plant debris, and in infected seed beneath the seed coat, even after acid delinted and treated with fungicide seed treatments. The fungus produces multiple types of spores and some types can survive in soil for many years. Spread within a field occurs when infested soil is moved by implements, vehicles or personnel, or when water carries infested soil or plant debris in irrigation or storm water to other fields. Management of Fusarium wilt in cotton is difficult because of the ability of *F. oxysporum f. sp. vasinfectum* to survive in soil for long periods. Crop rotation is often recommended to reduce the incidence of Fusarium wilt, but the ability of the fungus to survive in the soil for long periods in the absence of cotton limits the effectiveness of rotations.

Rust of wheat contains different rust disease in wheat such as stem rust (also known as black stem rust) is caused by *Puccinia graminis* f. sp. tritici. It is primarily a disease on wheat, though it can also cause minor infections on certain cultivars of barley and rye. Leaf rust is caused by *Puccinia recondita* f. sp. *tritici* (now known as Puccinia triticina). Stem rust is characterised by reddish-brown, powdery, oblong pustules. The pustules have a characteristic torn margin that can occur on both sides of the leaves, on the stems and the glumes. Stem rust spores are much darker in colour than leaf rust spores, which are light brown and don't have torn margins. Rust fungi have complex life cycles, which may require two specifically different host plants and up to five different spore stages. Rust diseases that require two host plants to complete the life cycle generally have what is known as an economic host and an alternate host. The economic host, in these diseases, is wheat. The alternate host is typically a weed or native plant. For example, barberry (Berberis vulgaris) is the primary alternate host for the stem rust fungus. Infection of barberry results in circular, yellow- to red-colored pustules on the underside of the leaves. Spores (aeciospores) produced on barberry plants infect wheat, and another type of spore (basidiospores) produced on wheat infects barberry plants. Although both hosts are necessary to complete the full life cycle, epidemics on wheat can develop rapidly because spores (uredospores) produced on wheat can cause auto-infection (spores infect the same plants on which they were produced). This spore stage of the life cycle is known as the repeating stage and is responsible for the rapid development of disease outbreaks.Management of rust of wheat is carried out by various methods such as variety choice, appling fungicides, cultutal practies and earlier wheat maturing varieties.

14.10 KEY WORDS

Anthracnose: A disease that appears as black, sunken leaf, stem, or fruit lesion, caused by fungi that produce their asexual spores in an acervulus.

Rust: A disease giving a “rusty” appearance to a plant and caused by one of the Uredinales (rust fungi).

Wilt: loss of rigidity and drooping of plant parts, generally caused by insufficient water in the plant.

Late blight: A highly destructive disease of solanaceous plants, especially potatoes and tomatoes, caused by the oomycete Phytophthora infestans, appearing in wet weather often late in the growing season, and characterized by decay of the foliage and tubers or fruits.

Leaf spot: any of a large number of fungal, bacterial, or viral plant diseases which cause leaves to develop discoloured spots.

14.11 SELF ASSESSMENT QUESTION AND EXERCISES

1. What is Anthracnose of mango?
2. Define Wilt of Cotton.
3. Give the name of causal organism of Leaf spot disease of Groundnut.
4. Give any two symptom of Rust of Wheat.
5. Wite essay on late blight of potato with reference to causal organism, symptoms, and disease cycle and control measures.