Course No. : PATH 231 **Title : Introductory Plant Pathology.**
Credits : 1 + 1 = 2 **Semester : III**

A. Teaching Schedule (Theory)

(As per Lettr No. HOD/PPAM/ UG/ Syl./637/ of 2000 dt. 16,29,30/8/20(from HOD/Path. M.P.K.V. Rahuri (Minutes of 2nd Meeting of reviewing sylla of U.G. courses in Plant Pathology & Agril. Microbiology (KKV, Dapoli 4-5 Au 2000)

Lecture No.	Topics to be covered
1	Plant disease concept and economic importance.
2&3	History and development of Plant Pathology.
4	Causes and types of plant diseases and their classification
5	Symptoms and signs of plant diseases.
6	Introduction to plant pathogens, morphology and ~~reproduction~~ i ~~fungi~~
7	Variability of plant pathogens.
8	Parasitism, perpetuation and disease development. (Pathogenesis)
9	Dissemination of plant pathogens.
10	Predisposition and effect of environment.
11	Introduction to epidemiology.
12	Plant disease forecasting.
13	Principles of plant disease control.
14	Exclusion including PEQ and Eradication.
15&16	Plant protection
17	Disease resistance.

Disease Assessment - Agri. III Gen Notes I D M

B. Teaching Schedule (Practical)

Exercise No.	Title of exercise
1	Study of a typical bread mold fungus.
2 & 3	Morphology of fungi (Vegetative structures).
4 & 5	Reproductive structures and spore fruits in fungi.
6 & 7	Symptoms and signs of plant diseases.
8 & 9	Examination and identification of plant diseases and plant pathogen
10	Isolation of plant pathogens on culture media.
11	Methods of inoculation and Koch's postulates.
12 & 13	Acquaintance with different groups of fungicides and physical mea of controlling plant diseases.
14 & 15	Study of fungicides, fungicidal formulations, preparation of comm fungicides and their application.
16	Study of plant protection appliances.
17	Methods of disease appraisal and crop loss assessment.

Text Book Recommended :

1. Introduction to Principles of Plant Pathology by R.S. Singh. Oxford & IBH. Publ. CO. New Delhi. 1996.
2. Essentials of Plant Pathology by V.N. Pathak. Prakash Publ. Jaipur, 1972.
3. Plant Pathology by G.N. Agrios. Fourth Edn. Acad. Press, New York, 1997.
4. Introductory Mycology by C.J. Alexopoulas, C.W. Mims and M. Blackwell. 4th Edn. Willey, New York, 1996.
5. Introductory Mycology by M.N. Kamat, Prakash Pub, Pune, 1967.

C. Lesson Plan

Lesson No.	Topics to be covered
1	Plant disease concept and economic importance : Introduction and development of concept of Plant Pathology. Definitions of plant disease by various scientists. Interaction between the host, pathogen and environment. Various branches of Plant Pathology. Economic importance. Wellknown epiphytotyics and losses caused by plant diseases.
2 & 3	History and development of Plant Pathology : I. History and development of Plant Pathology in ancient, dark, premodern, modern and present eras. II. Contributions made by : Surapal, Theophrastus, Pliny, Ibnalawan and others, Robert Hook, Anton van Leeuwenhoek, Needham, Linnaeus, Tillet, Prevost, Robert Koch, Marshai Ward, Millardet, Meyer, Jensen E.F. Smith, Erikson, Biffen, Iwanowasky, Stackman, Cragie, Luthra, Stanley, Bowden, and Pierie, Doi and Asuyama, Butler, Mehta, Mundkur, Dastur, Kulkarni, Bhide, Uppal, Thirumalachar, Patel, Rangaswamy and Patel.
4	Causes of Plant diseases and Their Classification : A Causes : I. Parasitic causes : Fungi, slime molds, bacteria, phytoplasmama phanerogamic parasites, protozoa, viruses and nematodes. II. Non-parasitic causes : Environment, deficiency, soil conditions and chemical injurics. B. Classification of plant diseases : I. On the basis of occurrence, spread, severity and periodicity : Endemic, epiphytotic, pandemic and sporadic. II. Group of hosts affected: cereal diseases, vegetable diseases III. Plant parts affected : Root diseases, stem diseases. IV. On the basis of pathological effect : Necrosis, hypoplasia, hyperplasia, wilt, cankers, rots, blights etc.
5	Symptoms and signs of plant diseases : A. Definitions of symptoms and signs. B. Types of symptoms : I. Hypoplasia II.

Hyperplasia and III. Necrosis.

6 Introduction of plant pathogens, morphology and reproduction : A. Introduction to plant pathogens viz. bacteria, phytoplasma, viruses, flowering plant, parasites, protoza and nematodes. B. Reproduction in fungi : Asexual and sexual methods. C. Spore fruits in fungi : asexual and sexual fruits.

7 Parasitism, perpetuation and disease development : A. Parasitism, types of parasitism. B Perpetuation : Mode of perpetuation of pathogen : facultatism by dormant mycelium, by sclerotia, polymorphism, physiologic specialization. C. mechanical presasure, production of cell wall degrading enzymes, avenues of penetration inside the host, direct avenues, indirect avenues.

8 Dissemination of plant pathogens : A. Continuous dissemination : Autonomous dissemination . Role of air or wind, water, animals, birds, insects, nematodes and mites in dissemination of plant pathogens. B. Discontinuous dissemination : Man, seed, soil and agricultural operations as agents of dissemination of plant pathogens.

9 Predisposition and effect of environment : A. Meteorological factors: Temperature, humidity, precipitation, dews, light, hail storms, etc. B. Soil factors : Soil temperature, soil moisture, soil conditions, pH, nutrient deficiencies in soil, etc. C: Biological factors : insect fauna and soil flora.

10 Epidemiology : A. Definition B. Simple interest and compound interest diseases. C. Essential conditions for epiphytotics 1. Distance of susceptible plant from the source of primary inoculum, 2. Abundance of distribution of susceptible host, 3. Disease proneness in the host, 4. Presence of suitable alternate or collateral hosts for survival, 5. Presence of aggressive isolate of pathogen, 6. High birth rate of the pathogen, 7. Low death rate, 8. Easy and rapid disposal of the pathogen, 9. Adaptability of the pathogen and 10. Optimum weather conditions. D. Decline of epidemics: Saturation of the pathogen in the host population, reduction in the aggression of the pathogen and decline in the host.

11 Plant disease forecasting : A. Methods of disease forecasting. B. survey and survellence. C. Forecasting model. D. Satellite imaginary forecasting.

12 Variability in plant pathogens : A. Variability among the pathogens (Viruses, bacteria, fungi). B. Viruses : Mutation, hybridization. C. Bacteria : Mulation, conjugation, transformation and transduction. D. Fungi : Mulation and hybridization.

13 Principles of plant disease control : A. Exclusion of inoculum. B. Avoidance of pathogen. C. Eradication. D. Protection. E. Disease resistance

14 Exclusion including PEQ and Eradication : A. Plant Quarantine : Domestic, international, seed certification B. PEQ. C. Eradication :

	1. Removal of plant parts and sanitation. 2. Eradication of alternate hosts. 3.Destruction of collateral host. 4.Rogueing and 5.Culltural Practices.
15&16	Plant Protection : A. By physical methods heat, steam, hot water, solar heat, etc. B. By chemical methods :Fungicides. C. Classification of fungicides: Sulphur fungicides, copper fungicides, mercury fungicides, organomercurial fungicides, systemic fungicides. D. Methods of application of fungicides : Seed treatment, soil application, spray/dust application. E. Plant protection by
17	biological means : biocontrol agents.
	Disease resistance : Types of resistasnce : Monogenic, oligogenic, polygenic, horizontal, vertical and environmental resistance.

D. WEIGHTAGES

Sr. No.	Topics	Marks
1	Plant disease concept and economic importance of plant diseases.	4-6
2	History and development of Plant Pathology.	4-6
3	Causes and types of plant diseases. Symptoms and signs of plant diseases.	6-7
4	Introduction to plant pathogens, morphology and reproduction in fungi and bacteria.	6-7
5	Parasitism, perpetuation, disease development and dissemination of plant pathogens.	4-5
6	Epidemiology, predisposition and effect of environment on plant disease development.	6-7
7	Plat disease forecasting and variability among the pathogens.	4-5
8	Principles of plant disease control	6-7
		40-50

NOTES ON- INTRODUCTRY PLANT PATHOLOGY

COURSE No. PATH-231 **Credits : 1 + 1 = 2**

(Notes prepared as per the Teaching Schedule Finalised during 2nd meeting of reviewing syllabi of U.G. courses in Plant Pathology & Agril. Microbiology held at K.K.V. Dapoli and minutes circulated vide letter No. HOD/PPAM/U.G./Syl./637/ of 2000 dt. 30-8-2000 from by the office of the HOD/Pl. Path. & Agricultural . Microbiology, M.P.K.V. Rahuri.)

INTRODUCTION

Plant Pathology: Plant Pathology or Phyotopathology is the branch of agricultural, botanical or biological science which deals with the **cause, etiology, resulting losses and control of plant diseases.** (Study of the causes of diseases about plants)

Objectives of Plant Pathology :

1. To study the living, non-living and environmental causes of disease or disorders in plants.
2. To study the mechanism(s) of disease development by pathogens.
3. To study the interaction between the plant and the pathogen in relation to the overall environment.
4. To develop systems of management of the diseases and reducing the losses caused by diseases.

BRANCHES OF PLANT PATHOLOGY

Microbiology : It is the science dealing with study of microorganisms from animals and protists kingdoms such as molds, yeasts, bacteria, viruses, algae, protozoa, nematodes, etc.

Mycology : It is the science that deals with the study of fungi and their life cycles.

Bacteriology : It is the science which deals with the study of bacteria.

Virology: It is the science that deals with the study of viruses.

Nematology: It is the science that deals with the study of nematodes.

What is Plant Disease?

When a plant is suffering, we call it diseased. However, this does not define the term 'disease' Often the symptoms produced by a disease, the cause of a disease, and the injuries caused to the plant have been considered synonymous with the term disease. However, they signify only the condition of the plant due to disease or the cause of the disease.

Plant disease: When something is functing poorly in the body, we come to the decision that we are sick, hence "disease is **malfunctioning process** that is caused by continuous irritation which results in suffering. It is a pathological process. (Horsfall and Diamond (Plant Pathology, Vol. I. 1959).

Plant disease: Disease is a malfunctioning process that is caused by continuous irritation, which results in some suffering producing symptoms. (Accepted by American Phytopathological Society and the British Mycological society).

When a plant is diseased, it is a " **dis--ease**". It means that the plant is **uneasy** due to some conditions to which its system is not accustomed.

THE IMPORTANCE OF PLANT DISEASES

Plant diseases are important because of the loss they cause. The loss can occur in the field or in the store and at any time between sowing and consumption of the harvest. In India alone crop diseases are destroying food grains and other products of the tune of more than Rs. 5,000 crores every year. The microorganisms destroy perishable food materials in the store.

In the history of mankind, plant diseases have been connected with a number of important events.

Late Blight epidemic of Potato : In 1845 this disease destroyed the potato crop of Ireland and caused famine as the potato crop was the staple diet of the England, Ireland and certain parts of continental Europe.
This famine reduced the population of 80 lacs to 60 lacs in Ireland.

Wheat rust epiphytotic : In many countries, wheat rust appeared in epiphytotic form time to time. This disease forced the farmers in many parts of the world to change their cropping patterns.

In Northern and southern Europe- wheat bread is more common, in central Europe, rye bread is common, in southern parts of U.S.A., people eat maize bread, while in northern U.S.A., wheat bread is more common. The reasons for this food habits is that in these parts **wheat does not grow well due to rust** and the farmers grow maize or rye.

Helminthosporium leaf spot of rice (Bengal Famine):

During 1943, Helminthosporium leaf spot disease caused a famine due to severe loss in yield of rice.

Coffee rust : In 1867 coffee rust attacked the plantations in Sri Lanka and reduced yield from 228 kg/acre to 101 kg/acre. By 1893 the export of coffee was reduced by 93%. The economic crisis forced the planters of cut down coffee plants and take to tea planting.

In addition to direct losses in yield and monetary returns, the plant diseases affect the society in many other ways.

When fodgrains are attacked by fungi, they may contain toxins (e.g. aflatoxin) which cause insanity, paralysis, stomach disorders, etc. in human beings and animals.

Causes of plant diseases

A pathogen is constantly associated with a disease. The world "Pathogen" can be broadly defined as any agent or factor that incites "Pathos" or disease in an organism.

The plant pathogens can be grouped under the following categories...

A. Abiotic Factors : (Non-living):

i) Deficiencies or excesses of nutrition- e.g. Khaira disease of rice
ii) Light
iii) Moisture
iv) Aeration- e.g. Hollow heart of potato
v) Abnormalities in soil conditions
vi) Atmospheric impurities- e.g. mango tip rot

B. Mesobiotic causes: (Neither living nor non-living)

i) **Viriods :** Naked, infectious strands of nucleic acid. e.g. spindle tuber of potato, cirtus exocortis.

ii) **Viruses :** These are infectious agents made up of one type of nucleic acid (RNA or DNA) enclosed in a protein coat e.g. Leaf roll of potato, Leaf curl of tomato and chilli, mosaic diseases of crops.

C. Biotic causes : (Living, Animate or cellular organisms)

I. Prokaryotes

1. **Mollicutes :** These are wall less prokaryotes that include Mycoplasma like organisms (MLO) and spiroplasmas. **Diseases caused by MLO-** Grassy shoot of sugarcane, little leaf of Brinjal, Sandal spike, bunchy top etc. Diseases cause by spiroplasmas : Citrus Stubborn, corn stunt.

2. **Rickettsia - like bacteria (RLB)**- These are very small sometimes sub-microscopic, walled bacteria e.g. Citrus greening.
3. **True bacteria:** e.g. Brown rot/ wilt of potato, soft rot of potato and vegetables, leaf blight and leaf streak of rice, citrus canker, s'cane ratoon stuning.

II. **Eukaryotes :**

1. **Fungi :** e.g. potato wart, club-root of cabbage, leaf blight of potato, downy and powdery mildew diseases.
2. **Protozoa :** e.g. Heart rot of coconut-palm, phloem necrosis of coffee.
3. **Alga :** e.g. Red rust of mango, papaya etc.
4. **Metazoan animals:** Nematodes- e.g. Root knot of vegetable crops, molya disease of barley and wheat, Ear cockle of wheat, citrus decline .
5. **Flowering plant parasites :** e.g. Dodder, Striga, Loranthus

DISEASES AND THEIR CLASSIFICATION

Disease is not the cause but it is the result due to cause. The diseases may be infections or noninfectious. Plant diseases can be classified on the basis of (1) cause, 2) Group of hosts affected, 3) Parts of plant affected 4) pathological effects on host 5) Gross effect produced on host (symptoms) 6) Occurrence, spread, periodicity and severity.

A. **According to major causal agents...**

1. **Non-infectious or Non-parasitic or Physiological diseases.**

These diseases remain non-infectious and cannot be transmitted from one diseased plant to another healthy plant. These diseases are due to disturbances in the plant body caused by...

(a) Lack of proper inherent qualities
(b) Improper environmental conditions of soil and air
(c) Injurious mechanical influences
(d) Low and high temperature
(e) Unfavorable temperature
(f) Unfavorable oxygen relations
(g) Unfavorable soil moisture and pH
(h) Presence of toxic gases in the atmosphere
(i) Lightning injury
(j) Mineral excesses and deficiencies in the soil
(k) Absence or excess of light

e.g. Tip rot/ Necrosis of mango fruits, Black heart of Potatoes, Tip burn, Dakhina, Khaira diseases of paddy.

2. **Infectious diseases:**
These are diseases which are incited by foreign organisms or viruses under a set of suitable environments. There is a definite association of pathogens with these type of diseases.

(a) Parasitic diseases : Fungi, bacteria, mycoplasma, algae, phanerogams (flowing plant parasites).

(b) Virus diseases : (Virus = Poison/Venum)

II. **Group of hosts affected:** e.g. Cereal diseases, vegetable diseases etc.

III **Plant parts affected :** e.g. Root diseases, stem diseases, leaf diseases.

IV **Pathological effects on hosts :** e.g.Necrotic, hypoplastic, hyperplasia diseases.

V **Gross effect produced on host:** e.g. wilts, cankers, rots, blights Necrosis Hypoplasia, Hyperplasia.

VI **Classification on the basis of occurrence, spread, periodicity and severity:** Parasitic and virus diseases are often classified in relation to their occurrence and severity.

1. **Endemic diseases:** When a disease is more or less constantly present from year to year in a **moderate** from, it is classified as **endemic** i.e. when the host and the pathogen reach a state of biological euilibrium and the association assumes apparently harmless characters. All such diseases which become persistent through their survival on alternate or wild hosts, (wart of potato is endemic in Darjeeling.) from one crop season to the next are also included in this group.
2. **Epiphytotic diseases:** when the diseases occur involving sudden outbreaks periodically e.g. wheat rust, powdery mildew of grapes, Downy mildew and ergot of Bajra. The pathogen may be more or less constantly present as in endemic disease but the environmental conditions favourable to the development of disease occur only periodically.
3. **Pandemic diseases:** when the epiphytotic disease spreads throughout the continents or sub-continents and results in mass mortality or damage. e.g. black stem rust of wheat in India in 1947, Late blight of Potato in Europe in 1845.
4. **Sporadic:** When the disease occurs at very irregular intervals and locations and in relatively few instances i.e. it affects individuals within a corp. e. g. Long smut of Jowar.

HISTORY AND DEVELOPMENT OF PLANT PATHOLOGY

The development of plant pathology is closely linked up with the progress of Botany. As a science, plant pathology had its beginning in middle of 19th Century. Until then the early workers concerned themselves more with observations and systematic arrangement of diseases.

History of plant pathology is divided into different five eras.....

1. Ancient Era : From ancient time to 5th century (476 A.D.)
2. Dark Era : 5th Century to 16th Century (476 A.D. to 1600)
3. Pr-modern Era: 17 th Centruty to 1853 (1600 to 1853)
4. Modern era: 1853 to 1906
5. Present era : 1906 onwards

1. **Ancient Era**: Diseases in plants have been known since ancient times. Rusts, blights, mildews, smuts were familiar to Hebrews, Greeks, Romans, Chinese and Indians. In this period though the observations on the plant disease accurrence were perfect, the interpretations about the causal agents and environmental relations were based on unsound analysis of facts and influenced by superstitions and religious dogmas. Rigveda, Atharveda (1500-500 B.C.), the Arthashashtra of Koutilya (32 HS6 B.C.), Sushrut Sanhita (200-500 A.D.), Vishnu Puran (500 A.D.), Agripuran (500-700 A.D.) Vishnu Darmottar (500-700 A.D.) etc. are ancient Indian Books where plant diseases and other enemies of plants have been mentioned along with the methods to control them.

The diseases were divided into two groups : Internal (Physiological diseases) and external (infectious diseases). Internal diseases were supposed to be due to disorders in the system of the plant while external diseases were supposed to be due to attack of micro-organisms and insects. Hygiene, tree surgery, protective covering with pastes and special culture of plants etc. were known in ancient India.

In chemical treatments use of honey, ghee, milk, barley flour, pastes made from herbs, plant extracts, etc. were recommended. For control of root diseases oil cakes of **Mahuva**, mustard, sesame, castor, etc. were used.

Symptoms of plant diseases are mentioned in the Bible, Shakespeare's pomes and dramas and other Christian literature in the same manner as they have been mentioned in Hindu mythology, Jataka of Buddhism, Raghuwansh of Kalidas and other ancient literature.

300 B.C. : Theophrastus : In his book "Enquiry in to Plants" had recorded his experiences about plant diseases which were based on imaginations and observations, but not on experimentation. He mentioned that diseases occur spontaniously or autonomously (i.e. without external cause).

Symptoms of diseases and control of diseases have been mentioned in "VRIKSHAYURVED" by Surapal in ancient India. Even mention of plant diseases has been made in Buddhist literature of 500 B.C.

100 A.D.- Anno domini : In the year of Lord Pliny observed and described plant diseases and suggested some remedies. He beleived that the disease origentated from the plants or from the enviroment. Romans had Rubigo and Rubigus as Rust gods.

2. Dark era : 476 A.D. to 16th Century.

Very little work was done in plant pathology in this period. The science and learning got a setback after the fall of Roman empire..

Some Arabians like Ibnal-awan described symptoms of many diseases and suggested some control mesures

1440: Printing was introduced in Europe and this reflected interest in learning of science, arts, literature and commerce.

3. Pre-modern Era : also known as Autogenic era (1600 to 1853) .

1645: Establishement of Royal society of London where great discoveries were communicated and discussed.

1675: Anton van Leeuwenhoek: A Dutch worker ground simple lenses and developed the first microscope.

1635-1703 : Robert Hook :saw sections of cork and plant tissues with the help of microscope and showed that plant tissues were made up of minute units called cells.

The invention of microscope in 17th century gave vision to the hitherto unknown microorganisms.

1683: Leeuwenhoek : observed bacteria and many other microorganisms new to the science of microbiology.

1705: Tournfort : classifed diseases on the basis of causes.

1729: P.A. Micheli : An Italian botanist, first time studied many fungi and their reproductive structures under the microscope and identified spores as seeds of fungi proving that the fungi originate from their spores.

1743 : Needham : reported plant parasitic nematodes in wheat galls.

1753: Carlous Linnaeus : established Latin Binomial System of nomenclature of plant and animals in his book **"Species Plantarum."**

1755: Tillet : A French botanist, proved that bunt of wheat is infectious and can be controlled by seed treatment. His seed treatment was widely Practiced.

1773: Zallingar : classified plant diseases in to 5 different classes.

1774: Fabricus : (Danish) classified plant diseases in to classes, families and genera.

1801: Persoon : published **"Synopsis Methodica Fungorum"** for classification of fungi and it is the starting point for nomenclature of Ustilaginales, Uredinales and Gastromycetes

1807: Prevost : A French scientist suggested Cu So4 seed treatment for bunt of wheat. Discovered life cycle of bunt fungus.

1821-32 : E.M. Fries : published **'Systema Mycologica'**– chief starting point for nomenclature of fungi.

1821: Robertson : first used sulphur as fungicide.

The theory of spotaneous generation continued to be dominant up to the first half of 19th Century. Workers like Unger, Mayen, Leibig, supported this theory. Unger believed that diseases in plants were the results of internal disturbances in the physiology of the plants. This is known as **Autogenic** or **physiologic period** since the thoughts of the period on plant diseases were distinctly physiologic with a tendency towards the mythology. At the end of the period, microorganisms then were considered as results and not the cause of the diseases. Later at the end of the period it was becoming increasing clear that fungi were very closely associated with disease lesions. This kept raising the question as to whether fungi might be the cause of the disease rather than the result of the diseases.

Increased use of microscope lead to questioning of physiologic theory. The existance of fungi as separate entities and cause was firmly established.

Late blight of potato occurred in devastating eoiphytotic form in the middle of 19th century. During 1830 – 45 in Europe this greately stimulated the work in plant pathology as it was the potato famine occurred in 1842 where the few scientists believed that late blight of potato was caused by fungi associated with it.

1846-48 M.J. Berkely : came out with the parasitic theory of plant diseses. He published several papers on diseases of vegetables, cereals and other crops. Berkely described *Oidium* as the cause of powdery mildew of vines. He (1855) observed root knot nematodes in cucumber.

1853: Anton de bary : Showed the fungous origin of the late blight of potatoes. His first paper on late blight of potato was published in 1861 and the last in 1876. The foundation of modern plant pathology was laid by him.

4. Modern era : (1853 to 1906) : This period was devoted to the study of the role of pathogens causing plant diseases. The theory of spontaneous generation was disproved by Louis Pasteur, Tyndall, Koch and others.

Anton de Bary (1831-1888) was born in Germany, Contributed a lot to the science of Botany. In 1853, he published a book " DIE BRAND PILZE" and established the relation of parasitic fungi. He studied the life cycle of many diseases caused by fungi. He showed the fungous origin of late blight of potato.

In 1864, he showed the heteroccious nature of rust fungus.

Heteroecism: The phenomenon where the fungus requires two urelated species of host plants for completion of its life cycle. He also suggested the role of enzymes in between pathogens and plant hosts. He trained many scientists from different counties among them to mention, Marshal Ward from England, Woronin (Russia), Farlow from U.S.A., Brefield from Germany and Millardet from France. He thus brought the science of plant pathology forefront. **He is known as the father of plant pathology.**

1840-50 : Tulsne brothers (R.L. & Charles Tulsane) : Have prepared mycological illustrations of rust and smut fungi. Most of the work dealt by them is on smut & rust fungi. They confirmed the work of Prevost.

1850-1875: Cunningham and A Barclay : Started identification of fungi in India. Cunninghdm made special study of rusts and smuts. K.R. Kirtikar was the first Indian scientist who collected and identified fungi in India.

1858: Julius Gotthels Kuhn: Wrote a book for farmers On plant pathology " The diseases of cultivated crops, their causes and their control" where fungi were considered as causal factor and he classified diseases as parsitic, unfavorable soil and climatic conditions. He first demonstrated the enterance of bunt mycelium into wheat seedlings. Kuhn (1857) described stem and bulb nematodes. Wetzel mentioned him as father of plant pathology.

1873: T.J. Burill : Started teaching of Plant pathology at University of Illinois.

1875-1883: Brefield : Introduced and developed methods of growing microorganisms in pure culture. He also studied life cycle of smut fungi and diseases of cereals.

1876: Robert Koch, and Louis Pasteur : Had proved bacterial nature of Anthrax disease in animals.

1878: T.J. Burrill : Showed bacterial nature of Fire blight of Apples.

1878: Woronin : In Russia studied the life cycle of club root of cabbage.

1881: Robert Koch : Developed the dilution plate method of isolation of bacteria and fungi.

1881: Marshal Ward : from England studied th life cycle of coffee rust in Sri. Lanka (Ceylon).

1882-85: P.A. Millardet : from France discovered the use of **Bordeaux mixture** (Cuso4 +Lime) for control of downy mildew of Grapes.

1886: Adolf Meyer : showed infectious nature of Tobacco Mosaic by injecting the juice from diseased plant into healthy plant.

1887: Jensan : developed hot water treatment to seeds for loose smut of wheat.

1888-91: E.F. Smith : transmitted Peach Yellows by budding. He also showed the bacterial nature of wilt of cucurbits.

1892: Ivanowski : passed the juice of Tobacco mosaic affected plant through chamber land filter and showed it as infectious i.e. filterable nature of viruses.

1894: Eriksson : a Swedish scientist showed biologic races in cereal rust. He also putforth the *mycoplasm* theory i.e. protoplam of fungus mixed with protoplasm of host he called as mycoplasm and according to him his mycoplasm was responsible for sudden outbreak of rust.

1899: Beijerinck : confirmed Ivanowski's work and called virus as Contagium vivum fluidum.(Infectious living fluid).

1900-1909 : Orton : studied *Fusarium* wilt of cotton, cowpea and watermelon and deveoped resistant varieties.

1902: Takami : showed insect transmission in Rice stunt virus.

1905: Biffen worked on cereal rusts and studied genetics of disease resistance and showed Mendelian Inheritance of rust resistance.

5. Present Era : 1906: onwards:

In 20th century, phytopathology expanded rapidly with remarkable discoveries. Only major lines of contributions are mentioned here -

1. Teaching of Plant Pathology as a subject has been started in Universities and Colleges. The information of research is being given to the cultivators through Extension agencies.
2. Genetics in relation to resistance of disease in host plants is being studied in order to develop resistant plants.
3. Environment in relation to plant disease is being studied which help in forecasting of diseases and ultimately to undertake prophylactic control measurs.
4. The nature and biological activities of plant viruses are being studied in detail.
5. Improvement of fungicides.
6. Prevention of diseases through regulation, quarantine, certification etc.
7. Namatode diseases are being studied.
8. Role of phytoplasmas as causal organisms in plant diseases is being studied.
9. **1907:** Establishment of American Phytopathological Society and publication of the Jurnal "Phytopathology" in 1911.
10. **1909 : L.R. Jones** : worked on enzymes secreted by soft rot bacteria. He worked on soil borne diseases in Wisconsin .
11. **1917: E.C. Stakman** : from Minnesota, demonstrated physiologic races in cereal rust of wheat.

E. J. Butler (1874-1943)- Before 1910, he initiated exhaustive study on Indian Fungi at Pusa. He is considered as the Father of Modern plant pathology in India. He trined scientists at I.A.R.I. in wilts of cotton and Tur, rice diseases, cereal rusts, He wrote monograph on "Pythiaceous fungi", Fungi and diseases in plants. He was appointed as the first Driector of I.A.R.I., New Delhi.

1927: Cragie : showed function of pycnia in rust fungi.

1930: Plant Pathology as a University subject came into being at the Universities of Madras (est. 1857), Allahabad (est. 1887) and Lucknow (est. 1921)

1931: J.C.G. Luthra : from Panjab in India advocated Solar-heat treatment to wheat seeds for controlling loose smut of wheat.

1935: W.M. Stanley : isolated virus from Tobacco mosaic affected plants, purified and showed the chemical nature as *crystalline protein*.

1936-37 Bawden and Pirie : showed that viruses are nucleo-proteins.

1945: Agra University had introduced P.G. Programme in plant pathology at Govt. Agril. College, Kanpur.

1960: Establishment of Agril. Universities in the country and teaching Plant Pathology with its supporting courses in mycology, bacteriology, virology, nematology, biochemistry, etc. has become important part of graduate and P.G. Programmes in agriculture.

1967: Japanese scientist Prof. Asuyama and his associates found mycoplasma like organisms were responsible for yellow disease of plants.

Flor 1955: Explained host- parasitic interaction in flax rust. He identified 25 such gene pairs in the flax rust. The gene for gene relationship has been found in some other host pathogen systems. e.g. *Phytophthora infestans* on potato. Several disease resistnt varieties have been and continuously being produced all over the world, but it is a continuous process as the fungi too breed more virulent races and the plant breeders always have to be on the alert.

IMPORTANT CONTRICUTIONS OF INDIAN PHYTOPATHOLOGISTS

Eriksson = cereal rust

K.C. Mehta (1892-1950): Of Agra College studied the Physiology and Epidemiology of cereal rusts in the country and showed that the rust spores die in plains of India during hot summer and infection is from hills where they over summer on self grown wheat and Barley plants and cause epidemics. He published a monograph on "Further studies on cereal rusts in India" in 1940.

B.B. Mundkur (1896-1952): He worked on cotton wilt in the then Bombay State. He published "Ustilagenales in India", (Genera of rusts)& suppliment to Fungi of India and published about 80 papers. He wrote the book Fungi and plant diseases' Dr. Mundkur was pioneer in establishment of Indian Phyto-Pathological Society in 1947 and its journal "Indian Phytopathology" in 1948.

E.J. Butter (1874-1943): Before 1910 he initiated exhustive study of Indian fungi at IARI Pusa. He is considered as Father of Modern Plant Pathology in India. He trained scientists in Cotton wilt, tur wilt, rice diseases, rusts of cereals, etc. He wrote Monograph on "Pythiaceous fungi", Fungi and Diseases in plants. He was the first Director of C.M.I. He left India in 1920. He published Papers on *Pythium, Chitridiales,*

J.F. Dastur (1886-1971): He worked on genus *Phytophthora*, Cotton Anthracnose, Pink disease of Citrus, Foot-rot of Betelvine. Cotton wilt is Important contribution to the science. He published about 36 original papers and 4 books. He was the first Indian Plant Pathologist who is credited with detailed study of fungi and plant diseases.

B.N. Uppal : He worked on Downy mildew of Maize, bajra and showed physiologic specialisation in *Sclerospora gaminicola*. He worked on several fungal, bacterial and viral diseases with M.K. Patel, and M. N. Kamat in the then Bombay State at Pune. He published ' Fungi of Bombay' with Patel and Kamat. He remained as a prihcipal of the Agril. College, Pune.

G.S. Kulkarni (1920): Worked on Downy mildew of Jowar and Bajra and smuts of Jowar.

S.L. Ajrekar: Studied cotton wilt, sugarcane smut and jowar ergot.

Padwick: He worked and wrote a book on Rice diseases.

M.J. Thirumalachar: He published about 500 research papers describing 20 new genera and 300 new species of fungi. He worked in Hindustan Antibiotics Ltd. Pimpri and has discovered Aureofungin, Hamycin and other antibiotics. He worked on smuts and Rust in India.

M.K. Patel (1899-1967): A Pioneer worker on bacterial plant pathogens in India. He published about 140 papers mainly on bacterial diseases. He

modified solar heat treatment for wheat smut. He advocated a new system of classification of Phytopathogenic bacteria.

S.N. Dasgupta and T.S. Sadasivan : Prof. Sadashivan at Madras contributed extensively explaining the role of enzymes and toxins in plant, diseases. Investigations on the role of enzymes and toxins in diseases started with a great zeal by Gaumann, Walker, Dimond. Schools of Fundamentals of Plant Pathology at Lucknow and Madras were started by them.

G. Rangaswami: His work on bacterial diseases of plants and their control was outstanding. He published 5 books on Microbiology and Pathology and over 300 scientific papers. He wrote books entitled" Diseases of crop plant in India, Bacterial Plant Diseases, Agricultural microbiology.

M.N. Kamat: Worked on number of plant diseases and fungi. He wrote books on Plant Pathology and Mycology. He trained several students leading to M.Sc. and Ph. D. in Mycology from 1953 onwards. He expired in December, 1980. He wrote books entitled "Introductory Plant Pathology, Handbook of mycology and Practical Plant. Pathology".

V.P. Bhide: He worked on fungal and bacterial diseases as well as on Nitrogen fixing bacteria in the soil. He worked on biological nitrogen fixation and use of bacterial fertilizers was initiated by him. He trained several students leading to M.Sc. and Ph.D. in Microbiology and Plant Pathology.

INTRODUCTION TO PLANT PATHOGENS

1. **Bacteria:** They belong to 'Procaryotes' of kingdom protists. The major subgroups of procaryotic protists are blue green algae, mycobacteria, spirochetes and eubacteria (true bacteria).

Bacteria: Bateria are defined as uniceilular micro organisms, devoid of chlorophyll and dividing by fission.

Most of the bacterial plant pathogens are in orders Pseudomonadales, Eubacteriales and Actionomycetales. Bacteria, though unicellular, comprise a large variety of morphological forms. Bacteria are rods, cylindrical, ellipsoidal, variously spherical, spiral or helicoidal. Spherical or ellipsoidal forms are called **cocci** and according, to grouping they are micrococcus, diplcocous, tetrads, sarcina, streptococcus, or staphylococcus. The rodshaped **Bacilli** bacteria are microbacillus, diplobacillus or streprobacillus. Certain rod shaped bacteial cells form **Endospores**. A slimy layer adhering and covering the bacterial cell is known as **capsule**. The **flagellum** or hairlike appendages, speific in number and location on the cell wall, is an organ of locomotion or motility. The bacteria may be **artichous** (nonmotile), monotrichous, amphitrichous, lophotrichous or peritrichous. Bacteria multiply by binary fission.

The genera *Xanthomonas*, *Pseudomonas*, *Erwinia*, *Corynebacterium*, *Agrobacterium* and *Streptomyces* are important plant pathogens.

Rhizobium and *Azotobacter* are nitrogen fixing bacterial genera.

2. **Bacteriophages** are viruses infecting bacteria. Bacteriophages are *obligate parasites* multiplying inside the host cell.
3. **Viruses:** Viruses are **obligate, parasites**, highly infectious and host specific, intracellular, ultramicrescopic entities made up of nucleic acid and protein, They are parasitic on may, animals, birds, insects and plants. Plant viruses are either rod or spherical in shape. They are mostly of DNA with protein coat while execptionally some plant viruses contain RNA. Viruses are connecting link between living and non-living organisms.
4. **Mycoplasma :** These are small unicellular, pocaryotic organisms, (size 0.1 to 1.0 micron) Mycoplasmas are quite wellknown in animal pathology. However, mycoplasma is new in plant pathology. Since 1967 , most of the plant diseases under yellows group of viruses have been reported to be caused by mycoplasmas. They are **pleuro pneumonia like organisms** or **PPLO** and can be cultured on highly specific cultural medium containing sterol for growth. The mycoplasma is non-motile, gram negative, **containing both DNA and RNA**. It has no rigid cell wall but has lipoproteinaceous membrane. It multiplies both by binary fission and budding. They are sensitive to antibiotics *viz.* Tetracycline and Chlorotetracycline.
5. **Nematodes:** These are the organisms resembling roundworms which are present is soil and water. They belong to animalia kingdgom. They are microscopic, multicellular usually colourless, elongated, cylindrical, unsegmented and found in moist places. Size vavies from 100 microns to 1mm. They have well developed digestive, nervous and excretory systems. Outer layer is of **chitin**, the mouth and head is provided with a needle like structure, the **'Stylet'**. They are generally unisexual but rarely bisexual. Female is capable of laying 500 to 800 eggs at a time and thus there are 10 to 12 generations in a year under favourable conditions. Plant parasitic nomatodes are either endoparasites or ectoparasites.

6. **Flowering plant parasites:** A few Phanerogams (seed plants) are parasitic on living plants. Parasites are those, living at the expenses of the other organisms. The parasitism of the phanerogams appears in varying degrees. The **parasite** depends physiologicaly for its existance on the host plant. While **epiphytes** are the plants which take physical support or protection of host plants and are not classed as parasites e.g. tropical orchids. The phanerogams or the flowering parasites attack some crops causing considerable damage or loss. Some parasites attack roots of the hosts while others parasitize on the stem. Some are devoid of chlorophyll and depend entirely upon the host plant for food supply. While others have chlorophyll and are, therefore, capable of manufacturing food from CO_2 and water, however, they obtain minerals from host. The phanerogamic plant parasites may be grouped as - - - -

1. **Stem parasites-**
a) Complete or Holoparasite e.g. **Cuscuta** sp. (doddar).
b) Partial or Semi-parasite e.g. Loranthus

2. **Root parasites-**
a) Complete parasite e.g. Orobanche on Tobacco.
b) Partial parasite e.g. Striga on Jowar.

Fungi: Fungi are the members of kingdom Fungi and are microscopic. Fungi do not possess chlorophyll. They are either uni or multicellular. Fungi reproduce by means of spores either by asexual or sexual means.

Fungi can be defined as eucaryotic microorganisms, chlorophyll-less (devoid of chlorophyll)), having a well defined nucleus, the vegetative structure being uni-or multicellular filamentous thallus with a firm wall and reproduce by division of vegetative cells, well defined asexual and sexual spores.

The thallus of fungi may be plasmodial, unicellular, pseudomycelial or mycelial. In Myxomycota or slime molds, the somatic structure ((thallus) is amoeboid plasmodium and lacks true cell wall. In true fungi (Eumycota) true cell wall is present, the thallus is uni-or multicellular, branching. Such branched filaments are known as **'Hyphae'**. A mass of such hyphae is the entire vegetative body and is known as **mycelium.** The hyphae are generally charcterised as **septate** or **aseptate.** The cells have definite cell-wall made up of **cellulose or chitin or both.**

When the hyphae is continuous with nuclei irregularly distributed throughout the thalllus, it is called **aseptate or coenocytic or non-septate.** If the hypha is partitioned into compartments i.e. cells by means of cross-septa; it is known as **septate.** Hyphae rarely occur singly. Neighbouring hyphae are generally interwoven into felt like masses and thick, myceial tissues are formed. They are known as **'Sclerotia' or 'Rhizomorphs'.** Fungial cells contain cytoplasm, nuclei and vacuoles. They are, as rule, uninucleate but 'aseptate' mycelium is mulinucleate. Fungi store the food reserve in the form of **oil** and **glycogen.** Glycogen is a carbohydrate taking place of starch in higher plants.

Mycelium: is collectively the entire thallus which is filamentous. Individually, the filament is called Hypha and a mass or hyphae is termed as **Mycelium**. There are two types of hyphae and accordingly, the mycelium is divided into two types.
Septate: divided by cross walls (Septa) into segments;
Non-septate: or **Aseptate** or **Coenocytic:** is unicellular not divided by cross walls. The nuclei are embedded into the cytoplasm without being separated by cross walls.

Nutrition of fungi : Fungi need N,P,K Carbon, Sulfur Mg, Fe, Cu, Vitamins. Fungi lack chlorophyll and, therefore, they are unable to synthesize their own food. They feed and grow on readymade food. Their mechanism of nourishment is **absorption,** which takes place by **osmosis** through the cell walls.

SYMPTOMS OF PLANT DISEASES

Symptoms are the expressions of the diseased condition. They help in general diagnosis of the disease. Symptoms on the plants are either due to the character and appearance of the visible pathogen or its structures or organs and due to some effects upon or change in the host plants. The control of plant diseases is only possible through the correct diagnosis of the disease.

'Symptoms' are expressed externally as well as internally. The appearance of symptoms is the result of interaction between the host and the parasite under favourable conditions. Symptoms are often deceptive and one symptom may appear in a number of diseases and one disease may exhibit a number of symptoms e.g. Chlorosis may be due to deficiency or virus or Downy mildew. Similarly, Anthracnose of cotton may show seedling blight necrotic spots on leaves, bolls, yellowing of lint. etc.

'Symptoms' are always confused with signs. Signs are more reliable than symptoms and help in accurate diagnosis of the disease. **'Signs'** are the actual presence of the pathogen or its structures on the host as a result of manifestation i.e. signs are incidental or experimental evidences of the disease causing pathogen. e.g. in case of Root diseases, rotting and shredding of the bark is a symptom but presence of sclerotial bodies on stem or inside the bark is a sign. Similarly, wilting is the symptom but vascular discoloration with fungus growth (the presence of mycelium) inside the vascular bundles is sign. In case of Downy mildew diseases, chlorosis is a symptom while downy growth of the fungus on the leaves is a sign.

Symptoms are broadly grouped under three categories:

I) **Hypoplasia:** (Hypo=under+plasia=Formation) **Atrophy** or **dwarfing** : These types of symptoms are due to under development of tissues or chlorophyll resulting in dwarfing or chlorosis respectively.

II) **Hyperplasia:** (Hyper=over+plasia=formation) or **Hypertrophy** or **Over-growth** : These types of symptoms are due to abnormal or over development of tissues due to increase in size and /or number of cells.

III) **Necrosis:** These types of symptoms are due to destruction of affected tissues involving decay or rotting of the epidermis, collenchyma, cambium and vascular tissues.

Symptoms: Symptoms are expressions of diseased condition. They are expressed internaly as well as externally and help in general diagnosis. With the help of symptoms, a diseased plant can be identified from a healthy one. However, symptoms alone are not helpful in ascertaining the exact nature of the disease. Similarly, symptoms may result from different causes, unrelated

to each other. e.g. Chlorosis may be due to downy mildew, viral infection or deficiency. Fever in human being may be due to wound, typhoid or malaria.

Signs: Signs are the experimental or scientific evidences of the diseases and generally confirmed under the microscope. Signs help in accurate diagnosis of the diseases. Signs are the actual presence of the pathogen or its structures on the host or in the host as a result of manifestaion e.g. presence of whitish growth on the leaves in downy mildew of grape or Jowar, bacterial ooze in Ring disease of potato etc.

Symptoms are grouped under four categories

HYPOPLASIA : (Hypo = under + plasia = Formation)

These type of symptoms, are due to under dvelopment or sub-normal levelopment of the tissue, or reduction in the chlorophyll content resulting in warfing or chlorosis. They may be due to virus infection, parasitic or non-arasitic agencies such as deficiencies, excess of minor elements etc.

Chlorosis :

It means destruction of the Chlorophyll from the tissue. Different terms are applied to describe chlorosis according to the degree of destruction of chlorophyll, such as-------

Yellowing : There is complete destruction of chlorophyll. When the olour becomes white it is known as **etiolation.** These symptoms are usually used by parasitic fungi, viruses deficienceies or by non parasitiec causes g. yellowing of sugarcane due to deficiency, of White aster yellows and llowing of double bean are due to viral infection.

Pallor: Partial destruction of chlorophyll in the form of streaks. There unhealthy appearance of the plant due to deficiency or excess of water or ck of light or reduction in chlorophyll content due to pathogenic organisms . bajra seedlings affected with downy mildew.

Mosaic: Mosaic is caused by virus infection and chemical deficiencies, t virus mosaics are highly infectious than the deficiency diseases. It is due partial loss of chlorophyll or chlorosis in uneven patches e.g. Papaya saic, Tomato mosaic, Chilli mosaic etc.

Yellow mosaic: Light green and yellow patches are observed in the leaf ina e.g. yellow mosaic of beans

Yellow vein mosaic : Veins become yellow and leaf lamina remains en e.g. yellow vein mosaic of Bhendi.

Mottling : Partial destruction of Chorophyll in the interveinal area e.g. tle leaf of cirturs due to zinc deficiency.

Dwarfing:

General underdevelopment of entire plant is brought about by reduction nternodes resulting in stunted growth or appearance e.g. Rice stunt, ssy shoot of sugarcane etc.

Small leaf or little leaf : The size of leaf is greatly reducd e.g. Little leaf injal, grassy shoot of sugarcane.

unchy top : The leaves emerge out in the form of a bunch at the crown of plant e.g. Bunchy top of banana caused by a virus.

II. HYPERPLASIA : [Hyper = Over + Plasia = formation]

It means over development or abnormal development of the affected tissues This over development may take place in two forms either due to a) Increase in size of individual cells of the affected tissues or b) increase in number of cells e.g. Stem galls, Club root of cabbage, hairy roots etc.

1) **Tumours and galls : Tumours** are knot like structures or over growths of the host tissues. They are bigger in size e.g. Tumours caused by a Parasite, **loranthus** on mango and other fruit trees.

Galls are abnormal swellings or blisters or pimples/knots formed on plant parts. The bacteria, fungi, nematodes and some viruses induce formation of galls in plants by stimulating mature cells to resume meristemetic growth; Galls are smaller in size than tumours.

a) Root galls : e.g. Root knot of Brinjal, caused by nematodes.
b) Stem galls : e.g. White rust of crucifers, Loranthus on mango.
c) Fruit galls : e.g. Malformation of fruits, Physiological disorders in Mango.
d) Club root : e.g. club root of Cabbage, caused by *Plasmodiophora brassicae.*
e) Tuber galls : e.g. wart of Potato.

2) **Hairy roots or Spindle tubers** : formation of numerous fine roots e.g. spindle tuber of potato due to virus.

3) **Witches broom** : Excessive malformation of floral parts due to stimulation of a large no. of buds. Numerous slender branches arise from limited region in close clusters appearing like broom e.g. Green ear of bajra caused by downy mildew.

4) **Leaf curl :** It is the curling or distortion of the leaves. In the leaves, an increase in cell numbers in either side of mid rib and stimulation of growth of the Palliasse cells occurs. The cells of spongy Parenchyma result in puckering and curling e.g. ~~leaf curl of chilli and tomato.~~

III. NECROTIC SYMPTOMS :

They involve death or destruction of the affected tissues. Either entire plant or plant parts may be affected, involving decaying or rotting of the epidermis, Collenchyma, Cambium and Vascular tissues e.g. Blights, rots, wilts, damping off, cankers, anthracnose etc.

1. Blights : Here, there is a general and rapid destruction of plant parts like shoots, leaves, blossoms, twigs etc. The dead organs turn brown to black showing burnt appearance e.g. Early and late blight of potato, Bacterial blight of Paddy.

2. Wilts : Wilting or drying of entire plant is observed in adult plants. The leaves and other succulent parts lose turgidity, become flaccid and drop. It is a typical vascular symptom due to plugging of water conducting vessels or toxic effect e.g. Tur wilt, cotton wilt, Pea wilt, gram wilt etc. *(Fusarium sp.)*

3) Rots : The term is applied in cases where affected tissues decay or rot. Infection of parenchyma and pith tissues of various parts takes. Rots impart different colour reactions and are designated accordingly.

A. Dry rot: Decay of tissues takes place. Even after rotting some times the infected tissues remain firm or hard e.g. Dry rot of potato and corn.

B. Soft rot : Decay of soft tissues, rotting is accompanied by softening of the tissues, e.g. soft rot of lemon, mango, tomato, banana etc.

C. Red rot : Affected tissues become red in colour e.g. Red Rot of Sugarcane.

D.Wet rot : In addition to softening, there is slimy oozing of liquid e.g. storage rot in potato, citrus and other fruits, usually due to bacteria.

E.Root rot : Destruction of parenchyma of underground stems e.g. *Rhizoctonia* root rot of cotton, hollow stem of jowar(charcoal rot of jowar).

Rots may be described some times according to plant parts affected e.g. Stem rot (Papaya)., Collar rot (Groundnut), Neck rot (Paddy), Foot rot (Ginger).

The rots are also described after the discoloration produced on infection e.g. Brown rot (Potato), Black rot (Cabbage), Red rot (Sugarcane) etc.

4. **Damping off** : Sudden wilting and collapse of seelings is observed commonly in seed beds. The stem near the soil is affected, becomes constricted and weak e.g. Damping-off of seedlings of tobacco, tomato, Cabbage, Chilli etc.

5. **Cankers** : Deep seated intection due to destruction of woody tissues and cambium tissues. Cankers are raised from epidermal surface of the tissues and are rough to touch e.g. Citrus canker, Guava canker etc.

6. **Spots** : Formation of spots is a localised destruction of the tissues in more or less circular manner. They are usually found on the leaves, and may develop on stems or fruits. The dead tissues which are in limited area, give shapes as angular, round or circular surrounded by yellow, purple, red margins e.g. eye spot of Jowar, tikka of Groundnut, angular leaf spot of cotton.

7. **Tar spots and Streaks or Stripes** : necrotic area become typically tar stained e.g. Forest trees, palms, grasses and jowar.

Streaks are elongations of necrosis e.g. bacterial streak of paddy and jowar.

8. **Blasts** : These are same as blights but here the spots are distinct e.g. Blast of Paddy.

9. **Die back** : Dying of plant organs, especially stem and branches, from the tip downard or backward e.g. Die back of citrus, chilli.

10. **Exudation** : Secretion of sticky gum like substance due to disease e.g. Gummosis of citrus.

11. **Anthracnose** : Destruction of collenchyma and cambium tissues. Lesions are sunken in the centre with raised and prominent margins e.g. Anthracnose of grapes, chillies and beans.

12. **Black heart**: Blackening of central portion is observed in potato tubers due to high temperature and poor ventilation in storage e.g. Black heart of potato.

13. **Scab** = destruction of epidermal tissues in the form of scars. Infection is not deep seated e.g. Scab of potato and apple.

14. **Shot holes** : Decayed leaf tissues are given away leaving holes or perforations e.g. shot hole of Ashok and Mango plants.

15.Mummification : These types of symptoms are observed in fruits. The skin of the fruit becomes hard and fruits get shrivelled such fruits are called as mummified fruits e.g. Downy mildew of grapes.

IV) TERATOLOGICAL PHENOMENON:

This is the phenomenon where the plant or plant parts lose their original appearance and take up different form. This may be due to genetical or pathological factors e.g. Stems turn into tendrils, sepals turn into petals etc.

1.Smuts : The floral parts are usually affected, the ovaries are destroyed and replaced by forming sori e.g. smut of jowar, loose smut of wheat etc.

2.Ergot: Normal grains are replaced by Sclerotia e.g. Ergot of Bajra.

3.Phyllody : Transformation of normal floral parts into abnormal structures e.g. sesamum phyllody. Floral parts transformed into green, leaf like structures resulting in complete sterility.

4. Green ear : Flowers are converted into green and elongated leafy structures e.g. Green ear of Bajra.

SIGNS:

1. **Mildews:** The presence of the pathogen is seen on the host surface e.g. **Downy mildew**: White cottony growth of the fungus is seen on the lower surface corresponding to chlorotic area on the upper surface e.g. Downy mildew of bajra, Grape, Jowar etc.
   Powdery mildew: Powdery growth consisting of mycelium and numerous oidia (conidia) is seen on the host surface e.g. powdery mildew of pea. appear on upper surface of leaf
2. **Rusts:** The pustules of spores usually protruding the epidermis are seen on the host. Pustules may be either dusty or compact and white, yellow, brown, red or black in colour e.g. white rust of crucifies, leaf rust and stem rust of wheat.

B Powdery mildew

REPRODUCTION IN FUNGI

Reproduction is the formation of new individuals having all characters typical of the species. Fungi reproduce by two ways-Sexual and Asexual.

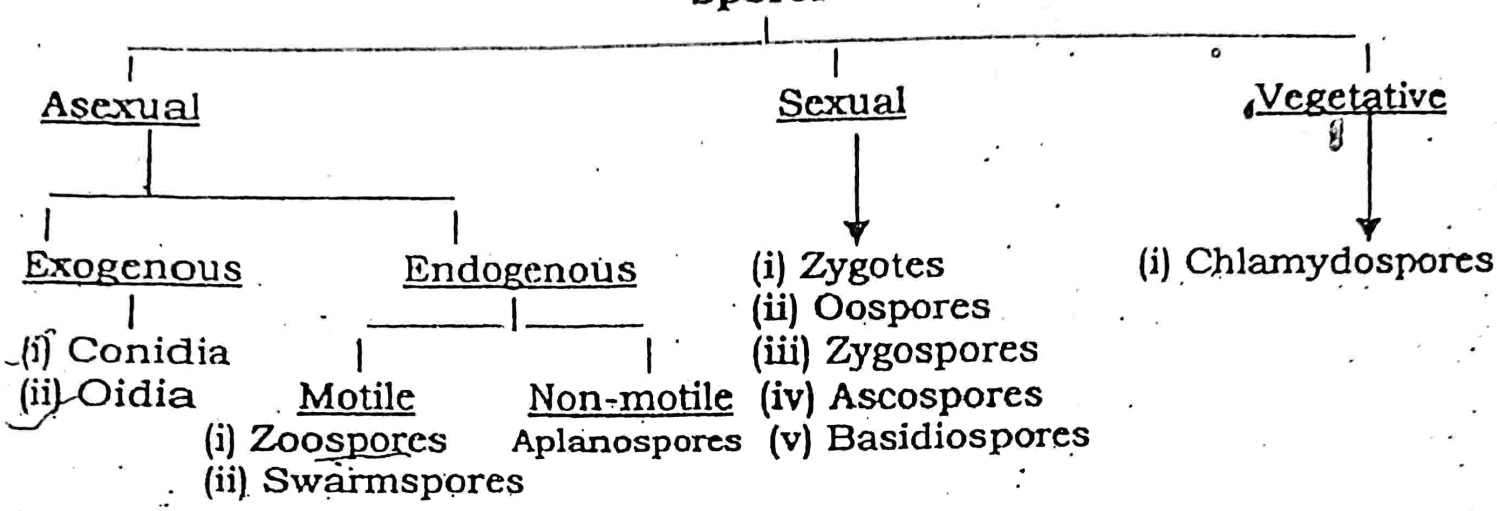
Fungi roproduce by spores. The spore is an unit of reproduction . Fungi reproduce by spores which are produced in 3 ways:

1. Asexual 2. Sexual 3. Vegetative.

Spores are specialised cells. One or more cells are set apart for reproduction. They are borne on specialised hyphae, the **sporophores** (Exogenous) or may be borne in special receptacle (Endogenous.) Endogenous spores are formed by division of the inner content of the mother cell into number of cells which become separated from one another and are liberated outside by breakage of the wall or through ostiole.

Exogenous spores appear as protrusions on the tip or at the side of the fertile hyphae and are cut off when they reach maturity.

CLASSIFICATION OF SPORES



Spore Fruits:

Spore fruit is an organisation of spores and spore bearing hyphae, arely naked but frequently enclosed in various types of receptacles. The pore fruit usually has thick covering known as **peridium.** The spore fruits re imortant in tiding over unfavourable conditions, multiplication and aintaenance of inoculum. They are utilised as taxonomic characters in etermining the broad line of various goups of fungi.

Stroma: The pseudoparenchymatous tissues into which sporophores :t embeded. Hyphae rarely occur singly. Neighboring hyphae are generally ter wound into felt like masses and form a thick hyphal tissue which is med as **plectenchyma.** When single hyphal element is still recognizable, it called **Prosenchyma** or **prosoplectenchyma.** When the hyphal elements ive lost their identity, the tissues bear a resemblance of parenchyma of gher plants, then the tissue is termed as **Pseudoparenchyma.**

CLASSIFICATION OF SPORE FRUITS IN FUNGI

A spore fruit is an aggregation or collection of spores and spore bearing hyphae [sporophores], sometimes naked but frequently enclosed in various types of containers or spore cases or receptacles. The spore fruits have a thick wall known as **peridium**, which protects the spores till the onset of favourable conditions. There are two types of spore fruits based on whether spore fruit contains or gives the sexual or asexual spores and thus, there are sexual or asexual spore fruits.

Spore fruits

I Asexual		**II Sexual**
1. Sporangium(a)	(a)Ascocarps.	1) Cleistothecium(a)
2. Coremium(a)		2) Apothecim(a)
3. Sporodochium(a)		3) Perithecium(a)
4. Sorus(sori)		4) Ascostroma (Ascostromata)
5. Pycnidium(a)		
6. Aecium(a)	(b)Basidiocarps	1) Puff balls
7. Acervulus (Acervuli)		2) Bracket fungi
8. Pycnium(a)		3) Mushrooms

I) Asexual spore fruits :

1. Sporangium [pleural. Sporangia]

This type of spore fruit is a charcteristic of the fungi belonging to the sub division **mastigomycotina** and **zygomycotina**. It may be of different shapes, the commonest being the elliptical and the round. The elliptical sporargia are formed by lower fungi of Mastigomycotina and Zygomycotina which are semi-acquatic in nature, while round sporangia are formed by terrestrial fungi belonging to above sub- divisions. The spores in sporangium are called **sporangiospores**. When sporangium gives motile spores, it is called **zoosporangium** or **swarmsporangium** and spores as **zoospores** or **swrmspores**. The round or spherical sporangia give non motile spores known as **Aplanospores**.

2. Coremium [Pleural-coremia]

The hyphae form erect conidiophores and are grouped together to form coremia. Each coremium consists of sterile stalk terminating into fertile hyphae bearing conidia e.g. *Stysannus thyrosoides*.

3. Sprodochium: [Pleural. Sporodochia]

A fruit having cushion shaped stroma covered with conidiophores is known as sporodochium. The conidia formed inside, ooze out in sticky mass e.g. Genus Necteria growing on the trunks.

4. Sorus: [Pleural. Sori]

It is a little heap like compact mass of sporophores and spores and spores are usually covered by epidermis. At maturity the epidermis breaks and all the spores are liberated in dust e.g. Smut and Rust fungi.

5. Pycnidium : [Pleural. Pycnidia]

It is a Spherical or oval shaped sporefruit with short conidiophores lining innerside which bear spores or conidia called Pycnidiospores. The spore fruit usually opens at the mouth and this opening is called as **ostiole**. *Phoma spp.* and *Phomopsis* sp.

6. Aecium [Pleural-Aecia]

It is cuplike or bell shaped structure usually formed on the lower surface of the leaf, consisting of binucleate, hyphal cells producing yellow or orange coloured spores in chain called **aeciospores**, which are formed in basipetal manner e.g. Rust fungi of wheat and Bajra.

7. Acervulus : (Pleural-Acervuli)

It is compact mass of hyphae giving rise to short, simple, hyaline conidiphores, closely packed together forming bed like mass with or without thorns, The thorns may be deep brown to black and are as called **setae**. It is also known is modified open sours e.g. *Colletotrichum* and *Gloeosporium*.

8. Pycniun : (Pleural-Pycnia)

It is a characteristic of rust fungi. It is flask shaped structure containing pycniospores or spermatia eg. Rust fungi

II. Sexual Spore Fruits :

a) Ascocarps :

These are the spore fruits produced by the fungi belonging to the sub division Ascomycotina. Sexual spores are produced endogenously and are known as **ascospores** in a sac called **ascus** (pl. Asci).

The spore fruits are of various forms i.e. Spherical, flask, cup-saucer and pod shaped . Following are the different types of ascocarps.

1. Cleistothecium : (Pleural-Cleistothecia)

It is closed without ostiole, round to oval ascocarp with irregularly arranged or scattered asci with dark brown to black colour and provided with appendages to anchor or to hold fast and to help in dissemination. e.g. powdery mildew fungi of order Erysiphales.

2. Perithecium : (pleural perithecia)

A flask shaped ascocarp with narrow neck like ostiole through which asci are released. The asci are arranged or lined to the inner wall of the perithecium. The sterile structures present in between the asci are known as **paraphyses** which help the asci in nutrition and dispersion e.g. *Claviceps* and *Glomerella* spp.

3. Apothecium : (pleural Apothecia)

A cup or saucer shaped spore fruit with broad opening is known as apothecium. The asic are arranged in palliasse layer(like straw-filled mattress) called hymenium. The apothecium is usually fleshy and leathery in nature e.g. *Sclerotinia* sp.

4.Ascostroma : (Pl-Ascostromata).

The asci are formed directly in a locule or cavity within the stroma. Thus, stroma forms the wall of the ascocarp.

(b) Basidiocarps:

These are the fructifications of sub-division Basidiomycotina and consist of Mushrooms, Toads-stools, bracket fungi and Puff balls. They are highly developed and have compound structures. They may be fleshy, leathery, woody or waxy in nature and bear special structures variously known as **gills, pores, needles** and **chambers.** The sexual spores are the **basdiosopores**, proeduced exogenously on the tip of enlarged cell of hypha known as ***Basidium*** **(Basidia).** The basidiospores are usually 4 in number. The basidia are intermingled with sterile structures called **paraphyses.** The cells slightly larger than basida intermingled with hymenial layer are known as **cyctidia.**

1. Puff balls:

These are round or spherical, very small to big basidiocarps, commonly found on dead organic matter. The basidiospores are produced in the hymenium which line the inner surface. On maturity, basidiosores are given-off in the form of puff or smokes. The puffballs have got small stalk at the base.

2. Bracket Fungi:

These are compound fructifications growing on dead tree trunks. These are woody and hard basidiocarps. They are typically bracket, hoof or saddle shaped and highly colored. They are borne on short stalks. The hymenial layer is found on the honey comb fashioned pores in which basida and basidiospores are observed.

3. *Mushrooms :*

These are the fleshy or leathery, compound fructifications with variously colored, commonly found on manure pits, dung heaps and on any rich organic matter. They are borne on stalks and provided with gills and pores to the underside which contain hymenial layer. The mushrooms may be edible and non-edible or poisonous e.g. *Agaricus sp.*

The sporefruits are important in tiding over unfavorable conditions and multiplication & maintenance of inoculum . The spore fruits are utilized as taxonomic characters in determining the broad lines of various groups of fungi.

TYPES OF REPRODUCTION IN FUNGI

Asexual reproduction: It is also called as somatic or vegetative reproduction and does not involve the union of two nuceli, sex or sex organs. Hyphae cut into minute spores

Sexual roproduction: It is chracterized by the union of nuclei or two gametes of oposite sex.

In the primitive forms of fungi at the time of reproduction the whole unicellular thallus is converted into reproductive structure, such fungi are called as **'holocarpic.**

In the majority of the fungi a protion of the multicellular thallus is utilized to form reproductive structure, such fungi are called as **'eucarpic'.**

METHODS OF ASEXUAL REPRODUCTION

1. **By the fragmentation of the hyphae** Mycelium and each fragment grows into a new individual. The hyphae break up into their component cells as oidia or arthrospores which behave like spores. If the cells become enveloped in a thick wall before they separate from each other from other hyphal cell adjoining them, they are called **chlamyospores.** Fragmentation may also occur accidentally by the tearing off of parts of the mycelium through external forces e.g. oidia of powdery mildew fungi.

2. **Fission of somatic cells into daughter cells (Binary fisson / simple splitting)** : by forming a cell wall each spore germinates and forms a germ tube which grows into the mycelium. It is the characteristic of the **bacteria** and some **yeasts** which are true fungi.

3. **Budding:** It is production of a small outgrowth (bud) from a parent cell. As the bud is formed, the nucleus of the parent cell divides and one daughter nucleus migrates into the bud. The bud increases in size and eventually breaks off and forms a new individual. It takes place in majority of **yeasts** and many other fungi like **Rust** and **smut fungi**. At certain phases (Budding of sporidia).

4. **Production of asexual spores :** Fungi produce spores asexually and two types of spores are commonly produced e.g. sporangiospores, conidia.

SEXUAL REPRODUTION IN FUNGI

Sexual reproduction in fungi involves a fusion between two gamets of opposite sex. A gamet is unisexual and, therefore, haploid (1n). The spore resulting from the sexual fusion is bisexual and, therefore, dipoid (2n).

Fungi may be **monoecious** where both sexes occur on the same thallus, i.e. some species produce distinguishable male and female sex organs on each thallus. A single thallus of monoecious or hermaphroditic species can reproduce sexually by itself if it is self compatible or self feritle, where as some fungi may be **dioecious** where the two sexes occur on different thalli. A single thallus of dioecious species cannot reproduce sexually by itself normally since it is either male or female.

Gametes may be naked and motile as in lower fungi (Archimycetes) and are called **'planogametes'** or they may be carried in special cells and are non-motile as in higher fungi. The cell carrying the gamet is called **gametangium.**

The sex organs of fungi are called **gametangia.** These may form differentiated sex cells called gametes or may contain one or more gamet-nuclei. The terms **'Isogametangia'** and **'Isogametes** respectively, are used to designate gametangia and gametes which are morphologically indistinguishable. and **'Heterogametangia'** and **'Heterogametes'** to designate male and female gemetangia. and gametes which are morphologically different. In the later case, the male gametangium is called the **Antheridium** and female gametangium the **'Oogonium'.**

The sexual process involves 3 typical phases:

I. Plasmogamy; Union of two **protoplasts** bringing two haploid nuclei together in the same cell.

II. Karyogamy: Fusion of two haploid nuclei brought together as a result of plasmogamy. Karyogamy immediately follows plasmogary but in some cases it is delayed and plasmogamy simply results in formation of binucleate cell containing one nucleus of each cell (sex). Such pair of nuclei is called **dikaryon** and stage of the fungus as **dikaryotic** condition.

III. Meiosis: Nuclear fusion takes paces in all sexually reproducting fungi forming diploid (2n) phase. Meiosis takes place sooner or later after the diploid phase i.e. after the karyogamy. Meosis is the **reduction division** bringing the diploid zygote nucleus to the original haploid number in daughter nuclei. Thus, the fungi like all other plants, have haploid and diploid phases. The former is known as **haplophase** and later as **diplophase** and hyphae are called **haplont** or **deplont** according to the chromosomes in their nuclei.

To summarize, plasmogamy brings two haploid nuceli together in one cell, karyogamy unites them into one diploid zygote nucleus and meiosis restores the haploid condition in the four nuclei which result from it.

Methods of sexual reproduction in fungi:

1. Planogamatic copulation.
2. Gametangial contact
3. Gametangial copulation.
4. Spermatization.
5. Somatogamy
6. Heterokaryosis
7. Dikaryotization.

Ascospores → Isogamous planogamates – Allomyces fungi
Monoblepharidales – Female gamete is non motile
→ zygospores
→ Pycnidiospores in receptive hyphae in rusts
→ Smuts
→ Fusarium, Alternaria
Basidiomycotina

1. **Planogamatic copulation:** This involves the fusion of two naked gametes one or both of which are motile. Motile gametes are called planogametes. The most primitive fungi produce isogamous planogametes which are morphologically similar but differ in size and are produced by only one group of lower fungi belonging to the genus ***Allomyces***. While in ***Monoblepharis*** of order ***Monoblepharidales***, the female gamete is non-motile where as the male gamete is motile. The later enters the oogonium and fertilizes the egg i.e. the mating gametes are heterogamous in characters.

2. **Gametengial contact:** In a large number of fungi, the gametes of the male or of both the male and female gametangia have been reduced to undifferentiated protoplasts consisting chiefly of nucleus. Such gametes are never released from the gametangia to the out side but are transferred directly from one gametangium into the other. In this method, two gametangia of opposite sex come in contact and one or more gamete nuclei migrate from the male to the female. In no case do the gametangia actually fuse or lose their identity during the sexual act. The male nuclei, in some species, enter the female gametangium through a pore developed by the dissolution of the gametangial walls at the point of contact while in other species, especially developed fertilization tube serves as a passage for the male nuclei. After the passing of the nuclei has been accomplished, the oogonium continues its development in various ways and the antheridium eventually disintegrates e.g. Ascospores.

3. **Gametangial copulation:** This method is characterized by the fusion of the entire contents of two contacting gametangia. Such fusion takes place in one or two ways - - - -

a) **Passage of the contents of one gametangium into the other through a pore developed in the gametangial walls at the point of contact :** This method is particularly characteristic of some **holocarpic** forms in which the entire thallus acts as a gamtangium, the male thallus attaching itself to and emptying its entire contents into the female thallus.

b) **Direct fusion of two gametangial cells into one:** This takes place by the dissolution of the contacting walls of the two gametangia resulting in a common cell in which the two protoplasts mix e.g. **Zygospore.**

4. **Spermatization:** Some fungi bear numerous, minute, uninucleate, spore-like male structures termed **spermatia** which are produced in various ways. The spermatia are carried by insects, wind, water, or in some other ways, to the female gametangium or to special receptive hyphae, or even to somatic hyphae to which they become attached. A pore develops at the point of contact and the contents of the spermatium passes into the particular receptive structure which serves as the female organ, e.g. **Pycniospore** and **receptive hypha in rusts.**

5. **Somatogamy:** No sex organs, wherever, are produced by many of the higher fungi, somatic cells taking over the sexual function e.g. **smuts.**

Parasexuality: Some fungi do not go through true sexual cycle but derive benefits of sexuality through parasexnality. In this process, plasmogamy karyogamy and diplodization takes place but not at specified point in the thallus.

6. **Dikaryotization:** This is a degenerate type of sexuality of common occurrence in **higher Basidiomycetes.** It is accomplished through migration of nuclei from one cell to another cell of the vegetative haphae; often through the mechanism of anastomosis or clamps. The two nuclei remain in paitr and divide as such conjugate and ony fuse prior to formation of spores. No special sex cells are produced e.g. Mushrooms.

7. **Heterokaryosis:** This process is in the nature of vegetative grafting so commonly met with in higher plants and is independent of sex. It is brought about by 'Hyphal fusion' and anastomosis' or 'bridging' of vegetative hyphae of carrying nuclei of different genetical reactions and is of common occurrence in Fungi imperfecti like ***Fusarium***, *Alternaria* giving rise to new nuclear combination and resulting in the origin of new forms. The phenomenon of existance of different kinds of nuclei in the same idividual is called Heterokaryosis.

8. **Nucler dissociation:** This is brought about by segregation or sectoring of nuclei from originally multiucleate condition giving to new **"Mutant"** or **'clones'**.

It will be observed from the above methods that the sexual process. i.e. sexuality in fungi is varied. The sexual process has a fixed pattern in lower fungi while it is highly flexible in the higher fungi. A single sexual act gives to a single sexual spore in lower groups of fungi, while it is followed by many sexual spores in higher fungi. The sexual phase i.e. karyogamy is delayed for a long period in some fungi.

Sexual compatibility: A great many fungi produe clearly distinguishable male and female sex organs on the same thallus. These two sex organs may be compatible, self-fertile or may be incompatible i.e. sexually self-sterile because their male organs are incompatible with their female organs. In some fungi the two sex organs may be produced on two different thalli.

Monoecious or Homothallic: Analogous to hermaphrodite nature in higher plants. Both sex organs are produced by the same mycelium and the sex organs are comatible.

Heterothallic: Analogous to dioecious in higher plants. One type of gametes are produced on one thallus or mycelium and the other type of gametes on the second thallus i.e. two different mycelia each producing one type of gamete, or if both the gametes are produced on the same thallus, they are self sterile.

Heterothallism: The condition in which the sexes are segregated in separate thalli i.e. two different thalli being required for sexual reproduction e.g. *Mucor sp.*

Dr.A.F. Blakaslee in 1904 discovered the phenomenon of sexual incomptibility in fungal species of *Mucor and Rhizopus*. The fungal species which could produce zygospores on single thallus are called **'Homothallic'** while Some species which require two different compatible thalli to form zygospores he called **'Heterothallic'.** Since the two compatible strains could not be distinguished morphologically, Blakaslee labelled them as (+) and (-). He showed the heterothallism in the **Mucorales** as representing a true segregation of sexes in different thalli.

Similarly, Cragie in Canada in 1927 showed the heterothallism in rust *Puccinia graminis* demonstrating the fuction of pycniospores which are haploid or + ve in sexual tendencies.

PARASITISM

Fungi are divided into three groups according to the manner they obtain their food as-------

i) Saprophytic ii) Symbiotic iii) Parasitic

Fungi lack chlorophyll and, therefore, they are unable to synthesize their own food. They feed and grow on readymade food. Their mechanism of nutrition is absorption which takes place by **osmosis** through the cell walls.

i)Saprophytic: These obtain their food from dead organic matter which may be in the form of rotten plants, debris or animal tissues. Along with bacteria they play important role in decomposing the organic matter of plant and animal origin into simple substances and restoring them in soil e.g. *Mucor, Rhizopus*

iii) Symbiotic- is the nature of co-oprative association of two dissimilar organisms which mutually benefit each other and are not antagonisitic e.g. Lichens, Mycorrhiza.

Lichens: They are formed due to the symbiotic association of fungi and Algae. The algae prepare carbohydrates by photosynthesis and the fungi absorb water and mineral elements in solution which is exchanged for sugar and products of photosynthesis.

Mycorrhiza? It is the symbiotic association of certain fungi with roots of higher forest plants.

iii) Parasitic – They obtain their food from living tissues of the plants or animals.

Among the parasites one can distinguish different degrees of parasitism as

(a) Obligate parasites, (b) Facultative saprophytes and (c) Facultative parasites.

(a) Obligate parasites: are the organisms **which live entirely on living tissues.** They pass their entire life on living plants only. They can never be grown on dead, artifcial food material. e.g. Rusts, powdery and downy mildews. They are also called as "BIOTROPHS"

(b) Facultative saprophytes: These are normally parasites but are **capable of switching over as saprophytes** e.g. Smuts.

(c) Facultative parasites: They are **originally saprophytes** but have the faculty or **ability to become parasites** when come in contact with the living host plants under favourable environmental conditions e.g. *Pythium*, *Fusarium* etc.

Depeding upon the nature of parasitism with the host plant, mycelium is either **'ectophytic or endophytic'.**

Ectophytic: Mycelium growing on the surface of the substrate and deriving nutrition from epidermal calls through haustoria e.g. powdery mildew fungi.

Endophytic: Mycelium growing **inside** the host tissues.

1. **Intercellular mycelium :** Mycelium growing in between the cells e.g. Rust fungi.
2. **Intracellular mycelium:** Mycelium growing within the host cells e. g. smut fungi.
3. **Vascular mycelium :** Mycelium growing in the vascular tissues of the plants is called vascular mycelium e.g. *Fusarium* spp. Causing wilts.

Haustoria: Ectophytic and intercellular mycelium obtain their nourishment from host cells through haustoria. Haustorium is a specialised fungous structure used in absorbing food.

Survival of plant pathogen

PERPETUATION OF PLANT PATHOGENS

Pathogen is defined as an entity or any agent that induces a disease. The term pathogen in generally used to denote living organism. The plant disease caused by pathogens are contagious. In order to become more plan ts diseased in a field, particles or propagules of the pathogen must reach healthy plants and establish new infecton. These particles are called the **inoculum.** (Inoculum is the portion of any pathgen capable of being disseminatd and potentially capable of initiating infection of the disease. The inoculum may consist of virus particles, bacterial cells, mycelial threads, spores, sclerotia, nematodes or any type of propagule.)

Perpetuation: means survival of the pathogen in different forms in the absence of the main host or under unfavorable conditions of environment.

In the absence of the host plant, the pathogens find some alternate source or mode of survival to have continuous chain of infection.

The survival of pathogens can be grouped as:

1. Infected host as a reservoir of inoculum - rice, mango, phytophthora, fusarium, ① Soil ② Infected
2. Saprophytic survival outide the host, - fallen plant ③ Alternal & collateral host.
3. Dormant spores and other structures in or on the host or outside the host.

The initial infection that occurs from these sources in the crop is **'Primary infection'** and the propagules that cause the primary infection are called as **primary inoculum.** After this infection, the spores or other structures produced, are the sources of **'Secondary infection'** spreading the disease in the field.

The primary inoculum may be brought by wind or through seed, tuber or soil. After the primary infection, the fungus produces spores on host parts and these spores are disseminated by wind, water or other agencies and cause new infection in healthy crops. This is secondary **infection.** The primary infection, thus, initiates the disease and the secondary infection spreads the disease.)

Inoculum may originate from a few diseased pants or from many. The source of inoculum may occur in the same location as a population on healthy susceptible plants or may be located else where. The incolum may be produced within the plant at the host surface, on the plant refuse or on organic matter in soil, depending upon the nature of the pathogen.

Many pathogens live and reproduce in soil, on weeds, or on alternate or collateral hosts of lesser value than a specified crop. Inoculum may also be harboured in such locations for a long time and constitute a reservoir of inoculum that is a threat to a crop.

It is important to know as to how the parasitic fungi and other microorgnisms lead their life when the hosts are absent. The typical life cycle of a plant pathogenic fungus has two stages 1. Imperfect (asexual) stage and 2. Perfect (Sexual) stage.

I. The fungal pathogen enters the host, establishes parasitic relationship either as ectophytic or ondophytic mycelia and at maturity, gives rise to condiophores which bear conidia. This is an asexual stage which may be repeated again and again under favourable conditions representing active parasitic phase.

II. With the onest of summer in tropics or winter in temperate zones, the pathogen initiates the resting phase with formation of sex organs which fuse to produce sexual spores which is the **perfect** or **sexual stage**. This sexual stage is usually helpful in tiding over the fungus under unfavorable conditions.

Some fungi produce resting vegetative bodies such as **sclerotia** and **Chlamydospores** . On return of the favorable conditions or season, these bodies or sexual spores resume activities and carry on the parasitic life cycle.

Various methods and devices are adopted by the pathogens to enable them to perpetuate themselves under adverse conditions. Knowledge of this aspect aids in timely and successful control of the diseases.

Perpetuation of Plant pathogens as - - -

1.**Facultatism:** Several parasitic fungi developed the ability to modify their mode of life as 'saprophytes'. Pathogens like *Pythium*, *Fusarium*, *Phytophthora*, *Cercospora* and others survive in soil. These fungi are normally saprophytes but resume parasitic activities under favourable conditions. They survve in the form of spores or mycelium in the soil. The soil is a reservoir of inoculum of soil borne pathogens e.g. Vascular wilt pathogens (*Fusarium*), root rot pathogens (*Rhizoctonia*), etc.

Spores of certain smuts remain attached to the seed surface (grain smut of Jowar) while few remain in soil and debris. (Head smut of Jowar, sugarcane smut etc.)

Bacterial pathogens such as *Pseudomonas solanacearum* (Brown or ring tot of Potato) and *Xanthomonas campestris* (Black rot of Cabbage) survive in seed material and in plant debris in soil.

2(i) **Perpetuation by persistant mycelium:** some fungi like *Phytophtora infestans* (late blight of potato) *Phytophthora urecae* (Koleroga of Arecanut), *Celloetotrichum falcatum* (Red rot of Sugarcane), *Fusarium oxysporum f.*

cubense (Banana wilt) etc. persist in dormant mycelial condition on perrenial parts of the host.

2(ii) Perpetuation in propagative parts: Viruses like Bunchy top of Banana, Chlorosis of Banana, Potato viruses, sugarcane viruses persist in vegetative parts of the host.

3. Perpetuation by dormant mycelium in seed: In certain cases the pathogens are carried internally in the seed and their plant parts in the form of dormant mycelium. In loose smut of wheat (*Ustilagonuda tritici*), Bean anthracnose (*Colletotrichum lindemuthianum*), powdery mildew of Pea (*Erysiphe polygoni*), the mycelium perpetuates within the seed. In mosaic of beans; the virus is carried in the seed.

4.Perpatuation by Sclerotia: Selerotia remain viable in manure and debris for a long time and in the host plants under favourable conditions. e.g. *Rhizoctonia* sclerotium causing root rot, *Claviceps fusiformis*, (Ergot of Bajra scelerotia of ergot disease).

5. Perpetuation on other hosts: 'Collateal or alternate' hosts help in the perpetuation of certain pathogens.

i. **Collateral or complementory hosts:** In this case, some stages of life cycle of the pathogen are found as in the main host plant. It is, generally, from the same botanical family. Many pathogens have more than one hosts and they perpetuate on their weed hosts or wild hosts in the absence of their main hosts e.g. Yellow vein mosaic of Bhendi has other collateral hosts like *Hibiscus sp, Holyhock*, etc. The cereal rust pathogen can survive on wild collateral weed hosts.

ii. **Perpetuation on alternate hosts:** Certain fungi, especially the rust fungi, require more than one host for completion of their life cycle. The other host, which is required for completion of the life cycle of the pathogen, is called' **alternate host'**. The phenomenon of requiring two unrelated species of host for completion of life cycle of certain parasitic fungi is called as ***Heteroecism*** e.g. *Puccinia graminis tritici* (Black stem rust of wheat) is heteroecious having Barberry as alternate host. Where as **'Autoecism'** is the phenomonon when the polymorophic fungus completes its life cycle on the same hot e.g. Linseed rust (*Melampsora lini*).

6. **Perpetuation by resistant spores:** Downy mildew fungi, *Albugo condida* (white rust), smuts, bunts, rust fungi, powdery mildews perpetuate by resistant spores like oospores, chlamydospores, teleutospores, ascospores in asci etc. These spores survive and remain viable for long periods.

7. **Polymorphism:** An organism having several (Many) spore-forms (more than two) in the life cycle is called polymerphic and the phenomenon of several spore forms by an organism is called **'Polymorphism'** e.g. a typical rust fungus has got five spore forms *viz.* Pycniospores, aeciospores, uredospores, teleutospores and basidiospores. *Fusarium* sp. have macro conidia, micro-condition and chlamydospores.

Polymorphism maintains infection chain and persistence of mycelium through periods of droughts, dessication and freezing The polymorphic fungi can adopt rigorous conditions and can have more successful life free from uncertainty and risk involved in monomorphic fungi.

8. **Physiologic specialization:** Fungi which are capable of attacking several different species of plant, develop many distinct forms or races. These forms are morphologically similar but differ greatly in their physiology and or in their ability to attack different hosts.A number of sub-species or biologic strains of a fungus which are similar or differ slightly in morphology but greatly differ in the ability to attack different species of the hosts, the phenomenon is called **'Physiologic specialization'.**

Schroeter (1879) for the first time noted varying degrees of parasitism in cereal rust fungus. However, **Eriksson** 1894 gave a very convincing experimental demonstration of this phenomenon in *Puccina graminis.* He termed these parasitic sub-species as **'formae speciales'** *Puccinia graminis* is divided into seven sub-species attacking a particular host.

Host specialization in *Puccinia graminis*:

Host (s)					
	Wheat	**Barley**	**Oat**	**Rye**	***Agrostidis* sp.**
Wheat	+	+	-	-	-
Barley	+	+	-	-	-
Oat	-	-	+	-	-
Rye	-	-	-	+	-
Agrostidis sp.	-	-	-	-	+

On the basis of the above results several physiologic forms or sub-species of *Puccinia graminis* are recognised: as -----

Puccinia graminis f. tritici On Wheat and Barley.

Puccinia graminis f. avenae on Oat

Puccinia graminis f.secalis On Rye

Puccinia graminis f. poae On Poae sp (Grass)

Puccinia graminis f. agrostidis On *Agrostidis* grass

Puccinia graminis f.agropyri on agropyron grass

Puccinia graminis f. airae on aria grass.

Each of the strains or sub-species are made of **Physiologic races**

which differ in parasitism on various agronomic varities of the same host. These varieties differentiating the races are known as **'differential hosts'**

Specialisation of parasitism is of an extreme type now known as **'Physiologic race'** or **'Physillogic form'** or **'Biotype'** has been demonstrated by **Stakman** and his co-workers (1917 onwards) at the University of Minnesota, in *Puccinia graminis* and other rust fungi.

Stakamn and others (1918) noted many races in *P. graminis*. Now nearly 300 races and their biotypes are known in Black rust of wheat and of which nearly 20 are known in India.

According to the agency bringing about the spread, in general, diseases in pants are groped as ------

1. Soil borne,
2. Seed borne,
3. Air borne and
4. Insect borne

1. **Soil borne diseases:** Are those, whose pathogens survive in soil for varying periods. Although, mostly these pathogens attack seeds, seedlings, and roots, etc. Soil is a reservoir of inoculum of soil borne pathogens. In soil borne diseases, the inoculum perpetuates through dormant spores and through other dormant structures in soil or in diseased pant debris in the soil. The diseases such as wilts, root rots, foot rots, seedling blights belong to this category.

2. **Seed borne diseases** : Are those, whose pathogens are carried on or in seed or vegetatively propagated parts used as seed. The pathogens perpetuate as dormant mycelium in the infected embryos. Some viruses are carried through infected embryos of true seed or in the vegetatively propagated parts. Such diseases are known as **internally seed borne** e. g. *Ustilago nuda trici* causing loose smut of wheat while the pathogen carried through spores on seed coat, it is known as **externally seed borne** e. g. *Sphaecelotheca sorghi* causing Grain smut of jowar.

3. **Air burn Diseases** : Are those whose pathogens are primarily disseminated through wind currents and are capable of long distance spread, e.g. The rusts of wheat, blast or rice, koleroga of arecanut, downy and Powdery mildews of grapes and other crops.

4. **Insects borne diseases** : Are mostly of virus origin. Viruses are highly infectious and are mostly carried through agency of insects, mostly the sucking types. The insect vectors are mostly leaf hoppers, aphids, white flies, thrips, beetles or other chewing insects. After the virus is acquired by its insect vector feeding on diseased plant, it may persist in the insect, where as in these instances, the virus-vector relationship is ephemeral lasting for a very short time or nonpersistant.

INFECTION AND PENETRATION

Infection consists of actual entry i.e. invasion of the pathogen into the host, its multiplication and establishment i.e. its stable parasitic relationship within or upon the host body.

The Phenomenon of infection can be studied under the head as

1. Mechanism of infection: Various processes by means infection is established
2. Avenues of infection i.e. penetration (Actual entry of pathogen)

1.Mechanism of infection: The machanism of infection differs accoring to the nature of the pathogen. The machanism is **active** in case of **fungi and flowering** parasites. It is aggressive type in these cases while it is **passive** type in bacteria and viruses.

In order to gain entry, the pathogen has to reach the host, and develop active growth on its suffface. After the pathogen comes in contact with the host, the earlier phase of penetration is concerned with spore germination on the host sirface. The spore lodges in a drop of moisture and is stimulated to germinate by secretions of the host. The germ tube grows into the direction of host surface and attaches it self to the host surface firmly by special

structures known as '**appresoira**'. The appresoria give rise to one or more thin finely pointed **infection threads** or hyphae which directly pierce the cuticle and gain entry into the interior. The process is mechanical and not chemical.

The infection hyphae meet with the epeidermal wall, consisting mainly of cellulose and secrete enzymes which dissolve the cell walls and gain further entry into the epidermal cells and deeper into the host tissues by its enzymatic activities. Thus, the process is chemical in later stage.

When there is direct contact of infection hyphae and their enzymatic activities, the contents of the hot cells undergo rapid degeneration, plasmolysis of the cytoplasm and nucleus sets in causing ultimately injury to and disfiguration of invaded tissues. The infection process is thus completely by establishment of close relationship between the pathgen and the host through the help of **haustoria**.

Parasitism is the phenomenon of growth of one organism, at the expence for other, Parasitism is accompanied in 2 ways:

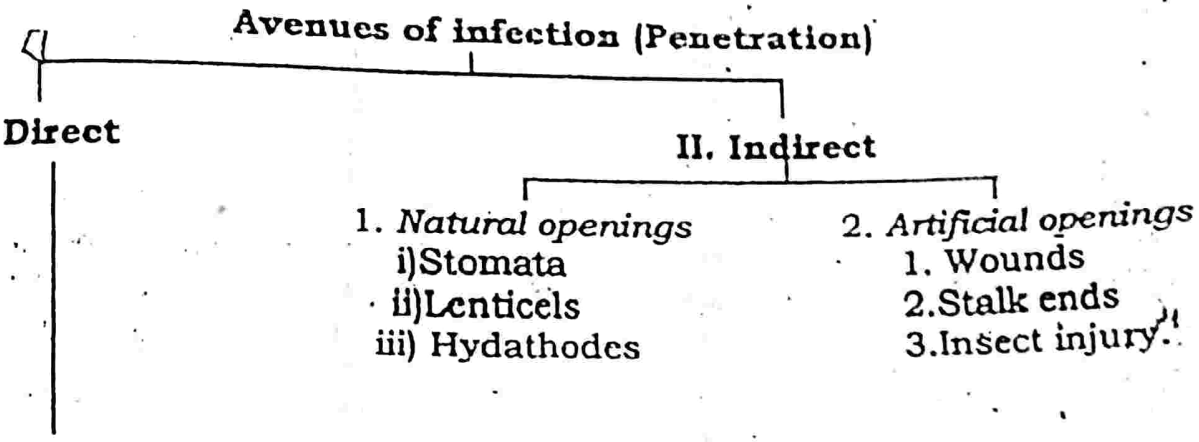
1. Destruction of host cells and
2. Closer reltionship between host and pathogen. e.g. Downy mildews, Powdery mildews, Rusts.

In the phanerogamic parsites like Loranthus, the process is just similar to fungi. The seed lodges on the host, production hypocotyl in contact with the host and sends down the primary root like structures which pierce the host tissues and get into cortexand xylem vessels from which it derives its nutrition. The infection, thus, established persists from year to year.

In Bacteria: The process of infection in bacterial pathogens, on the other hand, is of **passive type**. There is no direct attack and no machanical pressure is exerted. The action is more of **absorption** or **suction** and takes place through injuries, wounds, root hairs, nectaries and stigma ends.

In Viruses: In the plant viruses, the mechanism of infection is quite different. The viruses are highly infectious and filterable in nature and find their way through the sucking insects, which actually inject the virus into the host tisses by pointed stylets. The virus, then, multiplics into the host tissues and spreads in the entire plant. It induces host nucleic acid to producc virus nucleic acid.

2. Avenues of infection i.e. Penetration: After studying the mechanism of infection., it brings us the consideration of the next process., e.g. avenues of the penetration or the actual manner of entry of the pathogens into their hosts. Penetration may take place through several avenues depending upon the nature of the parasites.



1. Cutinised surface
i)Epidermis
2. Non-cutinised surface
1. Root hairs
2. Seedlings
3. Needle points
4. Nectaries
5. Buds
6. Stigma ends
7. Anthers

I. Dirct penetration:

1. Cutinesed surface-

i)Through epidermis: (Rusts, Powery mildews and Anthranose fungi). The basidiospores in rust fungi penetrate the host directly while the dikaryotic spores (uredospores) invariably penetrate through the stomata.

There are, however, several plant tissues which have no such cuticular defence line and make way for pathogen which use these organs as passage for migration into the deep seated tissues.

2. Non-cutinised surface-

1. **Root hairs:** As the root hairs are non-cutinised, they make an easy way for the parasites to enter through them. Several soil-borne bacterial pathogens and many root rotting fungi e.g. *Fusarium* wilt, *Rhizoctonia bataticola.*

2. **Seedlings:** It is known as systemic infection. Pathogen in such cases is carried on seed coat or comes in contact with germinating seed in soil. Infection of the young succulent tissue through hypocotyl occurs at the time of emergence of seedling and symptoms are exhibited in above ground

part e.g. D.M. at seedling stage or sometime at flowering stage e.g. Bunt and smut.-

3. **Needle points:** Infection through needle points is of special significance in the blister rust of pines caused by *Cronartium ribicole* which uses these points as avenues of entry for the basidiopores.

4.&5. **Nectaries and Buds:** These are favourable avenues for the infection in a few cases because of their non-cutinisied surface and sugary secretions, e.g. *Venturia inaequalis* causing scab of Apples, *Taphrina deformans* (Peach leaf curl) and bacterium *Erwinia amylovora* causing fire blight of Apples.

6. & 7. **Flower parts: Stigma and Anthers:** The stigma ends of the flowers are highly receptive organs for the entry of the pathogen because of their erect positions and the sugary secretions given out by their heads. The sugary secretions attract the flies and insets and infection takes place. The fungus or the pathogen enters through the succulent tissues of the stigma e.g. loose smut of Wheat (*Ustilago tritici*), Long smut, Ergot of Bajra (*Claviceps fusiformis*), Bunt of Rice (*Neovossia horrida*).

The penetrotion through anthers have been demonstrated in blossom blight of red clover caused by *Botrytis anthophila.*

II. INDIRECT PENETRATION

1. Entry through natural openings:

i) **Stomata:** This is a very common mode of entry for plant pathogens. The actual mechanism differs in different types of pathogens. The penetration of baterial pathogens takes place through action of absorption or suction and, therefore, passive in natue i.e. Xanthomonas *malvacearum.*

Many fungi utilise the stomatal opening for entry into their host plants. Late blight of Potato, Downy mildew of Grapes, white rust organisms and many leaf spotting fungi are common examples.

The uredospores of rusts infect primarily through the **stomata** but the basidiospores and the aeciospores make their entry directly through **cuticle.**

ii) **Lenticels:** (breathing pores): Entry through lenticels is similar to stomatal penetration and is usually common in diseases of vegetative parts such as stems, tubers and root crops, e.g. Guava canker. (*Pestalotia psidii*), Koleroga of Arecanut (*Phytophthora arecae*). Common scab (*Streptomyces scabis*), powdery scab (*Spongospora subterrenae*).

iii) **Hydathodes:** Hydathodes are water stomata i.e. water is exuded through them. Whenever they remain open the pathgens enter through them e.g. Black rot of Cabbage (*Xanthomonas campestris*).

2. Entry through artifical openings:

i) **Wounds:** This is a special mode of entry and is mainly utilised by wound parasites and weak parasites. Wounds or injuris caused by various agencies such as physical bruises resulting from animal, nematodes or storm, freezing injuries, sunburn.

Fungi like *Rhizobus, Penicillium, Diplodia, Botrytis Aspergillus, Gloeosporium* causing storage diseases and ripe rots have this mode of entry.

ii) **Stalk ends:** Storage and market fruits retaining organisms gain entry through stalk ends e.g. Blue mould of lemons, ripe rots of mangoes and banana, Black rot of Pineapple.

iii) **Insect injuries:** Entrance through insect injury is common. The classical example is Brown rot fungus (*Sclerotinia fructicola*), Injuries by bees or wasp which not only carry the fungus but deposit the conidia in the wounds caused by them as well as the birds. Insects also directly penetrate their stylets into host tissues and deposit the pahtogens deep inside.

The insect transmission of virus diseases is another classical example. The insect carrying and transmitting the virus is called **'Insect Veetor'.**

ENVIRONMENT IN RELATION TO DISEASE DEVELOPMENT

EIOPD

Predisposition : Is the action of a set of environmental factors prior to penetration and infection which makes the plant vulnerable (unguarded) to attack by the pathogen.

Environmental factors play an important role in the development of diseases in plants. Weather is one of the most important factors in the development of the disease because it directly affects the activities of the pathogen. The close relationship between diseases and weather is very obvious in many cases. Before it was accepted that diseases were caused by pathogens such as fungi and bacteria, it was believed that weather was the primary cause of disease. The spontaneous appearance of one or more pathogens in the lesions was regarded as the effect.

In order to have disease outbreak to reach epitphytotic proportions, three conditions or requirements must be met simultaneously *viz.*; (2) A soure of inoculum must susceptible plants must be growing in the area,

be present and (3) Weather conditions must be suitable for the development of the pathogen. If any one of the three conditions is not met, disease development cannot occur.

Many weather factors can control or affect the reaction of the pathogen and the host. **Temperature** and **moisture** are more important with air movement a close third. In addition, the frequency and amount of rainfall, dew, soil moisture and light also influence the pathogen development.

If weather conditions are favourable, the severtiy of disease out-break is also determined by number of other factors. Host susceptibility is affected by the crop types, degree of resistance, general health and vigour and the stage of development. Weather factors may interact e.g. the moisture may affect the temperature.

In general, no one weather factor is involved in determining disease development, but somewhat a combination of factors must interact to provide the necessary regime for disease development.

Besides, the normal definition of the disease as disturbances in the equilibrium of the host in respect of the structure, physiology or both and which may lead to the death of the plant or reduce the economic value of the product; the disease is defined as a complex phenomenon resulting from an interaction between the host, the pathogen and the environment. Environment is thus an important factor for development of disease. Disease assumes serious proportions only when the environment favours development of the causal organism. The diurnal and seasonal variations of weather in a particular locality may affect the composition of aerospores and this influence the incidence of disease by airborne pathogens. It is due to the environment that some diseases are prevalent in certain areas and their activity is restricted to certain parts of the year.

Environment comprises of three main factors :

I. **Meteorological** : 1. Temperature 2. Humidity 3. Precipitation 4. Topography 5. Dews 6. Light 7. Lightening and hail storms 8. Industrial by-products.

II. **Soil:** 1. Soil temperature, 2. Soil Moisture 3. Soil condition or texture 4. Soil-reaction 5. Deficiency or soil fertility.

III. **Biological factors:** 1. Insect fauna, 2. Soil flora.

I. Meteorological factors :

1. **Air temperature:** The influence of temperature on development of the disease need not be the same as that of the host or parasite. Conditions favourable to one may be unfavourable to the other. Pathogens differ in their optimum temperature for growth, sporulation and infection. Every organism has cardinal temperature for growth. The temperature determine the distribution of pathogenic fungi and the diseases caused by them.

Black stem rust of wheat (*Puccinia graminis tritici*) requires 21 to 26°C and hence is generally prevalent in south India. Leaf rust or Brown rust of

light, Humidity, dews, Topography

Wheat (*Puccinia graminis recondita*) requires 15 to 21°C and hence is prevalent in central India while, yellow or stripe rust (*P. graminis striformis*) requires 10 to 15°C i.e. cooler climate and hence is common in North India. For development of late blight of Potato in fields the cardinal temperature in Potato growing season is optimum.

Typically the tropical diseases require high optimum (25-27°C) while the temperate diseases 18-22 °C temperature.

Sr. No.	Disease	Minimum temp. (°C)	Optimum temp. (°C)	Maximum temp (°C)
1.	Late blight of Potato	12	22	27
2.	Early blight of Potato	12	25	35
3.	Downy mildew of Grape	12	22	30
4.	Powdery mildews	10	24	35
5.	Stem rust of wheat	15	25	33

2. Humidity : Humidity is one of the most important factors influencing the fungus growth. High humidity conditions help in germination and reproduction of fungi. Humidity may influence the rate of transpiration in plants. It also favors the stomatal opening. High humidity in combination with temperature has brought about epiphytotics. The koleroga disease is more severe in heavy rainfall area. Similarly heavy rainfall is favourable for bacterial blight, blast of paddy and angular leaf spot of cotton. Cloudy weather is conducive (helpful) to rusts where as bright light reduces the infection of rust but is necessary for condial production in powdery mildews. Some powdery mildews can thrive under less humid conditions.

3 & 4. Precipitation and Dews : They help in increasing the humidity and thus help in germination of spores and infection of diseases. These factors, in combination with the temperature, bring about epiphytotics of diseases like koleroga of arecanut, Downy mildew of Grapes, Blasts, *Alternaria blights*, rusts etc.

Karnal bunt of wheat caused by *Neovossia indica* develops best when rains are there at the time of flowering with low temperature (15-20°C). Coffee rust (*Hemilea vastatrics*) is influenced by rain and humidity.

5. Topography : The rusts of wheat are influenced by air temperature and ultimately the distribution depends upon the topography.

Sr. No.	Disease	Region	Air Temp. (°C)	Over Summers at
1.	Stem rust	South India	21-27	4,000 ft. above sea level
2.	Leaf rust or Brown rust	Central India	16-21	5,000 ft. above sea level
3.	Yellow rust	North India	10-18	7,000 ft. above sea level

6. **Light:** The influence of light, though important, has been difficult to separate from the influence of other climatic factors. Powdery mildew pathogens germinate better in light but ecologically they are more prevalent in shady localities.

7 & 8. **Lightening, Hail storms and Industrial fumes:** They are also responsible and of special significance in areas situated near towns and industries. Fumes like So2, HCL, Chlorine and fluorine etc. are highly toxic to the delicate foliage and blossoms. Lightening and hail-storm also cause crop injuries.

II **Soil factors :**

1. **Soil temperature :** In several soil or seed-borne diseases soil environment influences the incidence of the disease. Soil temperature acts directly on the host in some cases and also the pathogen in other cases and increases the disease development. This factor acts on the host, which is highly susceptible to the ever changing micro-climate of the soil. A general retardation in the growth of the host brings-in greater opportunities for the attack of the pathogens and higher incidence of the disease. Disease is, thus, a race between two opposing forces, the **host** and the **pathogen,** the one trying to overtake the other.

Low temperature at the time of wheat sowing is responsible for bunt of wheat as 10-15^{0}C is favourable for bunt-spore germination.

2. **Soil moisture :** Several damping-off fungi become severe in high soil-moisture. Since zoospores of *Phuytophthora* and *pythium* are more active in presence of abundant water because of their rapid dispersal. On the other hand, flag smut of Wheat and Scab of potato are favoured relatively at low soil moisture level. Flag smut in Punjab is more severe in moderate soil moisture level. High soil moisture has been found to favour the bacterial diseases : (Wet rot of potato) and the virus disease (Big vein of Lettuce). Panama wilt has been found to be severe at 25% soil moisture and decrease with increase in soil moisture content. Head smut of jowar is more severe at low soil moisture. Many non-parasitic diseases like blossom end-rot of tomato, corky spot of apple, mango and other fruits are variously influenced by soil conditions, with special reference to water relations.

3. **Soil condition or Texture :** Heavy soils retain more moisture while light soils retain less moisture. Nature of the soil affects the disease development. Heavy soils with poor drainage are more prone to cause root diseases.

4. **Soil reaction :** The growth of the organisms may be inhibited or prevented in too acidic or too alkaline media and so is the case in the soils. PH 5 to 6 (i.e. acidic) is most favourable for most of the fungi while bacteria and favoured in alkaline medium (above 7.5 PH). The best examples are the scab of potato and club root of cabbage. Alkaline soil favourus development of scab (*Streptomyces scabis*) while club-root of cabbage (*Plasmodiophora brassicae*) is favoured in acidic soil.

5. **Soil fertility or Deficiencies :** Generally excess of nitrogen causes the succulent growth, tissues become soft and pithy and makes the plants susceptible to the disease. While potash and phosphatic manures increase the resistance or immunity in plants against the pathogens. The effect of soil fertility is direct in case of fungi.

In soil, deficiency of some minor elements like Zn, Fe, Mn, Boron favours the development of deficiency diseases such as mottle leaf of citrus, chlorosis of sugarcane and khaira disease of paddy. Red leaf blight of cotton is belived to be due to deficiency of N, P and other minor elements like Mg.

III. Biological factors :

1. **Insect fauna:** Insect fauna and soil micro-flora are very important in many virus and other diseases. The insects are mainly responsible for carrying the virus infection and, therefore, the distribution and population of insects determines the severity of these diseases e.g. Aphids mostly carry mosaic viruses, Jassids transmit yellows types, white flies transmit curls; and thrips spread ring spots etc.

Some fungal and bacterial diseases are also transmitted by insects e.g. Citrus canker by leaf miner, Dutch elm disease by beetles, Mango malformation by mites etc.

2. **Soil flora:** Soil population comprises of fungi, bacteria, worms (Nematodes) which are either saprophytes or parasites. They influence the incidence of the diseases.

Some soil fungi act as antagonists (opponents) and do not allow the pathogen to grow. *Rhizobium, Azotobacter* fix the nitrogen and help in better stand of the crop.

Soils which are known to harbour the pathogenic organisms are known as **'Sick soils'**. The sick soils become unfit for cultivation of certain crops.

Short Note

DISSEMINATION OF PLANT PATHOGENS

Dissemination means spread. Many plant pathogens multiply with repidity and produce large number of spores in small space and within short time and have highly efficient methods of spore liberation. Some kinds of inoculum of the pathogens remain viable for a long time. The timely and wide spread dissemination of the pathogens is important in causing epiphytotics. Seasonal variations in dissemination may affect disease development in a single season only. Dissemination of inoculum may be local, discontinuous or rapid and continuous.

All parasitic diseases are transmissible i.e. they are infectious with an ability to spread. The transmission may be continuous or discontinuous. **Continuous** relates to the spread in a geographical unit from one part to

another part of the same host, host to host, field to field or locality to locality closely connected geographically. This ultimately covers long distances e.g. Black stem rust of wheat.

Discontinuous refers to long jump transmission and usually takes place between two geographical units separated by physical barriers such as oceans.

Dissemination may be effected through various agencies. Knowledge of dissemination of plant pathogens is essential to evolve suitable and effective control measures.

Agencies of Dissemination

I. Continuous dissemination: (Short distance dissemination) 1) Autonomous 2) Natural agencies like air or wind 3) Water 4) Animals 5) Birds 6) Insects 7) Nematodes 8) Mites 9) Agril. implements 10) Soil 11) Seed

Discontiuous dissemination(Long distance disseminatyion): **1) Man**

I. Continuous (Short Distance) dissemination:

1. **Autonomous** : The manner of spread of plant pathogen is restricted to short distance through Rhizomorphs and hyphal strands of fungus e.g. Wood rotting fungi and root rot fungi. (*Armillaria, Polyporus, Ganoderma, Fomes*). Some inoculum (the Zoospores and many bacteria) is motile in water and can swim ever short distances Sharma book P-31

2. **Anemochory or Wind**: This is the most common and efficient means of dissemination of air-borne diseases. The fungi have devices for easy discharge of spores and are disseminated by air or wind. The transfer of inoculum can be rapid.

Spores of Downy mildew, Powdery mildew, aeciospores, and basidiospores of rust fungi are unable to withstand long distances. Spores of blast and uredospores of rust are well adopted for long distance travel in viable condition and are responsible for epiphytotics. Resistant inoculum can withstand during long distance dissemination through air. Uredospores of *Puccinia graminis tritici* can spread north ward from Northern Mexico into Canada through the U.S.A. at least 3200 km in as little as 2 months. Similarly, the rust uredospores reach Maharashtra from Nilgiri Hills in Tamilnadu through air.

3. **Water:** Water serves an agency in dissemination in two ways: by runing water as a result of heavy showers and irrigation. Water Moulds, white rust and some Downy mildews produce motile spores and they migrate by the action of their flagella. All bacterial disease eg. angular leaf spot of cotton by Xanthomon

Flood or irrigation water carry many fungi from field to field or from one place to another e.g. *Colletortichum falcatum, Sclerotium oryzae, S. rolfsii, Fusarium, Rhizoctonia* and seeds of striga, orobanche, cuscuta etc.

The dropping of moisture from heavy dews, splashing-rain may transport spores from leaf or plant to adjustment plant e.g. *Phytophora* and downy mildew fungi;

Bacterial cells – X. malvacearum

4. **Animals:** The farm animals play some role in dissemination of plant pathogens which are mostly soil borne e.g. Sclerotia and rhizomorphs which may be carried by adhering to the legs of animals. The smut fungi can also be carried from field to field through alimentary canal of farm animals. Wood peckers carry spores of tree pathogens.

5. **Birds:** this mode of dissemination occurs in a few specific cases. Birds visit the diseased plant and get contaminated by spores. They transmit the spores to healthy plants e.g. Chestnut blight *(Endotheca parasitica)* and seeds of Loranthus. Birds are also known to carry Cleistothecia of Powdery mildew.

6. **Insects:** Insects plany a specific role in the transmission of virus and mycoplasma diseases of plants. Insects act as specific vectors. Depending on the virus, the insect vectora are leaf hoppers, aphids, white flies, thrips etc.

Insects also carry fungi externally or internally. Insects are attracted to sweet and honey secretions of fungi when they produce conidia, oidia and sepermatia. Spores of ergot disease and spermatia of *Puccina graminis* are transmitted externally by insects.

Insects also transmit bacterial disease e.g. *X. citri, Erwinia* etc.

7. **Nematodes:** Some nematodes transmit some viral, bacterial and fungal diseases. The *Xiphinema, Longidorus, Trichodorus* species of nematodes act as vectors. Nematode transmitted Polyhedral virus particles **(NEPO)** are tobacco ring spot, Tomato ring spot, Arabic mosaic, Cherry leaf roll, etc. While Nematode Transmitted Viruses with tubular particles **(NETU)** are Tobacco rattle, Pea early browning.

The NETU viruses are transmitted by the species of *Trycodorus*. Similarly, in bacterial diseases (Ear-rot of wheat) Yellow ear-rot (Tundu) of wheat, the bacterium *Corynebacterium tritici* is transmitted through the ear cockle nematodes (*Anguina tritica*)

8. **Seed:** The seed and vegetative propagating parts may carry internally the fungi (Mycelium and spores) or spores mixed with seed. Some fungi are carried on external surface of the seed. e.g. *Sphacelothea sorghi; Ustilago tritici, Colletotrichum lindemuthianum, Colletotrichum falcatum,*

Phytophthora infestans, *Claviceps purpurea*, *Claviceps fusiformis*, and seeds of Cuscuta in Lucerne seeds.

9. **Agricultural operations:** Plant pathogens may be transmitted by operations like ploughing, transplanting, watering, prunning, cutting etc. e.g. diseases of potato, Tomato, Sugarcane.

10. **Soil, Compost and manure:** By transport of these materials, fungi may be introduced into new area. Infected soils may reach other fields in various ways as on feet of farm workers, or on agricultural implemtns.

11. **Man:** man is an important agent in dissemination of plant pathogens. Due to export and import of seed, ornamental plants, tubers, fruits, cuttings, setts the spred of plant diseases are brought about e.g. late blight of Potato, Citrus Canker.

Transmission of Plant Pathology through man

Plant pathogen(s)	Transmitted		Year
	From	To	
P.M. Group	U.S.A.	Europe	1845
D. M. Group	U.S.A.	France	1878
Late blight Pathogen	U.S.A.	Europe	1845
Late blight Pathogen	Europe	India	1870
Synchytrium endobioticum	Europe	U.S.A.	1918
Piricularia oryzae (Blast of paddy)	South-East Asia	Madras (India)	1918
Panama disease of banana	Panama Canal	Bombay State (India)	1920
Xanthomonas citri (Citus canker)	Asia	U.S.A	1907
Bunchy top of Banana (Virus)	Ceylon (Sri Lanka)	India	
Ergot of Bajra	Africa	Bombay State (India)	1957

Many diseases caused by fungi, bacteria, Nematodes, viruses flowering plant parasities have been introduced in new regions through the gency of Man e.g. Chestnut blight, citrus canker, pine rust, Dutch elm disease, powdery mildew of grapes, maize rust.

EPIDEMIOLOGY

Epidemiology deals with outbreaks and spread of disease in a population. Epidemiology or epiphytology of plant diseases is essentially a study of the rate of multiplication of a pathogen which determines its capacity to spread a disease in a plant population.

Compound Interest Diseases

In such diseases the rate of increase is mathematically analogous to compound interest in money. The pathogen produces spores at a very rapid rate. These spores are disseminated by rapid means such as air. They infect other plants. The incubation period and sporulation period (period between inoculation and sporulation) is short. New crop of spores is produced, disseminated and the cycle is repeated. There are, thus, several or many generations of the pathogen in the life of the crop. Since the susceptible host area is fixed, by the acreage under the crop, amount of uninfected tissues continuously declines as the spread of the disease continues so that the rate of spread in terms of new infections also decreases. Pathogens spreading by means of air disseminated propagules, such as rusts of cereals and late blight of potato, show this type of spread. However, the spread is continuous only if the environment remains continuously favourable for spread, infection and disease development.

As an example of compound interest diseases the nature of spread of stem rust of wheat (*puccinia graminis tritici*) can be considered. In India, this rust does not have a local source of primary inoculum. It survives on alternate or collateral hosts in the form of active pustules of uredospores at remote places. These spores are brought by wind to the main crop. In the beginning only few pustules of rust develop in the crop. Each pustule is capable of producing roughly 50 to 400 thousand of uredospores which are disseminated by wind to other plants. Theoretically, the same number of the infections can be caused in the crop. Each new infection is capable of developing into a spore bearing pustule within 5-7 days at a temperature of 24°C. Thus, within a week of appearance of the first pustule in the crop several thousand new pustules are formed which could repeat the process within a week. If the weather conditions remain favourable for only few weeks the entire crop is severely affected by the disease.

Simple Interest Diseases

In these diseases the increase is mathematically analogous to simple interest in money. There is only one generation of the pathogen in the life of the affected crop. The primary inoculum is seed- or soil-borne and secondary infection rarely occurs during the season. All late infections noticed in the field are from the pre-existing inoculum in the soil. The simple interest diseases are exemplified by such soil borne fungi as causing wilt and root rots and seed- borne smuts such as loose smut of wheat, covered smut of barley, sorghum, etc. In the wilt disease there may be very slow secondary spread if the pathogen can sporulate on the host surface and other plants are in a position to be attacked. Normally it does not happen since the fungal propagules have to pass through the barrier of soil to reach the roots and most propagules are lost during this passage. In smuts, there is infection at seeding stage but the pathogen mostly sporulates in the inflorescence. These diseases are mostly systemic in nature, do not produce propagules external to the host during the active season, and the dispersal of propagules is restricted by climatic and biotic conditions.

susceptible host

Pathogen (aggressive)

Environmental cond. favourable

Essential Conditions for an Epiphytotic

For establishment of an epiphytotic the following conditions are necessary. These conditions are concerned with the host, the pathogen and the environment. (Host factor)

i) **Distance of susceptible plants from the source of primary inoculum:** The disease in an area is initiated by the primary inoculum surviving at some source. Longer the distance from the source of survival of the pathogen longer will be the time required for build up of epiphytotic in a susceptible crop. During dispersal in different direction the density of primary inoculum is diluted and as the distance increases fewer propagules are likely to reach the susceptible surface. This can occur in the same field, among different fields and in a larger area of the country.

ii) **Abundance and distribution of suceptible hosts:** Continuous cultivation of a susceptible variety in a given area, large areas under a similar susceptible variety, and distribution of the variety over large contiguous areas help in build up of inoculum and improve the chances of epiphytotics. Under these conditions the pathogen is able to use maximum number of its propagules effectively, increase the rate of multiplication many times and repeat the disease cycle quickly.

iii) **Disease proneness in the host due to environments :** Susceptibility is genetically controlled but proneness in the plant to get infected can be induced by environments and other factors. When such conditions exist the host is liable to more vigorous attack and successful infection by the pathogen. A susceptible variety becomes more susceptible and even a moderately resistant variety may tend towards susceptibility when conditions favouring proneness are prevalent. Under these conditions the pathogen has better chances of multiplying, causing infection and effectively use its propagules for secondary spread.

iv) **Presence of suitable alternate or collateral hosts:** This is required only for those pathogens which survive and multiply on wild hosts during absence of the cultivated host. For pathogens spreading through heterogenous infection chain presence of an alternate host is necessary for providing primary inoculum. The amount of inoculum thus available will determine the intensity of primary infection and subsequent spread. Presence of collateral hosts plays the same role for pathogens of homogenous or continuous infection chain. Grass hosts of *sclerospora sacchari*, *Sclerospora philippinensis* (sugarcane smut), *Pyricularia oryzae* (rice blast) may produce abundance of inoculum aiding in build up of epiphytotics of these diseases.

v) **Presence of aggressive isolate of the pathogen:** Only infectious diseases can take the form of epiphytotics. For any epiphytotic rapid cycle of infection is essential and successful infection can be caused only by aggressive isolates of the pathogen.

Alternate host belonging to diff. family

eg. Rust of wheat | Puccinia graminis tritici

main host - wheat

Alternate host

vi) **High birth rate of the pathogen:** Among animate causes of plant diseases the high birth rate of the pathogen is an important contributory factor for epiphytotics. The fungi that assume epiphytotic form invariably have the capacity to produce enormous quantity of spores that are adapted to quick and long distance dispersal in a short time so that they can take advantage of favourable weather conditions during that short period. These spores are asexually formed usually on the exposed surface of the host for dispersal by wind, water and insects.

vii) **Low death rate:** Among fungi the variations in circumstances of dispersal by wind, minute size of unprotected spores, possible chances of falling on wrong hosts, etc. are many factors that cause high death rate among the propagules. However, this weakness is offset by extremely high birth rate. Epiphytotics attributed to low death rate of pathogens are those in which the causal agent is systemic and protected by the plant tissues. Thus, the chances of high mortality are reduced to the minimum. The chief source for accumulation of inoculm for vegetative propagation and, therefore, the build up of epidemics is comparatively slow. When a particular area becomes saturated with diseased planting material chances of occurrence of epiphytotics are very high.

viii) **Easy and rapid dispersal of the pathogen:** The ability of a pathogen to cause epiphytotics is dependent as much on its dispersal as on high birth rate. The units of propagation produced by the pathogen are dispersed by external agencies which must be available if epiphytotics are to develop. Fungal spores are mostly disseminated by insects or, for becteria, by water. The velocity of the wind, its direction, moisture, number of suitable insect vectors, their rate of reproduction, feeding habits, etc. determine the degree of epidemics. The fungi have special mechanisms for making their spores wind-borne. Usually the spore discharge is with sufficient force to throw them in upper air currents. In addition these spores are light, minute and resistant to adverse conditions encountered in turbulent air.

ix) **Adaptability of the pathogen:** Weeds seldom die because they have the capacity to adapt to adverse conditions. Most of the pathogens causing epiphytotics can be placed in the same category. They can adapt themselves to various conditions listed above. However, exceptions can exist to these requirements. The necessity of adaptability can be substituted with other qualities such as high birth rate.

x) **Optimum weather:** Meteoropathology deals with the relationship between weather and epiphytotics. Optimum moisture, temperature, light, etc. are necessary for activity of animate pathogens. They are as much important as the nutrition for the pathogen. Assuming that a particular fungus meets all the above requirements for causing epidemic; it has high birth rate, high

aggressiveness, produces abundance of wind-borne spores, and the spores fall on susceptible hosts that are prone to infection; even then infection, invasion and development of epidemic may not occur if weather is not favourable for germination of spores or in absence of light stomata have not opened to permit entry of the infection tube or when the stomata open the moisture is so low that the germ tube has dried. The weather also affects the activity of the pathogen on the host surface. It may not permit sporulation on the host surface thus reducing amount of inoculum for secondary spread. The spores may be washed from leaf surface by rains before penetration has occurred.

Decline of the Epidemics

No epidemic remains for ever in a population. After development of the epidemic a stage is reached when it shows decline by itself. This stage is very common in epiphytotics of crop plants. The causes of decline is epiphytotics are as fllows:

i) **The saturation of the pathogen in the host population:** In a particular area when a disease assumes epidemic form, majority of the plants contact infection. The non-availability of more host plants limits further spread of the pathogen. This results in production of less inoculum, fewer secondary infections, and finally no new infections. The plants that escape infection are those that possessed resistance or in which resistance developed during the epidemic. It is possible that in future only these plants will be grown in that area. Thus, one of the advantages of an epidemic is that it eliminates susceptible individuals and permits only the resistant individuals of the population to survive and breed.

ii) **Decline of proneness in the host:** Most diseases attack the plant at a particular stage of its growth. When the plant has crossed this stage its proneness for contacting infection is reduced or completely lost. Under these conditions the epidemic will automatically decline. When the plant is receptive for infection throughout its life and its population has been infection throughout its life and its population has been affected by an epidemic, the weather conditions may not remain always favourable for disease development. As a result further spread of the disease will be checked and the epidemic will decline. Wheat crop in northern India usually gets the attack of rusts in January to March. Epidemics develop during these months. Although the plant remains prone to attack afterwards also, further development of the disease is checked because of rise in temperature which is unfavourable for the pathogen.

(ii) **Reduction in aggressiveness of the pathogen:** Due to above mentioned and other causes the aggressiveness of the pathogen may be reduced. When all susceptible individuals are destroyed by the pathogen, it may try to parasitise the remaining resistant

individuals of the same species. In these adverse conditions it may lose its power of successful infection, its reproduction may slow down, and thus it may not remain as aggressive as when the conditions were favourable.

PLANT DISEASE FORECASTING

Forecasting involves all the activities in ascertaining and notifying the farmers in a community that conditions are sufficiently favourable for certain diseases, that application of control measures will result in economic gain, or that the amount of disease expected is unlikely to be enough to justify the expenditure of time, energy and money for control.

This requires complete knowledge of epidemiology, that is, development of the disease in plant population under the influence of the factors associated with the host, the pathogen and weather. Forecasting is actually *applied epidemiology.*

Practical Advantages of Forecasting

Forecasting of disease contributes to prediction of crop yields. Although it is not the sole factor, without consideration of possible occurrence of disease no forecasts of crop yields can be true. The disease forecasting has two facets. It can be short term forecasting, during the crop season or just before the crop season, or it can be a long term forecasting, predictions of disease being made years in advance. Both are possible if sufficient data on weather, variety and response of disease cycle of the pathogen to these parameters, are available. Possibility of occurrence of diseases can be detected.

Thus, by detecting per cent infected or contaminated seeds in random samples and the extent of distribution of such seeds one can give a rough estimate of possible extent to which primary infection of a disease can occur in an area.

Primary inoculum of virus diseases of potato, loose smut of wheat, ergot of rye or bajra and ear cockle of wheat can be detected in seed lot by different methods and necessary warnings can be given to the growers against use of such seeds without proper precautions. By collecting data for several years on weather conditions during the crop season and pattern of the disease incidence, supported by laboratory studies, correlation between the two (weather and disease) can be determined and on that basis forecasts can be made whenever meteorological conditions tend to become favourable for serious disease incidence.

Some Examples of Disease Forecasting

Due to its devastating effects and its effective control with fungicides late blight of potato (*Phytophthora infestans*) is one of the diseases on which forecasting has been tried since long in Britain, Europe and the U.S.A. The complex relationship between weather and blight can be seen from the

following characters of the pathogen and this has been utilised in developing forecasting systems for the disease. Sporangia are formed at a relative humidity of nearly 100 per cent, or at least more than 90 per cent, at 18°-20°C for at least 6 hours or at 12°-15°C for at least 12 hours. Sporangia lose their viability in 1-2 hours at 20-40 per cent relative himidity (RH) or in 3-6 hours at 50-80 per cent RH. Thus, the favourable conditions for their germination must occur soon after their formation. These conditions are the presence of moisture and a fairly low temperature (10°-15°C) for 0.5 to 2 hours. Infection of the plant by zoospores liberated from sporangia requires at least 2-2.5 hours at 10°-25°C and the fungus there after develops most rapidly at 18°-21°C. The incubation period in 3-5 days under favourable conditions but must be longer at high temperatures.

The first successful warning system was developed in Holland where the four **Dutch** rules were formulated more than 50 years ago. The appearance of blight depends on 1) a night temperature below dew point for at least 4 hours (that is dew at least for this time):2) a minimum temperature of 10°C or above; 3) a mean cloudiness on the next day of at least 0.8; and 4) at least 0.1mm of rain during the next 24 hours. These rules worked well in Holland but when tried in South West they failed to be accurate. There, the rules were reduced to two, viz. a minimum temperature of 10°c and a relative humidity not falling below 75 per cent for at least 2 days. Seasonal and regional variations occur in inoculum density and weather and hence these rules may not be applicable everywhere.

In the United states two systems of forecasting were developed by **Hyre (1954)** and **Wallin (1962)**. The Hyre's system is based on records of daily rainfall and maximum and minimum temperatures. The initial appearance of late blight is forecast 7-14 days after the first occurrence of ten consecutive blight favourable days. A day is blight-favourable when the 5-day average temperature is below 25.5°c and the total rainfall for the last 10-days period is more than 3.5 cm. Days on which the minimum temperature falls below 7.2°C are considered unfavourable for blight development. This system does not require that data be collected in the crop canopy.

In Wallins system blight forecast is based on relative humidity (RH) and temperature. It takes into consideration the seasonal accumulation of "severity values". Severity values are numbers arbitrarily assigned to specific relationships between duration of RH periods of more than 90 per cent and the average temperature during those periods. The first occurrence of late blight is predicted 7-14 days after 18-20 severity values have been accumulated from the time of plant emergence. In this system data are required from within the crop canopy.

Although both systems in the U.S.A. were used for more than 18 years, they were not widely accepted and utilised by farmers because the systems were not readily available on a timely, regular and localised basis, this has led to the development of a computer programme which combines both these systems and provides the growers with a late blight control programme tailored to their local conditions.

Forecasting has been applied in case of many other diseases on the basis of (i) weather conditions during the intercrop period, (ii) weather during the crop season, (iii) amount of disease in the young crop, that is, the initial inoculum level, and (iv) amount of inoculum in air, soil, or planting material.

Prediction of downy mildew of grape (*Plasmopara viticola*) is made on the basis of weather favourable for germination of the oospores and infection of the plant. *Phytophthora phaseoli* is predicted on the basis of rainfall–temperature. A day is considered favourable for the disease when the 5 day mean moving temperature (recorded graphically) is less than 26°C with the minimum 7°C or above, and the 10 day total rainfall is 3.05 cm or more. The disease is likely to appear after about 8 consecutive favourable days. The spread of tikka or leaf spots of groundnut (*Cercosporidium personatum* and *Cercospora arachidicola*) in Georgia (U.S.A.) is favoured by diurnal periods of 10 hours or longer with RH at or above 95 per cent and with temperatures above 21°C during these periods.

Although not much work has been done on disease forecasting in India, except for finding out correlation between temperature-moisture conditions and fungal activity, there is good amount of data for forecasting blast of rice (*Pyricularia oryzae*). Forecasting can be made on the basis of minimum night temperature range of 20-26° C in association with a high relative humidity range of 90 per recent and above lasting for a period of a week or more during any of the three susceptible phases of crop growth, *viz.*, seedling stage, post-transplanting tillering stage, and neck emergence stage. Another approach is to plant highly susceptible variety in the locality and watch for occurrence of the disease. If the disease appears in this indicator crop and above weather conditions have been prevalent, a forecast of coming epidemic can be made.

The timings for spraying the crop with fungicides or antibiotics for direct control of the disease could be fixed on the basis of data for forecasting. In absence of such forecasting system the farmer has no other alternative but to start control operations at the approximate time of appearance of the disease and watch further developments. If disease appears and spreads he has to repeat the operations at short intervals. If accurate forecasts are available the farmer may not even have to start control operations or reduce the number of sprayings, thereby, saving money.

Computerised System of Disease Forecasting

The late blight development depends more on the crop climate than on the climate outside the crop. Thus, among the various systems mentioned earlier for late blight the Wallin's system seemed to be more reliable although other systems also had their local merit. A situation, encountered in the tarai area of north-west, U.P., exemplifies the role of climate in the crop canopy. In many fields the blight was present only in traces on the surface of the crop when inspected from outside. But when tubers were harvested, 50-60 per cent of them started rotting within a day or two before they could be transported to cold storage. A study of the problem revealed that actually blight was not in traces but in severe form, only it was not observable from

the top of the crop. The tubers were severely affected by late blight fungus and this pre-disposed them to soft rot bacteria. What actually happened was that the crops had luxuriant growth and very thick crop canopy due to ample irrigation and uses of fertilisers. This prevented examination of the under growth during inspection. Since the weather outside was not conductive to blight epidemic, no attention was paid to the examination of undergrowth. However, the thick crop canopy created ideal conditions for epidemic which existed around the lower leaves but not on the top leaves exposed to outer climate. The disease continued and zoospores caused infection of tubers.

The example brings to light following possibilities:

1. Crop canopy of a luxuriant growth in a particular area makes the crop climate favouable for epidemic and independent of macroclimate.
2. The meteorological data outside the field. (as used in Hyre's system and Dutch rules) may be deceptive if recorded as unfavourable for blight and data must be taken from within the canopy and in between the rows (as suggested in Wallin's system)
3. Since crop canopy resulting from luxuriant growth will depend on irrigation and ferilisers used in the cropping system, and these, in turn, will vary from field to field and farmer to farmer, the crop climate may vary accordingly among the fields.
4. The above possibilities imply that epidemics will vary from field to field wihin the same locality and, therefore, a generalised forecast for the entire locality may not give benefit to all categories of farmers in the locality.

Many of these questions have been answered by a system using computer and known as **Blitecast** in the U.S.A.

The Blitecast is a computer programme integrating both Hyre's system and Wallins system of forecasting based on rainfall, temperature and relative humidity. The system is a service relaying information on forecast from a central office to those who seek such forecasts. The data for the forecast are acquired on the following.

1. Maximum and minimum temperature for the day.
2. Number of hours of relative humidity more than 90
3. Maximum and minimum temperature during the period when the relative humidity was more than 90 per cent.
4. The rainfall reorded at 24-hour basis and measured to the nearest 0.1 cm.

The temperature and humidity data are recorded with a sheltered hygrothermograph located between the rows and within the crop canopy. Data recording must start early in the season when emerging plant foliage starts giving the look of green rows. In the early stages, there will not be much diference in data from within the crop and outside.

When, an American farmer desires a **Blitecast**, he telephones the **Blitecast** station giving most recently recorded environmental data. The

Blitecast operator feeds the data into a computer programmed to use the data which analyses them within a fraction of a second and returns the forecast and spray recommendations to the operator who relays the same to the farmer on the phone. The operation takes only about 3 minuets. The computer programming for Blitecast integrating the two systems mentioned above determines approximate severity of disease and accordingly determines whether spraying is needed or not and if needed what should be interval, i.e., 5 days or 7 days.

The programme has two parts, one determining the possible occurrence of the disease which it can do even with data from outside the crop canopy and the other determines severity and recommendation for spraying.

More expensive equipments based on the principle of Blitecast are being developed. These are self-contained units, programmed with parameters determining epidemics of late blight, and placed in the field where they continuously monitor the weather and as soon as a critical period has arrived, flash the warning which can be interpreted with the help of direction charts.

VARIABILITY IN PLANT PATHOGENS

No plant population can remain genetically pure for a long time. Genetically different individuals may develop due to self-pollination and mutation, etc. These self generated variations can make the plant resistant or susceptible. Similar causes induce variations in plant pathogens also. On the same host numerous spores formed by same parents may possess different characters including aggressiveness and adaptability for the host. Viruses, bacteria, and fungi undergo genetical changes by one or more methods,. Viruses and bacteria are not known to reproduce sexually.

Genetics and Variability of Viruses

Viruses are not considered micro-organisms. However, research in molecular biology that the molecular structure of viruses has the capacity of transferring its characters to its duplicates produced in the host cell. In other words, the principle of breeding true or like tends to beget like, applies to viruses as much as to living organisms. The laws of heredity apply to viruses and therefore they can also exhibit variability. When a small quantity of virus particles is inoculated in a suitable host the concentration of particles increases and the synthesized particles have the same harmful effects on the host which were caused by the original particles. However, in some viruses, in addition to immutable particles, virus particles having characters different from original can be synthesized. These are known as virus mutants. In this way the virus can produce several races, strains or variants. In Tobacco mosaic virus (TMV), Cucuber mosaic virus (CMV), sugarcane mosaic virus (SMV) and curly top of sugar beet several such strains are known to occur.

Hybridization can be one of the methods by which these new virus strains are formed. If two strains of a virus are inoculated into the same host

plant one or more new virus strains may be recovered with properties (aggressiveness, symptoms, etc.) different from either of the two strains originally used for inoculation. These new strains are probably hybrids and develop by recombination of the genetic material (RNA or DNA). The fact that virus particles are made up of mostly RNA or sometimes DNA molecules (the genetic materials in viruses) suggests that as in living organisms these materials from different sources may undergo recombinations and also determine the hereditary characters of the virus. However, the evolution of new strains in the above mentioned example may also be due to mutation.

The antigen properties of different strains of the same virus are identical. Antigens are proteins. Synthesis of proteins and enzymes takes place through line up of amino acids in specific sequence dictated by genes (in virus, nucleotide triplet in RNA). Therefore, if antigens are similar among different strains it proves that genetically they are closely related. This proof of affinity among virus strains is obtained by serological tests. In suitable medium each antigen produces specific antibodies.

Genetics and Variability of Bacteria

Strains differing in pathogenicity, colony characters, etc. occur in bacterial plant pathogens. For example, in the cotton blight bacterium, ***Xanthomonas malvacearum***, 17 strains or races are known to-date. Phenotypic variability is common among races of bacteria. These races can evolve by mutation or gene recombination. It has been now accepted that in bacteria the inheritance of genetic characters is on the same principle as in other organisms. The genetic material in absence of an organized nucleus in bacterial cell, is diffused throughout the cell and thus the entire cell functions as a nucleus. The transfer of this genetic material or characters coded in this material to daughter cells occurs at the time of binary fission.

The genetic variability among bacteria may be caused by following methods resembling sexual reproduction :

i) ***Conjugation :*** Two compatible bacterial cells come in contact and interchange of genetic material takes place between the two. In this way the genetic make-up of both cells is altered. Binary fission of these cells produces daughter cells of different genetic characters and these develop into new races by asexual reproduction.

ii) ***Transformation :*** Bacterial cells absorb the genetic material exuded by a compatible cell or freed by dissolution of the cell wall. The receiving cell then contains altered genetic material since new genes are added to it. This cell now reproduces to develop a new race.

iii) ***Transduction :*** The bacterial viruses (bacteriophage or phage) can transfer the genetic material from one cell to other attacked by them. If the attacked cell is not destroyed due to infection by the phage it reproduces to form new races having a different genetic character.

Genetics and Variability of Fungi :

The knowledge of variability in fungi is not only important for understanding pathogenesis, present or future, it is equally important for the success of any breeding programme for development of varieties resistant to the disease.

Fungi are uni-or multicellular micro-organisms. Like in other living organisms, the nucleus with its genetic material helps in determining the heredity of fungi. The fungi reproduce sexually as well as asexually. Some fungi have only asexual reproduction. Whatever the method of reproduction, due to presence of genetic materials the characters of the fungus are transferred from generation to generation. As mentioned for viruses and bacteria, mutation and hybridisation and, to some extent, other methods induce variability in genetic characters of fungal species and thus help in development of new strains or races. Although in laboratory two compatible fungi can easily be mated, in nature hybridisation and mutation are dependent on chance. However, enough chances are provided to them by nature to evolve new races as fast as, if not faster than, the varieties of higher plants evolved by man.

When the progeny shows variations in characters from the parents it is called a **variant**. The dissimilarities of characters in these progenies are hereditary. Progeny developed by a variant having similar heredity is called a **biotype**. Biotypes differ from the parent race only in few minor characters, mostly in the type of symptoms produced on specific host. A group of biotypes with identical characters forms a **race** or **strain**. In plant pathogens these races and biotypes are distinguished with the help of certain differential hosts. When a group of races of identical morphology attack and infect a number of different species of the host, the group is called **forma specialis** of variety. Several varieties of the fungus constitute a species (botanical species). All these variations in species at different levels are the results of genetic mutation or recombination.

Further explanation of variability is given below with few examples of pathogenic fungi which have been studied in detail.

The cereal rust fungus: Two categories of variants are recognized in *Puccinia graminis*-varieties and physiologic forms. The varieties differ from each other somewhat in shape and size of uredospores but the principal difference between them is their preference for groups of hosts in different members of graminae. Each variety can attack several species of one or more genera of the grass family, but it cannot attack the members of other genera which may be susceptible to other varieties of the same species (*P. graminis*). A variety, in turn, may contain several species of genus. Varieties are given **Latin names** while physiologic forms (races or strains) are designated by **Arabic numerals**. Thus, *Puccinia graminis tritici* race 15B means biotype B or race 15 of the variety *tritici* of the botanical species *P. graminis*. There are more than 200 such physiologic forms or races of *P. graminis tritici*. The

botanical species *P. graminis* consists of six varieties each having a large number of physiologic races.

From the above example it is clear that within the botanical species. *P. graminis* there was sequential variation leading to development of such biological units which gradually lost morphological differences and attained physiological difference such as choice of specific food or host. This type of variation from the original species is known as **physiologic specialisation** which has been defined as 'presence of entities within morphologic species, not readily distinguishable by structure, but differing from each other physiologically' including pahogenicity, host morphology, biochemical properties, cultural variability, spore germination and ecological relationships. These entities have been called **physiologic races** or **strains.**

The smut and other fungi: As in rusts, variability is quite common in smut fungi. As a matter of fact the smut fungi are more variable than any other group of plant pathogenic fungi. Variation abounds in the smut fungi, in both morphology and physiology. Morphologically, the variants within a species differ in spore markings, cultural characters and gross symptom expression on the host. Physiologically they differ in nutritional requirements, spore germination, compatibility relationships, nuclear behaviour and pathogenicity. Similar physiologic specialisation is known to occur in fungi causing late blight of potato, powdery mildew of cereals, wilt of pea, tomato, etc.

PLANT DISEASE MANAGEMENT

Measures taken to prevent incidence of a disease, reduce the amount of inoculum that initiates and spreads the disease, and finally minimise the loss caused by the disease have traditionally been called as control measures. During the past 10 years or so, however, there has been a general tendency to consider these measures under a system of disease management or pathogen management in place of using the term control. This approach has been followed by entomologist since long.

There is convincing logical basis to support the subsitution of the word control" with the word "management". As Apple (1977) has suggested the word control evokes the notion of finality, of having controlled or permanently settled a problem. But in reality this is not so. No plant disease has been permanently controlled. We have to repeat the operations in each crop season to ward off the disease. "Management" conveys the concept of a continuous process. It implies that diseases are inherent component of an agroecosytem that must be dealt with continuous knowledgeable basis. Management is based not on the principle of only eradication of the pathogen but mainly on the principle of maintaining the damage or loss below an economic injury level or at least minimizing occurrence of disease above that level. Management suggests need for continuous adjustments in crop cultivation systems.

General Principles of Disease Management

Plant disease management is based on following general principles:

1) **Avoidance of the pathogen:** Avoiding disease by planting at times when, or in areas where, inoculum is ineffective due to environmental conditions, or is rare or absent.

2) **Exclusion of inoculum:** Preventing the inoculum from entering or establishing in the field or area where it does not exist.

3) **Eradication:** Reducing, inactivating, eliminating, or destroying inoculum at the source, either from a region or from an individual plant in which it is already established.

4) **Protection:** Preventing infection by creating a chemical toxic barrier between the plant surface and the pathogen.

5) **Disease resistance:** Preventing infection or reducing effect of infection by managing the host through improvement of resistance in it by genetic manipulation or by chemotherapy.

6) **Therapy:** Reducing severity of disease in an infected individual.

The first five of these principles are mainly preventing (prophylactic) and constitute the major procedures for plant disease management. They are applied for the population of plants, i.e., the crop. The last, therapy, is a curative procedure and is applied to individuals. These procedures cross across the various approaches to disease management, viz., management of the environment, management of the associated mircobiota, managing host genes and management with chemicals.

1) Avoidance of the Pathogen:

Many diseases can be prevented by proper selection or change of land and alteration in time of sowing. The aim of these measures is to enable the host avoid contact with the pathogen or the susceptible stage of the plant and favourable conditions for the pathogen should not coincide. The main procedures under this group are:

1.i) Choice of geographic area: Selection of geographic area for any crop is made on the basis of suitability of prevailing temperature and humidity in the area for the crop. These factors influence incidence of the diseases also. Many fungal and bacterial diseases are more severe in wet areas than in dry areas. Crops susceptible to these diseases if grown in such areas are likely to fail due to serious disease incidence. If these crops are grown in dry areas with the help of irrigation disease incidence can be avoided. Smut and ergot diseases of bajra are serious in wet areas in regions where rains occur for long durations during flowering stage of the crop. Cultivation of bajra in wet areas is, therefore, not very profitable.

1.ii) Selection of field: Successful cultivation of a crop depends to a great extent on selection of proper field. Many soil-borne diseases can be avoided by proper selection of site. If the causal organism of soil-borne

disease of a crop is present in a field it is always advisable to avoid that field for the particular crop for some years. Presence of the pathogen in soil can be ascertained on the basis of previous history of the field and nature of the pathogen. Red rot fungus (*Colletotrichum falcatum*) can persist in soil for few months. Therefore, if sugarcane is planted in the same field immediately after harvest of the preceding diseased crop, chances of aggravation of the disease exist and the crop may fail. Similarly, bacterial wilt of potato, wilt of arhar, smut of bajra, ergot of bajra, ear cockle of wheat, root knot, etc. are such diseases whose causal organisms persist in soil for varying periods. Such fields should not be selected for susceptible crops.

In selection of field the management of drainage occupies a major place. Many diseases, such as red rot of sugarcane and downy mildew of bajra, occur more severely in fields where waterlogging is common.

Also in fruit orchards the selection of site is of special importance. The fruit trees remain standing on the same land for 40-50 years. If proper selection of land is not made at the time of planting the orchard, the trees show signs of many abnormalities after few years, especially when their root system grows deep in the soil. Apple orchards planted on land previously under oak forests usually show serious incidence of collar rot (*Rosellinia* sp.) when hard pan is present under the subsoil it prevents deeper growth of roots and trees suffer from malnutrition.

1.iii) **Choice of time of sowing:** In many diseases the incidence is most severe when the susceptible stage of plant growth and favourable conditions for the pathogen coincide. While choosing the time of sowing it should be taken into consideration that susceptible stage of plant growth and soil conditions and other environments favourable for maximum activity of the pathogen do not fall at the same time. Alteration in date of sowing can help in avoidance of favourable conditions for the pathogen. 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 (November-December) these diseases are also reduced. In areas where these diseases are serious late sowing helps in saving the crop. The stem rust of wheat damages the late sown crop more than the early sown crop because time of onset of disease and ear formation coincide. In areas where stem rust is a problem delay in sowing of the wheat crop should be avoided.

1.iv) **Disease escaping varieties :** In different crops certain varieties escape damage by diseases because of their growth characters. The disease escaping quality is not due to their genetic resistance but because of characteristics of growth and time of maturity. For example, varieties of pea which mature early (by January) usually escape much damage form powdery mildew and rust. These diseases normally become serious in January or later. If pods have developed before serious disease incidence the losses are considerably reduced.

ability in soil such as wilt-causing species of *Fusarium*. It is not so effective against diseases caused by pathogen having high degree of competitive survival ability and can persist in absence of host for long period.

3.iii) **Removal and destruction of diseased plants or plant organs:** The presence of diseased plants in the field or orchard is a source of continuous release of inoculum. Therefore, as far as practicable, such plants or their affected organs should be removed and destroyed to reduce the amount of inoculum. On the same basis, eradication of alternate and collateral host is also recommended.

3.iii.a) **Roguing:** The procedure known as roguing involves removal of diseased plants or their affected organs from the field. It checks spread of the disease to healthy plants and helps in production of disease-free seed. In orchards, where removal of the entire tree is not feasible unless it is very badly damaged, the affected organs can be cut (tree-surgery) and burnt. Roguing is employed in such diseases as loose smut of wheat, loose and covered smuts of barley, sorghum, maize, etc., red rot of sugarcane, wilt of arhar, etc. the procedure is practical only when size of the plots is small and number of diseased plants is not very high.

3.iii.b) **Eradication of alternate and collateral hosts:** Many diseases especially those with continuous infection chain, persist through alternate or collateral hosts of the pathogen. The primary inoculum is produced on and dispersed from these hosts. If these wild or uneconomic hosts of the pathogens are destroyed the source of primary inoculum is eliminated and chances of initiation of the disease in the crop host are reduced. The mosaic of cucurbits persists on wild cucurbit plants throughout the year which serve as a continuous source of inoculum of the virus. Insect vectors thansmit the virus from these hosts to the cultivated crop. In certain areas of the world cereal rusts can survive on their alternate hosts. By destruction of such hosts the life-cycle of pathogens or the infection chain is broken. Presence of weeds in and around the field may also help in perennation of certain pathogens. Detection and destruction of possible hosts of the pathogen among these weeds is an important step in plant disease control.

3.iii.c) **Sanitation:** Field sanitation, like plant sanitation, is essential for control of soil-borne and facultative parasites or saprophytes. Many obligate parasites also perennate through dormant structures in plant organs lying in the soil. Destruction of diseased crop debris by burning in the field decreases this type of survival of pathogens in the field. Burying the debris deep in the soil by soil turning ploughs also helps. Powdery mildews of wheat, barley and peas, downy mildews of peas and maize, and red rot of sugarcane are some examples of diseases checked by this method.

3.iv) **Heat and chemical treatment of diseased plants:** The pathogen present in the plant or in its special organs can be inactivated or killed by heat or chemical treatments. This method has been found useful

mostly in diseases of fruit trees. It can be employed to destroy resting structures or exposed growth of the pathogen present on the host. When these methods are able to affect the pathogen present in the internal tissues of the plant they are included in heat-or chemotherapy

3.v) **Soil treatments:** The aim of soil treatment is to inactivate or eradicate pathogens present in the soil. Soil treatment involves the use of chemicals, heat energy, flooding and fallowing, etc.

In **chemical treatment** of soil .many fungicidal dusts and nematicidal soil fumigants are used. The fungicidal dust can be used at the time of planting of the crop. Most of the fungicides used as soil treatment materials are selective in action and destroy only specific fungi. Therefore, by their use the development of other fungi due to decreased microbial antagonism can be enhanced. Majority of the soil fumigants are non-selective and destroy most micro-organisms in the soil. Reinfestation of such soil is easy and quick. The soil fumigants are applied usually few weeks before actual planting of the crop since they can harm the plant also. Many chemicals are now available in granule form which can be used in standing crops and which dissolve in water to produce the effect of soil fumigants.

In soil treatments the soil around the roots must be treated. Therefore, uniform treatment of soil all over the field is essential. As a result quantity of chemicals required is very high and the treatment becomes expensive. Such treatments are practicable in nurseries, small fields, and for cash crops where higher income in proportion to expenditure is ensured. Control of black scurf of potato, root knot of vegetable crops, nematode diseases of fruit trees, etc. has been recommended through the use of chemical soil treatments.

For small quantities of soil, **heat treatment** is an efficient method of eradication of pathogens. This treatment is practised for treating soil in pots, nurseries and greenhouses. Burning of crop deberis, burning of 12-18 inch thick layer of crop debris, grasses, and weeds spread over the field is one of the methods of applying principle of heat treatment. In many countries steam is also used for disinfestation of the soil.

In special conditions **flooding** of the field is a method of eradicating plant pathogens. If about 12 inch deep water is allowed to stand in the field for several weeks the anaerobic or low oxygen conditions and toxins produced by anaerobic bacteria destroy many fungi and plant parasitic nematodes. Resting structures of many pathogens float on the surface of the water and if the flood water is rapidly drained such structures are washed out of the field. Control of fusarium wilt of banana and root knot of vegetable crops by flooding has been demonstrated in some countries.

Fallowing as a practice to restore micronutrients in the soil has been used by farmers since the very beginning of agriculture. This procedure in crop production helps in reduction of inoculum of many soil-borne plant pathogens. However, in countries where culturable

land is in short supply, in proportion to population, fallowing of land is hardly recommended.

4) **Protective measures**

The inoculum of many fast spreading infectious diseases is brought by wind from neighbouring fields or any other distant place of survival. Principles of avoidance, exclusion and eradication may not be sufficient to prevent development of the disease in such cases. Protective measures are necessary to destroy or inactivate this inoculum. These measures include **use of chemical sprays** and **dusts** to create a toxic barrier between the host surface and the pathogen and necessary modification of the environment to make it unfavourable for development of the pathogen.

4.i) **Chemical treatments:** The aim of most chemical sprays, dusts and seed treatments is to form a protective toxic layer on the host surface so that when the pathogen comes in contact with the host surface it is killed or prevented from growth. The chemicals used for this type of protective covering are called **protective chemicals.** But when these chemicals destroy the already established fungus or other parasites from the host surface they are called **eradicant chemicals.** The same chemical may be protective as well as eradicant.

4.ii) **Chemical control of insect vectors:** The attack of insects on standing crop can be prevented by use of protective insecticides. Many species of insects are important vectors of viral and other diseases. Some virus diseases are transmitted only by their insect vectors. Therefore, timely and effective destruction of these insect vectors becomes the most important method of control of these diseases. If a large population of the vector attacks a crop and feeds on diseased plants which have been sprayed with an insecticide many of them will escape instant death and may visit healthy plants and transmit the disease before being killed. However, later spread of the disease is checked if majority of the insects have come in contact with the toxic chemical. The success of control of insect vectors depends to a great extent on the stage of plant growth and nature of the pathogen. It also depends on the speed with which the chemical can kill the insect. Those chemicals which kill the insects within few seconds are most effective in control of insect transmitted plant diseases.

4.iii) **Modification of environments:** Improvement of aeration reduces the humidity on leaves and other aerial parts of the plant and there by checks growth of fungi which flourish in humid atmosphere. This practice is recommended for control of downy mildew of grapevines (*Plasmopara viticola*). Reducing the number and amount of irrigation also helps in modification of environment against certain pathogens. Mixed cultivation of cotton and "moth" (*Phaseolus aconitifolius*) reduces incidence of root rot of the former crop (caused by *Rhizoctonia solani* and R. *bataticola*). The decrease in disease incidence has been attributed to temperature and high moisture in soil due to the cover provided by lush growth of moth. These temperature and moisture

conditions are not favourable for growth of the pathogens. Post-harvest rot of fruits is reduced by storing them in cool and dry rooms because warm and wet conditions favour the rot-causing fungi. Cold storage of potatoes is also an example of modification of environments against pathogen.

While such environmental factors as moisture and aeration are relatively easy to control or modify, alteration of temperature in the field or soil is difficult. Some soil-borne diseases are more pronounced in soil at high temperatures. Irrigation of the field lowers the temperature to some extent and protects the crop. Charcoal rot (*Macrophomina phaseoli*) of potato is an example.

4.iv) **Modification of host nutrition:** In some diseases of plants nutrients have been found effective in reducing disease intensity. Many leaf diseases are favoured by high nitrogen nutrition. Use of less nitrogen reduces incidence of such diseases. Deficiency of potash in plants renders the tissue susceptible to water soaking and the plant parts become susceptible to many diseases. High calcium increases resistance to wilt disease through strengthening of pectic substances in cell walls and obstructing the activity of pectic enzymes of the pathogens. Similarly, intensity of several other diseases is decreased by micronutrients such as boron, zinc, manganese, etc. By spraying these nutrients on the crop and increasing their level in the tissues protection can be given to the plants.

5) **Development of resistance in the host**

In any crop, resistance against a specific disease can be developed by selection or hybridisation. This type of resistance in genetic. Biochemical resistance of non-genetic nature can be developed in plants by chemotherapy or modification of nutrition. This type of resistance is induced and temporary, lasting till the chemical or nutrient is effective in the plant.

5.i) **Selection and hybrdisation for disease resistance: Genetic disease resistance** can be based on physiological, structural, or functional behaviour of the host. The principle of **physiological disease resistance** involves the presence or development of anti infection substances in the host cell or absence of suitable nutrients for the pathogen. In **structural resistance** different types of structural barriers are present or are formed in response to infection and growth of the pathogen is checked. In **functional resistance**, stomata do not open at a time when the pathogen produces its infection thread.

5.ii) **Resistance through chemotheraphy:** Temporary physiological resistance in plant can be developed through chemotherapy. Systemic fungicides and antibiotics when sprayed on the foliage or fed through the roots, persist in the protoplasm for some time and while their toxic level is maintained the pathogens cannot invade the tissues.

5.iii) **Resistance through host nutrition:** Making available the major and micronurients through spraying or through soil can provide resistance

to disease in the plant. Although the effectiveness of such measures is yet doubtful. A vigorous growth of the plant is always desirable. Vigorous plants with capacity to from new roots and shoots to take up the function of damaged roots and shoots tolerate the attack of many diseases.

Therapy of diseased plant

Although cure of the diseased plant or its organs in most crops is not possible, in some crops and fruit trees chemical and physical therapy has been applied to cure the plant by eradicating the pathogen.

i) **Chemotherapy** : Chemical treatments applied to eradicate the pathogen from tissues of the diseased plant and thus curing it are included in chemotherapy. The chemicals mainly include the systemic fungicides and antibiotics. The principle underlying chemotherapy is that **the chemicals used are absorbed by leaves and roots and on reaching the cell protoplasm act as a chemical defense mechanism by destroying the pathogen and inducing temporary resistance.** They can also act by detoxifying the toxins produced by the pathogen. In this manner the tissues not invaded by the pathogen are saved and the plant is cured.

The systemic fungicides when absorbed by roots are translocated through xylem to upper parts of the plant but when absorbed by levels they fail to be translocated downwards through phloem. They spread towards the margins of the leaf and stay cocentrated there. Antibiotics can be translocated from leaves downwards to roots and from roots upwards to leaves.

ii) **Heat therapy** : Plants which can tolerate the thermal inactivation death point of pathogens can be treated by heat to destroy the pathogens. These treatments are especially used for seeds, tubers, bulbs, grafts, etc. Grafts of fruit trees are exposed to high temperatures for inactivation of many viruses. For eradication of nematodes from roots of grafts heat therapy has been suggested. Ratoon stunting and other viral diseases of sugarcane are also eradicated by hot water or hot air treatment of setts.

iii) **Tree-surgery** : Large size fruit trees are cleaned of infection by cutting of the infected branches or scrapping of the diseased portion and covering the wound with a fungicidal paste. After removal of the infected portions the tree becomes free of infection. The only effective control of stem brown, stem black and pink diseases of apple is this type of surgery.