Introduction to Plant Pathology

Importance of the Plant Diseases:

Globally, enormous losses of the crops are caused by the plant diseases. The loss can occur from the time of seed sowing in the field to harvesting and storage. Important historical evidences of plant disease epidemics are Irish Famine due to late blight of potato (Ireland, 1845), Bengal famine due to brown spot of rice (India, 1942) and Coffee rust (Sri Lanka, 1967). Such epidemics had left their effect on the economy of the affected countries.

Plant Pathology or Phytopathology: Consist of three Greek words

-Phyton means plant, -Pathos means suffering / ailments or diseases and -Logus means knowledge /study/ science.

Plant Pathology or Phytopathology is **that branch of agricultural or biological science which deals with the study of plant diseases, their cause, etiology, resulting losses and management of plant diseases.**

OBJECTIVES OF PLANT PATHOLOGY:

The science of plant pathology has four main objectives:

i. The living entities that cause diseases in plants;
ii. The non-living entities and the environmental conditions that cause disorders in plants;
iii. The mechanisms by which the disease causing agents produce diseases;
iv. The interactions between the disease causing agents and host plant in relation to overall environment; and
v. The method of preventing or management the diseases and reducing the losses/damages caused by diseases.

Concept of Plant Disease:

The normal physiological functions of plants are disturbed when they are affected by pathogenic living organisms or by some environmental factors. Initially plants react to the disease causing agents, particularly in the site of infection. Later, the reaction becomes more widespread and histological changes take place. Such changes are expressed as different types of symptoms of the disease which can be visualized macroscopically. As a result of the disease, plant growth in reduced, deformed or even the plant dies.

When a plant is suffering, we call it diseased, i.e. it is at 'dis-ease'. Disease is a deviation from normal functioning of physiological processes due to continuous irritation caused by some biotic agents or disease is a condition that occurs in consequence of abnormal changes in the form, physiology, integrity or behaviour of the plant or disease is a deviation from normal functioning of physiological processes of sufficient duration or intensity to cause disturbance or cessation of vital activities (APS, 1940). The British Mycological Society in 1950 defined the disease as a harmful deviation from the normal functioning of process. Recently, Encyclopedia Britannica (2002)

forwarded a simplified definition of plant disease. A plant is diseased when it is continuously disturbed by some causal agent that results in abnormal physiological process that disrupts the plants normal structure, growth, function or other activities. This interference with one or more plant's essential physiological or biochemical systems elicites characteristic pathological conditions or symptoms.

Scope of Plant Pathology

Plant pathology comprises with the basic knowledge and technologies of Botany, Plant Anatomy, Plant Physiology, Mycology, Bacteriology, Virology, Nematology, Genetics, Molecular Biology, Genetic Engineering, Biochemistry, Horticulture, Tissue Culture, Soil Science, Forestry, Physics, Chemistry, Meteorology, Statistics and many other branches of applied science.

Importance of the Plant Diseases:

Plant diseases have been associated with many important events in the history of mankind on earth**.**

World scenario

1) **Irish Potato Famine**: Great Irish Famine, or Famine of 1845–49, famine that occurred in Ireland in 1845–49 when the potato crop failed in successive years. The crop failures were caused by late blight, a disease that destroys both the leaves and the edible roots, or tubers, of the potato plant. The causative agent of late blight is *Phytophthora infestans*. The Irish famine was the worst to occur in Europe in the 19th century (**Fig.1**).

   The Irish Famine of 1846-50 took as many as one million lives from hunger and disease, and changed the social and cultural structure of Ireland in profound ways. The Famine also spurred new waves of immigration, thus shaping the histories of the United States and Britain as well. The combined forces of famine, disease and emigration depopulated the island; Ireland's population dropped from 8 million before the Famine to 5 million years after. If Irish nationalism was dormant for the first half of the nineteenth-century, the Famine convinced Irish citizens and Irish-Americans of the urgent need for political change. The Famine also changed centuries-old agricultural practices, hastening the end of the division of family estates into tiny lots capable of sustaining life only with a potato crop.
2) Coffee rust in Sri Lanka (1869) took less than three decades to anhilate the entire coffee production of the island.
3) Downy mildew occurred in epiphytotic way during 1878-1882 in France.
4) Powdery mildew of grape - France (1851 and 1878).
5) Panama wilt of banana damaged the crop during 1910 in Panama and Surinam.
6) Sigatoka leaf spot of banana damaged the crop during 1930 in Central and South America.
7) Bunchy top of banana durin 1940 in Sri Lanka

Indian scenario

1) **Bengal famine:** In 1943, Bengal had to face a serious famine. One of the reasons to which this famine has been attributed is the loss of yield of rice due to attack of *Helminthosporium oryzae* causing leaf spot disease . At least, 2 million people died from starvation and malnutrition during a famine in the Indian province of Bengal.
2) In 1946-47 wheat rust epidemic caused food shortage.
3) In 1963 bacterial leaf blight of paddy in Bihar wiped the crop completely.
4) Tungro virus in 1985-87 and blast during 1985-88 caused havoc to rice crop in Tamil Nadu.
5) In 1938-39 red rot of sugarcane occurred in large scale in Bihar, Punjab and U. P.
6) Bunchy top of banana wiped out the whole banana plantation in Palani hills of Tamil Nadu.

Important Famines/epidemics in World and India and Their Importance

1. IRISH FAMINE

Country	Ireland
Location	Ireland
Period	1845 – 1852
Total population	8 million
Total deaths	1.5 million
Migration	1.6 million
Reason	Policy failure, potato blight
Impact on demographics	Population fell by 20-25% due to mortality and emigration
Consequences	Permanent change in the island's demographic, political and cultural landscape
Scientific Advancement	Birth of Plant Pathology as a science **Anto De Bary and Speer Schneider** (1861) proved Fungi as causal agent (*Phytophthora infestans*)

2. COFFE RUST

Country	Ceylon (Srilanka)
Period	1867 -1893
Cause	Cofffee Rust by *Hemileia vastatrix*
Impact	Economy ruined
Consequences	1. Yield reduced from 228 kg berries /acre to 101 kg/ acre 2. Export decline by 93 per cent by 1893 3. Coffee replaced by tea plantations 4. Drinking habit changed from coffee to tea

Scientific Advancement	**M.J Berkly** –Idetified the cause as *Hemileia vastatrix*
	H.M.Ward – Elucidated the Role of Environment and Monocropping in disease epidemic

4. DOWNY MILDEW

Country	France (Europe)
Period	1878
Reason/ Cause	Downy mildew caused by *Plasmopara viticola*
Consequences	1. Reduced grape yields and quality greatly 2. Killed young vines in many vineyreds 3. Wine industry affected and Economy ruined
Scientific Advancement	Discovery of **Bordeaux mixture (1885) by P.A.Millardet**

5. BENGAL FAMINE

Country	India
Location	Bengal state
Period	1943
Cause	Brown spot of paddy
Pathogen	*Drechslera oryzae*
Impact	1. Shortage of food 2. Raise in the price of rice 3. Death of 2 million people due to hunger 4. Migration of people to other states. 5. Change in the Agriculture policy.

Significance of Plant Disease/ Plant Pathogens

Of the 30,000 plant diseases recorded from different countries about 5,000 diseases are present in India. It is estimated that the total annual production for all agricultural crops worldwide is about 12000-13000 billion $. Of this, about 500 billion $ worth of produce is lost annually to diseases, insects and weeds.

Following are the significance of Plant diseases

1. **Famine:** Irish famine – Late blight of potato, Bengal famine – Brown spot of paddy
2. **LOSSES**

a) Quantity (Yield loss): During Wheat rust epidemic in Bihar (1956-57) yield reduced from 900kg/ha to 50 kg/ha.

b) Crop quality losses: Example: Citrus canker, Chiku rough storage, Apple scab, Groundnut – aflatoxin, Wheat – bunt, ear cockle.

3. **Human/ animal hazard /food poisoning:** Consumption of Ergot infected rye – causes gangrene, loss of fingers, limbs and death. (Known as Saint Antony's fire). Death of one Lakh turkeys in England due to consumption of Peanut meal contaminated with Aflatoxin. Aflatoxin is immune suppressive and causes Liver cancer in Human beings.
4. **Cropping pattern changed:** In Srilanka Coffee was replaced by Tea plantation due to Coffee Rust (1867).
5. **Limit the cultivation of variety:** Due to Downy mildew of Bajra, cultivation of GJ 104 variety was discouraged in Gujarat. Due to Wilt & red rot cultivation of co. 671, co. 8603 sugarcane variety was discouraged in south Gujarat.
6. **Diet change/ food habit change:** In Sri Lanka drinking habit changed from coffee to tea due to coffee rust. In Southern U.S.A food habit changed *i.e.* from wheat to corn due to wheat rust
7. **Agro- industries affected:** Due to Wilt and red rot diseases Sugar Industries in Gujarat affected. Wine industry in France was severly affected due to Downy mildew disease.
8. **People's migration:** Due to Irish Famine in more than 1.6 million people migrated from Ireland to North American continent.
9. **Sociological effect :**. Increase in Poverty due to close of Agro industries
10. **High/rise market price:** Shortage of food due to heavy crop loss leads to Rise in price of Agril. Commodities
11. **High cost of production:** Extra Expenditure to be incurred on pesticides, labour etc. for control of diseases
12. **Political effect:** Due to Irish famine, the free trade was allowed between England and Ireland
13. **Destroy the beauty of environment:** By damaging plants around houses, along street, in gardens & in forest.
14. **Ecological implications:** Environmental pollution due to fungicidal use or due to disease.

TERMINOLOGIES RELATED TO PLANT PATHOLOGY

1. **Host :** An organism harbouring a parasite or pathogen.
2. **Parasite:** An organism living on or in another living organism (host) and obtaining its food from the latter (host).
3. **Ectoparasite :** a parasite that lives and feeds from the exterior of its host's cells or tissues.
4. **Endoparasite :** a parasitic organism that lives and feeds within the cells or tissues of its host.
5. **Pathogen:** An entity, usually a micro-organism, that can incite disease.
6. **Abiotic :** pertaining to physical and inorganic components. For example, diseases/disorders in plants can be caused by abiotic factors such as extremes of heat, light, moisture, lack of nutrients etc.
7. **Biotic :** pertaining to life and therefore living organisms. For example, plant diseases of a biotic origin are caused by living organisms such as fungi, bacteria, virus, viroids, algae, insects, nematodes, etc.

8. **Disease:** Any malfunctioning of host cells and tissues that results from continuous irritation by a pathogenic agent or environmental factor and leads to development of symptoms.
9. **Disorder:** Non-infectious plant diseases due to abiotic causes such as adverse soil and environmental conditions are termed disorders.
10. **Saprophyte:** An organism that derive their nutrition from dead organic matter by absorbing soluble organic compounds. Example: *Cellulomonas*
11. **Pathogenicity:** The capacity of a pathogen to cause disease.
12. **Symptom:** The external or internal reactions or alterations of a plant as a result of a disease.
13. **Spore:** A specialised reproductive body in fungi (and some other organisms), containing one or more cells, capable of developing into an adult.
14. **Hypha (pl. Hyphae):** A single tubular thread-like filament of a fungal mycelium. The hypha is the basic structural unit of a fungus.
15. **Mycelium (pl. Mycelia):** A mass of hyphae that forms the body (thallus) of a fungus.
16. **Appressorium:** The swollen tip of a hypha or germ tube that facilitates attachment and penetration of the host by the fungus.
17. **Haustorium:** slender lateral outgrowth of intercellular hyphae of pathogen which involved in nutrient absorption from host cell.

Plant Pathology branches

1. **Mycology:** The study of fungi is called mycology. OR It is the science which deals with the study of fungi is known as mycology.
2. **Nematology:** It is the science which deals with the study of nematodes.
3. **Virology:** It is the science which deals with the study of viruses.
4. **Bacteriology:** It is the science which deals with the study of bacteria.

Causes of Plant Diseases

Plant diseases are caused by pathogens. Hence a pathogen is always associated with a disease. In other way, disease is a symptom caused by the invasion of a pathogen that is able to survive, perpetuate and spread. Further, the word “pathogen” can be broadly defined as any agent or factor that incites 'pathos or disease in an organism or host. In strict sense, the causes of plant diseases are grouped under following categories:

1. Animate or biotic causes: Pathogens of living nature are categorized into the following groups.

(i) Fungi : Powdery mildew, Late blight of Potato, Rust and smuts, blights, spots, Club root of cabbage etc.
(ii) Algae : Red rust of mango and tea.
(iii) Bacteria : Bacterial blight of rice, black arm of cotton.
(iv) Phanerogamic parasites:

a) Total stem parasite : *Cuscuta*
b) Total root parasite : *Orobanche*
c) Partial stem parasite : *Loranthus*
d) Partial root parasite : *Striga*

(v) Phytoplasma : Sesame phyllody, Little leaf of Brinjal
(vi) Protozoa: Heart rot of Coconut
(vii) Nematodes : Root knot of vegetables, seed gall of wheat, Molya of wheat and barley.

2. Mesobiotic causes: These disease incitants are neither living or non-living, e.g.
(i) Viruses: Tobacco mosaic, Leaf curl
(ii) Viroides: Potato spindle tuber disease, Coconut cadang cadang.

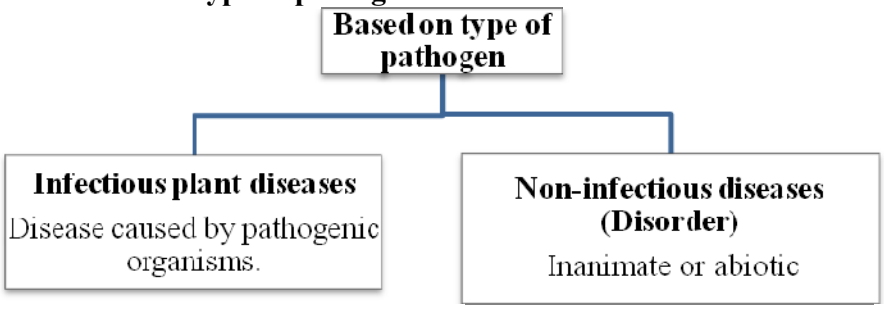
3. Inanimate or abiotic causes: In true sense these factors cause damages (any reduction in the quality or quantity of yield or loss of revenue) to the plants rather than causing disease. The causes are:

(i) Deficiencies or excess of nutrients: 'Khaira' disease of rice due to Zn deficiency, Chlorosis in Groundnut due to Ca deficiency.
(ii) Light: Damage to potato plants due to lightening
(iii) Moisture: High moisture cause 'edema' in cabbage, Damping off in vegetables.
(iv) Temperature: High temperature cause tip burn in betel vine, Dry root rot, charcoal rot
(v) Air pollutants: Black tip of mango, apple scald
(vi) Lack of oxygen: Hollow and black heart of potato
(vii) Mineral toxicity: Leaf crinkle of cotton due to manganese toxicity, Akiochi in Paddy due to H_2S (Hydrogen sulphide) toxicity.
(viii) Improper cultural practices or Iatrogenic diseases: Zineb controls downy mildew but enhance the incidence of powdery mildew of grapes.

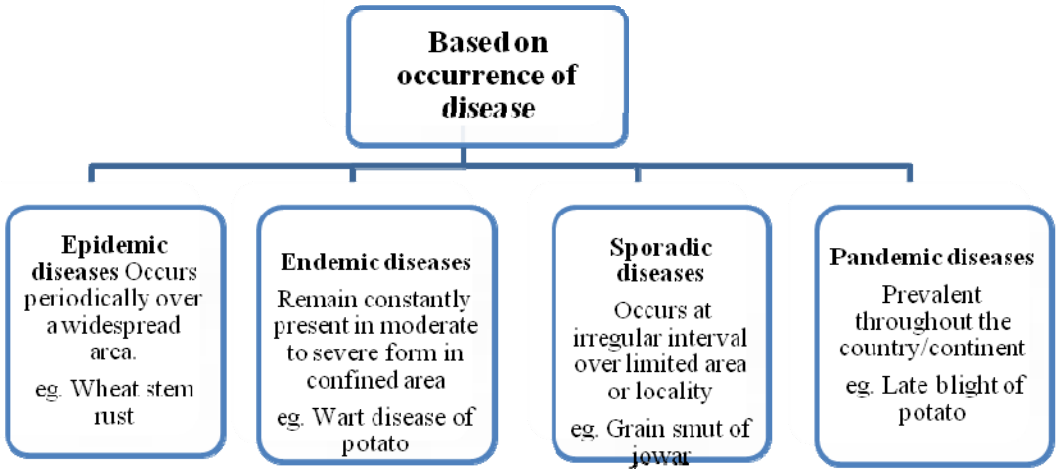
Classification of Plant Disease

To facilitate the study of plant diseases they are needed to be grouped in some orderly fashion. Plant diseases can be grouped in various ways based on the symptoms or signs (rust, smut, blight etc.), nature of infection (systemic or localized), habitat of the pathogens, mode of perpetuation and spread (soil-, seed- and air-borne etc.), affected parts of the host (aerial, root disease etc.), types of the plants (cereals, pulses, oilseed, ornamental, vegetable, forest diseases etc.). But the most useful classification has been made based on the type of pathogens that cause plant diseases. Since this type of classification indicates not only the cause of the disease, but also the knowledge and information that suggest the probable development and spread of disease along with their possible control measures.

I. The classification based on type of pathogen is as follows:



II. The classification based on occurrence of disease is as follows:



HISTORY OF PLANT PATHOLOGY

LANDMARKS IN THE DEVELOPMENT OF PLANT PATHOLOGY

1. Ancient period:

A literature of European and vedic eras will give us some information on the plant diseases and their control measures. Earlier people were aware about plant diseases but they aware unable explain scientifically hence they believed that plant diseases were a manifestation of the wrath of God and, therefore, that avoidance or control of the disease depended on people doing things that would please that same superpower.

In the fourth century BC.; the Romans suffered so much from hunger caused by the repeated destruction of cereal crops by rusts and other diseases that they created a separate god, whom they named **Robigus**. To please Robigus, the Romans offered prayers and sacrifices in the belief that he would protect them from the dreaded rusts. The Romans even established a special holiday for Robigus, the **Robigalia**, during which they sacrificed red dogs, foxes, and cows in an attempt to please and pacify Robigus so he would not send the rusts to destroy their crops.

The first person to study and write about the plant diseases is the Greek philosopher **Theophrastus.** He made observations on the plant diseases in his book **enquiry into plants**. His experiences were mostly based on imagination and observation but not on experimentation.

Theophrastus (370 B.C. – 286 B.C)

- First botanist to study and write about the diseases of trees, cereals and legumes.
- He divided the plant diseases into **external and internal.** External diseases are caused by external factors like Temperature, Moisture etc; internal diseases are caused by internal conditions of plant.
- He stated that different plants are affected by different types of diseases
- Some plants are prone to infection than others. Ex: Cereals are most affected by Rust than pulses. Among cereals Barley is more attacked than any other crop.
- First to recognize influence of environment and topography of the land on plant diseases. Example: Plant grown in elevated areas are less affected than plant grown in low land.
- **He wrote two important books**: 1**. Historia plantarum** (the enquiry into plants)

2. **Decauses Plantarum**

- Regarded as **Father of Botany**

In India, the information on plant diseases is available in ancient literature such as rigveda, atharveda (1500-500BC), arthasashtra of Kautilya (321-186 BC), Sushruta sanhita (200-500AD), Vishnupuran (500AD), Agnipuran (500-700AD), Vishnu dharmottar (500-700AD), etc. In Rigveda, not only the classification of plant diseases has been given but the germ theory of disease was also advocated.

Vriksha ayurveda by **Surpal** in ancient India is the first book in which lot of information on plant diseases is available. In this book, plant diseases were categorized into two groups, internal (probably physiological diseases) and external (probably infectious diseases). External diseases were supposed to be due to attack of microorganisms and insects. In this book, a mention of treatments for different diseases caused by different agencies was prescribed which were based on superstition as well as scientific observation. Hygiene, tree surgery, protective covering with pastes and special culture of plants are practices which are still recommended. In chemical treatments, use of honey, ghee, milk, barley flour, pastes made from herbs, plant extracts, etc., were recommended. For the control of root diseases, oilcakes of mahuva, mustard, sesame, castor, etc., were used.

Symptoms of plant diseases such as rust, downy mildew, powdery mildew and blight are often mentioned in the bible, Shakesphere's poems and dramas of other Christian literature. In Jataka of Buddhism, Raghuvansh of Kalidas there was also a mention about different symptoms of plant diseases.

(1) P.A. Micheli (Pier Antonio Micheli) (1729):

- He was first to study the fungi and saw the spores of fungi.
- First man to attempt pure culture of fungi growing artificially on Melon pieces
- He described the 1900 plants among awhich 900 were fungi
- He published a Monograph – **"Nova Plantarum Genera"**
- He is considered as "Father **of Mycology**"

(2) **Buller: Mechanism of spore discharge in Fungi (Buller phenomenon)**

(3) Carl Linnaeus (1753):

- He was using latin binomial system for naming the plants & fungi.
- He published – "Species Plantarum"
- He is considered as "Father of Botany"

(4) Tillet (1755):

- He published a paper on "Bunt or stinking smut of wheat".
- Reported wheat seeds that containing black powder on their surface produced more diseasesd plants than the clean seeds.
- Beleived that the toxin present in the black powder caused the disease rather than the fungus.
- He showed **contagious (infectious) nature of "wheat bunt" disease.**

(5) Persoon, C.H. (1801):

- First to gave real systematic of fungi and laid the **foundation for taxonomy of fungi**
- He published a Book – "**Synopsis Methodica Fungorum".**
- He classified many fungi using binomial nomenclature as Uredinales, Ustilaginales and Gasteromycetes.

(6) Fries Elias Magnus (E.M. Fries) (1821-1832):

- He published "Systema Mycologicum".
- He regarded as "Linnaeus" of Mycology. "Father of Systematic mycology"
- He considered rust and smut fungi as a products of the diseased plants.

(7) Prevost (1807):

- He proved that diseases are caused by microorganisms.
- He was first man to show microorganism as disease causing agent.
- He discovered the life cycle of "Bunt fungus".

- He observed the spore germination in bunt fungus.
- He was the first man to suggest or use of chemical copper sulphate for seed treatment to control seed borne disease.

(8) Anton De Bary (1831-1888):

- German Scientist born in 1831
- Experimentally proved that the ***Phytophthora infestans*** was the cause of late blight.
- He studied late blight of potato fungus, its nomenclature and renamed it as *Phytophthora infestans* (infectious plant destroyer)
- Studied the other diseases also such as rust, smut, downy mildews.
- First to study the **physiology of plant disease** and reported the role of enzymes and toxin in tissue degradation caused by *Sclerotinia sclerotiorum* in carrot.
- Reported **heteroecious** nature of Rust fungi.
- Introduced the terms **Biotrophs, Facultative parasite and Facultative saprophytes**
- Reported the **plasmodial** stage of Myxomycetes
- Authored two books:
  1. Morphology and Physiology of Fungi, lichens and Myxomycetes (1867)
  2. Comparative Mophology and biology of the Fungi, Mycetozoa and Bacteria (1884)
- For all his contributions regarded as
  1. Founder of "**Modern Experimental Plant Pathology".**
  2. Father of "**Plant Pathology"**
- **Guided the no. students among them**

Woronin (Russia), Brefeld (Germany), Millardet (France), Ward (England), Farlow (USA)

(9) M. J. Berkeley (1855) :

- Observed root knot nematodes in galls of cucumber.
- Founder of Mycology in Bristish - described 6000 species of fungi
- Studied the Late blight of potato pathogen, Rust of coffee and Grape downy mildew.

Authored books: 1. Outlines of British Fungology

2. Introdcution to cryptogamic botany

(10) Julius Gotthelf Kuhn (1858) :

- Published a text book in Plant Pathology entitled "**The Diseases of Cultivated Crops, Their Causes and Their Control".**

- Demonstrated the penetration of Bunt fungus into wheat and its development in the plant.
- Copper sulphate seed treatment was standardized and brought into usage at farmers level.
- He studied the Rusts, Smuts of cereals, ergot sooty mold and diseases of Mustard.

(11) Robert Harting (1935-1901):

- Worked on Forest Pathology
- He wrote two important books in German on :
  - (1) Important Diseases of Forest Trees (1874)
  - (2) Text Book of Tree Diseases (1882)
- He is called the "**Father of Forest Pathology".**

(12) Brefeld & its colleague (1875-1912):

- Discovered the methods of artificial culture of micro-organisms i.e. gave "**Pure culture Techniques".**
- Given the **Single spore isolation technique**
- Studied the life cycle of Cereal smut

(13) Pierre Marie Alexis Millardet (P.M.A. Millardet) (1882) :

- He discovered the **Bordeaux mixture in 1882** for the control of downy mildew of grape caused by *Plasmopara viticola*.
- studied the root aphid phylloxera of the grape vine and hybridization experiments in order to have resistant plants.

(14) M.S.Woronin (Russia)

- Discovered the cause of **Club root of Cabbage (*Plasmodiophora brassicae*)**
- Studied its **life cycle**
- Suggested the **removal of infected plants** and burning them to destroy the resting spore

(15) R. H. Biffen (1905):

- Resistance to yellow rust in wheat **was inherited and Follow Mendelian Fashion**.
- First to develop Rust resistance variety "**Ribet**" against Yellow rust of wheat (*Puccinia striiformis*)

(16) Flor (1955):

- Given "**Gene for Gene Hypothesis**" concept in Flax against Rust (*Melampsora lini*). "For each gene confers resistance in the host there is a corresponding gene in the pathogen that confers Avirulence and vice-versa".

(17) Vander Plank (1963):

- Given the concept - **Horizontal and vertical resistance**, Threshold theorem, **Vertifolia effect**, **AUDPC** (Area under Disease Progress Curve)
- He written books on

  (i) Disease Resistance in Plants (1968)

  (ii) Principles of Plant Infections (1975).

  (iii) **Plant Diseases: Epidemics and Control (1963)**
- Regard as **Father of Epidemiology**

(18) H.M.Ward (1901)

- Worked on coffee **rust and life cycle of pathogen.**
- Elucidated the role of environment in coffee rust epidemics.
- Demonstrated the **Hypersensitive phenomenon** in Borme grass infected with brown rust.
- Wrote book: Diseases in plants
- Regarded as **Father of Tropical Plant Pathology**

(19) J. H. Craigie (1927)

- Discovered the function of Pycniospore of *Puccinia graminis tritici*
- Discovered the sexual stage of Rust fungi.

20**. E.C. Stakman:**

- ✓ Showed existence of physiological races in wheat rust.
- ✓ Coined term Hypersensitivity.
- ✓ Wrote book: Principles of plant pathology.

21. **Blackslee**: Gave concept of Heterothallism in Fungi.

22. J.C.Walker:

- ✓ Reported the role of Phenolic compound in plant defense mechanism
- ✓ Reported the resistance of coloured onion bulbs to smudge fungus (*Colletotrichum circinans*) was due to **catechol and Protochatecuic acid** present in the dry scale
- ✓ He wrote two books on
  - Plant Pathology (1968)
  - Diseases of Vegetable Crops (1952).

23. Muller and Cruckshank:

- ✓ Involvement of Phytoallexin in plant disease resistance.

Bacteriology:

1. Anton Van Leeuwenhoek:

- ✓ He was a "cloth merchant" living in Delft Holland.
- ✓ Developed magnifying lenses (300- 500 times magnification).
- ✓ He developed simple microscope
- ✓ First to discover the micro-organism like – Bacteria, Protozoa, alage and named them as "Little animalcules".
- ✓ He is regarded **as Father of 'bacteriological world'.**

2. Luis Pasteur:

- ➢ Proved the **germ theory**
- ➢ coined the terms **Aerobic, anaerobic organisms**
- ➢ Proved **fermentation** is biological process
- ➢ Developed **Pasteurization** process
- ➢ Developed vaccine for Rabies disease
- ➢ Regarded as "**Father of Microbiology"**

3. Robert Koch (1876) :

- ➢ He proved that the anthrax disease of cattle is caused by a bacterium (*Bacillus anthracis*).
- ➢ He gave **Koch's rules or Koch's postulates** or proof of pathogenicity.
- ➢ Use of **Gelatin** as a solidifying agent
- ➢ Found causal organism of Tuberculosis disease.
- ➢ He considered as "**Father of Medical bacteriology".**
- ➢ Regarded as **father of bacteriological techniques**

Koch's postulates:

1. The suspected micro-organism must be present in every diseased host.
2. The suspected causal agent must isolated from the diseased host and grown in pure culture.
3. The specific disease must be produced when a pure culture of the suspected causal agent is inoculated into a healthy susceptible host.
4. The same causal agent must be recovered again from the experimentally inoculated and infected host.

4. T.J. Burrill (1878) :

- ➢ He first time proved that the **fireblight of apple & pear was caused by a bacterium (*Erwinia amylovora*).**
- ➢ First to Show **Bacteria can cause plant disease**

5. E. F. Smith (1900)

- ➢ Final proof for bacteria can cause plant disease

- Studied bacterial wilt of cucumber, Crown gall of apple
- Gave **Fourth Step** of Koch's postulates
- Wrote books : 'Bacterial diseases of plants', 'Bacteria in relation to plant diseases'
- Father of "**Phyto bacteriology"**
- He first time demonstrated that budding or grafting could be another method of transmission of plant viruses.

6. Walther and Frau Hessey (1881)

- **Used Agar –Agar as a solidifying agent in media.**

7. R.J.Petri

- **Developed the petriplates (two circular plates holding the media)**

8. Doi and Ishi (1967):

- **Discovered Mycoplasma like organisms (MLO's)** causing yellows type diseases (Mulberry dwarf, Aster yellows) in plants.
- Ishi *et al* Reported **Tetracycline** can be used for Management

9. Davis and Worley, J.E. (1972) :

- He first time discovered "**Spiroplasma**" in corn stunt disease.

10. Windsor, I.M. and Black, L.M. (1972) :

- He observed first occurrence of **Rickettsia like organisms (RLO's)** in clover club leaf disease.

Virology:

1. Carolus Clusius

- **Recorded breaking of tulips due to viral infection**

(2) Adolph Mayer (1886):

- First time discovered the **tobacco mosaic virus** (TMV) disease.
- Named the symptom as "**Mosaic**"
- Showed that **sap transmission** of disease.
- He demonstrated that this disease (TMV) could be transmitted from diseased to healthy plants by the sap from leaves showing mosaic symptoms.

(3) Dimitrii Ivanowski (1892) :

- He demonstrated that tobacco mosaic virus (TMV) could pass through even those **filters that retained (to keep) bacterial cells.** So he suggested that viruses were smaller than smallest known bacterium.

(4) M.W. Beijerinck (1898) :

- He concluded that Tobacco Mosaic Virus (TMV) was caused by **"*Contagium Vivum Fluidum*" (infectious living fluid) which he called a "VIRUS".**
- He discovered the causal agent of Tobacco mosaic disease.
- **Coined the term 'Virus'**
- **Father of Plant Virology**

(5) Stanley, W.M. (1935) :

- He **purified** and **crystallized Tobacco Mosaic Virus** (TMV).
- Reported Viruses are protenacious molecules
- He got Noble Prize.

(6) **F. C. Bawden and N. W. Pirree (1938)**

- Proved that Viruses were **Nucleo-proteins**.
- Showed that TMV consist of **95% protein and 5% Nucleic acid**

(7) Gierrer and Schramm:

- ✓ **Showed RNA of Tobacco mosaic virus (TMV) is infectious not the protein part.**

(8) Knoll and Ruska (1932)

- **First invented electron microscope**

(9) Diener, T.O. (1971) :

- **Discovered the 'VIROIDS'**
- Determined that potato spindle tuber disease is caused by small molecules of infectious ribonucleic acid (RNA) which he called "VIROIDS".

(10) Stanley Prsuiner

- **Discovered the Prions causing Scrapie disease in Sheep**

(11) A.N.Fire and C.C.Mello (2006)

- **Awarded Nobel Prize for their work on RNA Interference gene silencing by ds RNA.**

<u>NEMATOLOGY:</u>

(1) Nathan Augustus Cobb (N.A. Cobb) (1913-1932) :

- He contributed a lot to nematode morphology, taxonomy and methodology.

- Given the term "Nematology"
- Given techniques for seperation nematodes from soil - **Cobbe Seive technique**
- He is considered as **"Father of Plant Nematology".**

(2) T. Needham (1743):

- He reported first plant parasitic nematodes in wheat-galls-Ear cockle of wheat (*Anguina tritici*).

History of Plant Pathology in India:

D. D. Kunnighham (1850-1875)

- collection and Identification of Fungi of India
- **Identified the causal agent of Red rust of tea in Assam (*Cephaleurous virescens*).**
- Studied the Rust and Smuts

K.R. Kirthikar

- First Indian Scientist to collect fungi and Indentify in India

E.J. Butler (1905):

- First cryptogamist to India
- worked on red rot of Sugarcane, Wilt of Red gram, Rice diseases
- studied the life cycle of smut diseases.
- He wrote book on "**Fungi and Disease in Plants" in 1918.**
- Monograph "Pythiaceous and Allied Fungi"
- Established Herbarium At IARI
- He considered as **"Father of Indian Plant Pathology".**
- He became **First Director of Imperial Mycological Institute (1929)**

J.F. Dastur (1886-1971) :

- First Indian plant Pathologist credited for Detailed study of Fungi and Plant diseases.
- He was studied the genus *Phytophthora* and diseases of castor.
- He is internationally known for establishing the species of *Phytophthora parasitica* from castor.

Mehta, K.C. (1929):

- Worked on **Epidemiology of Wheat rust**
- He also proved Barbery has no role in perpetuation of rust fungi in India.
- Discovering the disease cycle of cereal rusts in India.
- Wrote Monograph : **Further studies on Cereal Rust In India (1940)**

- **Regarded as Father of cereal Rusts in India**

B.B. Mundkur (1948) :

- He worked on **Managment of cotton wilt through Resistance breeding**
- Worked on Smut fungi, wilt diseases.
- He published a textbook **"Fungi and Plant Diseases",**
- Wrote Monograph: Ustilaginales in India
- He started single handed **"Indian Phytopathological Society"** with its journal **"Indian Phytopathology" in 1948**.

M.J. Thirumalachar

- He successfully introduced the use of antibiotics in control of plant diseases eg. Aureofungin and Streptocycline.
- Extensively studied the **Smuts and Rusts**
- Wrote Monograph: **Uredinales of world, Cercosporae**

J.C. Luthra and his associates:

- He developed the solar treatments of wheat seed for control of loose smut (internally seed borne disease).

T.S.Sadasivan:

- Worked on biochemistry of Host- Parasite Relation
- Developed the concept of **Vivotoxin**
- Worked on the mechanism of Cotton wilt (due to Fusaric acid)

S. N. Dasgupta:

- Worked on biochemistry of Host- Parasite Relation
- Studied the **Black tip of Mango**

M.K. Patel (1948-51):

- Studied the bacterial plant diseases in cotton, paddy.
- Proposed new family **Phytobacteriaceae** to include all the phytopathogenic bacteria
- Devised a synthetic medium known as **PATEL's MEDIUM** to cultivate bacterium.
- Discovered several *Xanthomonas* and *Psuedomonas* species causing plant diseases.
- Regarded as **Father of Phytobacteriology**.

Nene Y.L.

- worked on pigeonpea sterility mosaic disease
- Reported **Khaira Disease of Paddy due to Zn deficiency**
- published books on: 1. Fungicides in plant disease control
  2. Viral diseases of pulse crops in U.P.

G.S. Kulkarni: a student of Butler, generated detail information on downy mildew and smut of jowar and bajra.

S.L. Ajrekar: a student of Butler, studied wilt disease of cotton, sugarcane smut and ergot of jowar

Manoranjan Mitra: He was considered as one of the most critical plant pathologist worked on *Helminthosporium*. He first reported Karnal bunt of wheat in 1931 from Karnal in Haryana.

T.S. Ramakrishnan: a mycologist to Madras Government cultivated ergot diseased rye for toxin production. He published two books entitled Diseases of Millets (1963) and Diseases of Rice (1971). Renowned plant pathologists viz., G Rangaswami and R. Ramakrishnan were his students.

Study questions:

1. What is meant by Robigalia and write its significance?
2. Name the rust God and Godess created by Romans
3. Write the scientific contributions of following scientists

Theophrastus, P.A.Micheli, Anton De Bary, Prevost, E.J.Butler, B.B.Mundkur, M.W.Beijerinck, K.C.Mehta, Robert Koch, M.J.Berkly, Oscar brefeld, N.A.Cobb, W.M. Stanly, E.F.smith, Vander Plank.

4. Answer the following questions.

a) What is meant by Gene for Gene Hypothesis and name the scientist proposed it.
b) Name the scientist who discovered that 'Resistance in wheat is inherited in Mendalian fashion'.
c) Name the scientist who first time attempted for culturing of Fungi artificially on Melon pieces.
d) Name scientist reported first plant bacterial disease?
e) The final proof for bacteria can cause plant disease was shown by…….
f) Name the scientist who discovered the Bordeaux mixture and write its use.
g) The bunt of Wheat is contagious in nature was first time shown by?
h) Name the scientist first to study plant diseases and write the name of the book in which he described about them?
i) Name the scientists discovered causal agent of Irish famine potato disease
k) Name the scientist discovered solar heat treatment for the management of loose smut of wheat
l) The vector for the rice dwarf disease was first time shown by…….
m) Name the first Indian Scientist to collect fungi and indentify them in India
n) The Indian Phytopathological society was established by B.B.Mundkur in the year…..

5. Write the name of the scientist/s who discovered the following ones

a) Virus	b) Viroid	c) MLO's	d) Spore of Fungi
f) Bacteria	g) Khaira disease	h) Vivotoxin	j) Satelite virus
k) Virusoids	l) Prions		

6) Name the author/s for the following books

a) Fungi and Plant diaseases
b) Systema Mycologium
c) Synopsis Methodica fungorum
d) Nova Plantarum genera
e) Species platarum
f) Decauses plantarum
g) Fungi and disease in plants
h) Diseases of cultivated plants cuases and their control

7. Name the scientists who discovered following methods/ chemicals for control of plant diseases

a) Solar heat treatment
b) Meristem tip culture
c) Hypovirulence
d) Horizontal and vertical resistance
e) Use of Antibiotics
f) Hot water treatment
g) Cross protection
h) Bordeaux mixture
i) RNA interference

Important Plant Pathogenic micro-organisms

Nematodes:

- ✓ Microscopic in nature, the length of the nematode may vary from 0.2 mm (*Paratylenchus*) to about 11.0mm (*Paralongidorus maximus*).
- ✓ Inhabit marine, freshwater and terrestrial environments as free – livers and parasites.
- ✓ Vermiform Bilateraly symmetrical, triploblastic, unsegmented and pseudocoelomates.
- ✓ Posses stylet and oesophagus is tripartite
- ✓ Growth accompanied by molting of juvenile stages, usually four juvenile stages.
- ✓ Oral opening surrounded by 6 lips and 16 sensory structures.
- ✓ Four systems are present - Digestive, Nervous, excretory and reproductive systems.
- ✓ Circulatory and respiratory systems are absent.

Importance:

- ✓ Cause disease in Plants, animals and human beings.
- ✓ Used as biocontrol agents for the management of Insect pest and diseases.
- ✓ Used in biological studies Eg: *Cnerorhabditis elegans* in Gene silencing.
- ✓ Important plant diseases caused by nematodes: Wheat gall (*Anguina tritici*), Molya disease of wheat and barley (*Heterodera avenae*).

Bacteria:

- ✓ They are prokaryotic, unicellular organism.
- ✓ The cell wall is made up of N-acetyl glusoamine and N-acetyl muramic acid.
- ✓ Most of them are aerobic, few are facultative anaerobe (*Erwinia*).
- ✓ They may be rod, cocci, cylindrical, spiral and vibrios shape.
- ✓ Some bacteria are motile with flagella
- ✓ It can be grown artificially on media in laboratory.
- ✓ They reproduce asexually by binary fission.
- ✓ They may be Gram positive or Gram negative.
- ✓ Some having extra chromosomal DNA called 'Plasmid' in their cell.

Importance:

- ✓ They cause diseases in plants Ex: Brown rot of potato by *Ralstonia,* Bacterial blight of rice by *Xanthomonas oryzae* pv. *oryzae.*

- ✓ They play important role in natural cycling of elements (Nitrogen, sulphur etc;).
- ✓ Play important role in organic matter decomposition.
- ✓ Used in industry for the preparation of valuable compounds.
- ✓ Used as biocontrol agents. Eg: *Pseudomonas fluoroscens, Bacillus subtilis.*

Phytoplasmas: Discovered by **Doi *et al* and Ishii *et al* in 1967.**

- ✓ They are prokaryotic organisms lacking cell wall but covered by cytoplasmic membrane.
- ✓ They contain both DNA and RNA.
- ✓ They are pleomorphic and smaller in size…
- ✓ They are obligate parasites and cause disease only in plants.
- ✓ They are present in the phloem of the plants
- ✓ They are resistant to penicillin and sensitive to tetracycline.
- ✓ They transmitted by Leaf hoppers and Psyllids.
- ✓ They can be cultivated in laboratory conditions.
- ✓ Reproduce by binary fission.

Diseases caused by phytoplasmas: Little leaf of Brinjal, Sesamum phyllody, Aster yellows,

Spiroplasma:

- ✓ Discovered by **Davis** *et al.*
- ✓ They are prokaryotes, Lack cell wall, bounded by a single triple layered membrane.
- ✓ They are **helical shape and move by screw motion of the helix.**
- ✓ They contain both DNA and RNA.
- ✓ They are present in the phloem of the plants.
- ✓ They are resistant to penicillin and sensitive to tetracycline.
- ✓ They can be cultivated in laboratory conditions.
- ✓ The colonies look **Fried egg shape** on solid media.
- ✓ Reproduce by binary fission.
- ✓ They cause disease only in plant and Transmitted by Leaf hoppers.

Diseases caused by Spiroplasmas: Citrus stubborn, Corn stunt.

Rickettsia:

Obligate parasitic bacteria of Arthropods. It causes diseases in animals and Human beings. They have cell wall. They very small, non-motile, rod shaped or coccoid shaped. It contains nucleic acid DNA and RNA. Multiplication by binary fission. They can be cultured on egg yolk artificially. Sensitive to antibiotic penicillin. They are transmitted by as arthropod vector like Lice, mites, fleas.

Human Diseases: Rocky mountain fever (Ticks), Typhus fever (lice), Murin typhus fever (Fleas)

Fastidious vascular bacteria: Fastidious vascular bacteria earlier called as Rickettsia Like organisms (RLO's). They are prokaryotes, bounded by cell wall and cell membrane. They have no flagella and most of them are gram negative. They are present either in the phloem or xylem of the

plant. They are transmitted by leaf hoppers. They are sensitive to antibiotics such as penicillin, tetracyclines.

They are two types:

Xylem Limited FVB	Phloem Limited FVB
Can be cultivated artificially	Cannot be cultivated artificially
They are limited to Xylem of the Plant	They are Limited to phloem of the plant
Ex: Pierces disease of grapevine (*Xyllella fastidiosa*), Sugarcane stunt (*Clavibacter xyli*)	**Ex: Citrus greening,**

Virus:

- ✓ Non-cellular, sub microscopic entities which replicate only in living cells and consist of either DNA or RNA and surrounded by protein coat.
- ✓ Protein coat of virus particle is called capsid.
- ✓ They can be visualized only by Electron microscope.
- ✓ Size of plant viruses ranged from 20-200nm.
- ✓ They are obligate intracellular parasites.
- ✓ The virus particles can pass through the bacterial filter chamber.
- ✓ They reproduce only in living hosts.

Importance: They can cause disease in Animals, plants and human beings. They are used in genetic engineer studies (Gemini virus), used in the management

Plant diseases: Tobacco Mosaic virus (TMV), Potato leaf roll virus, Tomato leaf curl, Papaya ring spot, citrus tristeza.

Viroids:

- ✓ First discovered by **T.O.Diener** in Potato spindle tuber viroid.
- ✓ They are smaller than viruses. They are called **Naked viruses**.
- ✓ They consist of **Nucleic acid only without protein coat**.
- ✓ Nucleic acid is circular, low molecular weight ssRNA (ss RNA - single stranded RNA).
- ✓ They are very small ranging from 246-467 nucleotides.
- ✓ Viroids do not code for any protein.
- ✓ They replicate by rolling circle method.
- ✓ They transmit mechanically.
- ✓ They are resistant to heat, UV radiation and ioninizing radiations.
- ✓ They cause disease only in plants.

Importance: viroids cause diseases in crop plants: Potato spindle tuber, citrus exocortis, coconut cadang cadang.

Prions:

- ✓ Discovered by **Pruisner, in 1982.**
- ✓ These are **infectious protein particles**.
- ✓ They consist of only proteins.
- ✓ They are smaller than viroids.
- ✓ They infect and cause diseases only in animals.
- ✓ **Example: Mad cow disease in cow, scrapie disease in Sheep.**

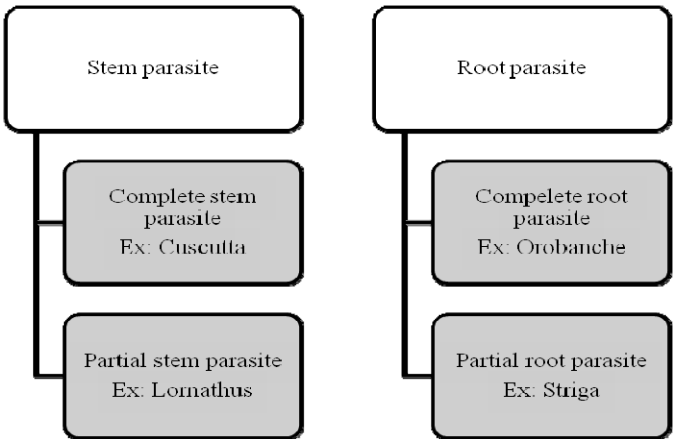
Bacteriophage:

- ✓ First discovered by **F.W. Twort and Fellix D'Herelle**.
- ✓ It is a virus that infects the bacteria.
- ✓ Bacterial viruses are widely distributed in nature.
- ✓ Phages exist for most of the bacteria.
- ✓ Phages can be isolated in laboratory by phage plaque technique.
- ✓ They are composed of nucleic acid surrounded by nucleic acid.
- ✓ The nucleic acid may be either DNA or RNA.

Flowering plant parasites:

A parasitic plant as an angiosperm (flowering plant) that directly attaches to another plant via a haustorium.

Types of flowering plant parasites:



Cuscutta:

- ✓ It is also called as dodder fireweed, devil's guts, devil's hair, goldthread, hairweed, love vine, strangle weed, angel hair, and witch's hair.
- ✓ It is complete stem parasite.
- ✓ It non chlorophyll, bearing leaflets, parasitic plant. They attach their yellow, orange or pink stem to other plant. It produces haustoria which penetrate the plant and absorbs nutrient form

host plant. It produces yellow, pink flowers and sett seeds. Seed remain viable in for several years in soil.

- ✓ Ex: It parasites clover, berseem ,flax, alfalfa, potatoes.

Loranthus:

- ✓ It partial stem parasite.
- ✓ It produce cluster of small twigs bearing broad leaves , orange coloured flowers with red berries.
- ✓ It forms haustoria which penetrate deep into the stem and absorbs food and water from the host plant.
- ✓ It also produce seed which disseminated by birds.
- ✓ Ex: Mango, citrus, conifers.

Orobanche:

- ✓ It is complete root parasite.
- ✓ It appears as yellowish brownish stem arising from base of the host plant. The flowers are white and produce millions of seeds.
- ✓ Ex: Tobacco, Brinjal, tomato, cabbage, cauliflower.

Striga:

- ✓ It is partial root parasite
- ✓ It possesses leaves having chlorophyll. It produces seed and remains viable for several years.
- ✓ Ex: It parasites Sorghum, maize, sugarcane.

Study questions:

2. Write the characters of Fungi, Bacteria, Virus, Viroid, Phytoplasma.
3. Give the example of Xylem Limited FVB
4. Give the example of Phloem Limited FVB
5. Write the meaning or definition of: Virus, Viroid, Bacteriophage, Prions.
6. Which organism is referred as Naked virus.
7. Infectious proteinacious particle is……..
8. Name disease caused by Prions
9. Name the disease caused by viroid.
10. Name the type of parasitism with example of crop infecting for following: Cuscutta, loranthus, orobanche, striga.
11. Circular, low molecular weight ssRNA without protein coat is called as…
12. Wheat gall and Molya disease of wheat and barley are caused by…..

GENERAL, MORPHOLOGICAL CHARACTERS, CLASSIFICATION , METHODS OF REPRODUCTION OF BACTERIA

BACTERIA: Unicellular, and lack of organised nucleus. Bacteria are mostly rod shaped , non motile or motile by means of one or more flagella on their body.Plant pathogenic bacteria are usually gram negative, bacteria are mainly dispersed through seed , soil air and water Bacteria are weak parasite, enter the host through natural opening such as stomata, lenticels and hydathodes.

MORPHOLOGICAL TYPES OF BACTERIA :

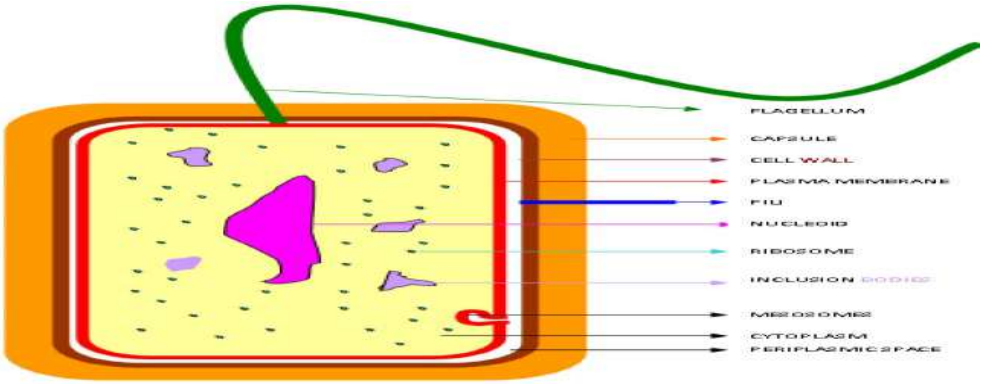
The knowledge of the morphology and internal structure of bacteria will be useful in classifying the bacteria and also to understand various physiological processes that are taking place in bacteria

(1) SIZE:

0.5 to 1.0 µm in diameter, surface area/ volume ratio is exceedingly high favoring unusually high rate of growth and metabolism of bacteria. No circulatory mechanism is needed to distribute the nutrients that are taken in, due to this high surface to volume ratio.

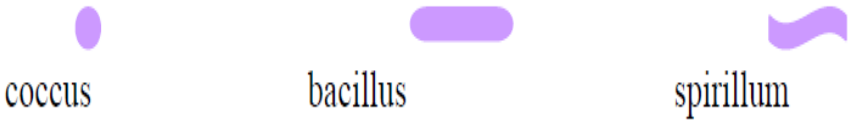
Micron (µ) is the unit for measurements of microorganisms. Hesting has proposed the unit micron (µ).

TYPICAL BACTERIAL CELL STRUCTURE AND FUNCTIONS

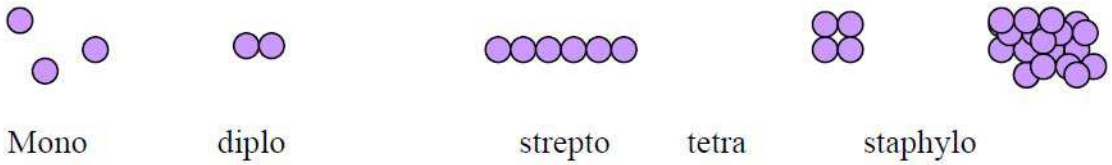


(2)SHAPE AND ARRANGEMENT

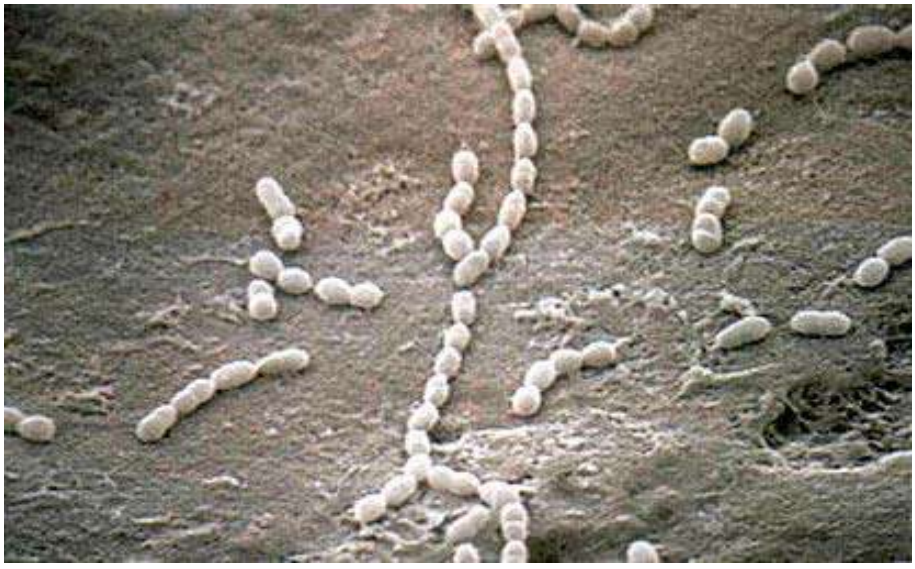
The shape of bacterial cell is governed by rigid cell wall. They may be spherical (Coccus – Cocci), straight rods (Bacillus – Bacilli), or rods that are helically curved (Spirillum – Spirilli) or they may be pleomorphic (exhibit a variety of shapes).



• The Cocci are further grouped into Diplococci, Streptococci, Tetracocci and Staphylococci based on the characteristic arrangement of the cells. Bacilli are mostly singular or in pairs (Diplococci). But some species may be Streptobacilli (Ex: *Bacillus subtilis) or trichomes (Ex: Beggiatoa) or may have palisade arrangement* (*Corynebacterium diphtheria). Some other bacilli may form long, branched* multinucleated filaments called hyphae, which collectively form mycelium (Ex: *Streptomyces)*



Streptococci



Shape of Bacteria:

The shape of bacteria governed by its rigid cell wall. The typical bacteria cells are rods, spherical, curved, comma and pleomorphic.

Rod shape: (Bacillus Pl. Bacilli)

The cells are rod shape.

e.g. *Bacillus substilis*

Xanthomonas citri

Spherical (Coccus Pl. Cocci) Round:

The bacterial cells are typical round shape.

e.g. *Streptococcus lactis*

Spiral Curved Shape (Spirillum):

The cells are vermiform spiral.

e.g. *Spirillum serpens*

Vibrios comma shape:

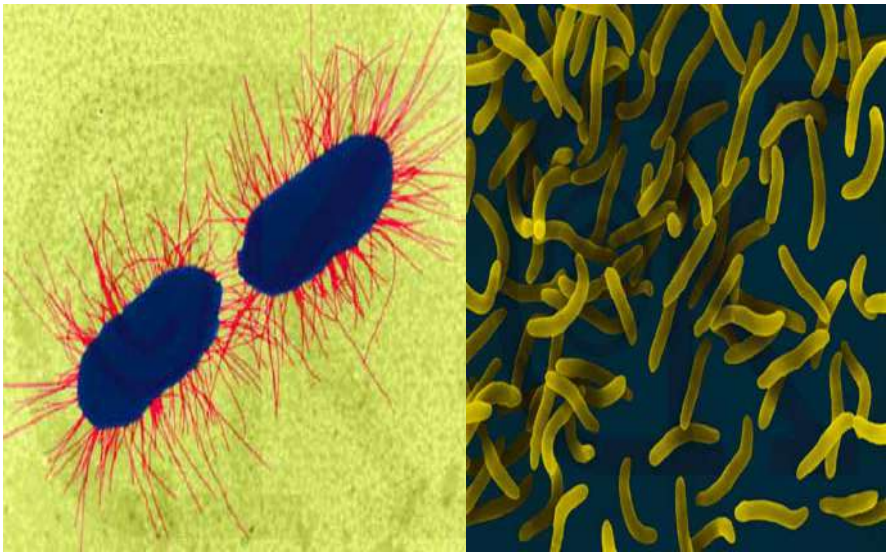
The cells are comma shape.

e.g. *Vibrio comma*

Pleomorphic:

Some bacterial species are exhibits a variety of shapes.

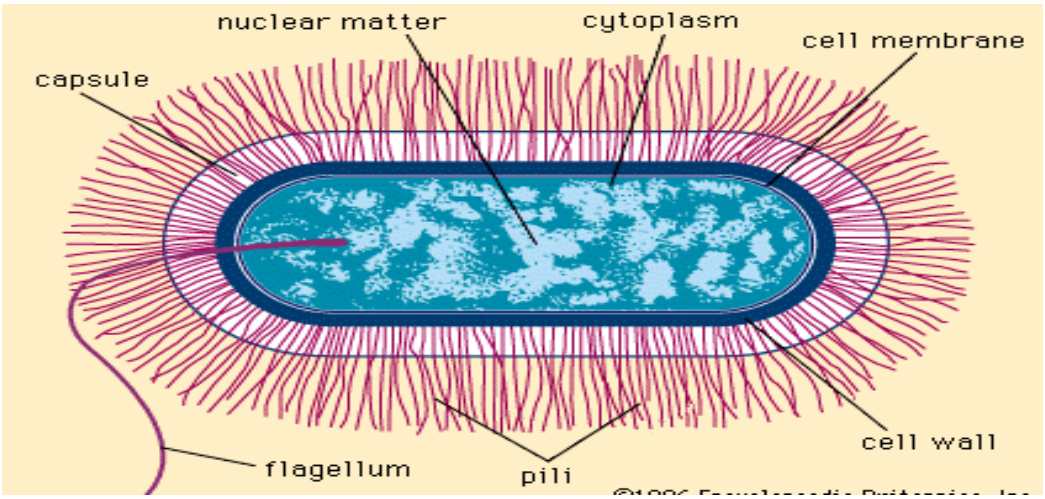
e.g. *Arthrobacter* spp.



E. coli (pathogenic) **Cholera *Vibrio cholera* Gram (-) comma shaped rods**

(3) Structure of Bacteria:

Examination of a bacterial cell reveals various component structures. Some of these are extended to the cell wall. E.g. Capsules, pili and flagella, other are internal to the cell wall e.g. Cytoplasmic membrane, cytoplasm, nuclear material etc. Some structures are present in only certain species than of others and still other cellular parts such as cell wall are naturally common to almost all bacteria.



Cell wall:

It is very rigid structure located in cell, between external structure (flagella and capsule) and internal structure of cell, i.e. cytoplasm and cytoplasmic membrane.

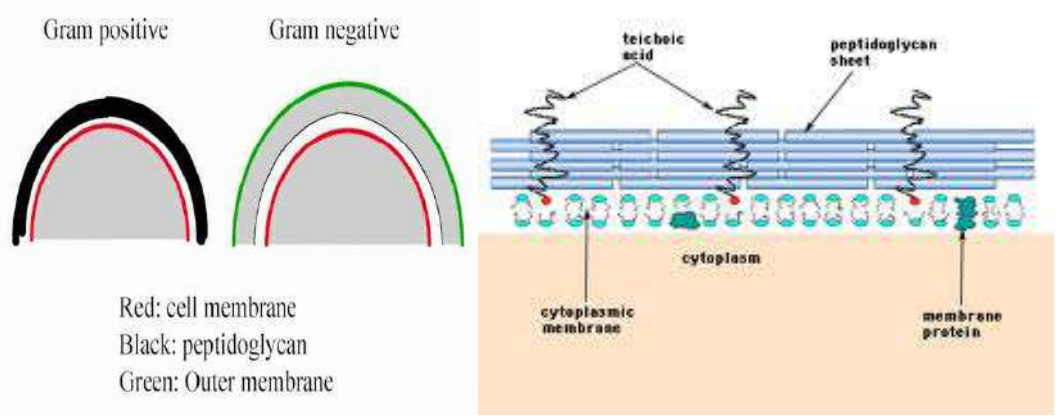
Chemical composition of cell wall:

It is made up of peptidoglycan, polysaccharides, teichoic acid and lipids & lipoproteins.

Chemical composition of cell wall may be the major function in the Gram reaction and the bacteria classified in two main group as Gram positive and Gram negative.

Function:

- It gives shape to the cell.
- It prevents the cell from expending and brusting.
- It gives physical protection to cytoplasm in which most of vital activities are performed.
- It is essential for bacterial growth and division.
- Due to permeability, cell can absorb dissolved nutrients.



- Gram staining is one of the most important and widely used differential staining introduced by Christian Gram in 1884. Bacteria stained by Gram's staining method fall into two groups –Gram positive, (which appear deep violet in color) and Gram negative (which appear red in color). Gram staining is generally not applicable to other microorganisms. However, yeasts consistently stain gram positive.

- **Differences in the cell wall of Gram positive and Gram negative eubacteria :**

CHARACTER	GRAM POSITIVE	GRAM NEGATIVE
1. Thickness	-Thicker wall (20 – 25ηm)	-Thinner (10-15 ηm)
2.Layers	-A single thick. layer	Two layers (a Peptidoglycan layer and outer membrane).
3.Peptidoglycan	Account for 50% dry weight of cell wall	Only about 10% of cell wall.
4.Other constituents	Polysaccharides and Techoic acids	Outer membrane is rich in phospholipids, proteins or lip polysaccharides. Peptidoglycan layer is linked to outer –membrane by Braun's lipoprotein.
5. Susceptibility to a) Penicillin b) Mechanical disintegration	More susceptible Less susceptible	Less susceptible More susceptible

A. Component external to the cell wall:

1. **Capsule or Slime layer:**

Not all but some bacterial species develop capsule. Cells are surrounded by a viscous substance secretion from cell, which form a covering layer or envelope around the cell wall.

If this layer can be visualized by light microscope using special capsule staining technique, it is termed as a "**Capsule".** If it is too thin to be seen microscope than it is termed as a **'microcapsule',**

if it is abundant that may cells are embedded in a common matrix, the material is called '**slime layer**'.

Function:

1. It provides protecting covering of cell and retain moisture in cell.
2.The function of the slime layer is to protect the bacteria cells from environmental dangers such as antibiotics and desiccation.
3. It serves as a reserved stored food or for disposal of waste substances.
4. In disease producing bacteria, it increases infection capacity

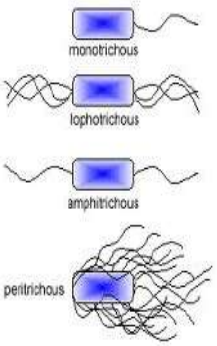
Capsule	**Slime layer**
1. Thick hard layer	1. Thin layer
2. It is a part of cell	2. It is secretion of cell.
3. Cell secretion sharply defined	3. Cell secretion diffused, invisible & readily visible.
4. Uniform density & definite shape	4. Not definite shape and density.
eg *Bacillus* & *Azotobacter*	eg, *Streptococcus*

A bacterial cell secretes materials in forms of capsules or a slime layers. There are some differences between a slime layer and a capsule. A capsule is attached to a bacterium and has defined boundaries. Slime layers can be easily washed off and are loosely attached to bacterium.

The cell capsule is a very large structure of bacterial cells. It is a layer that lies outside the cell wall of bacteria. It is a well organized layer, not easily washed off, and it can be the cause of various diseases.

When the amorphous viscid secretion (that makes up the capsule) diffuses (spread out, not concentrated) into the surrounding medium and remains as a loose secretion, it is known as slime layer.

(2)The bacteria may be classified on the basis of number and arrangement of flagella.



Bacterial flagella are hair like helical appendages that protrude through the cell wall and are responsible for swimming motility. It grows at the tip unlike hair, which grows at the bottom.

A. Monotrichous: A single flagellum at one end of the cell

e.g. *Xanthomonas citri*

B. Amphitrichous: One flagella at each end of the cell.

e.g. *Thiobacillus thioparus*

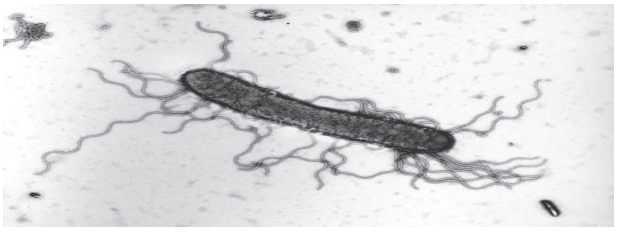
C. Lophotrichous: Two or more flagella at one or both ends of the cell.

e.g. *Spirillum serpens*

D. Peritrichous: Large number of flagella surrounding the cell.
e.g. *Proteus vulgaris*, *Salmonella typhi*

E. Atrichous: The cells are without flagella.
e.g. *Mycobacterium tuberculosis.*

F. Peritrichous: Flagella protruding from all portion of its surface
e.g. *Erwinia spp.*



- A flagellum is composed of 3 parts

(a) Basal body associated with cytoplasmic membrane and cell wall

(b)a short hook and c). A helical filament which is usually several times longer than the bacterial cell. The hook and filament are made up of protein whereas the composition of basal body is not known. The protein of the filament is known as flagellin.

(3) Pili or Fimbriae:

- Any filamentous appendages other than flagella on bacterial cell known as Pili.
- They are thinner, shorter and more numerous than flagella. They do not function in motility.
- The term fimbreae is used for common pili.

Function:

- Attachment to substrate.
- Useful for sexual reproduction as the part of entry of genetic material during bacterial mating.
- Some pili play a major role in human infection by pathogenic bacteria.

B. Component internal to the cell wall:

(1) Cytoplasmic membrane:

- In the cell it is immediately beneath the cell wall.
- This structure is approximately 7.5 nm (0.0075 μm) thick and is composed primarily of phospholipids (20 to 30 %).
- It is in intimate contact with the inside of the cell wall.

Function:

- It has tremendous importance in life processes of cell.
- It controls the exchange of material between the cell and its environment.

- It acts as a true semi permeable membrane (low molecular weight) substance can pass through it.

(2) Cytoplasm:

- The liquid material within the cytoplasmic membrane.
- It is usually clear watery or slightly viscous substances such as stored food.
- It is divided in three parts.
    - Cytoplasm (materials around nucleus)
    - Nucleus or Naked DNA
    - Other inclusions
- Cytoplasm composed of organic and inorganic chemicals, carbon, nitrogen, hydrogen, sulphur, protein, fats and carbohydrates.

(3) Nucleus (Genetic material or naked DNA):

- Bacteria are prokaryotic microorganism hence; do not exhibit a well defined nucleus.
- It contains a diffused nucleus, chromatin, granules are spread out in cytoplasm, which contain circular loop of DNA and attached at one or more points to mesosome.
- The nucleus has been the hereditary characters.

(4) Granules:

They are considered to be a reserve source of food substances, highly refractive hence, easily observed, important for differentiation of bacterial species, their presence and absence may be significant in disease diagnosis of diphtheria.

Reproduction in bacteria

- Bacteria may either be in an active growing stage (vegetative stage) or in the form of resting cells.
- Actively growing bacterial cells may increase in numbers in several different ways, most commonly they multiplied by splitting cross walls i.e. by transvers fission.
- All true bacteria reproduce by this method, an asexual process known as ‘binary fission’.
- This method of multiplication is so common and characteristic of the bacteria.

Spore Production: Some genera of bacteria produce reproductive spores called conidiospores or sporangiospores at the tip of filamentous growth, each of these spores give rise to a new organism. Eg: *Streptomyces*

Physical Requirements

- **Temperature**
    - psychrophiles (cold loving microbes)
        - range 0 C - 20 C

- mesophiles (moderate temp. loving microbes)
    - range 20 C - 40 C
- thermophiles (heat loving microbes)
    - range 40 C - 100 C

- **pH**

Most bacteria grow between pH 6.5 - pH 7.5

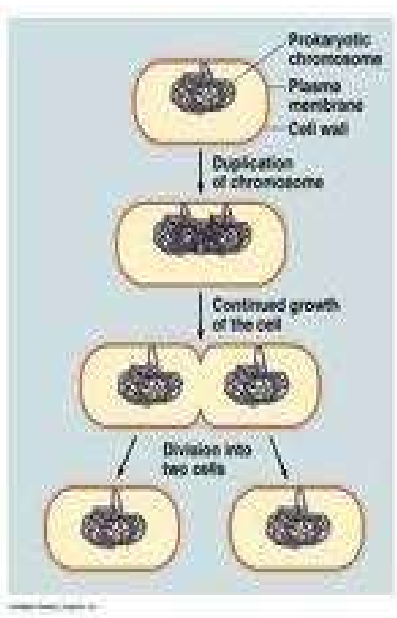
Very few can grow at below pH 4.0

Many foods, such as pickles, and cheeses are preserved from spoilage by acids produced during fermentation

Asexual reproduction:

1. Binary fission

Under favourable conditions the simple cells divide into two, after the critical size of cell and development of a transverse cell wall to separate the intracellular contents.

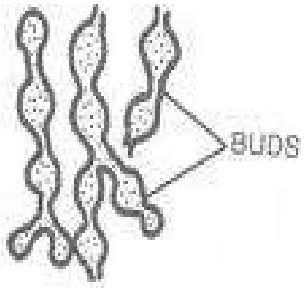


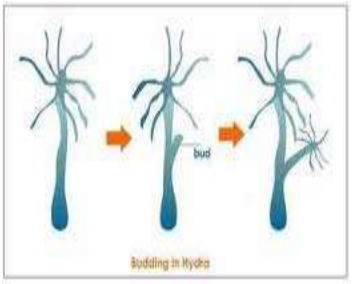
2. Budding

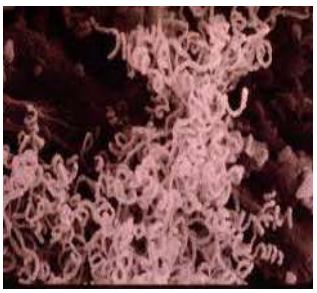
Some bacteria such as *Rhodopseudomonas acidophila* reproduce by budding in which small bud develops at one end of the cell. It enlarges and develops into new cell, which separate from parent cell.

3. Fragmentation:

Bacteria that produce extensive filamentous growth, such as *Nocrdia* spp. reproduce by fragmentation of the filamentous into small bacillary or cocci cell.







Generation time:

The time required for a bacterium to give rise to two daughter cells under optimum conditions is known as "Generation time" or population doubling time. It is different for different bacteria.

e.g. Bacteria **Generation time**

Escherichia coli	17 minutes
Bacillus mycodies	28 minutes
Mycobacterium tuberculosis	792-932 minutes

Growth of fungi is different from other unicellular organisms that divide either by binary fission or by budding. In filamentous fungi growth occurs only at the tips of filament and therefore is not exponential (Increasing very rapidly). Moreover, extensive branching occurs in fungi and new ends are generated at which growth occurs. Thus, fungi growth is difficult to describe in simple mathematical terms.

GROWTH CYCLE OF BACTERIA:

When a liquid medium is inoculated with unicellular bacteria or yeasts or other budding organisms, the population undergoes a characteristic sequence of events during the increase in cell number. When the number of cell /ml is determined periodically and plotted against time, a curve is formed showing four distinct phases of growth.

A- Lag phase / Acclimatization phase
B- Log phase / exponential phase / Logarithmic phase
C- Stationary phase
D- Death phase / Declined phase

Lag Phase

Immediately after transferring into a fresh medium growth does not take place but only after a period of time called the 'lag phase'. This period may be brief or extended. There is no significant increase in the number of cells. However, cell growth occurs as indicated by increase in cell mass. This stage represents a period of active growth without cell division. The length of the lag phase depends on a variety of factors such as the age of the inoculum, the composition of the growth medium and the environmental factors such as temperature, pH and aeration etc. At the end of lag phase each organism divides. However, since not all organisms do not complete the lag period simultaneously, there is a gradual increase in the population until the end of this period when all cells are capable of dividing at regular intervals.

Log Phase

The rate of exponential growth is influenced by environmental conditions as well as by characteristics of the organism itself. During exponential phase cells are in a steady state. New cell material is synthesized at a constant rate but the new material is itself catalytic and the mass increases in an exponential manner. This continues upto a point when one or more nutrients in the medium become exhausted or toxic metabolic products accumulate and inhibit growth. For aerobes, once cell population reaches 1 X 107 cells /ml, the growth rate will decrease unless O2 is forced into the medium by agitation or bubbling in air. When the cell concentration reaches 5 X 109 / ml the rate of oxygen diffusion cannot meet the O2 demand even in an aerated medium and growth is progressively slowed

The time taken for log phase is different for different microorganisms. Some bacteria take 20-30 minutes to grow, some soil bacteria take about 60-50minutes. Bacteria like *Nitrisomonas, Nitrobacter normal few hours, Mycobacterium tuberculosis take about* 12-24 hours to grow. Metabolites which are produced during log phase are known as Primary metabolites. Primary metabolites are producing only during this phase and are required for cell division and growth.

Stationary Phase

There is a slow loss of cells through death, which is just balanced by the formation of new cell through the growth and division. When this occurs, the total cell count slowly increases although the viable count stays constant. Some spore forming bacteria, form endospore when they reach stationary phase if they are resistant to lysis or death. In such cases the number of viable cells will remain constant after attaining the stationary phase and a phase of decline or death may not be seen. Certain cell metabolites called secondary metabolites are produced primarily in the stationary phase. Secondary metabolites include toxins, antibiotics, sterols etc. These products inhibit the growth and survival of other microorganisms and may give competitive advantage to the producer of Secondary metabolites

Death Phase

If incubation continues after a population reaches the stationary phase, the cells may remain alive and continue to metabolize, but often they die. If the latter occurs, the population is said to be in the death phase. The total count may remain constant while the viable count slowly

decreases. In some cases death is accompanied by cell lysis leading to decrease in total count. As there is no food materials available, accumulation of toxic material leads to death of microorganisms. The cells start dying exponentially and hence we can see a sharp declined in the growth curve. After a majority of cells are dead, a small number of survivors may persist for months or even years which may be due to the growth of a few cells at the expense of nutrients released from cells that die and lyse.

Introduction to plant viruses

(Viruses dominate biosphere: there are 10-100 viruses per each living cell)

Defination:

Viruses: The word '**virus**'is derived from Latin language and it means **poison**, **venom** or **infectious matter**. Viruses are very small (submicroscopic) infectious particles (virions) composed of a protein coat and a nucleic acid core.

Viruses are considered as mesobiotic (in between living and non-living) agents.

Size of virus: Viruses are the smallest among all known organisms. The typical diameter of a spherical plant virus is 30 nm. Some of the filamentous viruses reach the length of 2000 nm or 2 μm.

Viral Structure: Virions are complete, fully developed viral particles composed of nucleic acid surrounded by a protein coat. Some viruses have an envelope composed of a phospholipid bilayer with viral glycoproteins.

Plant virus particles consist of infectious nucleic acid (the genome), which is encapsidated within a protective protein coat or shell. The genome, essential for virus replication, is composed of ribonucleic acid (RNA in most groups of viruses) and deoxyribonucleic acid (DNA in the caulimovirus and geminivirus groups). The RNA and DNA may be single stranded (ss) or double stranded (ds) Besides these two basic components, an envelop of lipid or lipoprotein membrane is present in some plant viruses. Other components are metallic ions and polyamines present in varying amounts. Some enzymes are found in reoviruses and rhabdoviruses. Water constitutes 10-50 percent of the mass of virus particle.

The nucleic acid may be present as a single continuous strand (single molecular species) in a particle. It is called mono-partite genome. Some nucleic acid genomes have two or more pieces (molecular species) in different particles; usually they are not always encapsidated within separate protein shells. Such genomes are termed as bi-, tri-, or multi-partite or the viruses with divided genome.

In some RNA viruses, the genetic information is divided into two or more parts. They are called multi-component viruses and the individual components are not infectious alone. Hence two or more genomic elements are needed to cause infection and replication.

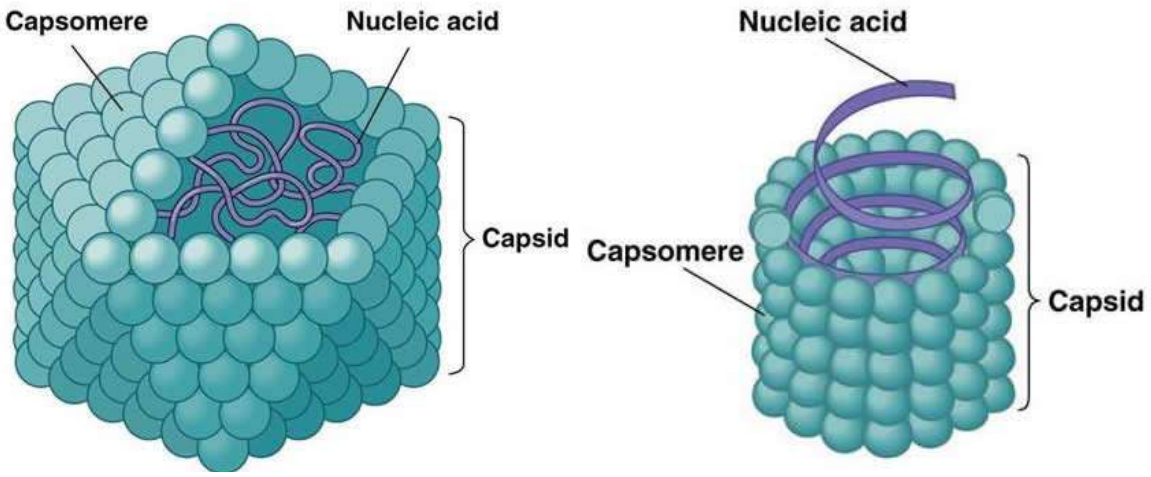
The genomic organization of viruses depicts structure and function of genes or cistrons. Some triplet bases called codons are responsible for expression of genes. There are two types of codons, (i) initiation codons (AUG, GUG) and (ii) termination codons (UAG, UAA, UGA) and they control functions of genes and translation products.

1) Nucleic acid

- Viral genomes are either DNA or RNA (not both).
- Nucleic acid may be single- or double-stranded
- Nucleic acid may be circular or linear or separate molecules.
- Nucleic acid ranges from about 5- 40%.

2) Capsid

- Capsid - protein coat
- Capsomeres are subunits of the capsid



Icosahedral **Helical**

Morphology : The protein shells of plant viruses (capsids) are assembled in different types of symmetry.

a) Helical (roughly elongated). The elongated viruses come in two major variants, rigid rods **(Figure 1)** and flexuous filaments **(Figure 2)**. In both of these variants, the nucleic acid is highly ordered: it assumes the same helical conformation as the proteinaceous capsid.

b) Icosahedral (roughly spherical; **Figure 3**)

c) Bacilliform virions **(Figure 4)** and

d) Twin virions composed of two joined incomplete icosahedra **(Figure 5)**.

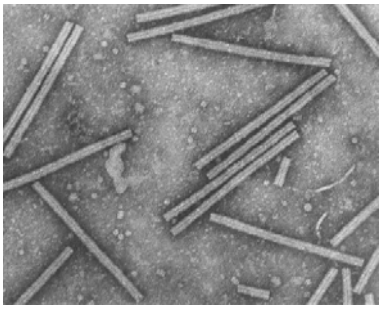


Fig 1. Rod-shaped particles

Fig 2. Flexuous filamentous particles

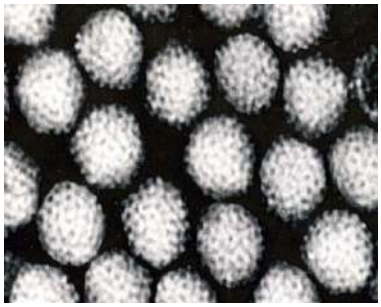


Fig 3. Icosahedral particles

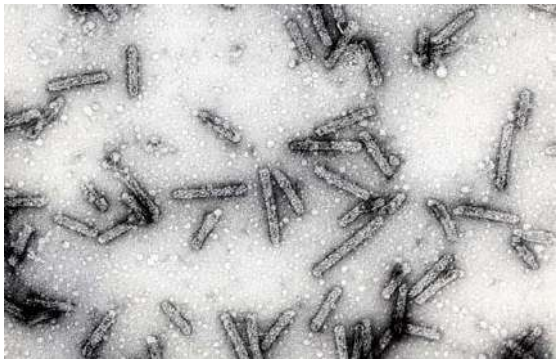


Fig 4. Bacilliform particles

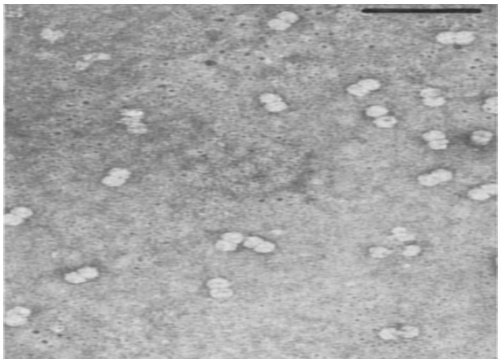
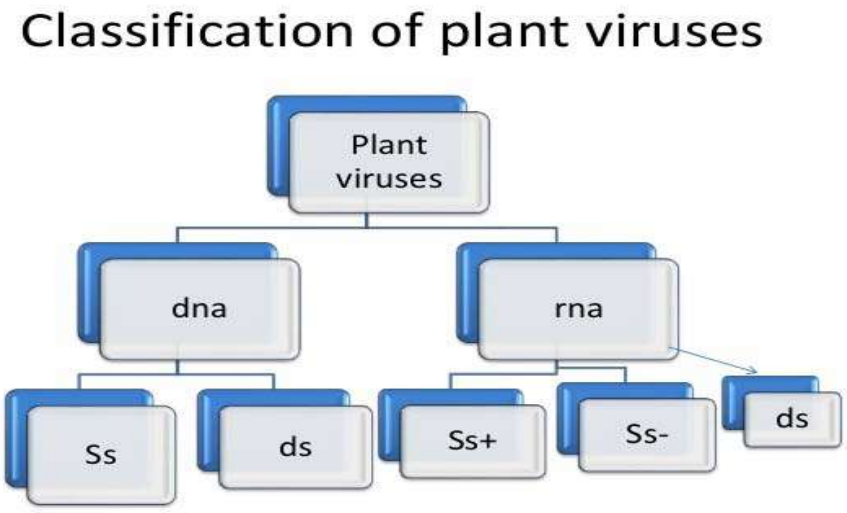


Figure 5. Twin virus particles

Classification of plant viruses :

Scientists made several attempts to device a rational, taxonomically sound classification system. Originally the virus are recognized by the symptom they caused and name of host to which it is associated.

The most useful classification is by the type of nucleic acid, the particular virus contains and its mode of expression. This classification was proposed by Nobel prize **winner David** Baltimore. It should be noted that none of these classifications are expected to be phylogenetic, as viruses may not share common origin. Following flowchart depicts the classification based on type of nucleic acid and strandedness.



Example of different groups of virus:

a) ds DNA virus : Cauliflower mosaic virus
b) ss DNA virus : Tomato leaf curl virus
c) (+) sense ss RNA virus : Rice tungro spherical virus
d) (-)sense ss RNA virus : Potato yellow dwarf virus
e) ds RNA virus : Wound tumour virus

Study questions:

1. Name the scientist who discovered virus.
2. Name the first virus discovered
3. what is the meaning of virus
4. Name the double stranded DNA virus.

Introduction to Fungi

What is a fungus?

A fungus is a eukaryote that digests food externally and absorbs nutrients directly through its cell walls. Most fungi reproduce by spores and have a body (thallus) composed of microscopic tubular cells called hyphae. Fungi are heterotrophs and, like animals, obtain their carbon and energy from other organisms. Some fungi obtain their nutrients from a living host (plant or animal) and are called biotrophs; others obtain their nutrients from dead plants or animals and are called saprotrophs (saprophytes, saprobes). Some fungi infect a living host, but kill host cells in order to obtain their nutrients; these are called necrotrophs.

How old are fungi?

Fungi are an ancient group—not as old as bacteria, which fossil evidence suggests may be 3. 5 billion years old—but the earliest fungal fossils are from the Ordovician, 460 to 455 million years old.

Economic importance of fungi : The *fungi* are of great *economic importance* on account of their both harmful as well as beneficial affects. A large number of fungi cause destructive havoc to our valuable crop and timber plants, various lines of food products. They also attack the live-stock as well as human beings. But, all of them are not harmful to the mankind, as most of the species bring about decomposition of dead bodies of plants and animals as well as of animal dung.

a). Harmful economic aspects of fungi:

i) ***Spoilage of food, stored grains and other essential commodities:*** Fungi (mould fungi) are the chief agents responsible for most of the disintegration of fruits, vegetables and other done stuffs, e.g. jams, jelly, pickles, bread, various cooked-foods eat. if not properly stored. Common food damaging fungi are *Aspergillus*, *Penicillium*, *Mucor* and *Rhizopus*. About 25 -50% of harvested fruits and vegetables are destroyed by fungi.

ii) **Induce diseases of plants**: Fungi are also responsible for millions of rupees worth of damage to crops by causing plast diseases. Of the serious diseases of crop plants, late blight disease of potato plant caused by *Phytophthora infestans*; *Claviceps purpurea* (Ascomycotina) causes the 'ergot' disease of rye. The members of 'rust' (*Puccinia*) and 'smut' (*Ustilago*, *Tilletia*, etc.) are also highly destructive to various different crop plants like wheat, maize, oct, barley etc. Many fungi cause immense loss to timber trees by causing wood-rot disease.

iii) Induce diseases of animals and human being**:** Fungi are responsible for the diseases of animals as well as human beings. Some of the serious diseases of human being, i.e. the diseases of skin, far, throat, nose and as well as bronchial and intestinal disorders are caused by various groups of fungi. A few species of *Mucor* and *Rhizopus* are common fungi infecting lungs, brain and gastric system, *Neurospora* and *Fusarium* infect corneal tissue of eye. Several species of *Aspergillus* are human pathogens, causing a group of diseases collectively called Aspergilloses.

iv) Toxic substances obtained from fungi: Several toxins have been extracted from mushroom like *Amantia phalloides*, *Aspergillus flavous* and *Aspergillus niger* may infest dried foods and ground out meal and produce a dangerous toxins called aflatoxin-- this toxin has carginogenic effect.

v)Wood-rotting fungi: Many wood rotting fungi e.g. a few species of *Lentinus*, *Serpula lacrymans*, *Coniophora cerebella*,etc. cause a great monetary loss. Dry rot and wet rot are most dangerous diseases of the timber.

vi)*Fungi growing in Aircraft fuel tanks:* *Amorphotheca resinae* is the main organism of the kerosene-based fuel tanks of aircrafts. This fungus grows in storage and fuel tanks profusely as a result fungal hyphae may plug the valves and pipes of the aircraft and cause disastrous effect during flight.

B. Beneficial economic aspects of fungi:

i) ***In the production of antibiotic substances, alkaloids and organic acids*****:** Most of the fungi produce enzymes, alkaloids and various other organic compounds as their metabolic products; there metabolic products are of great economic importance. One of the important metabolic products produced by fungi is the antibiotics. In 1929 Sir Alexander Fleming for the first time established the fact that certain fungi could produce antibiotics. The most wonder drug 'penicillin' is an antibiotic substance extracted from Penicillium notatum.

ii) ***Fungi used as food*****:** Some fungi e.g. 'mushrooms' (Agaricus campestris, A. bisporus, Volvariella volvacea, Pleurotus sajar-caju, etc.), 'morels' (Morchella esculenta) etc. are used as human food. Well known yeast cake is obtained from Saccharomyces cerevisiae - yeast protein is called commonly 'single cell protein', and its protein is above 40% ; yeast cake is produced from Saccharomyces cerevisiae. A popular food known as 'sufu' is produced from Mucor, Antimucor, etc.

iii) ***To maintain the soil fertility*****:** Many saprophytic fungi along with bacteria decompose the dead organic matters and thereby hey help in returning the nutrients (derived from the organic matters) to the soil in a form available to green plants.

v) ***In biological control of soil-borne pathogens and insects*****:** Some fungi (Arthobotrys musiformis, Dactylaria thaumasia, etc.) are known are predaceous fungi, because they parasitise amoebae, nematodes etc. - such fungi are used in large scale for controlling soil borne pathogens, reagak nematodes.

GENERAL FEATURES OF FUNGI:

- ✓ Fungi are cosmopolitan in nature.
- ✓ They are eukaryotic
- ✓ They are heterotrophic in nutrition
- ✓ They may be saprophytes, parasite or symbionts.
- ✓ Thallus may be plasmodium, pseudoplsmodium or mycelia.
- ✓ They may be unicellur or filamentous multicelluar.
- ✓ Cell wall is primarily composed of chitin and glucans, although the walls of some species contain cellulose.
- ✓ They lack chlorophyll pigments and are incapable of photosynthesis.
- ✓ Life cycle may be simple to complex.
- ✓ Reproduce by Sexually, asexually, parasexually.

MORPHOLOGICAL CHARACTERISTICS OF FUNGI:

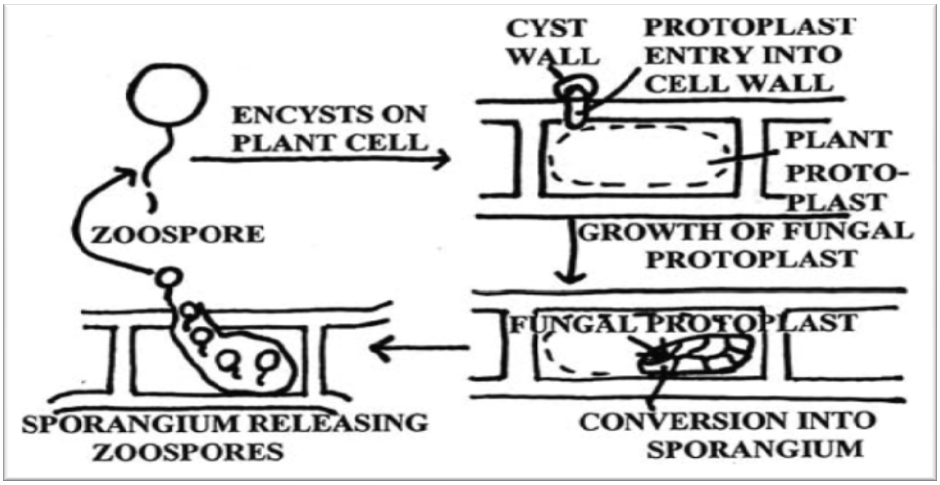
Characters	Remarks
Eukaryotic	Possess membrane-bound nuclei (containing chromosomes) and a range of membrane-bound cytoplasmic organelles (e.g. mitochondria, vacuoles, endoplasmic reticulum).

Distribution	Cosmopolitan
Habit	May be free-living or may form intimate relationships with other organisms i.e. free-living, parasitic or mutualistic (symbiotic).
Filamentous /Unicellular	Most are filamentous, composed of individual microscopic filaments called hyphae, which exhibit apical growth and which branch to form a network of hyphae called a mycelium. Some are unicellular : e.g. yeasts.
Thallus	Plasmodial, pseudoplasmodial, pseudomycelial,mycelial.
Cell Wall	Composed primarily of chitin and glucans, although the walls of some species contain cellulose (Oomycetes).
Nuclear Status	Multinucleate, homo or heterokaryotic, haploid (n), diploid (2n), dikaryotic (n+n).
Achlorophyllous	They lack chlorophyll pigments and are incapable of photosynthesis
Chemoheterotrophic (chemo-organotrophic)	They utilise pre-existing organic sources of carbon in their environment and the energy from chemical reactions to sythesise the organic compounds they require for growth and energy.
Storage compounds	Trehalose, glycogen, sugar alcohols and lipids.
Life cycle	Simple and complex
Reproduction	Sexual, asexual, parasexual reproduction often result in the production of spores.

Somatic structure

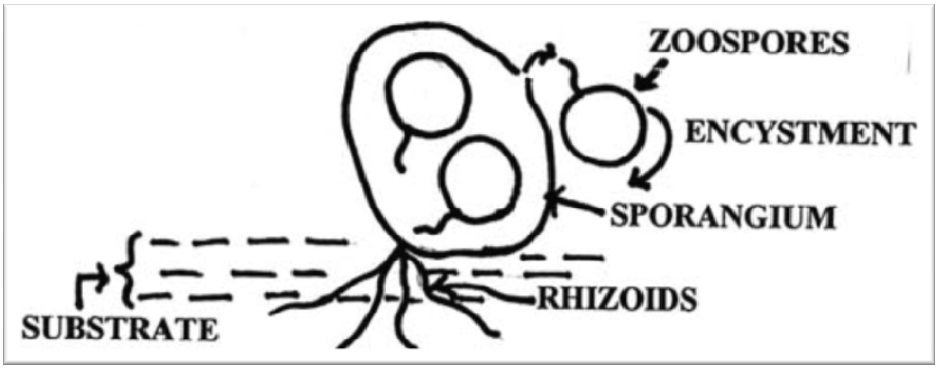
1) **Thallus :** Entire body of fungus is called thallus (pl. thalli; Gr.thallus = shoot or sprout). The vegetative/somatic thallus gives rise to reproductive structures, from which the spores/propagules are produced either sexually or asexually. Thallus may be:
   1. Plasmodium
   2. Pseudoplasmodium
   3. Mycelium
   4. Pseudomycelium

a) **Holocarpic/Unicellular thallus:** Holocarpic (Gr. *holos* = entire+ *karpos* = fruit) thallus comprises of single, spherical, ellipsoidal or irregular shape microscopic cell with true cell wall. Whole thallus is converted into one or more reproductive structures and hence the reproductive and vegetative don't occur in the same thallus. Eg. *Yeast*, *Synchytrium* and *Olpidium*.

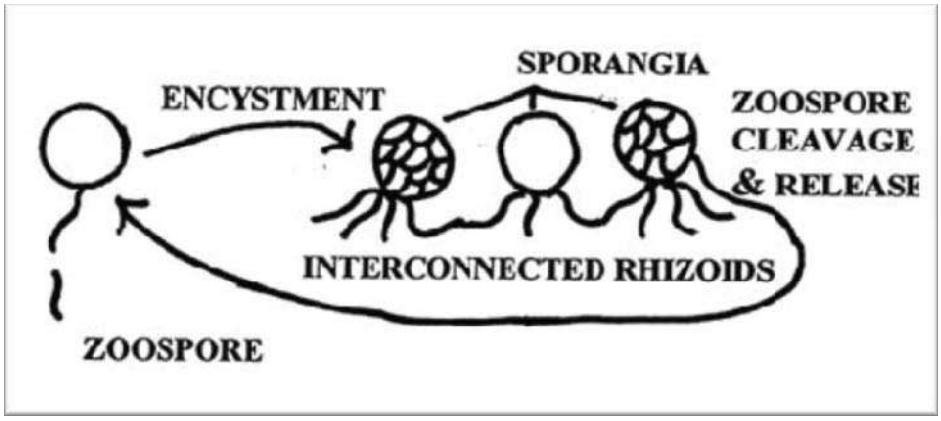


b) **Eucarpic/Filamentous thallus :**In eucarpic (Gr. *Eu* = good + *karpos* = fruit) thallus**,** only a portion gives rise to reproductive organ, while remaining portions continue to grow vegetatively. Hence vegetative and reproductive phases occur together. Eg. Oomycota, Mucoromyccota, Ascomycota, and Basidiomycota.

i) Monocentric (monocentrism) :Thallus consists of single reproductive cell attached to substrate hyphae.



ii) **Polycentric (polycentrism)** : Thallus has many reproductive centers.



c) **Plasmodium :** Thalli consisting of naked motile multinucleate mass of protoplasm that feeds in an amoeboid fashion, called plasmodium (pl. plasmodia; Gr. *Plasma* = moulded object). **Eg. *Plasmodium brassicae***

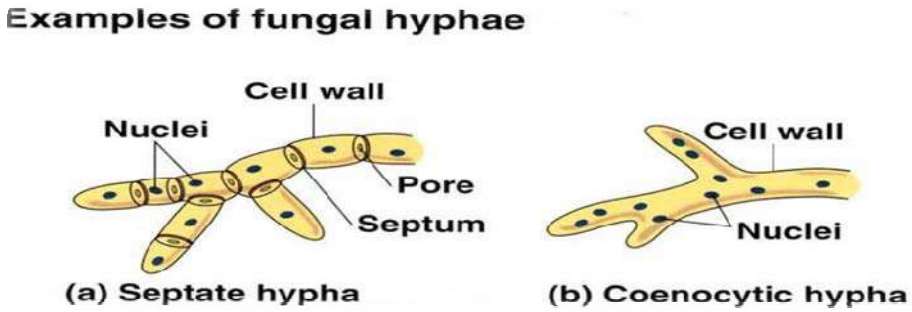
Another type of amoeboid plasmodium formed by aggregation of unicellular, uninucleate naked cells are called as **pseudoplasmodium**.

d) **Pseudomycelium (pl. pseudomycelia; Gr. *pseudo* = false+mycelium):** Thallus with series of cells adhering end to end due to incomplete cell wall separation after budding are called pseudomycelium. eg. Yeast

2) **Hypha (pl. hyphae;Gr. 'hyphe' means web):** It is a long, tubular, thread like filamentous and branched structure. Hypha may be:

a) Septate : With cross wall or septum (pl. septa; L. *septum* = partition) that divide the hypha into segments having single nuclei in each segment. Transverse or longitudinal septum forms at regular interval behind the hyphal tip divide the hypha into segments.

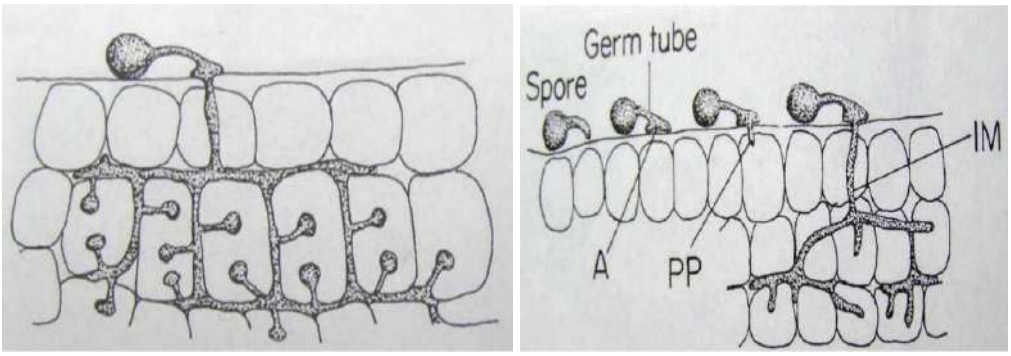
b) Aseptate : Without cross wall having multiple nuclei throughout the segment. Aseptate and multinucleate hyphae are also called 'coenocytic'(Gr. *Koinos* =common + *kyotos* = a hollow vessels).



3) **Mycelium (pl. Mycelia, Gr. *myke* = mushroom) :** The hyphal mass or network of hyphae constituting the body (thallus) of the fungus is called as mycelium. It may be :

a) **Intercellular mycelium** : The mycelium of parasitic fungi grows on the surface of the host and spread between the cells and it is called intercellular mycelium.

b) **Intracellular mycelium** : The mycelium of parasitic fungi, which grows on the surface of the host and penetrates into the host cells and is called intracellular mycelium.



c) **Hyaline (Gr. *Hyalinos* = made of glass) :** Mycelium is colourless, transparent.

d) **Coloured mycelium :** Mycelium is pigmented.

e) **Monokaryotic mycelium (uninucleate) :** Mycelium contains single nucleus that usually forms part of haplophase in the life cycle of fungi.

f) **Dikaryotic mycelium (binucleate) :** Mycelium contains pair of nuclei (dikaryon), which denotes the diplophase in the life cycle of fungi.

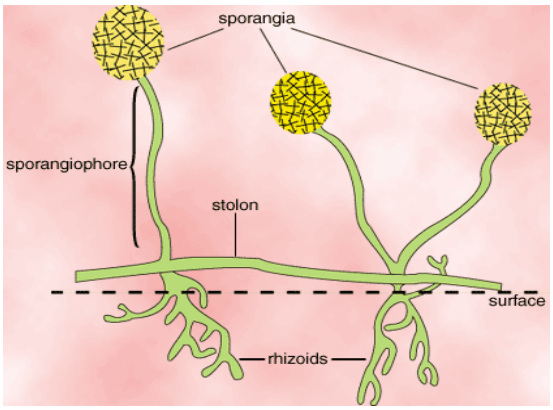
4) **Specialized Somatic Structures :**

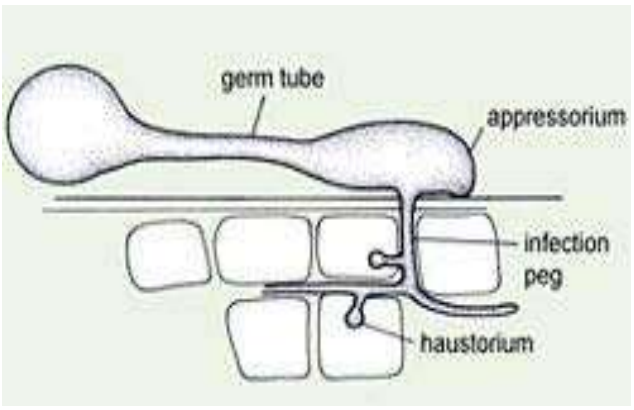
a) **Rhizoid** :A rhizoid (Gr. *rhiza* = root + *oeides* = like) is a short, root-like filamentous outgrowth of the thallus generally formed in tufts at the base of small unicellular thalli or small porophores. Rhizoid serves as anchoring or attachment organ to the substratum and also as an organ of absorption of nutrients from substratum. Rhizoids are short, delicate filaments that contain protoplasm but no nuclei. Rhizoids are common in lower fungi like Chytridiomycetes, Oomycetes and Zygomycetes.

b) **Appressorium :** Appressorium (p1. appressorium; L. *apprimere* = to press against) is a simple or lobed structure of hyphal or germ tube and a pressing organ from which a minute infection peg usually grow and enter the epidermal cell of the host. It helps germ tube or hypha to attach to the surface of the host or substrates. In addition to giving anchorage, appressoria help the penetrating hyphae, branches to pierce the host cuticle.

c) **Haustoria :** Haustoria (sing. haustorium; L. *haustor* = drinker) are special hyphal structures or outgrowths of somatic hyphae sent into the cell to absorb nutrients. Haustoria may be knob-like or balloon – like in shape, elongated or branched like a miniature root system. The hyphae of obligate parasites of plants like downy mildew, powdery mildew or rust fungi late blight fungus etc ., produce haustoria.

d) **Hyphopodia** : Hyphopodium (pl. hyphopodia Gr. *hyphe* = web + *pous* = foot) is a small appendage with one or two cells in length on an external hypha and function as absorbing structures. The terminal cell of hyphopodium is expanded and rounded or pointed.





5) Aggregations and modifications of hyphae and tissues :

a) Rhizomorph : Rhizomorph (Gr. *rhiza*=root + *morphe* = shape) is the aggregation of highly differentiated hyphae with a well defined apical meristem, a central core of larger, thin walled, cells which are often darkly pigmented. These root-like aggregation is found in the honey fungus or honey agaric *Armillariella mellea (=Armillaria mellea).*

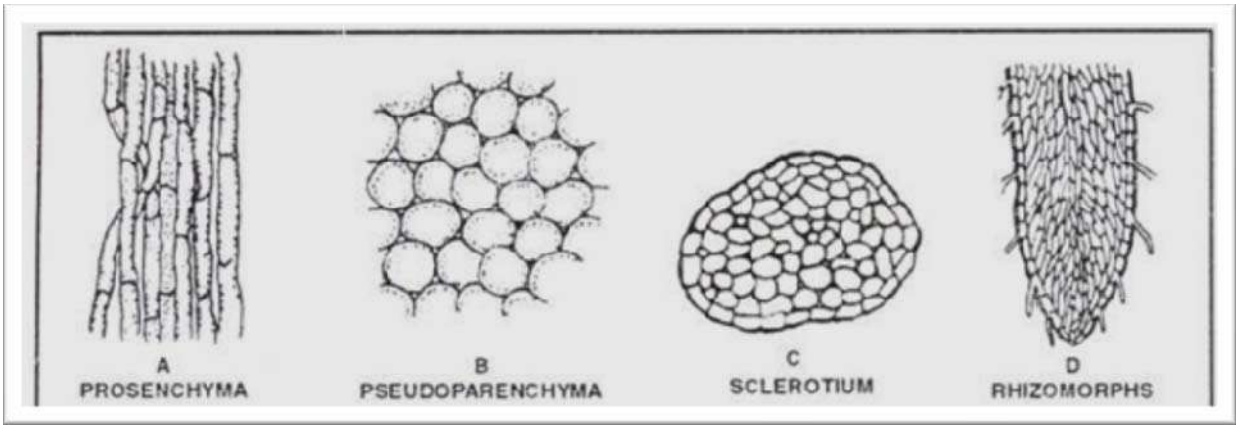
b) Plectenchyma : Fungal mycelium becomes organized into loosely or compactly woven tissues. These organized fungal tissues are called plectenchyma (Gr. *plekein* = to weave + *encyma* = infusion i.e., a woven tissue). There are two types of plectenchyma viz.,

i) Prosenchyma : When the tissue is loosely woven and the hyphae lie parallel to one another it is called prosenchyma (Gr. *pros* = toward + *enchyma* = infusion, i.e., approaching a tissue). These tissues have distinguishable and typical elongated cells.

ii) Pseudoparenchyma (Gr. *Pseudo* = false): Consists of closely packed, more or less isodiametric or oval cells resembling the parenchyma cells of vascular plants. In this type of tissues hyphae lose their individuality and are not distinguishable.

c) Stroma (Gr. *stroma* = mattress) : A stroma is a compact, somatic structure or hyphal aggregation similar to a mattress or a cushion, on which or in which fructifications of fungi are usually formed. They may be of various shapes and sizes. Hyphal masses like acervuli, sporodochia, pionnotes etc. are the fertile stromata, which bear sporophores producing spores.

d) Sclerotium (Gr. *Skleros*=hard): These are hard dormant bodies consist of compact hyphae protected by external thickened hyphae. Each Sclerotium germinates into a mycelium, on return of favourable condition, e.g., *Slcerotium*, *Penicillium.*



Study questions:

1. List the characters of fungi
2. Write in detail the economic importance of Fungi.
3. List the somatic structures of Fungi along with examples and explain any one.
4. List the Different types of Types thallus with example and explain anyone
5. Define: Mycelium, Appressorium, Holocarpic, Hypha, Haustorium, Rhizomoprh, sclerotia, Rhizoid, , Plasmodium, Eucarpic, ectophytic, endophtyic, endobiotic, stroma, coenocytic hypha,
6. Give the examples for following of organism producing following structures: coenocytic hypa, Rhizoid, Septate hypha, Holocarpic, Eucarpic, Ectophytic, endobiotic, plasmodium, Pseudoplasmodium, Sclerotium, haustoria.
7. Write the function of following structures: Rhizoid, Aprressorium, Haustorium, Hyphopodia, rhizomorph, rhizomycelium, sclerotia.
8. Differentiate

    1. Holocarpic and Eucrpic
    2. Moncentric and polycentric
    3. Ectophytic and endophytic
    4. Intercellular and intracellular
    5. Rhizoid and Rhizomycelium

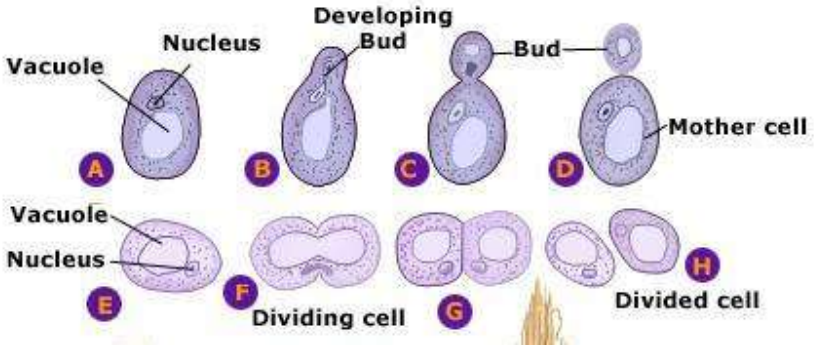
6) REPRODUCTION: Reproduction is the formation of new individuals having all the characteristics typical of a species. The fungi reproduce by means of asexual and sexual or parasexual reproduction. Asexual reproduction is sometimes called somatic or vegetative and it does not involve union of nuclei, sex cells or sex organs. The union of two nuclei characterizes sexual reproduction.

a) Asexual reproduction: In fungi, asexual reproduction is more important for the propagation of species. Asexual reproduction does not involve union of sex organs (gametangia) or sex cells (gametes) or nuclei. In fungi the following are the common methods of asexual reproduction.

i). Fragmentation of mycelium: Mycelial fragments from any part of the thallus may grow into new individuals when suitable conditions are provided.

ii) Fission of unicellular thalli: It is also known as transverse cell division. Reproduction by the method of fission is in fungi. Fission is simple splitting of cells into two daughter cells by constriction and the formation of a cell wall. It is observed in *Schizosaccharomyces* spp.

iii) Budding: Budding is the 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, while still attached to the parent cell and eventually breaks off and forms a new individual. It is common in yeasts.(*Saccharomyces* sp).



iv) Production of asexual spores : Reproduction by the production of spores is very common in many fungi. The term 'spore'(Gr. spora=seed, spore) is applied to any small propagative, reproductive or survival unit, which separates from a hypha or sporogenous cell and can grow independently.

There are different types of asexual spores produced by the fungi viz. endogenous (Sporangiospores) and exogenous (Conidiospores and chlamydospores).

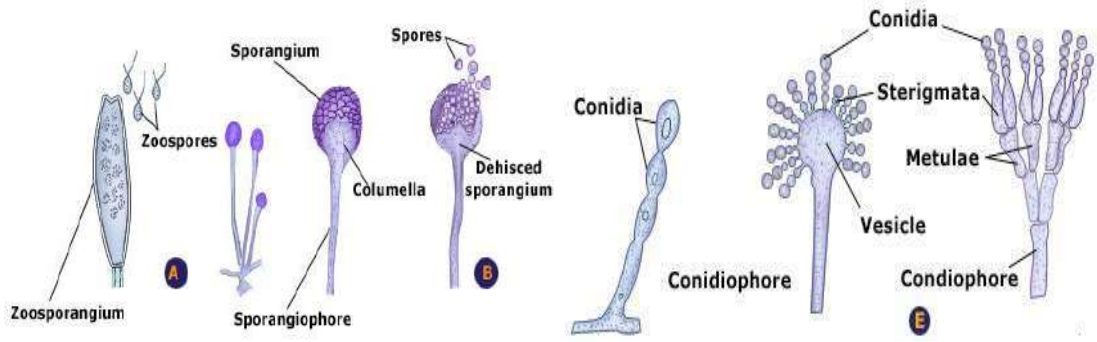
- **Sporangiospores:** Sporangiospores are formed in globose or sac-like structure called sporangium (pl. sporangia; Gr. Spora = *seed*, spore + *angeion* = vessel). Sporangiospores may be motile (planospores) also known as zoospores or non- motile spores (aplanospores). The zoospores are provided with one or two flagella (sing. flagellum, L. *flagellum*=whip) for its movement in the surrounding film of water. Flagellum is a hair like structucre that serves to propel a motile cell. These flagella may be anterior, posterior or laterally attached to a groove in the body. There are two types of flagella in zoospores. They are whiplash and tinsel types.

The whiplash flagellum has a long rigid base composed of all the eleven fibrils and a short flexible end formed of the two central fibrils only. The tinsel flagellum has a rachis, which is covered on all sides along its centre length with short fibrils.

- **Conidiospores:** Conidiospores or conidia (sing. Conidium; Gr. *konis*= dust)) are asexual reproductive structures borne on special spore bearing hyphae conidiophores. They are found in many different groups of fungi, but especially in ascomycotina, Basidiomycotina and Deuteromycotina. Conidia may be formed in acropetal (oldest conidium at the base and the youngest at the apex) or basipetal (oldest conidium at the apex and youngest at the base) succession. Generally the term 'conidia'is used for any asexual spores other than sporangia and spores formed directly by hyphal cells. When the spore is not much differentiated from the cells of the conidiophore in shape the term oidium is often used for conidia.

- **Arthrospores or oidia** (Gr. *Arthron* = joint): Produced by fragmentation of hyphae from apex to base. Each cell thus formed round off and separates as a spore. Eg. *Erysiphe* spp. Powdery mildew fungus.
- **Chlamydospores** Chlamydospore (Gr. *Chlamys* = mantle + *spora* = seed, spore) is a thick-walled thallic conidium that generally function as a resting spore. Terminal or intercalary segments or mycelium may become packed with food reserves and develop thick walls. The walls may be colourless or pigmented with dark melanin pigment. These structures are known as chlamydospores. e.g. *Fusarium, Mucor racemosus, Saprolegnia.* Generally there is no mechanism for detachment and dispersal of chlamydospores. They become separated from each other by the disintegration of intervening hyphae.

They are the important organs or asexual survival in soil fungi. When chlamydospores are found in between fungal cells they are called 'intercalary chlamydospores'. Chlamydospores produced at the apex of the hypha are called 'apical or terminal chlamydospores'.



b) Sexual reproduction: Sexual reproduction in fungi involves union of two compatible nuclei. The nuclei may be carried in motile or non-motile gametes, in gametangia or in somatic cells of the thallus.

Phases of sexual reproduction: Three typical phases occur in sequence during the sexual reproduction.

i). Plasmogamy: In plasmogamy (Gr. *plasma*=a molded object, i.e. a being + *gamos* = marriage, union) anastomosis of two cells or gametes and fusion of their protoplasts take place. In the process the two haploid nuclei of opposite sexes (compatible nuclei) are brought together but eh nuclei will not fuse.

ii). Karyogamy : The fusion of two haploid nuclei brought together as a result of plasmogamy is called karyogamy (Gr. *karyon* = nut, nucleus + *gamos* = marriage). This stage follows immediately after plasmogamy in many of the lower fungi or may be delayed in higher fungi.

iii). Meiosis : Karyogamy results in the formation of a diploid (2n) nucleus. Meiosis (Gr. *meiosis*=reduction) reduces the number of chromosomes to haploid and constitutes the third phase of the sexual reproduction. This nucleus undergoes a reduction division to form two haploid nuclei each with 'n'chromosomes. A mitotic division follows and four nuclei are formed. In ascomycetes another nuclear division takes place resulting in the formation eight nuclei. The nuclei get surrounded by a small amount of cytoplasm and secrete a wall to become spores.

Organs involved in sexual reproduction

Fungi, which produce morphologically distinguishable male and female sex organs in each thallus, are called hermaphroditic (Gr. *hermes* = the messenger of the Gods, symbol of the male sex + aphrodite = the Goddess of love, symbol of female sex) or monoecious or *unisexual* (Gr. *monos* = single, one + *oikos* = dwelling, home). A single thallus of a monoecious fungi can reproduce sexually by itself if it is self-compatible. In a fungus when the female and male organs are produced on two different thalli it is said to be dioecious or bisexual. (Gr. *dis* = twice, two + *oikos* = home; i.e.; the sexes separated into two different individuals). Normally a single thallus of a dioecious fungus cannot reproduce sexually by itself as the thallus is either male or female. The sex organs of fungi are called gametangia (sing. gametangium; Gr. *gametes* = husband + *angeion* = vessel, container). Sex cells are called gametes and the mother cells (sex organs) are called gametangia. If the gametes and gametangia produced are morphologically identical or similar they are called as isogametes (Gr. *ison* = equal) and isogametangia respectively. When the gametes and gametangia produced differ in size and structure (morphologically different) they are called heterogametes (Gr. *heteros* = other, different) and heterogametangia respectively. In the latter case, the male gametangium is called **antheridium**

(pl. antheridia; Gr. antheros = flowery + *idion,* dimin. suffix) and the female gametangium is called **oogonium** (pl. oogonia; Gr. *oon* = egg + *gonos* = offspring). The male gamete is known as antherozoid or sperm and the female as an egg or oosphere.

Methods of sexual reproduction:

The following are the five methods, which the fungi employ to bring the compatible nuclei together for fusion.

1.Planogametic copulation

2.Gametangial contact (Gametangy)

3.Gametangial copulation (Gametangiogamy)

4.Spermatization

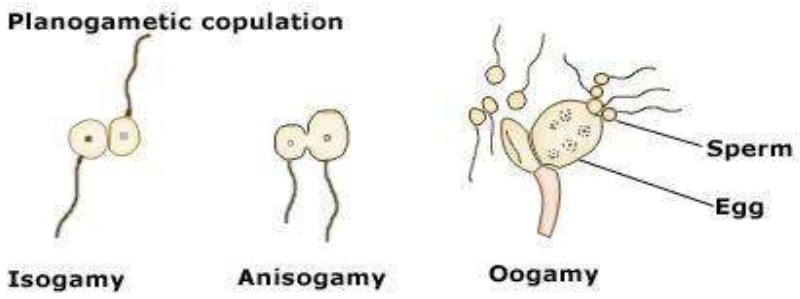
5. Somatogamy

1. **Planogametic copulation or conjugation** : A planogamate is a motile gamete or sex cell. The fusion of two gametes, one or both of which are motile is called planogametic copulation. This type of sexual reproduction is common in aquatic fungi. There are three different types of planogametic copulation.

a. **Copulation of isogamous motile gametes:** In this type morphologically similar but compatible type of mating type of gametes unite to form a motile zygote. e.g. *Synchytrium.*

b. **Copulation in anisogamous motile gametes** : It involves union of one larger gamete with another smaller gamete. The resultant zygote is motile. The zygote resulting from isogamous or anisogamous planogametic copulation forms a 'resting sporangium'. On further development it functions as sporangium by differentiating zoospores internally. Eg. *Allomyces* sp.

c. **Heterogamous planogametic copulation:** In this type, a non-motile female gamete (oosphere) is fertilized by a motile male gamete. This results in the formation of oospores, a resistant structure and resting spore. Oospores germinate and produce mycelium directly. Eg. *Monoblepharis.*

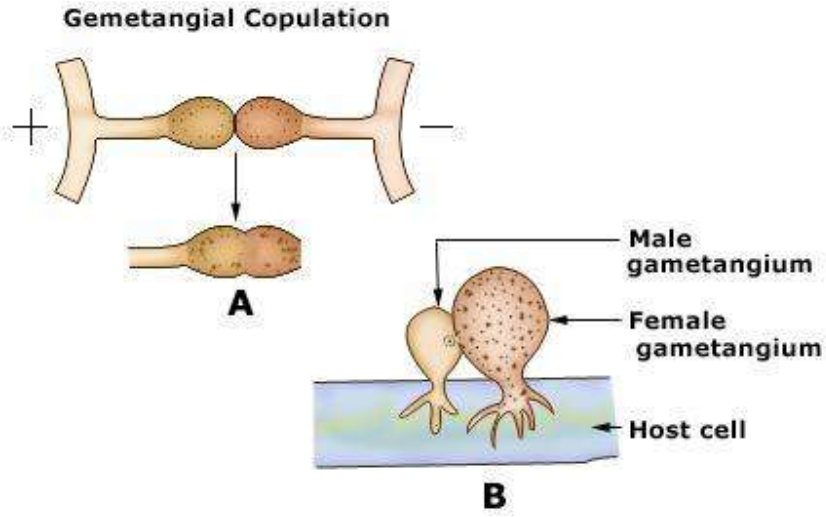


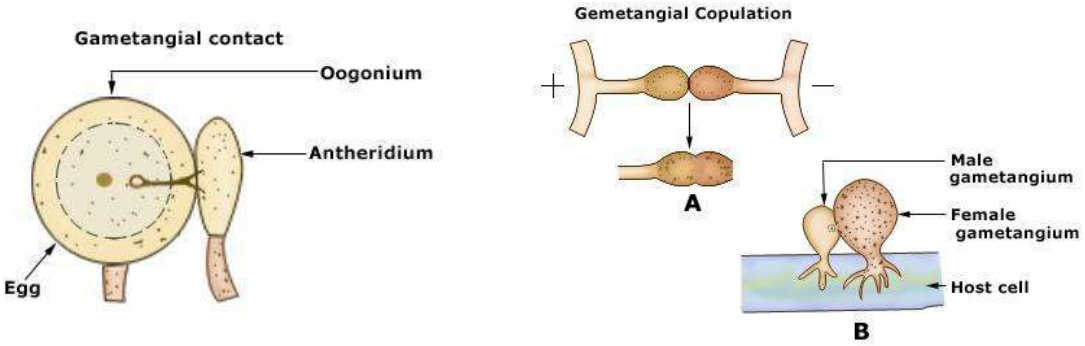
2. Gametangial contact : In this method the male gamete (antheridium) and the female gamete (oogonium) come in contact and one or more nuclei from the male gamete enter the female gamete, oogonium dissolved in the intervening wall through a pore or through a fertilization tube. In no case the gametangia actually fuse or lose their identify during the sexual act. e.g. Fungi in Peronosporales. Gametangial contact is also common in some Ascomycotina where antheridia and female organs (ascogonia) may or may not be well defined. Eg. *Pythium, Albugo, Phytophthora* etc

3. Gametangial copulation: This is a process of fusion of entire contents of the two mating gametangia. There are two types.

a. Fusion of entire protoplasm of male and female gametangia : Two gametangia meet and their entire contents fuse in the female gametangium leading to formation of a zygote. The zygote forms a resting sporangium. e.g.. *Mucor*

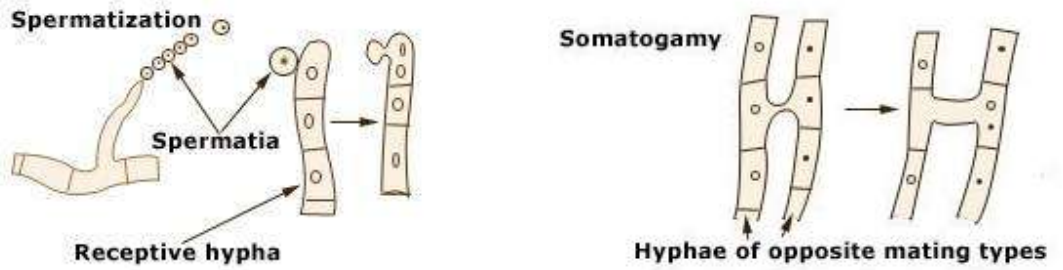
b. Migration of entire protoplast of one gametangium into other through a pore : The entire gametangia act as gametes. The recipient gametangium is called the female, while the gametangium that empties its content is the antheridium. Eg. *Rhizophidium*





4. Spermatization: Some fungi like rusts bear numerous minute, non-motile uninucleate, male cells called spermatia. (sing. spermatium; Gr. *spermation*=little seed) Spermatia are produced in spiral receptacles called spermagonia (sing. spermagonium; Gr. Sperma =seed, sperm+ gennao=I give birth) or pycnia (sing. pycnium; Gr. *pycnos*=concentrated). Insects, wind or water to the female gametangium carries them, which is usually a special receptive hypha (or trichogyne) to which they become attached. A pore develops at the point of contact and the contents of spermatium pass into the particular respective hyphae. This results in plasmogamy and initiation of the dikaryotic stage of the cell.

5. Somatogamy: In somatogamy to sex organs are produced and somatic cells function as gametes somatogamy (Gr. *soma* = body + gamos = marriage, union) hyphae anastomose and the nuclei of opposite matting type are brought together in one cell. Somatogamy is common in Ascomycotina and Basidiomycotina fungi.



Heterokaryosis

The phenomenon of existence of different kinds of nuclei in the same individual is known as heterokaryosis. (Gr. *heteros* = other+ *karyon*=nut, nucleus). The individual which exhibit heterokaryosis is called heterokaryon or heterokaryotic. It has been demonstrated in numerous Ascomycetes, Basidiomycetes and Fungi Imperfecti (Davis, 1966). In a heterokaryotic individual, each nucleus is independent of all other nuclei, but the structure and behaviour of the individual appear to be controlled by the kinds of genes it contains and the proportion of each kind. Heterokaryosis may arise in a fungal thallus in four ways:

1. By the germination of a heterokaryotic spore, which will give rise to a heterokaryotic soma.

2. By the introduction of genetically different nuclei into homokaryon (Gr. homo=same + karyon

= nut, nucleus), a soma in which all nuclei are similar.

3. By mutation, in a multinucleate homokaryon.The mutant nuclei subsequently survive, multiply and spread among the wild-type nuclei.

4. By fusion of some nuclei in a haploid homokaryon to form diploid nuclei which subsequently survive, multiply and spread among the haploid nuclei.

Parasexuality or parasexual cycle

Some fungi (Deuteromycetes) do not go through a sexual cycle but derive many of the benefits of sexuality through parasexuality (Gr. *para* =beside+sex). This is a process in which plasmogamy, karyogamy and haploidization takes place, but not at specified points in the thallus or the life cycle of an organism. Parasexual cycle is very important in Deuteromycetes where sexual reproduction does not take place. Some fungi, which reproduce sexually, also exhibit parasexuality. Pontecorvo and Roper from the University of Glasgow in Aspergillus nidulans, the imperfect stage of Emericella nidulans first discovered parasexuality in 1952. Since then it has been reported in number of fungi in Ascomycotina. The sequence of events in a complete parasexual cycle is as follows :

1. Formation of heterokaryotic mycelium,
2. Fusion between two nuclei
a. Fusion between like nuclei
b. Fusion between unlike nuclei
3. Multiplication of diploid nuclei side by side with the haploid nuclei
4. Occasional mitotic crossing-over during the multiplication of the diploid nuclei
5. Sorting out of diploid nuclei
6. Occasional haploidization of the diploid nuclei
7. Sorting out of new haploid strains

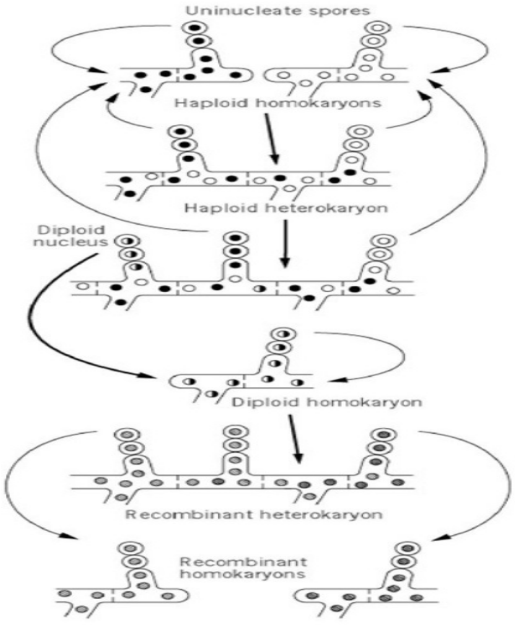


Fig.Parasexual life cycle

Sexual spores : The sexual spores are formed as a result of fusion between two opposite sex gametes. They are resting spores, incapable of germination immediately after formation. Sexual spores are oospores, zygospores, ascospores and basidiospores desiring their names from the class to which the fungi belong.

1. Oospores : An oospore (Gr.*oon* = egg + *spora* = seed, spore) is a sexually produced spore, which develops from unequal gametangial copulation or markedly unequal gametic fusion. It is the characteristic sexually produced spore of oomycetes.

2. Zygospores :Zygospores (Gr. *zygos* = yoke + *spora* = seed, spore) are sexually produced resting spores or structures formed as a result of plasmogamy between two gametangia, which are usually equal in size. They are resting structures. Zygospores are the typical sexually produced spores of Zygomycetes e.g. Mucorales and Entomophthorales.

Fig. Oospore

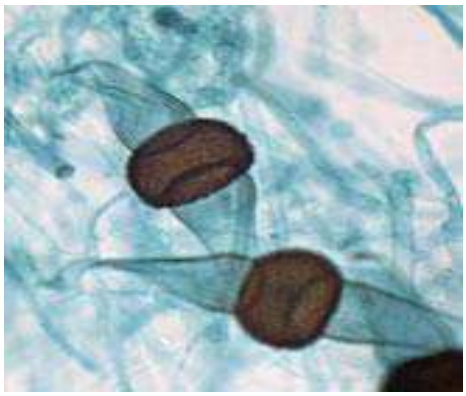


Fig. Zygospore

2. **Ascospores** : Ascospores (Gr. *askos* = sac + *spora* = seed, spore) are the characteristic spores of the large group of fungi known as Ascomycotina.

3. **Basidiospores :** Basidiospore (Gr. *Basidion* = small base + *spora* = seed, spore) is a spore borne on the outside of a basidium, following karyogamy and meiosis.

Fructifications and fruit bodies : The sporophores bear fruiting bodies or form fructifications, which may be asexual or sexual in nature. In lower fungi Plasmodiophoromycetes, Chytridiomycetes, Oomycetes and Zygomycotina) asexual spores are usually enclosed in simple sacs called sporangia or zoosporangia. In higher fungi (Ascomycetes and Basidiomycetes) complex aggregates of spore bearing hyphae are formed and supporting and protective tissues surround it. These complex structures are called as spore fruits or fructifications (L.*fructus* = fruit).

Asexual fructifications : In fungi conidiophores are grouped together to form specialized structures such as synnemata (sing. synnema) and sporodochia (sing. sporodochium) or produced in fructifications known as pycnidia (sing. pycnidium) and acervuli (sing. acervulus).

synnema: (pl. synnemata; syn. coremium) : Compact or fused, generally upright conidiophores, with branches and spores forming a headlike cluster [synnemata of *Ophiostoma (Graphium) ulmi]*.

Sporodochium : Sporodochium is a fruiting body in which conidiophores arise from a central cushion-like aggregation of hyphae. The conidiophores are packed tightly together and are generally shorter than those composing a synnema. e.g. *Epicoccum, Nectria.*

Pycnidium : Pycnidium is a globose or flask-shaped body, which is lined on the inside with conidiophores. e.g. *Septoria ,Phoma, Ascochyta, Leptosphaeria.*

Acervulus : Acervulus (pl. acervuli) is a fruiting structure commonly found in the order Melanconiales (Deuteromycotina). It is typically a flat or saucer-shaped mass of aggregated hyphae bearing short conidiophores in a compact layer. Intermingled with the conidiophores, setae (sing. seta; L. *seta* = bristle) are found. Setae are long, pointed, dark coloured, sterile structures. In nature acervuli are produced on plant tissues subepidermally or subcuticularly and becomes erumpent on maturity. e.g. *Colletotrichum*.

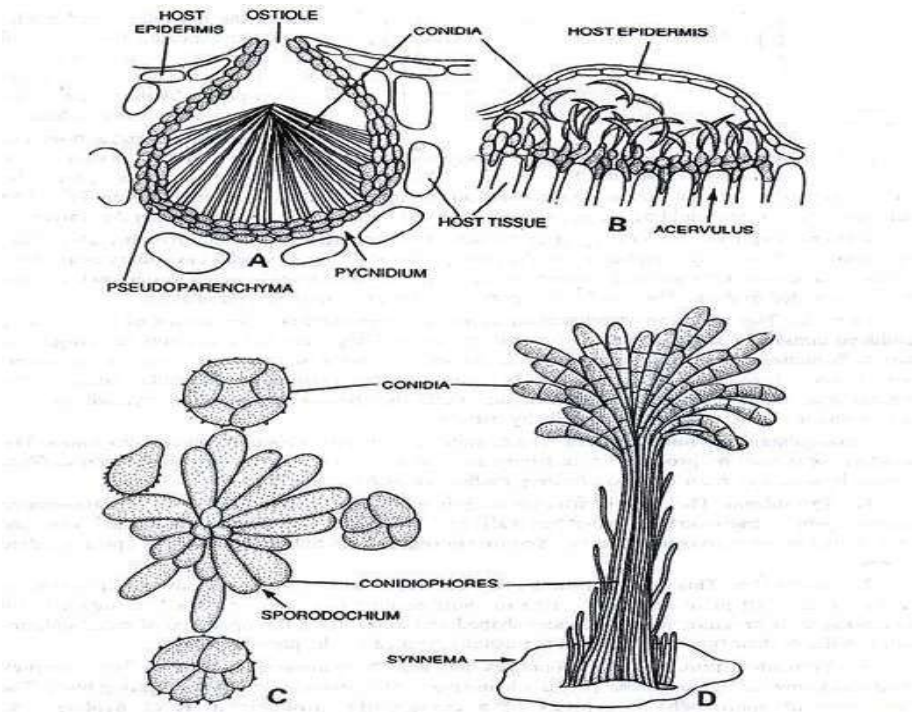


Fig. Asexual fruiting bodies of fungi

Sexual fruiting bodies

1) Ascocarp : Ascocarp (Gr. askos = sac+ *karpos* =fruit) is a fruiting body that contains asci and ascospores. Ascomycetes fungi with few exceptions produce ascocarps. They are in various forms like spherical, flask-shaped, cup-and saucer shaped and pod-shaped. They may be closed in some, and provided with a narrow wide opening in others. Ascocarps may formed singly or in groups. They may be superficial, erumpent or deeply embedded in the

substratum. The substratum may be composed entirely of hose tissue, or it may be a hyphal stroma or in which the ascocarps form. Basicaly, there are three types of ascocarps :

Perithecia : Perithecium (pl. perithecia, Gr. *peri* = around + *theke* = a case) is a flask -shaped ascocarp with a wall of its own. It is provided with a narrow ostiole and may possess a short or a long neck through which the asci are released at maturity.

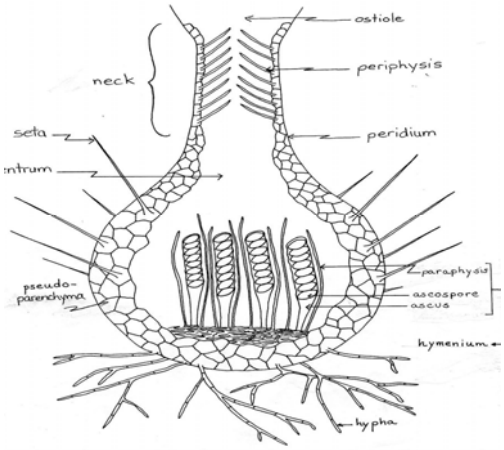
The asci are arranged in a regular manner and are lined the inside wall. The asci are intermingled with sterile filaments called *paraphyses,* which help the asci in nutrition and dispersal. The *paraphyses, which* are rigid and appear in the ostiole are called periphyses. The perithecia may be borne singly or in groups. These are found in *Cordyceps* and *Claviceps*.

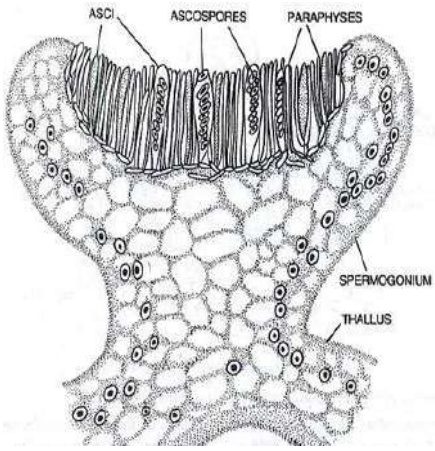
Apothecium : Apothecium (pl. Apothecia; Gr. *apotheke*=store house) is an open ascocarp. It has a broad opening and is either cup or saucer shaped with asci arranged in a palisade layer within.

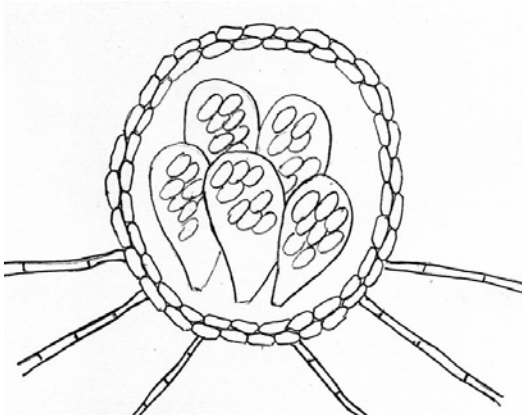
It is usually fleshy or leathery in nature. It contains club-shaped or cylindrical asci, usually with many or few paraphyses among them. These are found in Pezizales, *Colletotrichum* spp.

Cleistothecium: (pl. cleistothecia; Gr. *kleistos* = closed + *theke* = case), is a closed ascocarp and has no ostiole. It is deep brown to black in colour, more or less spherical and often provided with appendages on its body, which serve as organs of anchorage and help in dissemination.

They may contain one to several asci, which discharge their spores violently. Each cleistothecium of *Sphaerotheca* and *Podosphaera* contains a single ascus whereas each cleistothecium in *Erysiphe, Microsphaera, Uncinula, Leveillula* and *Phyllactinia* contain several asci.







2) **Basidiocarp** : Basidiocarp (Gr. *basidion*=small base + *karps* = fruit) is a fruiting body, which bears basidia and basidiospores. Basidia are borne on the under surface of fruit body. Basidia bear basidiospores exogenously usually on projections called sterigmata.

They are highly developed and have compound structure. Basidiocarps may be thin and crust-like, gelatinous, cartilaginous, papery, fleshy, spongy, corky or woody. They may vary in size from microscopic to a metre or more in dia. Most fungi in basidiomycotina except smuts (Ustilaginales) and rusts (Uredinales) form basidiocarps.

Nutrition: The fungi lack chlorophyll. Therefore, they cannot synthesiz their own food.

Depending on from where and how they get nutrition, fungi are of following types:

(a) Saprotrophs (= saprobes): They obtain food from dead and decaying organic matter. They secrete digesting enzymes to outside which digest the substratum and then absorb nutrients, e.g., Mucor, Agarious. Rhizopus (bread mould) etc.

(b) Parasitic: They obtain food from living .They maybe facultative or obligate. Facultative parasites grow on a variety of tissues and often cause 'soft rot' of the tissue, e.g., Ustilago.

The obiigate- parasites absorb through specialized haustoria. The parasitic fungi that grow on surface of host cells and absorb food through haustoria are called ectoparasites or ectophytic parasites (e.g., Mucor). When parasitic fungi grow inside the host tissue arc called endoparasites or endophytic parasites (e.g., Pythium, Puccinia).

(c) Predacious: Some soil fungi develop ring-like noses to trap annelids, nematodes etc. e.g., Arthrobotrys, Zoophagus, Dactylella etc.

(d) Symbiotic: They live in mutualistic relationship with another organism by which both are benefited. The two common examples are lichens and mycorrhiza. Lichens are symbiotic associations between fungi and algae. The fungal partner is a member of ascomycetes or basidiomycetes that provides water and nutrients, while the algal partner is a green alga or cyanobacteria that prepares food by photosynthesis.Mycorrhizas or mycorrhizae (fungus roots in Greek) are the mutualistic symbiotic associations between soil fungi and the roots of most plant species (95% of all plant families are predominantly mycorrhizal).

Study questions:

9. List the sexual fruiting bodies of fungi and give examples.

10. List the asexual fruiting bodies of Fungi and give example.

11. List the methods of sexual reproduction in fungi and explain any one

12. List the methods of asexual reproduction in fungi and explain any one.

13. What is parasexuality and list the steps in parasexual cycle.

Taxonomy:

Kingdom Fungi is a monophyletic group (meaning that all modern fungi can be traced back to a single ancestral organism) that diverged from a common ancestor with the animals about 800 to 900 million years ago. The most recently published classification divides the kingdom into seven phyla, ten subphyla, 36 classes, 12 subclasses, and 140 orders.

Kingdom: Fungi 1**. Phylum: Chytridiomycota** Class: Chytridiomycetes 1. Order: Chytridiales Family: Chytridiaceae Genera: Chytridium, Synchytrium, Ex: *S.endobioticum* causing black wart of potato. 2. Order: spizellomycetales Family: Olpidiaceae Genus: *Olpidium* Ex: *Olpidium brassicae* causing damping off of crucifers.	2. **Phylum: Blastocladiomycota** **Class**: Blastocladiomycetes **Order**: Blastocladiales Family: Physodermataceae Genera: *Physoderma* *P.zea maydis* –physodemra leaf spot of Maize 3**. Phylum: Glomeromycota** **Class**: Glomeromycetes **Order**: Glomerales Genus: *Glomus* (Endo-Mycorrhizal fungi) Subphylum: Mucoromycotina 1. Order: Mucorales 1. Family: Mucoraceae Genera: *Mucor, Rhizopus* 2. Family: Choanephoraceae Genera: *Choanephora, Blakeslea* 3. Family: Pilobolaceae Genus: *Pilobolus* (Shot gun fungus)
Subkingdom: Dikarya **4. Phylum**: Ascomycota **1. Subphylum**: Taphrinomycotina **1. Class**: Taphrinomycetes **Order**: Taphrinales 1. Family: Taphrinaceae Genus: Taphrina *T.deformans* –peach leaf curl 2. Family: Protomycetaceae Genus: Protomyces *P.macrosporus* – Stem gall of coriander. 2. **Class**: Schizosaccharomycetes **Order**: Schizosaccharomycetales Genus: *Schizosaccharomyces* (Fission fungi)	**2. Subphylum**: Saccharomycotina **Class**: Saccharomycetes **Order**: Saccharomycetales Family: Saccharomycetaceae Genus: *Saccharomyces* (Bakers yeast fungi) *S.cerevisiae* var.*Elliposides* – used in brewing industry. **3. Subphylum**: Pezizomycotina **1. Class**: Dothideomycetes **1. Subclass**: Dothideomycetidae **1. Order**: Capnodiales Family: Capnodiaceae Genus: *Capnodium* Family: *Mycosphaerellaceae* Genus: *Mycosphaerella* *M.musicola* – Sigatoka leaf spot of Banana

2. Order: Dothideales
Family: Dothideaceae
Genus: *Dothidea, Sydowia*

3. Order: Myriangiales
Family: *Elsinoeaceae*
Genus: *Elsinoe*

2. Subclass: Pleosporomycetidae
Order: Pleosporales
Family: Pleosporaceae
Genus: *Pleospora*
Family: Venturiaceae
Genus: *Venturia*
V.inaequalis – Apple scab

3. Dothiodeomycetes Incertae sedis (without any subclass)
Order: Botryosphaeriales;
Genera: *Botryosphaeria*, *Guignardia*.

2. Class: Eurotiomycetes
Subclass: Eurotiomycetidae
Order: Eurotiales
Genera: *Eurotium*, *Emericella*, *Talaromyces*

3. Class: Leotiomycetes
1. Order: Erysiphales **(Powdery mildews)**
Family: Erysiphaceae
Genera: *Erysiphe*, *Blumeria*, *Uncinula*

2. **Order**: Helotiales
Family: Sclerotiniaceae
Genera: *Sclerotinia, Monilinia*

3. **Order**: Rhytismatales
Genera: *Rhytisma*

4. Class: Pezizomycetes (Apothecial fungi)
Order: Pezizales
Genera: *Peziza*, *Morchella*, *Pyronema*

5. Class: Sordariomycetes
1. Subclass: Hypocreomycetidae
1. Order: Hypocreales
Family:Claviceptaceae
Genus: *Claviceps*

Family: Hypocreaceae
Genus: *Hypocrea*

Family: Nectriaceae
Genus: *Nectria*

2. Order: Microascales
Family: Ceratocystidaceae
Genus: *Ceratocystis*

2. Subclass: Sordariomycetidae
Order: Diaporthales
Family: Diaporthaceae
Genera: *Diaportha*

Family: Cryphonectriaceae
Genus: *Cryphonectria.*

Order: Ophiostomatales
Family: Ophisotmataceae
Genus: *Ophiostoma*

Order: Sordariales
Family: Sordariaceae
Genera: *Sordaria*, *Neurospora*, *Chaetomium*

3. Subclass: Xylariomycetidae
Order: Xylariales
Family: Xylariaceae
Genera: *Xylaria*, *Hypoxylon*.

Sordariomycetes *incertae sedis*
(not placed in any subclass)
Order: Meliolales
Family: Meliolaceae
Genus: *Meliola*

Order: Phyllachorales
Family: Phyllachoraceae
Genus: *Phyllachora*.

Order: Glomerellales
Family: Glomerellaceae
Genus: *Glomerella*

5. Phylum: Basidiomycota **1. Subphylum**: Pucciniomycotina **Class**: Pucciniomycetes **1. Order**: Septobasidiales Family: Septobasidiaceae Genus: *Septobasidium* **2. Order**: Pucciniales 1. Family: Pucciniaceae Genera: *Puccinia*, *Gymnosporangia, Uromyces* 2. Family: Melmpsoraceae Genus: Melampsora 3. Family: Phakpsoraceae Genus: *Phakopsora* **2. Subphylum**: Ustilaginomycotina **1. Class**: Ustilaginomycetes **i) Order**: Urocystales Family: Urocystaceae Genus: *Urocystis* **ii) Order**: Ustilaginales Family : Ustilaginaceae Genus: *Ustilago*, *Tolyposporium* **2. Class**: Exobasidiomycetes **i) Order**: Entylomatales **Family: Entylomataceae** Genus: *Entyloma* **ii) Order**: Exobasidiales Family: Exobasidiaceae Genus: *Exobasidium* **iii) Order**: Tilletiales Family: Tillatiaceae Genus: *Tilletia* **Class**: Agaricomycetes **Subclass**: Agaricomycetidae **Order**: Agaricales Genera: *Agaricus*, *Coprinus*, *Pleurotus*	**Order**: Auriculariales; example genera: *Auricularia*, *Exidia*, *Bourdotia*. **Order**: Corticiales Example genera: *Corticium*, *Vuilleminia*, *Punctularia*. **Order**: Polyporales; example genera: *Polyporus*, *Fomitopsis*, *Phanerochaete* **Anamorphic fugi/ Former Deuteromycetes fungi** **Devided into three groups** 1.Hyphomycetes anamorphic fungi 2. Acervular imperfect fungi 3. Pycnidial imperfect fungi 1. **Hyphomycetes anamorphic fungi** **Order: Moniliales** **Family: Dematiaceae** Examples of Genera: *Alternaria, Botrytis, Cercospora, Curvularia, Helminthosporium, Bipolaris, Drechslera, Pyricularia, , Trichothecium* Family: Moniliaceae Ex: *Verticilium,* Family: Tuberculariaceae Conidia and conidiophore produced in Sporodochium. Ex: *Fusarium* 2. **Acervular imperfect fungi:** **Order: Melanconiales** **Family: Melanconiaceae** Examples of Genera: *Colletotrichum, Pestalotiopsis, Sphaceloma* 3. **Pycnidial Imperfect Fungi:** **Order: Sphaeropsidales** **Family: Sphaeropsidaceae** Examples of Genera: *Ascochyta, Lasiodiplodia, Diplodia, Phyllosticta, Phoma, Phomopsis, Septoria.*

Kingdom Chromista	**Kingdom Protozoa**
1. Phylum Hyphochytriomycota **Class: Hyphochytriomycetes** **Order** Hyphochytriales. Genus: *Hyphochytrium* **2. Phylum** Oomycota **Class: Oomycetes** **Subclass: Peronosporomycetidae** **i) . Order** Pythiales Family: Pythiaceae Genera: *Pythium* **ii) Order:** Peronosporales Family: Peronosporaceae Genera: *Phytophthora, Peronospora*, *Bremia*, *Plasmopara*. **iii) Order: Albuginales** Family: Albiginaceae Genus: *Albugo* **Order** Saprolegniales; example genera: *Achlya*, *Saprolegnia*.	**Lineage: Plasmodiophorids** **Class:** Plasmodiophorea **Order:** Plasmodiophorida Example genera: *Plasmodiophora*, *Polymyxa* **Phylum:** Mycetozoa **Class:** Myxogastrea (Myxomycetes) Members are called **slime molds.**

Kingdom: Protozoa

- ✓ They are simple eukaryotic organisms.
- ✓ They have a membrane bound nucleus and endomembrane systems.
- ✓ Most of the organisms are unicellular, some are colonial and some are multicellular like algae.
- ✓ Most of the members live in water, some in moist soil or even the body of human and plants.
- ✓ Nutrition - they can be both hetreotrophic or autotrophic.
- ✓ Thallus may be plasmodium, pseudoplasmodium, aphanoplasmodium
- ✓ Movement is often by flagella or cilia.
- ✓ They have mitochondria for cellular respiration and some have chloroplasts for photosynthesis.
- ✓ Reproduction - some species have complex life cycle involving multiple organisms. Example: Plasmodium.
- ✓ Some reproduce sexually and others asexually.
- ✓ They form cysts in adverse conditions.

- ✓ They are pathogens of both animals and plants. Example: *Plasmodium vivax* causes malaria in humans.

Lineage (formerly phylum): Plasmodiophorids

Class: Plasmodiophorea **(**Plasmodiophoromycetes)

- ✓ Members are obligate endoparasite in plants.
- ✓ Lack of phagotrophic nutrition.
- ✓ Thallus is plasmodium which is endobiotic.
- ✓ Plasmodium may be haploid or diploid is holocarpic.
- ✓ Plasmodium develops into thin walled sporangia.
- ✓ The plasmodium is designated as zoosporogenous or cystogenous depending on whether it produces zoospores or cysts.
- ✓ The zoospores are uninucleate, anteriorly biflagellate having whiplash type.
- ✓ Presence of cruciform nuclear division.

Order Plasmodiophorida (Plasmodiophoriales)

Family: Plasmodiophoraceae

- ✓ They are obligate endoparasites of flowering plants.
- ✓ They induce hypertrophy in infected cells.

Genera: *Plasmodiophora*, *Spongospora*, *Polymyxa*

Plasmodiphora brassicae - cause disease called Club root of crucifers.

Spongospora subterranea – cause disease Powdery scab of potato

Polymyxa graminis is a vector of Wheat mosaic virus, oat mosaic virus and Barley yellow mosaic virus.

Kingdom: Chromista

- ✓ Unicellular, filamentous or colonial nature
- ✓ Presence of tinsel type flagellum
- ✓ Thallus diploid
- ✓ Phtosynthetic or heterotrophic nutrition
- ✓ Cell wall made up of cellulose and glucans
- ✓ Mitochondria with tubular cristae
- ✓ Storage product Mycolaminarin

Phylum : Oomycota

- ✓ Unicellular to filamentous mycelium
- ✓ Thallus is diploid
- ✓ Asexual spores: Zoospores
- ✓ Zoospores are biflagellated, heterokont and anisokont
- ✓ Cell wall made up of β 1-3 glucans and cellulose
- ✓ Synthesis of lysine via diaminopimelic acid pathway
- ✓ Presence of fucosterol

Class : Oomycetes

- ✓ Cell wall made up of β 1-3 glucans and cellulose
- ✓ Thallus is diploid
- ✓ Coenocytic mycelium (mycelium without cross walls)
- ✓ Asexual spores: Zoospores produced in zoosporangium
- ✓ Zoospores are biflagellated
- ✓ Sexual spore: Oospore
- ✓ Sexual reproduction: Gametangial contact
- ✓ They found in both aquatic and terrestrial environment
- ✓ They are saprobes as well as plant parasites.

Order: Pythiales

- ✓ Members are saprobic, and plant parasitic
- ✓ Sporangiophores are indistinguishable from hyphae and also of indeterminate growth

Genus: *Pythium*

- ✓ Facultative parasite
- ✓ Coenocytic hyphae with inter or intracellular
- ✓ Haustoria are not produced
- ✓ Globose or oval sporangia apically produced from unspecialized hyphae
- ✓ Zoospore discharged through Vessicle
- ✓ Sexual spore : oospore
- ✓ Sexual reproduction: by gametangial contact (paragynous)
- ✓ Cause damping off disease in vegetable, papaya, tobacco etc;

Order: Peronosporales

Family: *Peronosporaceae*

- ✓ Facultatative saprophyte or obligate plant parasites
- ✓ Coenocytic mycelium with intercellular hyphae
- ✓ Produce haustoria

- ✓ Sporangiophore is determinate and distinct from hyphae
- ✓ Members cause blight, Downy mildew diseases
- ✓ Important Genera: *Phytophthora, Basidiophora, peronospora, sclerospora, sclerophthora, bremia,* etc;

Phytophthora

- ✓ Faculatative saprophyte
- ✓ Intercellular mycelium with haustoria
- ✓ Sympodial branching of sporangiophore
- ✓ Sporangia may be lemon shaped or globose with or without papilla.
- ✓ Ex: *P.infestans* – Late blight of Potato

Downy mildew genera: They are obligate plant parasites. Sporangiophore is determinate and distinct from hyphae. They are mainly identified based on their sporangiophore branching

Plasmopara

- ✓ Sporangiophores arise at right angles and are irregularly spaced.
- ✓ Sporangia germinate producing zoospores
- ✓ Ex: *P.viticola* – Downy mildew of grape
- ✓ Caused epidemic in France in 1870's

Peronospora

- ✓ Dichotomously branched Sporangiophores taper to fine points bearing conidia.
- ✓ Direct germination (sporangia germinate producing conidia)
- ✓ Ex: *P. tabacina* – Blue mold of tobacco.

Pseudoperonospora

- ✓ Dichotomously branched Sporangiophores taper to fine points bearing sporangia.
- ✓ Indirect germination (sporangia germinate producing Zoospores)
- ✓ Ex: *P.cubensis* – Downy mildew of cucurbits.

Sclerospora

- ✓ Sporangiophore is swollen, stout with many upright branches bearing sporangia at the tips.
- ✓ Sporangia germinate indirectly by producing zoospores.
- ✓ Ex: *S. graminicola* – Downy mildew (green ear) of Bajra.

Peronosclerospora

- ✓ It combines the character of *Peronospora* and *Sclerospora*
- ✓ Sporangia germinate directly by producing germ tube like in *Peronospora*.
- ✓ The sporangiophores are branched like *sclerospora i.e.* swollen and stout with many upright branches.
- ✓ Ex: *P.sorghi* – Downy mildew of sorghum

Sclerophthora

- ✓ It combines the character of *Sclerospora* and *Phytophthora.*
- ✓ The sporangiophores are branched like *sclerospora i.e.* swollen and stout with many upright branches.
- ✓ Sporangiophores are indeterminate in growth and have bigger sporangia like *Phytophthora.*
- ✓ Ex: *S.rayssiae* var. *zeae* – brown stripe downy mildew of Maize

Bremia

- ✓ Sporangophores are dichotomously branched at acute angle in which tip of the branches are expanded into cup shaped apophyses with four sterigmata each bearing sporangia at their tips.
- ✓ Ex: *B.lactucae* – causing Downy mildew of Lettuce.

Bremiella

- ✓ Similar to *Bremia* except that tip of the branches are inflated into bulbous shaped apophyses on which sterigmata bearing sporangia are produced.

Basidiophora

- ✓ Sporangipohre is unbranched, basidium like club shaped and bears sporangia on short sterigmata.

Order: Albuginales

Family: Albuginaceae

Genus: *Albugo*

- ✓ Obligate plant parasite
- ✓ coenocytic intercellular mycelium with haustoria
- ✓ sporangiophores are sort club shaped bearing sporangia in chains
- ✓ The oldest sporangia lie at the top and youngest at the base of the chain (Basipetal)
- ✓ The two sporangia are attached by a disc like structure called isthmus or disjunctor.

- ✓ They cause hypertrophy of floral parts and forms galls.
- ✓ Ex: *Albugo candida* – white rust / pseudo rust of crucifers.

Phylum: Chytridiomycota

- ✓ Considered to be the most ancestral group of fungi
- ✓ Water moulds that live as aquatic saprotrophs or parasites in fresh water and soils; a few are marine.
- ✓ Chytrids produce motile asexual zoospores with a single posterior flagellum,
- ✓ Thallus may be unicellular or filamentous, and holocarpic or eucarpic, monocentric, polycentric or filamentous.
- ✓ Golgi apparatus with stacked cisternae
- ✓ Sexual reproduction with zygotic meiosis or sometimes produce motile sexual zoogametes.

Class: Chytridiomycetes

Order: Chytridiales

Family: synchytriaceae

Genus: *Synchytrium*

- ✓ *S.endobioticum* cause disease black wart of potato.
- ✓ The diases is quarantine importance, in India it is restricted to Dargelling district of Westbengal.
- ✓ Pathogen is obligate endoparasite.
- ✓ Thallus is holocarpic and endobiotic
- ✓ It also act as vector of Potato mop top virus and Potato virus X.

Phylum: Zygomycota

- ✓ Members form coenocytic mycelium
- ✓ Cell wall is made up of chitosan chitin
- ✓ Asexaul reproduction by forming Aplanospores in sporangium
- ✓ Sexual spore is Zygospore
- ✓ Mode of sexual reproduction is gametangial copulation
- ✓ Members are saprobic, facultative parasites
- ✓ Some members are of great value in industries such as fermentation, production of acids etc; Ex: *Rhizopus* sp. Used in preparation off cheese and Temph (fermented food from soybean).
- ✓ Some members are parasites of Plant and animals. Ex: *Rhizopus stolonifer* – Soft rot of Sweet potatoes.

Subphylum: Mucromycotina

Order: Mucorales

Family: Mucoraceae

Genera: *Mucor, Rhizopus*

Mucor	*Rhizopus*
Stolons are absent	Stolons are present
Root like structure called Rhizoids are absent	Rhizoids are present
Sporangiophores arise singly from the hyphae	Sporangiophores arise in groups from the junction of the stolon and rhizoid
Sporangiospores firmly adhere to collumella in a drop of fluid not easily blown off by wind	Sporangiospores are dry and easily blow off by wind
Do not cause contamination of air and laboratory cultures	Cause contamination of air and laboratory cultures.

Family: Pilobolaceae

Genus: *Pilobolus*

- ✓ It produces Coenocytic hyphae which feed on dung.
- ✓ It is **coprophilus** fungi *i.e.* it gets stimulus compound (coprogen) in dung for its growth hence it is called dung loving fungi.
- ✓ Sporangiophore is phtotrophic in nature and produced from a carotene rich trophocyst
- ✓ Sporangium is violently discharged to a distance of 2-2.5m hence called **Hat thrower or Shot gun fungus.**

Phylum: Glomeromycota

- ✓ Mycelium is coenocytic
- ✓ They are **obligate symbionts (Endomycorrhizal fungi)**
- ✓ They form association with roots of higher plants (Agriculture and Horticulture plants)
- ✓ Forms arbuscules in plant root cells
- ✓ Large multinucleate spores with layered walls
- ✓ They do not produce Zygospores

Order: Glomerales

Family: Glomeraceae

Genus: *Glomus*

Phylum : Ascomycota

- ✓ It is the largest phylum of the fungi having the 75% of all the known fungi.
- ✓ Forms Septate mycelium
- ✓ Cell wall contains Chitin and it is double layered.
- ✓ The means of Asexual Reproduction are budding, fragmentation, fission, Chlamydospores and conidia.
- ✓ Conidia are produced on specialized hyphae called Conidiophore or in specialized cells called condioma.
- ✓ Sexual spore is Ascospore formed in a sac like structure called Ascus.
- ✓ Ascus is a formed nakedly or in a structure called Ascoma or Ascocarp
- ✓ Each ascus contains Eight Ascospores.
- ✓ Ascospores are produced by free cell formation process.
- ✓ Presence of dikaryotic phase in life cycle.
- ✓ The means of sexual reproduction are gametangial contact, gametangial copulation, spermatization and somatogamy.
- ✓ Most of the ascomycetes members produce Sexual fruiting body called Ascocarp which may cleistothecium, perithecium or apothecium.
- ✓ Female gametangium called Ascogonia and male gametangium called Anthredia are formed. Nuclei from antheridium moves into ascogonium through trichogyne.

Ascomycota devided into Three subphyla

Subphylum: Taphrinomycotina.

1. Class : Taphrinomycetes

Order: Taphrinales

- ✓ Asci is produced from dikaryotic ascogenous cells
- ✓ There is no Ascocarp formation
- ✓ Ascus is formed directly on the stroma without any covering hence called **Naked Ascomycetes.**
- ✓ Members are **dimorphic in nature** *i.e.* formation of mycelium in parasitic phase and yeast like colonies in saprobic phase.

Genera: *Taphrina, Protomyces*

Taphrnia: it cause necrotic spots, galls and witches broom symptoms in plants

Ex: *T.deformans* cause peach leaf curl disease

T. maculans cause turmeric leaf blotch

Genus: *Protomyces*

- ✓ It cause leaf lesions and galls on plant parts.
- ✓ It forms asexual resting spore called **Chlamydospore**
- ✓ It forms a structure called **Synascus or compound ascus** with in which Ascospores are produced.
- ✓ Ex: ***P. macrosporus* cause stem gall of coriander.**

2. Class: schizosaccharomycetes.

Order; schizosaccharomycetales

Family: Schizosaccharomycetaceae

Genus: *Schizosaccharomyces*

- ✓ It is unicellular yeast characterized by its asexual reproduction.
- ✓ It reproduces asexually by fission hence called Fission Yeast.
- ✓ Asci produced from fusion of haploid cells.

2. **subphylum: Saccharomycotina**

- ✓ Members are unicellular
- ✓ They lack mycelium or it is poorly developed
- ✓ Cell wall is made up of β 1,3 glucans
- ✓ Direct development of Asci from Zygote or somatic cells.
- ✓ Absence of Ascoma or Ascocarp.
- ✓ Life cycle is halpobiontic diploid type.

Class: Saccharomycetes

Order: saccharomycetales

Family: Saccharomycetaceae

Genus: *Saccharomyces*

- ✓ *S. cerevisiae* is called yeast of commerce or Bakers and brewers yeast.
- ✓ It has been used for baking and brewing since ancient times.
- ✓ It is also used as a probiotic (live organisms which when administered through in adequate amounts, confers benefit on the host).
- ✓ It was first eukaryotic organism whose genome was completely sequenced.

3. Subphylum: Pezizomycotina

- ✓ Mycelium is septate, branched and filamentous.
- ✓ They form sexual fruiting bodies

- ✓ Asci is pro or bitunicate

1. Class: Eurotiomycetes

Order: Eurotiales

- ✓ Form sexual fruiting body called Cleistothecia
- ✓ Asci are protunicate
- ✓ Unseptate Ascospores
- ✓ Absence of Inter ascul tissues.

Family: Trichocomaceae

Genera: *Eurotium, Emericella* (anamorph is *Aspergillus* spp.), *Talaromyces, Eupenicillim* (anamorph is *Penicillium*)

Anamorph	**Teleopmorph**
Aspergillus glaucans group	*Erotium*
Aspergillus nidulans	*Emericella*
Aspergillus fumigates group	*Neosartorya*
Penicillium	*Talaromyces*

Aspergillus: members are saprobic, and plant parasites.

They play key role in biodegradation, biodeterioration, genetic studies, toxin production in food-feed stuffs and industrial usage.

Importance of *Aspergillus*

- ✓ Cause disease in humans and animals called Aspergillosis (*A. fumigatus*).
- ✓ Produce mycotoxin in food and feed stuff: Example: *Aspergillus flavus* produce Aflatoxin in groundnut. In England in 1960 about 1, 00, 00 turkey were died by consumption of groundnut cake infested with *A.flavus* which produced Aflatoxin in groundnut.
- ✓ They are used in industry for commercial production of citric acid, gluconic acid etc;
- ✓ Used as a tool in biological studies, example: parasexual phenomenon was first discovered in 1952 by Pontecarvo and Roper in *A. nidulans.*
- ✓ Cause disease in Plants, Example: Crown rot of groundnut caused by *A.niger*.

Importance of *Penicillium*

- ✓ Antibiotic production: **Alexander Flemming** first discovered antibiotic 'penicillin' from *Penicillium notatum.* Now *P.chrysogenum* is used for commercial production of penicillin.
- ✓ Used in preparation of cheese. Example: *P.roquefortii.*
- ✓ Cause diseases in plants. Example: blue mold in citrus (*P. italicum*)
- ✓ Produce mycotoxin in food. Example: patulin, penicillic acid etc;

Aspergillus	***Penicillium***
Conidiophores are unseptate and unbranched	Conidiophores are septate and branched
Conidiophores develop from specialized thick walled cell called Foot cell	Conidiophores develop from any vegetative cell of mycelium, foot cell is absent
Each conidiophore enlarge into a swollen vesicle at its tip.	Vesicle is not formed at the tip of conidiophore.
Mature conidia are yellow, green or black in colour	Mature conidia are green, blue or yellow in colour
Wall of cleistothecium is loose	Wall of cleistothecium is comparatively thick
Produce mycotoxin such as Aflatoxin	Produce mycotoxins such as patulin, penicillic acid.

Class: Leotiomycetes

Order: Erysiphales

- ✓ They cause powdery mildew disease in plants.
- ✓ They are obligate plant parasites.
- ✓ Mycelium is septate and filamentous
- ✓ Mycelium may be ectophytic or endophytic
- ✓ They form sexual fruiting body such as cleistothecia (Now called Chasmothecium)
- ✓ Cleistothecia is having external appendages.
- ✓ Each cleistothecia may contain one or more than one ascus.
- ✓ There are no paraphyses in cleistothecia.
- ✓ Each ascus contains eight Ascospores.
- ✓ Ascus is inoperculate, unitunicate.
- ✓ Asexual spore is conidia, which may oidium type, oidiopsis type or ovulariopsis type.
- ✓ Conidia is large and does not require external moisture for germination and infection
- ✓ The superficial mycelium produces enormous number of conidia on infected plant parts which appear like a mass of white powder, hence they named as **powdery mildew.**

Family: Erysiphaceae

Genera: *Erysiphe, Podosphaera, Uncinulla, Phyllactinia, Leveillula, Blumeria, Sphaerotheca* and *Microsphaera.*

Powdery mildew members can be differentiated based on their growth habit (ecto, endo), cleistothecial appendages, Numbers of ascus in cleistothecium and the anamorph.

Characters of Powdery mildew Genera

Genus	**Mycelium growth**	**Anamorph**	**Number of asci in Cleistothecium**	**Appenndage morphology**	**Disease**
Erysiphe	Epiphytic	Oidium	Several	Myceloid (simple hypha like)	*E.polygoni* – P.M. of clusterbean
Microsphaera	Epiphytic	Oidium	Several	Branched dichotomously at tip	
Uncinula	Epiphytic	Oidium	Several	Coiled or hooked at tip	*U.necator* – P.M. of Grape
Blumeria	Epiphytic	Oidium	Several	Myceloid	*B.graminis* – P.M. of Wheat
Golovinomyces	Epiphytic	Oidium	Several	Hypha like	
Podosphaera	Epiphytic	Oidium	Single Ascus	Branched dichotomously at tip	*P.leucotricha* – P.M. of Apple
Sphaerotheca	Epiphytic	Oidium	Single Ascus	Simple hypha like	*S.pennosa* – P.M. of rose
Phyllactinia	Partially endophytic	Ovulariopsis	Several	Straight with bulbous base	*P.corylea* – P.M of Mulberry
Leveillula	Partially endophytic	Oidiopsis	Several	Simple hypha like	*L.taurica* – P.M. of chilli, Redgram

Class : Dothideomycetes

Subclass: Dothidoemmycetidae

Order: capnodiales

Family: Mycosphaerellaceae

Genus: *Mycosphaerella*

- ✓ It is largest and wide spread genus with enormously varied anamorphs like *Cercospora, Ramularia* and *Septoria.*
- ✓ Produce sexual fruiting body called perithecium (pseudothecium)
- ✓ Perithecium produced in a stroma containing locules.
- ✓ Asci are ovoid to saccate and fissitunicate.

✓ Ascospores are transversely septate.

✓ Important diseases caused by Mycosphaerella, Sigatoka of Banana (*M.musicola*), Tikka leaf spot of groundnut (*M.arachidicola, M.berkeleyii*).

Subclass: pleosporomycetidae

Order: pleosporales

Family : Venturiaceae

Genus: *Venturia*

- ✓ Mycelium septate and internal
- ✓ Mycelium concentrated just below the cuticle hence called subcuticular pathogen.
- ✓ Sexual fruiting body : Pseudothecia produced in a locule.
- ✓ Interascual tissues like Pesudoparaphyses are present in between the asci.
- ✓ Ascocpore are yellowish unequally bicelled.
- ✓ Asexual spore: conidiaproduced on a conidiophore.
- ✓ It cause scab disease in plants. Example: Apple scab caused by *V. inaequalis.*

Class: sordariomycetes

- ✓ Produce Perithecium as fruiting body
- ✓ Asci are inoperculate, unitunicate or prototunicate
- ✓ Strom is present in some genera
- ✓ Some are plant parasites (*Cryphonectria*, *ophiostoma*, *Claviceps*, *xylaria*), some are Biocontrol agents (*Trichoderma*), some used in genetic studies (*Neurospora*).

Genus: *Nuerospora* – used in Genetic studies by Tatum and beadle.

Genus*: Cryphonectria*

- ✓ Caused epidemic in U.S. by destroying the chestnut trees.
- ✓ *C.parasitica* causes disease called Chestnut blight.
- ✓ Conidia and Ascospores are transmitted by Beetle and birds.

Genus: *Ophiostoma* (O: Ophiostomatales, F: Ophiostomataceae)

- ✓ It caused pandemic in 1920's and 1940's by devastating elm trees.
- ✓ Pathogen forms long necked ostiolate perithecia.

- ✓ Presence of setae in the ostiolar portion.
- ✓ Interascul tissue is absent
- ✓ Transmitted by Bark beetle (*Scolytus*)
- ✓ Anamorph: *Graphium, Sporothrix*
- ✓ Dutch elm disease caused by two species, *O.ulmi* and *o.novoulmi.*

Genus: *Claviceps* (Sub cl.: Hypocreomycetidae, O: Hypocreales, F: Claviceptaceae)

- ✓ They are plant parasite infecting the grasses (Bajra, Rye, Sorghum).
- ✓ They infect the inflorescence and cause disease known as 'Ergot'.
- ✓ Produces perithecium in a stroma
- ✓ Asci are long and narrow
- ✓ Ascospores are filiform
- ✓ It produces sclerotium
- ✓ It produces a toxin called Ergotin, ergotamine which upon consumption causes Ergotism in Animals and Human beings.
- ✓ Ergot (from the French "argot," which means a spur) is the fruiting structure produced by *Claviceps* in place of the seed of the plant and contaminates the grain after harvest.
- ✓ It also produces an alkaloid used in medicine *i.e.* LSD (d-Lysergic acid diethylamide).

Genus: Hypocrea

- ✓ I is teleomorph of *Trichoderma.*
- ✓ *Trchoderma* is used as a biocontrol agent for management of soil borne plant pathogens.

Genus: *Ceratocystis*

- ✓ Long necked perithecia
- ✓ *C.paradoxa* causes pineapple disease or sett rot of sugarcane.

Genus: *Xylaria*

- ✓ Well developed storma
- ✓ Perithecia is black thick walled and globose
- ✓ Paraphyses are present
- ✓ Cuase root rot disease in Apple, peach and other stone fruits.
- ✓ *X.hypoxylon* - root rot of apple.

Genus : Meliola (Order: Meliolaes)

- ✓ They form dark coloured hypha hence named as **Black mildew.**
- ✓ They produce Hyphopodia structures

Genus: *Glomerella* (O: Glomerellales, F: Glomerellaceae)

- ✓ Cause bitter rot and anthracnose disease
- ✓ Anamorphic stage is ***Colletotrichum*.**
- ✓ It produces beaked perithecia in strom .
- ✓ Asci short stalked with Ascospores.
- ✓ In anamophic stage produces asexual fruiting body called **Acervulus**.

Phylum: Basidiomycota

- ✓ They are highly evolved fungi, comprising about 30, 000 species.
- ✓ Members are saprobic as well as plant parasites.
- ✓ Used as food (Mushroom), cause diseases in plants such as Rust (Pucciniales) and smuts (Ustilaginales).
- ✓ Thallus is typically mycelium except some yeast.
- ✓ Mycelium is filamentous, branched with septa.
- ✓ The cell wall is double layered composed of chitin.
- ✓ They form Dolypore septa.
- ✓ Sexual spore is basidiospore formed externally on a Basidium.
- ✓ Basidium may be holo or fragmented type.
- ✓ Most of the members form fruiting body called basidiocarp, which may microscopic to large macroscopic.
- ✓ Based on the shape of basidiocarp organisms are named as Button mushroom, birds nest fungi, Gelly fungi stink horn, bracket fungi etc.
- ✓ They form a hook like structure called Clamp connection on dikaryotic hyphae.
- ✓ Presence of long lived dikaryotic phase which is nutritionally independent.
- ✓ Asexual reproduction by budding, fragmentation, conidia, arthrospores.
- ✓ Sexual reproduction by somatogamy.
- ✓ Life cycle is Dikaryotic.

Subphylum: Ustilaginomyctoina

Class: Ustilagnimycetes

Order: Ustilaginales

- ✓ They are ecologically obligate plant parasites causing ‘smut’diseases.
- ✓ Smut means sooty black powdery mass of spores formed on the infected plants.
- ✓ They form well developed mycelium which is intercellular producing haustoria.
- ✓ They form dolypore septum without parenthosome.
- ✓ Presence of dikyaryotic phase which is long lived and nutrionally independent.
- ✓ They produce only Basidiospores and teliospores.
- ✓ Teliospores on germination produce septate promycelium (basidium) on which Basidiospores will be produced terminally and laterally.

- ✓ Teliospores are formed in sori.
- ✓ Teliospores may be free, in pairs or aggregated in more or less persistent spore balls.

Important genera: *Ustilago, Sphaecelotheca, Sporosorium, Tolyposporium*

Loose smut of wheat – *Ustilago nuda* var. *tritici*

Grain smut of sorghum – *Sporosorium sorghi*

Long smut of Sorghum – *Tolyposporium ehrhenberghii*

Order: Urocystidiales

- ✓ Promycelium is hollow bearing two to six Basidiospores.
- ✓ Teliospores produced in balls consist of 1-4 fertile central cells surrounded by a peripheral layer of hyaline sterile cells.

Eg: *Urocystis cepule* – onion smut, *Urocystis agropyri* – flag smut of wheat

Class: Exobasidiomycetes

Order: Tillatiales

- ✓ Members cause disease called 'Bunt'.
- ✓ Promycelium is hollow unseptate bearing filiform or elongated Basidiospores.
- ✓ Basidiospores are produce terminally.
- ✓ Members produce trimethyl amine chemical which imparts foul or fishy smell to the infected kernels.
- ✓ Eg: *Tilletia*
- ✓ *Neovossia indica* – Karnal bunt of wheat.
- ✓ *Tilletia foetida* – Smooth spore bunt of wheat

Order: *Pucciniales* (Earleir called as Uredinales)

- ✓ Members cause disease called 'Rust'.
- ✓ Mycelium is branched filamentous septate and intercellular forming haustoria.
- ✓ Presence of dolypore septa but without parenthosomes.
- ✓ They are obligate plant parasite.
- ✓ They may be Autoecious or Heteroecious in nature.
- ✓ They are polymorphic producing various types of spores (Pycniospore, Aeciospore, Uredospore, Teliospore and Basidiopsore).
- ✓ Sexual reproduction is through spermatization where in spermatia fertilize special receptive hypha in spermagonia.
- ✓ Basidiospores discharge by actively or explosively.
- ✓ Genera: *Puccinia, Uromyces, Melampsora, Phakpsora, Gymnosporangium.*

Subphylum: Agaricmycotina

Class: Agaricomycetes

- ✓ Members are saprobes, symbionts and parasites
- ✓ They produce well developed mycelium.
- ✓ Presence of dolypore septa with parenthosome
- ✓ Clamp connections may be present or absent.
- ✓ Plasmogamy is through samotagamous.
- ✓ They produce fruiting body called basidiocarp/ basidiomata.
- ✓ Basidiocarp vary in size, shape and structure.
- ✓ Basidiospores may be ballistospore or statismospores.

Order: Agaricales

- ✓ It includes gilled and non-gilled fungi.
- ✓ Edible fungi are called 'Mushroom' and poisonous ones called 'Toadstools'
- ✓ Genera used as Edible fungi are: *Pleurotus* (Oyster mushroom), *Agaricus* (White button mushroom), *Volveriella* (Paddy straw mushroom).
- ✓ Example of Poisonous fungi: *Amanita phalloides* (Death chair or death cap), *A.verna* (destroying angel), *A.muscaria* (fly agaric).
- ✓ They produce poiosonous compound called **Amanitin** and **Phalloidin.**

MECHANISM OF GENETICAL VARIABILITY IN PATHOGENS

The characteristic of individuals within biological species are not fixed because the members of a population may be similar but not identical. The progenies produced involving sexual reproduction will differ among themselves as well as from the parents. In addition to sexual reproduction organism employ other means of multiplication, which also provide some mechanism for promoting variability among the population. Mutation and recombition are common mechanism employed by various organisms. Fungi and bacteria have some specialized methods of generating variability.

Fungal pathogen:

Fungal populations are genetically flexible. Various mechanisms operating in different group of fungi and are responsible for developing variability in phenotypic characters including virulence.

1) **Mutation:** It is universal mechanism of creating alternation in genetical material of a living organism. Mutation occurs due to the change in the genetic material, which is stable and inherited by the progeny. Mutation occurs spontaneously, but they can be induced also by physical and chemical mutagens. Frequency of spontaneous is very low about 10-7 to 10-4. Mutations are brought by any change in base sequence in genetic material.

2) **Recombination:** The fusion of two haploid nuclei results in formation of zygote i. e. diploid state of life cycle. During further division of zygote there are chances of crossover of parts of chromatids. This results in exchange of genetical material and formation of recombinants. Recombination is most potent tools for the creation of variability.

3) **Heterokaryosis:** A fungal cell containing two genetically different nuclei in the same cytoplasm is known as heterokaryon, which may arise due to fertilization or anastomosis between genetically different cell due to mutation in one of the nucleus of bi or multinucleate cell. It is common mechanism for fungi and particularly in *Basidiomycota*, important for causing infection. E.g. Rust, smut, *Alternaria solnai* and *Botritis cineria*.

4) **Parasexuality:** Certain fungi enjoy the benefits of recombination without undergoing normal sexual cycle. No coordination or sequence in fertilization, segregation and recombination is observed. The chances of variability created through parasexuality are great as it may originate heterokaryon. Parasexuality are great as it may originate heterokaryon.

5) **Heteroploidy:** Spore or cells of the same fungus may contain variable numbers of chromosomes depending upon the number of for that organism.

Bacterial pathogen:

In bacteria and probably in mollecutes, following sexual like process are responsible for creating variability for various characters

1) **Conjugation:** Two bacterial cells come in contact and genetical material is transferred from one bacterial cell to another, through conjugation bridge i.e. pilus. Once foreign DNA is inside recipient cell, it may results in formation of recombinant.

2) **Transformation:** Bacterial cells absorb genetical material secreted by other, without coming in physical contact. This genetical material gets integrated in its own genome and may lead to variability for certain characters.

3) **Transduction:** This is bacteriophage mediated transfer for genetical material from one bacterial cell when the phage particle infects another bacterial. This is highly potential tool for horizontal gene transfer. *Agrobacterium* is often is used to transmit genetical material crossing various taxonomic barrier, sometimes across kingdom also.

Viral pathogen:

1) **Mutation:** When small quantity of virus particle is inoculated in a suitable host the particles show immutability and newly synthesized particles produce the same effect on the host as the original particle. These are known as virus mutants.

2) **Mixed inoculation:** Natural inoculation often consist of mixture of virus strain which can be demonstrated by inoculating a local lesion host with dilute sap and then sub-culturing the

virus from individuals lesion. Presence of many strains or genetical material in the same environment may lead to hybridization.

3) **Pseudo-recombination:** In mixed infection of viruses having split genome, fragments of the genome may aggregate and re-assemble to form a new type of progeny. This mechanism differs from true genetic recombination found in cellular organisms where the recombination occurs from the exchange of genetic information between parental genomes at crossing over chromosomes during meiosis.

4) **Genome masking (Heterologous encapsidation):** This can result from direct interaction between different viruses in mixed inoculations. The genome of one virus is encapsidated in the protein of another strain of virus. Since the effect of a virus on the host or the vector is mediated through its protein. This may assist virus in infecting a host not normally susceptible to it or being transmitted by vector or vector species that normally does not do so. If two strains of virus inoculate or vector introduce in the same host one or more new strain may be recovered with properties different either of both virus. These new strain can be called as hybrid.

INTRODUCTION TO PLANT PATHOLOGY : DEFINITION AND OBJECTIVES OF PLANT PATHOLOGY

Why Plant Pathology?

Plants are essential for maintenance of life. Plants not only sustain the man and animals, they are also the source of food for multitudes of micro-organisms living in the ecosystem. Thus, while man has been able to subjugate plants and animals for his own use, the competing micro-organisms still defy his efforts and claim a major share of resources which man would like to use for himself. It is in this context that the need for fighting the competing micro-organisms and other agencies that lack loss of productivity has been felt. The attack on plants by these micro-organisms changed the appearance and productivity of the crop and this observed change was called a disease. Plant diseases have been considered as stubborn barriers to the rapid progress of food production. We call a plant healthy only so long as it continues to perform all its normal physiological activities and give the expected yield according to its genetic potentiality.

Physiological activities of a healthy plant

1. Normal cell division, differentiation and development.
2. Uptake of water and nutrients from the soil.
3. Synthesis of food from sunlight by photosynthesis.
4. Translocation of water and food to the sites of necessity through xylem and phloem.
5. Metabolism of synthesized material
6. Reproduction

A diseased plant fails to perform one or more of these functions. The effect of a disease on functioning of an organ depends on which cells or tissues were first attacked by the pathogen.

For example, rotting of root tissues will affect the absorption of water and minerals from soil and if vascular tissues have been affected, the translocation of water and photosynthetates will be stopped or reduced. If leaf tissues are attacked by a pathogen, photosynthesis is affected and plant suffers from deficiency of carbohydrates essential for supplying energy for other activities. Thus, disease can be defined as malfunctioning process that is caused by continuous irritation by a pathogen (Dimond, 1959).

Definitions:

1. **Disease** is a malfunctioning process that is caused by continuous irritation which results in some suffering producing symptoms (American Phytopathological society & British Mycological society).
2. **Disease** is an alteration in one or more of the ordered sequential series of physiological processes culminating in a loss of coordination of energy utilization in a plant as a result of continuous irritation from the presence or absence of some agent or factor.
3. **Disease:** Any malfunctioning of host cells and tissues that result from continuous irritation by a pathogenic agent or environmental factor and leads to development of symptoms (G.N.Agrios, 1997).

Pathogens bring about these irritating processes through different but inter-related pathways

1. by utilizing the host cell contents,
2. by causing death of cells or by interfering with their metabolic activities through their enzymes, toxins and growth regulators,
3. by weakening of tissues due to continuous loss of nutrients, and
4. by interfering with translocation of food, minerals and water.

OBJECTIVES OF PLANT PATHOLOGY:

The science of plant pathology has four main objectives:

1. to study the living, non-living and environmental causes of plant diseases,
2. to study the mechanisms of disease development by pathogens,
3. to study the interactions between the plants and the pathogen, and
4. to develop the methods of controlling the diseases and reducing the losses caused by them.

Ingredients of Plant Pathology:

Plant Pathology consists of following ingredients:

1. Plant pathology is an applied science.
2. It is a branch of agricultural, biological or botanical science.
3. Science of plant pathology includes systemic study of disease causing agents, symptoms, etiology, host-parasite relationship, pathogenesis, predisposing factors and mode of perpetuation of disease.
4. Assessment of losses from diseases.
5. Management of diseases preferably of economic crops which aims at better crop production.

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HISTORY OF PLANT PATHOLOGY

Ancient period:

A literature of European and vedic eras will give us some information on the plant diseases and their control measures. Greek philosopher **Theophrastus** recorded some observations on the plant diseases in his book **enquiry into plants**. His experiences were mostly based on imagination and observation but not on experimentation. He had mentioned that plants of different groups have different diseases which were autonomous
or spontaneous, i.e., no external cause was associated.

In India, the information on plant diseases is available in ancient literature such as rigveda, atharveda (1500-500BC), arthasashtra of Kautilya (321-186 BC), Sushruta sanhita (200-500AD), Vishnupuran (500AD), Agnipuran (500-700AD), Vishnu dharmottar (500-700AD), etc. In Rigveda, not only the classification of plant diseases has been given but the germ theory of disease was also advocated.

Vriksha ayurveda by **Surpal** in ancient India is the first book in which lot of information on plant diseases is available. In this book, plant diseases were categorized into two groups, internal (probably physiological diseases) and external (probably infectious diseases). External diseases were supposed to be due to attack of microorganisms and insects. In this book, a mention of treatments for different diseases caused by different agencies was prescribed which were based on superstition as well as scientific observation. Hygiene, tree surgery, protective covering with pastes and special culture of plants are practices which are still recommended. In chemical treatments, use of honey, ghee, milk, barley flour, pastes made from herbs, plant extracts, etc., were recommended. For the control of root diseases, oilcakes of mahuva, mustard, sesame, castor, etc., were used.

Symptoms of plant diseases such as rust, downy mildew, powdery mildew and blight are often mentioned in the bible, Shakesphere's poems and dramas of other Christian literature. In Jataka of Buddhism, Raghuvansh of Kalidas there was also a mention about different symptoms of plant diseases.

In Europe and other western countries, after the time of Theophrastus (about 286 BC) no definite opinion could be formed about plant disease for the next 2000 years. In ancient

period, the plant diseases were attributed to many causes which include divine power, religious belief, superstition, effect of stars and moon, bad wind and wrath of god, etc.

(1) P.A. Micheli (Pier Antonio Micheli) (1729):

- He was to study the fungi and saw the spores of fungi.
- He published a book – "Nova Plantarum Genera"
- He is considered as " Father of Mycology"

(2) T. Needham (1743):

- He reported first plant parasitic nematodes in wheat-galls-Ear cockle of wheat (*Anguina tritici*).

(3) Linnaeus (1753):

- He was using latin binomial system for naming the plants & fungi.
- He published – "Species Plantarum"
- He is considered as "Father of Botany"

(4) Tillet (1755):

- He published a paper on "Bunt or stinking smut of wheat".
-He showed contagious (infectious) nature of "wheat bunt" disease.

(5) Persoon, C.H. (1801):

- He published a Book – "Synopsis Methodica Fungorum".
- He classified many fungi using binomial nomenclature as Uredinales, Ustilaginales etc.

(6) Fries Elias Magnus (E.M. Fries) (1821-1832):

- He published "Systema Mycologicum".
- He regarded as "Linnaeus" of Mycology.
- He considered rust and smut fungi as a products of the diseased plants.

(7) Prevost (1807):

- He proved that diseases are caused by microorganisms.
- He was first man to show microorganism as disease causing agent.
- He discovered the life cycle of "Bunt fungus".
- He observed the spore germination in bunt fungus.
- He was the first man to suggest or use of chemical copper sulphate for seed treatment to control seed borne disease.

(8) Anton De Bary (1831-1888):

- He is considered as "Father of Modern Plant Pathology".

- He studied late blight of potato fungus, its nomenclature and finally renamed the pathogen as *Phytophthora infestans*.

(9) Berkeley (1855) :

- Observed root knot nematodes in galls of cucumber.

(10) Julius Kuhn (1858) :

- Published a text book in Plant Pathology entitled " The Diseases of Cultivated Crops, Their Causes and Their Control".

(11) Robert Harting (1935-1901):

- Worked on Forest Pathology

- He wrote two important books in German on :

    (1) Important Diseases of Forest Trees (1874)

    (2) Text Book of Tree Diseases (1882)

- He is called the "Father of Forest Pathology".

(12) T.J. Burrill (1878) :

- He first time proved that the fireblight of apple & pear was caused by a bacterium.

(13) Brefeld & its colleague (1875-1912) :

- Discovered the methods of artificial culture of micro-organisms i.e. gave "Pure culture Techniques".

(14) Pierre Marie Alexis Millardet (P.M.A. Millardet) (1882) :

- He discovered the Bordeaux mixture in 1882 for the control of downy mildew of grape caused by *Plasmopara viticola*.

(15) Robert Koch (1876) :

- He considered as "Father of Medical bacteriology".

- He proved that the anthrax disease of cattle is caused by a bacterium.

- He gave Koch's rules or Koch's postulates or proof of pathogenicity.

(16) J.C. Walker (1883):

- He wrote two books on

    (i) Plant Pathology (1968)

    (ii) Diseases of Vegetable Crops (1952).

(17) Adolph Mayer (1886):

- First time discovered the tobacco mosaic virus (TMV).

- He demonstrated that this disease (TMV) could be transmitted from diseased to healthy plants by the sap from leaves showing mosaic symptoms.

(18) E.F. Smith (1891) :

- He first time demonstrated that budding or grafting could be another method of transmission of plant viruses.

(19) Dimitrii Ivanowski (1892) :

- He demonstrated that tobacco mosaic virus (TMV) could pass through even those filters that retained (to keep) bacterial cells. So he suggested that viruses were smaller than smallest known bacterium.

(20) M.W. Beijerinck (1898) :

- He concluded that Tobacco Mosaic Virus (TMV) was caused by "*Contagium Vivum Fluidum*" (infectious living fluid) which he called a "VIRUS".

(21) Stakman Elvin Charles :

- He wrote a book on " Principles of Plant Pathology" in association with Harrar, J.G.

(22) Vander Plank (1963) :

- He written book on

  (i) Disease Resistance in Plants (1968)

  (ii) Principles of Plant Infections (1975).

(23) Stanley, W.M. (1935) :

- He got Noble Prize. He purified and crystallized Tobacco Mosaic Virus (TMV).

(24) Kausche and his associates (1939) :

- He observed first time virus through electron microscope.

(25) Nathan Augustus Cobb (N.A. Cobb) (1913-1932) :

- He contributed a lot to nematode morphology, taxonomy and methodology.

- He considered as "Father of Plant Nematology".

(26) Doi and Ishi (1967):

- Mycoplasma like organisms (MLO's) can cause yellows type diseases, very much similar to diseases caused by viruses.

(27) White. P.R. (1968) :

- He formulated a synthetic medium as White's medium, to raise plants *in vitro* for tissue culture.

(28) Diener, T.O. (1971) :

- Determined that potato spindle tuber disease is caused by small molecules of infectious ribonucleic acid (RNA) which he called "VIROIDS".

(29) Davis and Worley, J.E. (1972) :

- He first time discovered "Spiroplasma" in corn stunt disease.

(30) Windsor, I.M. and Black, L.M. (1972) :

- He observed first occurrence of Rickettsia like organisms (RLO's) in clover club leaf disease.

History of Plant Pathology in India:

(1) E.J. Butler (1905) :

- He considered as "Father of Indian Plant Pathology".
- He wrote book on "Fungi and Disease in Plants" in 1918.

(2) Mehta, K.C. (1929):

- Discovering the disease cycle of cereal rusts in India.

(3) B.B. Mundkur (1948) :

- He published a textbook "Fungi and Plant Diseases".
- He started single handed "Indian Phytopathological Society" with its journal "Indian Phytopathology" in 1948.

(4) J.F. Dastur (1886-1971) :

- He was studied the genus *Phytophthora* and diseases of castor.
- He is internationally known for establishing the species of *Phytophthora parasitica* from castor.

(5) M.J. Thirumalachar:

- He successfully introduced the use of antibiotics in control of plant diseases eg. Aureofungin and Streptocycline.

(6) J.C. Luthra and his associates:

- He developed the solar treatments of wheat seed for control of loose smut (internally seed borne disease).

(7) M.K. Patel (1948-51):

- Devised a synthetic medium known as PATEL's MEDIUM to cultivate bacterium.

(8) R.S. Singh (1950 to onwards):

- He wrote very useful textbook on
  - (i) Introduction to Principles of Plant Pathology (1975)
  - (ii) Plant Pathogens, The Fungi (1982)
  - (iii) Plant Diseases

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TERMS AND CONCEPTS USED IN PLANT PATHOLOGY

1. **Plant Pathology or Phytopathology:**

   Consist of three Greek words

   -Phyton means plant,

   -Pathos means suffering / ailments or diseases and

   -Logus means knowledge / science

   Plant Pathology or Phytopathology is that branch of agricultural, botanical or biological science which deals with the study of plant diseases, their cause, etiology, resulting losses and control of plant diseases.

   It is a science similar to that of human medicine and veterinary which deals with diseases of man and animals, respectively.

2. **Parasite:** An organism living on or in another living organism (host) and obtaining its food from the latter (host).
3. **Ectoparasite :** a parasite that lives and feeds from the exterior of its host's cells or tissues.
4. **Endoparasite :** a parasitic organism that lives and feeds within the cells or tissues of its host.
5. **Pathogen:** An entity, usually a micro-organism, that can incite disease.
   - Most pathogens are also parasites in that they derive the nutrients for growth from a living plant, the host or suscept.
   - All parasites may not be pathogens but all pathogens are parasite. eg. Cuscutta (dodder / amarvel) is parasite on Lucerne crop but it is not pathogen.
6. **Host :** an organism harbouring a parasite or pathogen.
7. **Abiotic :** pertaining to physical and inorganic components. For example, diseases/disorders in plants can be caused by abiotic factors such as extremes of heat, light, moisture, lack of nutrients etc.
8. **Biotic :** pertaining to life and therefore living organisms. For example, plant diseases of a biotic origin are caused by living organisms such as fungi, bacteria, virus, viroids, algae, insects, nematodes, etc.

9. **Disease:** Any malfunctioning of host cells and tissues that results from continuous irritation by a pathogenic agent or environmental factor and leads to development of symptoms.

   When something is functioning poorly in the body "we come to decision that we are sick".

11. **Plant Disease:** A physiological disorder or structural abnormality that is harmful to the plant or only it's parts or products that reduced the economic value.
12. **Disorder:** Non-infectious plant diseases due to abiotic causes such as adverse soil and environmental conditions are termed disorders. The common characteristic of noninfectious diseases of plants is that they are caused by the lack or excess of something (temperature, soil moisture, soil nutrients, light, air and soil pollutants, air humidity, soil structure and pH) that supports life. Non-infectious plant diseases occur in the absence of pathogens, and cannot, therefore, be transmitted from diseased to healthy plants.
13. **Disease Incidence :** the number of plants affected by a disease within a population. eg. For soil borne diseases, nematodes
14. **Disease Severity :** the measure of damage done by a disease. OR Amount of disease present in a population. eg. For Leaf, stem, seed diseases
15. **Hypha (pl. Hyphae) :** a single tubular thread-like filament of a fungal mycelium. The hypha is the basic structural unit of a fungus.
16. **Mycelium (pl. Mycelia) :** a mass of hyphae that forms the body (thallus) of a fungus.
17. **Spore:** a specialised reproductive body in fungi (and some other organisms), containing one or more cells, capable of developing into an adult.
18. **Biotroph:** an organism that can live and reproduce only on another living organism. A biotroph is completely dependent on the host organism as a source of nutrients, i.e. it is an obligate parasite.
19. **Necrotroph :** an organism (parasite) that causes the death of host tissues as it grows through them, obtaining its energy from the dead cells.
20. **Saprophyte :** an organism that obtains nourishment from non-living organic matter (usually dead and decaying plant or animal matter) by absorbing soluble organic compounds.
21. **Pathogenicity:** The capacity of a pathogen to cause disease.

22. **Pathogenesis** is the chain of events that lead to development of disease in the host (or) sequence of progress in disease development from the initial contact between the pathogen and its host to the completion of the syndrome.
23. **Sign:** The pathogen or its parts or products seen on a host plant.
24. **Symptom:** The external or internal reactions or alterations of a plant as a result of a disease.
25. **Syndrome:** The set of varying symptoms characterizing a disease are collectively called a syndrome. **OR** The series of symptoms of a disease collectively known as syndrome.
26. **Virulence:** The degree of pathogenicity of a given pathogen.
27. **Virulent:** Capable of causing a severe disease; strongly pathogenic.
28. **Avirulent (non-virulent):** Non-pathogenic or Lacking virulence.
29. **Infection:** The establishment of a parasite within a host plant.
30. **Latent Infection :** where the host is infected with a pathogen but does not show any symptoms.
31. **Infectious disease:** A disease that is caused by a pathogen which can spread or transfer from a diseased to a healthy plant.
32. **Non-infectious disease:** A disease that is caused by an abiotic agent, that is, by an environmental factor, not by a pathogen.
33. **Inoculum:** The Pathogen or its parts that can cause infection. That portion of individual pathogens that are brought into contact with the host.
34. **Inoculum potential:** Which consists of the number of propagules and their capacity to cause infection.
35. **Inoculate:** To bring a pathogen into contact with a host plant or plant organ.
36. **Inoculation:** The arrival or transfer of a pathogen onto a host.
37. **Isolation:** The separation of a pathogen from its host and its culture on a nutrient medium.
38. **Isolate:** A single spore or culture and the subcultures derived from it.
39. **Microscopic:** Very small; can be seen only with the aid/use of a microscope.
40. **Penetration:** The initial invasion of a host by a pathogen.
41. **Primary infection:** The first infection of a plant by the overwintering or oversummering pathogen.

42. **Primary inoculum:** The overwintering or oversummering pathogen, or its spores that cause primary infection.
43. **Secondary infection:** Any infection caused by inoculum produced as a result of a primary or a subsequent infection. **OR** An infection caused by secondary inoculum.
44. **Secondary inoculum:** Inoculum produced by infections that took place during the same growing season.
45. **Transmission:** The transfer or spread of a pathogen from one plant to another.
46. **Culture:** To artificially grow micro-organisms on a prepared food material.
47. **Immune:** Cannot be infected by a given pathogen.
48. **Resistant:** Possessing qualities that hinder the development of a given pathogen. **OR** Infected little or not at all.
49. **Susceptible:** Lacking the inherent ability to resist disease or attack by a given pathogen; non-immune.
50. **Tolerance:** The ability of a plant to sustain the effects of a disease without dying or suffering serious injury or crop loss.
51. **Disinfectant:** A physical or chemical agent that frees a plant, organ or tissue from infection.
52. **Disinfestant:** The chemical agent that kills or suppress or inactivates pathogens in the environment or on the surface of a plant or plant organ before infection takes place.
53. **Fungicide:** Chemicals used to kill fungi **OR** A compound toxic to fungi.
54. **Nematicides:** A chemical compound that kills nematodes.
55. **Bactericide:** A chemical compound that kills bacteria.
56. **Mycology:** The study of fungi is called mycology. OR It is the science which deals with the study of fungi is known as mycology.
57. **Nematology:** It is the science which deals with the study of nematodes.
58. **Virology:** It is the science which deals with the study of viruses.
59. **Bacteriology:** It is the science which deals with the study of bacteria.
60. **Eradication :** the control of plant disease by eliminating the pathogen after it is established or by eliminating all of the plants that carry the pathogen.
61. **Exclusion :** a method of disease prevention in which the pathogen or infected plant material is excluded from crop production areas. See quarantine.

62. **Quarantine :** legal restriction of the transport of plants and/or plant parts in order to prevent the spread of pests and pathogens.

63. **Race :** a subgroup of pathogens within a species that infect a given set of plant varieties. Races may be distinguished from each other by virulence or symptom expression but not by morphology.

64. **Systemic : (1)** pertaining to a disease in which the pathogen spreads generally throughout the plant **OR** The growth of pathogen from the point of entry to varying extents without showing adverse effect on tissues through which it passes.; **(2)** pertaining to a chemical absorbed into the plant through root or foliage and transported internally throughout the plant.
- Systemic fungicide: a chemical agent that spreads internally through the plant and eradicates established fungal infections.

65. **Vector:** any living organism (e.g., insect, mite, bird, nematode, parasitic plant, human, etc.) that transmits a pathogen from an infected organism to an uninfected one.

66. **Virus :** a submicroscopic, non-cellular structure consisting of a core of infectious nucleic acid (either RNA or DNA) within a protein coat.

67. **Epidemic or Epiphytotic disease:** A disease usually occurs widely but periodically in a destructive form is referred as epidemic or Epiphytotic disease. Ex: Late blight of potato – Irish famine (1845)

68. **Sporadic disease**: Occur at very irregular intervals and locations and in relatively fewer instances. Ex: Udbatta disease of rice, Angular leaf spot of cucumber – *Pseudomonas lachrymans.*

69. **Endemic**: Constantly present in a moderate to severe form and is confined to a particular country or district.
Ex: Club root of cabbage in Nilgiris
Black wart of potato – *Synchytrium endobioticum*
Onion smut – *Urocystis cepulae*

70. **Necrosis:** Death of tissue, cells or organ is known as necrosis.

71. **Phyllody:** Floral part converted into leafy structure is called as phyllody.

72. **Hypertrophy:** Excessive growth of host tissues due to increase in size of affected organs or cells of a particular tissues.

73. **Hyperplasia:** It is the abnormal increase in the size of a plant organ due to increase in number of cells of which the organ is composed.
74. **Appressorium:** The swollen tip of a hypha or germ tube that facilitates attachment and penetration of the host by the fungus.
75. **Disease cycle:** The chain of events as the transmission of pathogen, attack on the host, infection, invasion of tissues and development and reproduction of pathogen on the host to completing the process of pathogenesis.
76. **Antagonism:** Injury, killing or inhibition of the growth of one species of microorganism to another microorganism.
77. **Antagonists:** An organism having the capacity or ability to inhibit the growth or interfere with the activity of another microorganism.
78. **Antibiosis:** Inhibition or destruction of one microorganism by a metabolic products of another microorganism.
79. **Competition:** The ability of one organism to utilize a substrate more efficiently than another microorganism.
80. **Hyper parasitism:** The parasitism of a parasite by another parasite. (*Rhizoctonia solani* on *Pythium* spp., *Fusarium maxima* on Rust fungi).
81. **Hypersensitivity:** Excessive sensitivity of plant tissue to certain pathogens. Affected cells are killed quickly blocking the advancement of obligate parasite.
82. **Adjutants:** The inert materials added to a fungicide to improve the physical characteristics of the toxicant and its carrier. Most of them are surface active agents and cause variations in surface tension.
83. **Compatibility:** It is a combing ability of a chemical with each other without any adverse effect.
84. **Phytotoxicity:** The capacity of a compound to produce injury to plant.
85. **Tenacity:** It is an ability of chemicals/pesticides for holding them on plant surface.

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SURVIVAL OF PLANT PATHOGENS

The means of survival are the **first link** in infection chain or disease cycle. The initial infection that occurs from the sources of pathogen survival (Infected host as a reservoir of inoculum, saprophytic survival outside the host or dormant spores and other structures in or on the host or outside the host) in the crop is *primary infection* and the propagules that cause this infection are called *primary inoculum*. After initiation of the disease in the crop, the spores or other structures of the pathogen are sources of *secondary inoculum* and cause *secondary infection*, thereby spreading the disease in the field.

Ex: The fungus (*Phytophthora infestans*) causing late blight of potato survives in seed tubers or in soil. ***Infected tubers*** bring the ***primary infection*** in the field while primary inoculum (PI) present in soil causes primary infection of the crop from healthy seed. The PI may also be brought by wind from neighboring fields or long distances. Then the fungus produces spores on leaves. These spores are dispersed by wind and water and reach healthy plant surfaces to cause new infections. This is ***secondary infection***. **The primary infection initiates the disease and secondary infection spreads the disease.**

SOURCES OF SURVIVAL OF PATHOGENS:

1) Infected host as reservoir of inoculum (or) survival in vital association with living plants.
2) Survival as saprophytes outside the host.
3) Survival by means of specialized resting structures in or on the host or outside the host.
4) Survival in association with insects, nematodes and fungi.

1) Infected host as reservoir of inoculum:

The infected host serving as reservoir of active inoculum is grouped into

a) **Seed:** Seed may be externally or internally infected by plant pathogens during the course of development and maturation in fruit or pod. Most seed borne pathogens survive as long as seed remains viable.

Ex 1: The pathogen of loose smut of wheat, *Ustilago nuda tritici*, enters the stigma and style and infects the young seed, in which it survives as mycelium. So it is internally seed borne disease.

Ex 2: Grain smut of sorhum (Sphacelotheca sorghi) is externally seed borne disease.

Ex 3: *Pseudomonas syringae pv. tomato* has been shown to survive in dried tomato seed for 20 years.

b) **Collateral hosts / Alternative hosts (wild hosts of same families)**: Collateral hosts are those which are susceptible to the plant pathogens of crop plants and provide adequate facilities for their growth and reproduction of these pathogens during off-season. Weeds which survive and live during non-cropping season provide for the continuous growth and multiplication of the pathogen. Thus the weed hosts help to bridge the gap between two crop seasons.

Ex: The fungal pathogen for blast disease of rice, *Pyricularia grisea* (Teleomorph: *Magnaporthe grisea*) can infect the grass weeds like *Brachiaria mutica*, *Dinebra retroflexa*, *Leersia hexandra*, *Panicum repens* etc., and survive during off-season of rice crop. As soon as a fresh rice crop is raised, the conidia (inoculum) liberated from the weed host disseminated by wind infects the fresh rice crop.

c) **Alternate hosts (Wild hosts of other families):** The role of alternate hosts is not as important as of collateral hosts. However, when a pathogen has very wide host range (as *Sclerotium rolfsii, Rhizoctonia solani, Fusarium moniliforme* etc.) and is tolerant to wide range of weather conditions the alternate hosts become very important source of survival of the pathogen. These alternate hosts are very important for the completion of the life cycle of **heteroecious rust** pathogens. For example in temperate regions the alternate host of *Puccinia graminis tritici* (black or stem rust pathogen of wheat), the barberry bush (*Berberis vulgaris*) grows side by side with the cultivated host. In such areas this wild host belonging to a different family is important for survival of the fungus.

Differetiate between the Alternate host and Collateral host:

Sr. No.	Alternate host	Collateral host
1	Wild host of different families	Wild host of same families
2	Pathogen has very wide host range	Pathogen has narrow host range
3	Heterogenous infection chain	Homogenous or continuous infection chain
4	-Root knot nematode on potato, tomato, brinjal of solanaceae family, okra and cotton malvaceae family, cucurbits in cucurbitaceae family - *Sclerotium rolfsii* wide host range - *Rhizoctonia solani* etc.	eg. -Powdery mildew and mosaic of cucurbits pathogen survive on wild cucurbit plants. - Cereal rust in India pathogen over summer on other members of the host families i.e. grasses & self sown wheat on hills at different altitude. - Grass host of *Sclerospora sacchari*, *Ustilago scitaminea* (sugarcane smut), *Pyricularia oryzae* (rice blast).

Differentiate between Autoecious rust and Heteroecious rust:

Sr. No.	Autoecious rust	Heteroecious rust
1	A parasitic fungus that can complete its entire life cycle on the same host is called as **Autoecious rust**	Requiring two different kinds of hosts to complete its life cycle is called as **Heteroecious rust**
2	All the spores (Pycniospores, Aeciospores, Uredospores, Teliospores, Basidiospores) states may be formed on a single host as in autoecious rust	All the spores states may be formed on two alternate host as in heteroecious rust
3	eg. Linseed rust (*Melampsora lini*) , Pea rust (Uromyces fabae), Sunflower rust (Puccinia helianthi), Alfalfa rust (Uromyces striatus), Bean rust (Uromyces phaseoli) all are exaples of autoecious rust.	eg. – Alternate host of black stem rust of wheat (*Puccinia graminis tritici*) is berberry - Alternate host of bajra rust (*P. penniseti*) is brinjal.

d) **Self sown crops:** Self sown crops, voluntary crops and early sown crops are reservoirs of many plant pathogens. Ex: Self sown rice plants harbour the pathogen (*Rice tungro virus*) as well as vector (*Nephottetix virescens*).

e) **Ratoon crops:** Sometimes ratoon crops also harbour the plant pathogens.
Ex: Sugarcane mosaic.

f) **Survival by latent infection:** Latent infection refers to the conditions in which the plant pathogens may survive for a long time in plant tissue without development of visual symptoms. Ex: *Xylella fastidiosa*, the causal agent of pierce's disease of grapevine infect different weeds without developing visible symptoms.

2) Saprophytic survival outside the host:

The ability to live saprophytically enables many plant pathogens to survive in the absence of growing susceptible plants. Saprophytic survival usually occurs in or on the soil. Waksman (1971) distinguished between soil inhabitants and soil invaders; the former comprise the basic fungal flora of the soil, whereas the later are short lived exotics.

In the absence of the cultivated host plant, fungi are capable of surviving as saprophytes and can be studied under three categories:

1) **Soil inhabitants**: Those organisms which survive indefinitely in the soil as saprophytes in the absence of the host plant. Ex: Species of *Pythium*, *Rhizoctonia* and *Sclerotium*

2) **Root inhabitants**: These are more specialized parasites that survive in soils in close association with their hosts. The active saprophytic phase remains as long as the host tissue in which they are living as parasites is not completely decomposed. Ex: Species of *Fusarium*, *Verticillium* (vascular wilt causing fungi) and root rot of cotton (*Phymatotrichum omnivorum*)

3) **Rhizosphere colonizers**: Those organisms which colonize the dead substrates in the root region and continue to live like that for a longer period which are more tolerant to soil antagonism. Ex: Leaf mold in tomato: *Cladosporium fulvum*

Differentiate Soil inhabitants and soil invaders:

Soil inhabitants		Soil invaders
1. These are unspecialized parasites with a wide host range that are able to survive indefinitely in the soil as saprophytes.		1. These are more specialized parasites that survive in soils in close association with their hosts.
2. Pathogen remain in soil for more than 4-5 years		2. Pathogen remain in soil for 2-3 years
3. Pathogen survive in soil without plant debris		3. Pathogen survive in diseased plant debris in soil
4. Produced dormant structures like oospore, sclerotia and chlamydospores		4. Not produced dormant structures
5. Difficult to manage as long duration crop rotation is required		5. Easy to manage by short crop rotation
6. Fast multiplication		6. Slow multiplication
7. eg. Pythium, Fusarium, Sclerotium, Macrophomina		7. eg. Colletotrichum, Alternaria, Pyricularia etc.

3) Survival as dormant spores or specialized resting structures:

Plant viruses have no resting stage and are transmitted through a continuous infection chain.

Phytopathogenic bacteria: The plant **bacteria** also do not produce resting spores or similar structures. They continuously live in their active parasitic stage in the living host or as active saprophytes on dead plant debris.

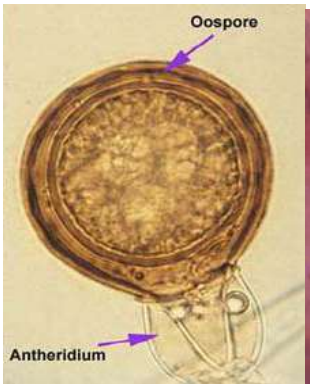
Nematodes: They survive in the form of active parasitic phase on a living host and also survive through dormant structures, i.e., eggs, cysts, galls, formed in host tissues. These structures may be present in soil or in seed lots.

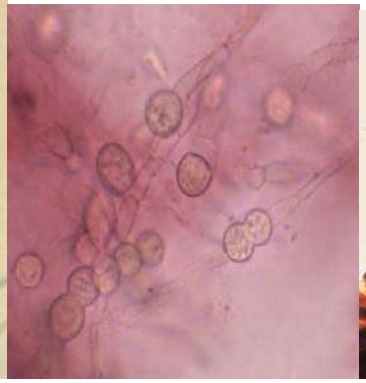
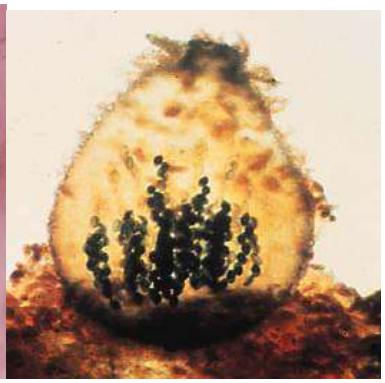
Phanerogamic parasites: They survive in dormant state for many years through seeds. Ex; Seeds of Orobanchae survive in soil for more than 7 years.

Among plant pathogens, fungi are the only organisms that produce spores, analogous to eggs of nematodes, and other resting structures for their inactive survival. These dormant structures of survival can be classified in the following categories.

1) ***Soil borne fungi***:

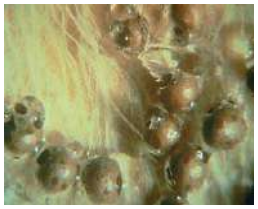
a) Dormant spores {Conidia (Peach leaf curl pathogen, *Taphrina deformans*), Chlamydospores (Wilt pathogen, *Fusarium* sp.), oospores (Downy mildew fungi), perithecia (Apple scab pathogen, *Venturia inaequalis*) etc.}.



Oospore Chlamydospores Perithecium

b) Other dormant structures such as thickened hypha, sclerotia (Cottony rot fungus, *Sclerotinia sclerotiorum*), microsclerotia (Verticillium), Rhizomorphs (Armillaria mellea), etc.



Thickened hyphae Sclerotia Rhizomorphs Microsclerotia

c) Factors affecting the survival of pathogen in the soil are **a)** physical factors (high temperature, irradiation, dessication and anaerobiosis), **b)** chemical factors (antibiotics, antagonistic chemicals produced by other microbes) and **c)** biotic factors (parasitism, predation by microflora and microfauna).

2) Seed borne fungi:

a. **Externally seed borne**: Dormant spores on seed coat Ex: Covered smut of barley, grain smut of jowar, bunt of wheat, etc.
b. **Internally seed borne**: Dormant mycelium under the seed coat or in the embryo Ex: Loose smut of wheat (*Ustilago nuda tritici*)
c. Factors affecting the survival of the pathogen on/in the seed are temperature and moisture.

3) ***Dormant fungal structures on dormant or active host***

Ex: In downy mildew of grapevine, powdery mildew of grapevine, apple etc., The fungus mycelium may be present in dormant state in the affected twigs or its oospores or perithecia may be embedded in the tissues of the affected organs.

Parasitic phanerogams survive in the form of seeds, and in plant parasitic nematodes eggs, cysts and larvae serve as over seasoning structures.

4) *Survival in association with insects, nematodes and fungi*

Several important plant pathogens may survive within the insect body and over winter therein. The corn flea beetle, *Cheatocnema pulicaria* carries inside its body, the corn wilt pathogen, *Xanthomonas stewartii* and thus helps in over wintering.

Plant viruses like wheat mosaic, tobacco necrosis, tobacco rattle and tobacco ringspot viruses survive with nematodes or fungi found in the soil between crop seasons. Tobacco ringspot is associated with the nematode *Xiphinema americana*. The fungi, *Polymyxa graminis* (Wheat soil borne mosaic & Barley yellow mosaic) and *Spongospora subterranea* (Potato mop top virus) carry the viruses internally and transmit them through the resting spore.

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DISPERSAL OF PLANT PATHOGENS

The **second link** in infection chain is the dissemination of plant pathogens. Transport of spores or infectious bodies, acting as inoculum, from one host to another host at various distances resulting in the spread of the disease, is called **dispersal, dissemination** or **transmission** of plant pathogens. The dispersal of the pathogen or disease is important not only for spread of plant diseases but also for continuity of the life cycle and evolution of the pathogen. The knowledge of these methods of dispersal is essential for effective control of plant diseases because possibilities of preventing dispersal and thereby breaking the infection chain exist.

In **fungi**, productions of asexual and sexual spores follow the active vegetative growth of the fungus in or on the host tissues and are dispersed mechanically in time and space by various means. In **bacterial diseases**, the bacterial cells come out on the host surface as ooze or the tissues may be disintegrated so that the bacterial mass is exposed and then dispersed by various physical and biological agencies. **Viral diseases** which have no such organs are transmitted by insects, mites, phanerogamic parasites, nematodes and human beings.

The two links in the infection chain of an animate pathogen, *Viz.*, survival through dormant structures and the dispersal of the pathogen are very closely bound with each other. Actually the dormant structures provide means of *dispersal in time*, i.e., the pathogen is retained viable over a period of time enabling it to be transported through physical agencies without being harmed.

The dispersal of infectious plant pathogens in *space* occurs through two ways:

1. Autonomous or direct or active dispersal.
2. Indirect or passive dispersal.

I) Autonomous or direct or active dispersal:

In this method the dispersal of plant pathogens takes place through soil, seed and planting material during normal agronomic operations. There is no major role of external agencies like insects, wind, water, etc. in this type of dispersal.

1) *Seed as the source of autonomous dispersal:*

Since most of the cultivated crops are raised from seed the transmission of diseases and transport of pathogens has much importance. The dormant structures of the pathogen (Ex: seeds of Cuscuta, Sclerotia of ergot fungus, smut sori, etc.) are found mixed with seed lots and they are dispersed as **seed contaminants**. The bacterial cells or spores of fungi present on the seed coat (such as in smuts of barley, sorghum, etc.) are transported to long distances. Dormant mycelium of many fungi present in the seed is transmitted to long distances. There are three types of dispersal by seed, *viz*., contamination of the seed, externally seed borne and internally seed borne.

a) *Contamination of the seed:* Seed borne pathogens move in seed lot as **separate contaminants** without being in intimate contact with the viable crop seeds. The seeds of the pathogen or parasite and the host are mixed during harvest of the crop. In many cases, the identity of the seeds of the two entities (host and the pathogens) is difficult to separate. Ex: Smut of pearlmillet and ergot of rye. Smut sori and ergots mix easily with the seed lots during harvest and threshing.

b) *Externally seed borne*: **Close contact between structure of the pathogen and seeds** is established where the pathogen gets lodged in the form of dormant spores or bacteria on the seed coat during growth of the crop or at the time of harvest and threshing. Ex: Short smut of sorghum, bacterial blight of cotton, loose smut of barley etc.

In many pathogens the externally seed borne structures such as smut spores can persist for many years due to their inherent capacity for long survival. Ex: The spores of *Tilletia caries* (Stinking smut of wheat) remain viable even after 18 years and those of *Ustilago avenae* (Oat smut) for 13 years.

c) *Internally seed borne*: The pathogen may penetrate into the ovary and cause **infection of the embryo** while it is developing. They become internally seed borne. Ex: **Loose smut of wheat**.

Differentiate Seed infection and infestation

Seed infection: The seed in infected only when the pathogen has grown in or on it for sometime and established its relationship with the seed tissues. Ex: Loose smut of wheat, where the fungus grows in the embryonic tissues and becomes dormant when the seed enters dormancy.

Seed infestation*:* When the fungus or the pathogen is present on the seed coat and in the seed lot, it is only transport of the pathogen and the seed is infested.

2) ***Soil as a means of autonomous dispersal:*** Soil borne facultative saprophytes or facultative parasites may survive through soil. The dispersal may be by movement of pathogen in the soil or by its growth in soil or by movement of the soil containing the pathogen. The former is known as *dispersal in soil (i.e. movement of the pathogen in soil)* while the later is called *dispersal by soil (i.e. movement of soil containing the pathogen).*

a) <u>Dispersal in soil:</u> The following are the three stages of dispersal in soil

i) *Contamination of soil*: Contamination of the soil takes place by gradual spread of the pathogen from an infested area to a new area.

ii) *Growth and spread of a pathogen in soil*: Once the pathogen has reached the soil it can grow and spread based on its ability to multiply and spread. Among characters of the pathogen its adaptability to soil environment including its **saprophytic survival ability** are most important. The survival ability of the pathogen is governed by high growth rate, rapid spore germination, better enzymatic activity, capability to produce antibiotics and tolerance to antibiotics produced by other soil-microorganisms.

On the basis of this competitive saprophytic ability the pathogens in soil can be of three types. **Specialized facultative parasites** (Saprophytes) can pass their life in soil in the absence of host plants, but they depend more on the residues of the host plant (ex: *Armillariella mellea* (wood rooting fungi), *Ophiobolous graminis* (brown leaf spot of rice) etc.). **Unspecialized facultative parasites** can pass their entire life in the soil (*Pythium* sp., and *Phytophthora* sp.). The soil borne **obligate parasites** such as *Plasmodiophora brassicae* (clubroot of crucifers), *Synchytrium endobioticum* (wart disease of potato) require the presence of active host.

Differentiate between Biotroph and Necrotroph:

Sr. No.	Biotroph	Necrotroph
1	Biotroph are those organisms who obtain their food from living tissues.	Necrotroph are the organisms who obtain their food from dead tissues also.
2	Affected host tissues are not killed immediately.	Affected host tissues are killed immediately.
3	Toxins and cytolytic enzymes are rarely produced	Toxins and cytolytic enzymes are plently produced
4	Haustoria are usually produced	Haustoria are not produced
5	Pathogen penetration through direct or natural opening	Pathogen penetration through wounds or natural opening
6	Pathogen may not survive out of host	Pathogen may survive out of host
7	Healthy tissues of plants are usually affected	Healthy tissues of plants are rarely affected
8	Biotrophs have restricted host range i.e. narrow host range	Necrotroph have wide host range
9	Eg. Rust fungi, Powdery mildew fungi, Downey mildew fungi and viruses are common examples of biotroph	Eg. Pythium, Rhizoctonia and soft rot bacteria (*Erwinia* spp) are common examples of necrotroph.

iii) *Persistence of the pathogen in soil*: The pathogens persist in the soil as dormant structures like **oospores** (*Pythium, Phytophthora, Sclerospora* etc.), **Chlamydospores** (*Fusarium*), smut spores (*Ustilago*) and **sclerotia** (*Rhizoctonia*, *Sclerotium*).

b) <u>Dispersal by the soil:</u> The pathogen is dispersed by the soil during cultural operations through the agricultural implements, irrigation water, workers feet etc. Propagules of fungi and the plant debris containing the fungal and bacterial pathogens thus spread through out the field. The transfer of soil from one place to another along with propagating materials is the most important method of dispersal of pathogen. For example transfer of papaya seedlings from a nursery infested with *Pythium aphanidermatum* (causal agent of stem or foot rot of papaya) can introduce the pathogen in new pits for transplanting the seedlings.

Similarly grafts of fruit trees transported with soil around their roots can transmit pathogens present in the nursery to the orchards.

3) *The plant and the plant organs as a means of autonomous dispersal:*

The plants, plant parts **other than seed** that are used for vegetative propagation, raw field produce and plant debris that accumulates during the course of cropping constitute the third method of autonomous dispersal. Ex: Late blight of potato was introduced in North America and in Europe through seed tubers brought from the native source of the in South America. Citrus canker was introduced into California from Asia. The climatic conditions favoured its epidemic in California. Bunchy top of banana was introduced from Ceylon to Kerala and it was also introduced from Maharashtra to Gujarat. Black scurf of potato was introduced in Gujarat through seed tubers brought from the source of the Himachal Pradesh.

II) <u>Passive or Indirect dispersal:</u>

Passive dispersal of plant pathogens happens through animate (biotic) and inanimate (abiotic) agents.

1) Animate (biotic)agents:

a) *Insects*: Insects carry plant pathogens either externally (**epizoic**) or internally (**endozoic**). They can disseminate bacteria, fungi, viruses, mycoplasmas, spiroplasmas, rickettsia, etc.

Fungal diseases: The external transmission is of special interest in those fungi which produce **conidia, oidia and spermatia in honey secretions** having attractive odours. Ex: Sugary disease of sorghum. The spermatial oozings at the mouth of spermagonia in the ascomycetes attract various type of insects, flies, pollinating bees and wasps which play a dual role, *viz*., pollination and transmission of plant pathogens. Dutch elm disease (*Ceratostomella ulmi*) is transmitted internally by elm bark beetles.

Bacterial diseases: The fire blight organism (*Erwinia amylovora*) and citrus canker bacterium (*Xanthomonas axonopodis pv. citri*) are transmitted by flies (bees) and ants and the later by leaf miner respectively. The cucumber wilt bacterium, *Erwinia tracheiphila* is spread by the stripped cucumber beetles (*Acalymma vittata*) and the spotted cucumber

beetle (*Diabrotica undecimipunctata*). When the beetles are feeding on the diseased plant, the bacterium contaminates the mouth parts and passes into the gut (intestine) of the beetle and over winters inside the beetle during the winter season. Thus the beetle helps the bacteria in two ways, *i.e.*, in their transmission and survival.

Viral diseases: More than 80 per cent of the viral and phytoplasmal diseases are spread by different types of insects. The insect which acts as specific carriers in disseminating the diseases are called *insect vectors*. Both **Aphids** (Aphididae) and **leaf hoppers** (Cicadellidae or Jassidae) in the order **Homoptera** contain largest number and the most important insect vectors of plant viruses. Certain species of mealy bugs and scale insects (Coccoidae), whiteflies (Aleurodidae) and tree hoppers (Membracidae) in Homoptera also transmit virus diseases. Insect vectors of plant viruses are few in true bugs (Hemiptera), thrips (Thysanoptera), beetles (Coleoptera) and grasshoppers (Orthoptera).

S.No.	Vector	Virus
1.	**Aphid transmitted viruses**	
	Myzus persicae	Beet mosaic, Lettuce mosaic, Potato virus Y, Turnip mosaic, Beet yellows, **Potato leaf roll virus**
	Acyrthosiphon pisum	Bean common mosaic, Bean yellow mosaic, Soybean mosaic, Pea enation mosaic
	Toxoptera citricidus	Citrus tristeza
2.	**Leaf hopper transmitted viruses**	
	Nephotettix impicticeps, N. nigropictus, N. virescens	Rice tungro virus
	Nephotettix cincticeps, N. nigropictus	Rice dwarf virus
	Circulifer tenellus	Beet curly top
	Agallia contricta	Potato yellow dwarf
	Graminella nigrifrons	Maize chlorotic dwarf
3.	**Tree hopper transmitted viruses**	
	Micrutalis malleifera	Tomato-pseudo curly top

4.	**Plant hopper transmitted viruses**	
	Perigrinus maidis	Maize mosaic
	Sogatodes oryzicola	Rice hoja blanca
5.	**Whitefly transmitted viruses**	
	Bamesia tabaci	Bhendi / Okra yellow vein mosaic, Bhendi leaf curl, Chilli leaf curl, Cotton leaf curl, Papaya leaf curl, Tomato leaf curl, Mungbean yellow mosaic
6.	**Thrips transmitted viruses**	
	Thrips tabaci, Frankliniella schultzei, Scirtothrips dorsalis	Tomato spotted wilt virus
	Frankliniella schultzei	Bud necrosis of groundnut
7.	**Mealy bugs transmitted viruses**	
	Planococcoides njalensis	Cocoa swollen shoot
	Pseudococcus saccharifolii	Sugarcane spike (Phytoplasma)
8.	**Grass hoppers transmitted viruses**	
	Melanophus differentialis	Potato virus X, Tobacco mosaic virus (Mechanical transmission)
9.	**Lace bugs transmitted viruses**	
	Piesma quadratum	Beet leaf curl virus
	Stephanites typicus	Root (wilt) disease of coconut (Phytoplasma)
10.	**Beetle transmitted viruses**	
	Ceratoma trifurcata	Cowpea mosaic
	Acalymma trivitata	Squash mosaic
	Diabrotica longicornis	Brome mosaic

Mycoplasma diseases: Plant MLO's are phloem inhabitants and those insects which are feeding on phloem of plants transfer the MLO's. Mycoplasmal diseases are mostly transmitted by leaf hoppers. Ex: Sesamum phyllody (*Orosious albicinctus*) and little leaf of brinjal (*Hishimonas phycitis*) are transmitted by MLO's.

b) *Mites*: Mites belonging to the families Eryophyiidae (eryophyiid mite) and Tetranychidae (spider mite) of class Arachnida transmit plant viruses. The genera *Abacarus, Aceria, Eriophyes* and *Brevipalpus* are important.

Ex: *Aceria cajani* (eryophyiid mite) transmits Pigeonpea sterility mosaic virus

Aceria tulipae transmits wheat streak mosaic virus.

c) *Fungi*: Some soil borne fungal plant pathogens carry plant viruses in or on their resting spores and zoospores, and transmit them to susceptible hosts during the infection process. *Tobacco necrosis virus* and *Cucumber mosaic virus* are carried outside the fungi, while lettuce big vein virus is carried inside the zoospores. Many soil borne viruses are transmitted by the members of Chytridiales and Plasmodiophorales.

Fungal transmitted viruses

S.No.	**Fungal vector**	**Disease**
1.	*Olpidium brassicae*	**Tobacco necrosis**, Tobacco stunt, Lettuce big vein
2.	*Olpidium cucurbitacearum*	Cucumber necrosis
3.	*Polymyxa graminis*	Barley yellow dwarf mosaic, Wheat soil borne mosaic, Peanut clump
4.	*Polymyxa betae*	Beet necrotic yellow vein
5.	*Spongospora subterranea*	**Potato mop top**
6.	*Synchytrium endobioticum*	**Potato virus X**

d) *Nematodes*: Several nematodes act as vectors for transmission of fungi, bacteria and viruses.

Bacterial diseases: The bacterium which causes **yellow ear rot** of wheat (*Corynebacterium tritici* or *Clavibacter tritici*) is disseminated by **ear cockle nematode**, *Anguina tritici.* If these two diseases appear together, a complex disease called **tundu** of wheat occurs. *Corynebacterium tritici* is not capable of dispersal and infection unless it is carried by *Anguina tritici.*

Fungal diseases: Similarly, root rot and wilt pathogens such as *Phytophthora, Fusarium, Rhizoctonia, Verticillium*, etc., are disseminated by nematodes.

Viral diseases: Plant nematodes play a vital role in transmitting certain virus diseases. Many soil borne viruses are known to be transmitted by the nematodes. *Xiphenema, Longidorous, Trichodorus and Paratrichodorus* are the nematode genera belonging to Dorylaimoidea which are known to transmit plant viruses. The nematode transmitted viruses are divided into two groups on the basis of shape of their particles: nematode transmitted polyhedral viruses (NEPO) and nematode transmitted tubular (NETU) viruses.

NEPO viruses: These are nematode transmitted viruses with **polyhedral particles**. These are generally transmitted by species of *Xiphenema* and *Longidorus.* Ex: Tobacco ringspot virus, Tomato ringspot virus, Tomato black ring virus, Arabis mosaic virus

NETU viruses: These are nematode transmitted viruses with **tubular particles**. NETU viruses are transmitted by *Trichodorus* and *Paratrichodorous*. Ex; Pea early browning virus (*Trichodorus* sp.), Tobacco rattle virus (*Trichodorus pachydermis*).

Nematode transmitted viruses:

S.No.	Nematode vector	Virus	Virus group
1.	*Paratrichodorus* sp. & *Trichodorus* sp.	Pea early browning, **Tobacco rattle**	NETU group
2.	*Xiphenema index (Dagger nematode)*	**Grapevine fan leaf** OR **Fan leaf virus of Grape**	NEPO virus
3.	*Xiphenema americanum*	Tobacco ringspot, Tomato ringspot	NEPO virus
4.	*Longidorous elongatus*	Raspberry ringspot	NEPO virus

e) ***Human beings:*** Human beings role in dissemination of plant pathogens is more direct than indirect. The ways and means in which human beings help in dispersal are as follows.

- **Transportation of seeds (seed trade):** The import and export of contaminated seeds without proper precautions lead to movement of pathogens from one country to another or from one continent to another. The diseases which are amenable to such transmission are mainly those that are carried in or on the propagative parts

and seed. Ex: Late blight of potato, Downy mildew of grapevine, Citrus canker, *Fusarium* wilt of banana, etc.

- **Planting diseased seed materials:** Planting diseased bulbs, bulbils, corms, tubers, rhizomes, cuttings, etc., of **vegetatively propagated** plants such as potato, sweet potato, cassava, sugarcane, banana, many ornamentals and fruit trees etc., help in dispersal of pathogens from field to field, orchard to orchard, locality to locality or from one country to another.
- **During adoption of normal farming practices:** Human beings engaged in preparatory cultivation, planting, irrigation, weeding, pruning etc., help in dispersal of plant pathogens. Spores and other external structures of fungi can be carried by workers clothing's, shoes, and hands etc., from plant to plant and from field to field.
- **By use of contaminated implements:** Pathogens are transferred from one area to another through implements used in various cultural operations (weeding, thinning, hoeing etc.) in the field. Ex: Soil borne diseases such as root rot, wilt etc. Cutting knives and pruning knives also help in dispersal from one plant to another. Ex: Bunchy top of banana.
- **By use of diseased grafting and budding material:** Grafting and budding between healthy and diseased plants is the most effective method of distribution of pathogens of horticultural crops.

f) *Dispersal by phanerogamic parasites:* Phanerogamic parasites transmit the viruses by acting as a bridge between the diseased and healthy plants. Ex: **Dodder** (*Cuscuta California, C. campesris, C. subinclusa* etc.)

Cuscuta subinclusa	–	Cucumber mosaic virus
Cuscuta California	–	Tobacco mosaic virus
		Tobacco rattle virus
		Tomato spotted wilt virus
Cuscuta campestris	–	Tomato bushy stunt virus

g) ***Dispersal by birds:*** This mode of dispersal is important in dissemination of seeds of flowering parasites and certain fungi. The dispersal of phanerogamic plants by bird is an established fact. eg. Loranthus, Dodder (amarvel). In tropics, crows feeding on the fleshy, sticky and gelatinous berries of gaint mistletoe (*Dendrophthoe* sp.) deposit the seeds on the

other trees with excreta. Seeds of *Loranthus* are disseminated by birds by sticking on their beaks and also through excreta. Stem segments of dodder are carried by birds for preparing their nests and thus get transported to new areas. Moreover, spores of chestnut blight fungus, *Endothea parasitica* are disseminated by more than 18 species of birds. Cleistothecia of many powdery mildew fungi are carried by feathers of birds.

h) ***Farm and wild animals:*** Farm animals (cattle) while feeding on diseased fodder ingest the viable fungal propagules (spores or oospores or sclerotia) and pass out as such in the dung. This dung when used as manure spread in the field and act as source of inoculum. Further, soil inhabiting fungi especially sclerotia adhere to the hoofs and legs of animals and get transported to other places.

2) Inanimate agents:

a) Wind: The dispersal of pathogens by wind is known as **anemochory**. Wind transmission involves the upward air currents, velocity and the downward movements of the wind. Wind acts as a potent carrier of propagules of fungi, bacteria and viruses.

Fungi: Usually the fungal pathogens are light in weight and are well adapted to wind dispersal. The **adaptations for wind dispersal** in fungal pathogens include production of numerous spores and conidia, discharge of spores with sufficient force, production of very small and light spores so that they can move to long distances. Ex: Powdery mildew, downy mildew, rusts, smuts etc.

Both short and long distance dissemination is possible by means of wind.

i) Spores adopted for **short distance** dissemination- sporangia of downy mildew fungi, conidia of powdery mildew fungi and basidiospores of rust fungi. In the plains of northern India the annual recurrence of cereal rusts is solely due to uredospores brought by wind from the source of survival in the hills in the far north (Himalayas) and south (Nilgiris).

ii) Spores adapted to **long distance** dispersal – uredospores of rust fungi, Chlamydospores of smut fungi and conidia of *Alternaria*, *Helminthosporium* and *Pyricularia.*

Uredial stages of the rust fungi travel long distances through air currents and thus are responsible for destructive epidemics over wide areas. Ex: The uredospores of

Puccinia graminis var. tritici have been detected as high as **14000** feet above infected wheat fields (Stackman and Christensen). Similarly, *Alternaria* spores at 8000 feet, *Puccinia recondita* and *Cronartium ribicola* spores at 12500 feet were reported.

Dispersal distance: In USA, uredospores of this fungus are blown from the far south (Mexico) into Dakota and Minnesota (far north) travelling more than 1000 miles in about two days without losing their viability. If the uredospores reach an altitude of 5000 feet, their distance dispersal in a 30 mile per hour wind could be about **1100 miles**, without loosing viability.

Nematodes: In addition to fungi, it also helps in the dissemination of the cysts of nematodes and also the seeds of phanerogamic parasites. Ex: Cysts of the nematode *Heterodera major*, which causes **molya disease of wheat and barley,** are carried by dust storms from **Rajasthan** to **Haryana**

Bacteria: Some pathogenic bacteria are carried along with the infected material to short distances by wind. Ex: *Erwinia amylovora*, the causal agent of fire blight of apple and pear, produces fine strands of dried bacterial exudates which may be broken off and are transmitted by wind.

Viruses and **phytoplasmas** are not directly transmitted by wind, but the insect and mite vectors that carry the viruses move to different directions and distances based on the direction and speed of the air.

b) **Water**: Transmission of plant pathogens by water is called as **hydrochory**. Water is less important than air in long distance transport of pathogens, but it is more efficient as the pathogens land on the wet surface and can germinate immediately. Water dissemination occurs mainly through **surface running water** and **rain splash**.

The **surface flow of water** after heavy rains or during irrigation from canals and wells arries the pathogens to short distances. Ex: The mycelial fragments, spores or sclerotia of fungi, *Colletotrichum falcatum* **(red rot of sugaecane)**, *Fusarium, Ganoderma, Macrophomina, Pythium, Phytophthora, Sclerotium,* etc., are transmitted through rain or

irrigation water. Long distance dispersal is also possible by water only when the floods cover larger areas or when the water flows from the sources of survival of pathogens to longer distances.

Dissemination by rain splash is also called as **splash dispersal**. It is one of the efficient methods of dispersal of **bacterial plant pathogens**. Rain drops falling with force on sori, pustules, cankers or even soil surface may splash the propagules in small droplets and enable them to land on neighbouring healthy susceptible surfaces or the water droplets may be carried to long distances by air. Ex: Bacterial leaf spot of rice (*Xanthomonas campestris pv. oryzae*), Bacterial leaf streak of rice (*Xanthomonas campestris pv. oryzicola*), Green ear of bajra (*Sclerospora graminicola*).

Fungal spores and bacteria present in the air or plant surface are washed downward by rain splash or drops from overhead irrigation and are deposited on susceptible healthy plants. Water not only plays an important role in the dissemination of plant pathogens, but also helps in the growth and spore discharge of many fungi. It also helps in the spore germination and infection process.

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PHENOMENON OF INFECTION/ INFECTION PROCESS

It is the **third link** in the infection chain after survival and dispersal of inoculum. Infection process means establishment of pathogen in the host plant. Entry and colonization of pathogen in the host tissues is known as establishment and the infective propagules coming in contact with the host are known as **inoculum**.

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

Def: It is defined as the resultant of the action of environment, the vigour of pathogen to establish an infection, susceptibility of the host and amount of inoculum present (Dimond and Horsfall, 1960).

OR

It is defined as the energy of growth of a parasite available for infection of a host at the surface of the host organ to be infected (Garret, 1960).

In case of specialized pathogens as rusts and powdery mildews, very few or even one spore is capable of causing infection successfully. In case of non-specialized pathogens such as *Pythium, Phytophthora, Rhizoctonia* and *Sclerotium* require high density of inoculum on the surface of susceptible host for successful infection.

The success of process of infection depends on

1. **Host factors**

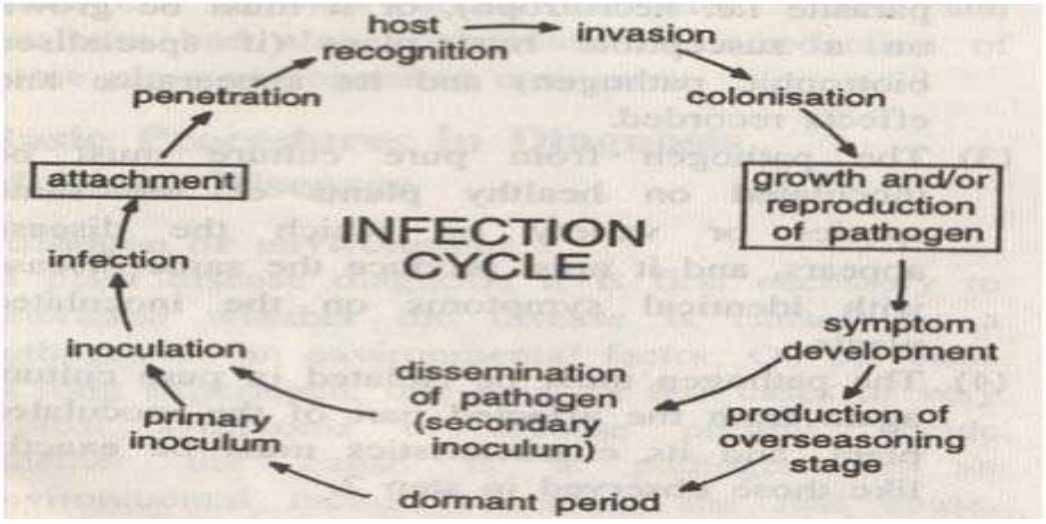
- *Susceptibility of host*: It is genetically controlled by DNA and it is an inheritable character which is transmitted from parents to off springs.
- *Disease proneness of the host*: It is decided by the external factors such as host nutrition, i.e., more nitrogen application makes the host more susceptible and more potash application leads to less susceptibility.

2. **Pathogen factors**

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

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

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



Stages in the development of infection or disease cycle

1. PRE-PENETRATION: Depending upon the plant pathogen activity, the plant pathogens are classified in to 2 categories

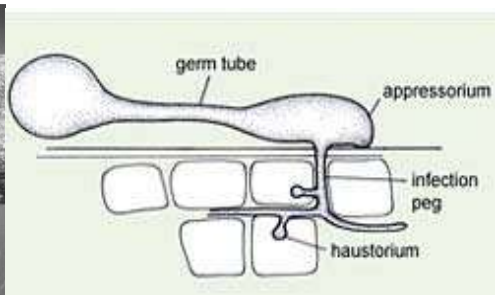
1. Active invaders and 2.Passive invaders

Active Invaders	Passive Invaders
1. Pathogens which make an aggressive effort to gain entry into intact host cells.	1.No aggressive effort
2. They do not require help of any external agency to gain entry into host cells.	2. Require help of external agencies like insect vectors or wounds caused by agricultural implements.
3. Eg. Phyto-pathogenic fungi Phanerogamic parasites	3. Eg. Plant viruses Phyto-pathogenic bacteria

Plant viruses are particulate in nature and they do not have any capacity to enter the host cell so they do not make any aggressive effort for entry, but depend on different insect vectors for their entry into host cell. Bacteria have no dormant structures; hence no repenetration activity except for multiplication in infection drops on the natural openings. However, nematodes show some orientation towards root surface before actual penetration.

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

Rhizomorphs



Appressorium

2**. PENETRATION:** Pathogens penetrate plant surfaces by direct penetration or indirectly through wounds or natural openings. Bacteria enter plants mostly through wounds and less frequently through natural openings. Viruses, viroids, mollicutes, fastidious bacteria enter through wounds made by vectors. Fungi, nematodes and parasitic higher plants enter through direct penetration and less frequently through natural openings and wounds.

A. Indirect Penetration

1. **Wounds**: Wounds caused by farm operations, hail storms, or insect punctures, etc., will help in the entry of different plant pathogens into the host cells. Organisms which cause storage diseases and ripe rots will enter through the wounds caused by farm operations.
Ex. *Rhizopus, Gloeosporium, Aspergillus, Penicilium, Colletotrichum, Diplodia*, etc. Weak parasites enter through the wounds caused by hail storms and freezing.
Ex. *Macrophomina phaseolina*

Pathogen causing brown rot of fruits (*Sclerotinia fructicola*) enters through the wounds caused by insect punctures. Similarly, causal organism of Dutch elm disease (*Ceratostomella ulmi*) enters through the wounds caused by elm bark beetle.

2. Natural openings

a) Stomata: There is variation in the behaviour of germ tube at the time of penetration through the stomata. In *Puccinia graminis tritici*, the uredospore germinates and forms a germ tube which on approaching stoma swells at the tip to form an appressorium in the stomatal aperture. From the appressorium a blade like wedge grows through the stomatal slits and swells inside to form a sub-stomatal vesicle from which the haustoria enetrating the cells are produced.

In *Peronospora destructor* infecting onion leaves, the germ tube continues to grow after the formation of first appressorium. In *Pseudoperonospora cubensis*, the hyphae penetrate the stomatal aperture and swell to form a sub-stomatal vesicle from which in turn other hyphae grow to form haustoria in the adjacent cells of the leaves. *Mycosphaerella musicola* forms a small structure called **stomatopodium** over the pore of the stoma after growing for few days on the surface of the leaf. A hypha then arises from it which grows into the sub-

stomatal chamber and swells to form a vesicle, which in turn gives rise to hyphae which invade palaside tissues.

Other examples: *Xanthomonas campestris* pv. *malvacearum* (Black arm of cotton), *Xanthomonas phaseoli* (Bacterial leaf spot of green gram), *Phytophthora infestans* (Late blight of potato), *Albugo candida* (White rust of crucifers) and uredospores of *Puccinia graminis tritici* (Black stem rust of wheat).

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

c) Hydathodes: ***Xanthomonas campestris pv. campestris*** (Black rot of crucifers)

B) Direct penetration: Most fungi, nematodes and parasitic higher plants are capable of penetrating the host surface directly. However, the plants are provided with different mechanisms of defense which include structural features of the host, presence of chemical coverings on the cell walls, and anti-infection biochemical nature of the protoplasm. Hence, the pathogen should have mechanisms to overcome these barriers for direct penetration.

a) *Breakdown of physical barriers*: Viruses have no physical force or enzyme system of their own to overcome structural or chemical barriers of the host and therefore come in contact with the host protoplasm only through wounds. Bacteria are mostly weak parasites and cannot employ force to effect penetration. Fungi and nematodes are the only group of plant pathogens that employ force for direct penetration of the host. Fungi penetrate host plants directly through a fine hypha produced directly by the spore or mycelium or through a penetration peg produced by an appressorium. These structures exert pressure on the surface which results in stretching of the epidermis which becomes thin. Then the infection peg punctures it and effects its entry.

b) *Breakdown of chemical barriers*: the host is provided with defense mechanisms against invasion which include i) presence of cuticular layer on the epidermis, ii) lack of suitable nutrients for the pathogen in the host cells, iii) presence of inhibitory or toxic substances in the host cells, iv) exudation of substances toxic to pathogen or stimulatory to antagonists of

the pathogen. Ex: The glands in leaf hairs of begalgram contain maleic acidwhich is antifungal and provide resistance to infection by the rust fungus (*Uromyces ciceris arietini*). Similarly, protocatecheuic acid and catechol in the red scales of onion provide resistance to onion smudge pathogen, *Colletotrichum circinans*. To overcome these physical and chemical barriers, the fungi produce various enzymes, toxins organic acids and growth regulators.

Through non-cutinized surfaces:

a) Seedlings: Grain smut of jowar (*Sphacelotheca sorghi*), Loose smut of jowar (*Sphacelotheca cruenta*), Downy mildew of jowar and bajra (*Sclerospora graminicola*), Wheat bunt disease (*Tilletia caries*, *Tilletia foetida*)

b) Root hairs: Wilt causing fungi (*Fusarium* sp.), Club root of cabbage (*Plasmodiophora brassicae*), Root rot of cotton (*Phymatotrichum omnivorum*)

c) Buds: Pea rust fungi (*Uromyces pisi*), Witches broom of cherries (*Taphrina cerasi*)

d) Flowers: Loose smut of wheat (*Ustilago nuda tritici*), Long smut of jowar (*Tolyposporium ehrenbergi*), Bunt of rice (*Neovossia horrida*), **Ergot of rye** (*Claviceps purpurea*)

e) Leaves: Basidiospores of white pine blister rust fungus (*Cronartium ribicola*) germinate and grow down into branches and leaves, where aecia are produced.

d) Nectaries: **Fire blight of apple** (*Erwinia amylovora*)

e) Stalk ends: *Penicillium italicum, Theilaviopsis paradoxa* (Post harvest disease fungi)

Through cutinized surfaces:

a) Cuticle: Leaf spot of spinach (***Cercospora beticola***), early blight of solanaceous plants (*Alternaria solani*), Tikka disease of groundnut (***Cercospora personata***)

3. POST PENETRATION

Invasion and colonization: Infection is the process by which pathogens establish contact with the susceptible cells or tissues of the host and derive nutrients from them. A parasitic relationship is formed between host cytoplasm and parasite cytoplasm. During infection, pathogens grow and multiply within the plant tissues. **Invasion** of plant tissues by the pathogen, and growth and reproduction of the pathogen (**colonization**) are two concurrent stages of disease development.

Fungi spread into all parts of host organs, either by growing directly through the cells as an intracellular mycelium or by growing between the cells as an intercellular mycelium. During establishment, pathogen produces different substances which include enzymes, toxins, growth hormones and polysaccharides which will help in colonization of the host.

In **ectoparasites** the main body of the pathogen lies on the surface of the host with only feeding organs (haustoria) penetrating the tissues Ex: Most of the powdery mildew fungi. Some fungal parasites develop both external and internal mycelium Ex: *Rhizoctonia solani*. The endophytic parasites or **endoparasites** grow subcuticularly (*Diplocarpon rosae*, black spot of rose), in parenchyma tissues (most fungal and bacterial pathogens as well as many nematodes) or in vascular tissues (vascular wilt parasites). Some pathogens are **endobiotic**, *i.e.,* mycelium is not produced and the thallus is entirely present within a host cell Ex: *Synchytrium endobioticum*.

Differentiate between Ectoparasites and Endoparasites:

Sr.No.	**Ectoparasites**	**Endoparasites**
1	A parasite or pathogen feeding on a host from exterior	A parasite or pathogen which enters a host and feeds from within the host
2	eg. Powdery mildew fungi, Rust - fungi, Grain smut of sorghum	eg. Downy mildew fungi, loose smut of wheat
3	Superficial mycelium having internal haustoria in epidermal cells	The mycelium is internal as intercellular without haustoria

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

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

Exit of the pathogen

After invasion and colonization of the host, the pathogens come out of the host to maintain the continuity of the infection chain or disease cycle and escape death due to overcrowding. Once the pathogens exit from the host, they survive and are disseminated to other hosts and continue the infection cycle.

Viruses can exist only with the living protoplasm and hence disseminated through their animate vectors like insects, fungi, nematodes, etc. The **bacteria** ooze out in the form of slime on the host surface from where they can be disseminated through water and insects. However, the **fungi** have the most elaborate system of exit. Most plant pathogenic fungi grow out on the host surface and produce repeating spores (secondary inoculum), usually asexually, under favourable conditions. The spores thus formed are disseminated through wind, water, soil, seed, vegetative propagating material, agricultural implements, etc.

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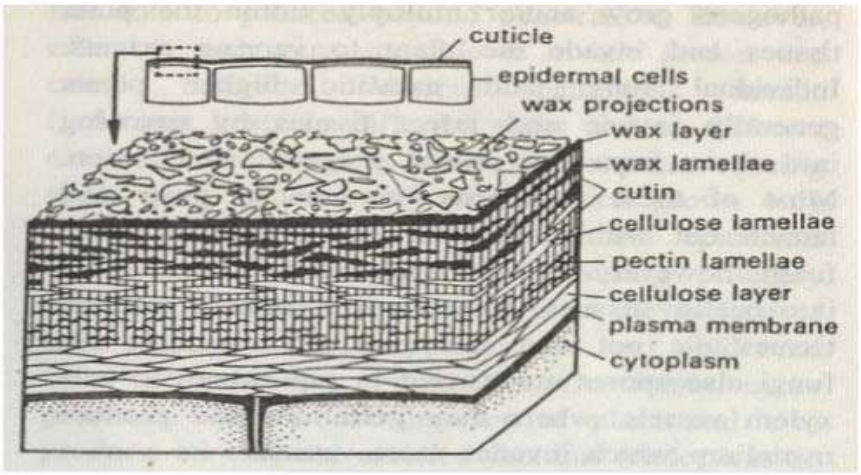
ROLE OF ENZYMES IN PATHOGENESIS

Pathogenesis: A process or chain of events by which disease development takes place. The events of pathogenesis are Prepenetration, Penetration and Post penetration.

Enzymes are large protein molecules which catalyze all inter-related reactions in the living cell. Most pathogens derive energy principally from enzymatic break down of food materials from host tissue.

Role of enzymes: Plant pathogenic enzymes disintegrate the structural components of host cells, break down inert food substances in the host cell or affect the protoplast directly and interfers with its functioning system.

Composition of the cell wall: Functionally cell wall is divided into 3 regions, *viz*., middle lamella (made of pectins), primary wall (cellulose, pectic substances) and secondary cell wall (entirely cellulose).



Middle lamella acts as intercellular cement which binds the cells together in tissue system. Pectin or pectic substances are major chemical constituents of wall layers and entire middle lamella, where as in other layers, cellulose is found in good amounts.

Besides these two major components, other components such as hemicelluloses, lignin and some amount of protein is also present. Main components of cell wall are pectic substances, cellulose, hemicelluloses, lignin and small quantity of protein.

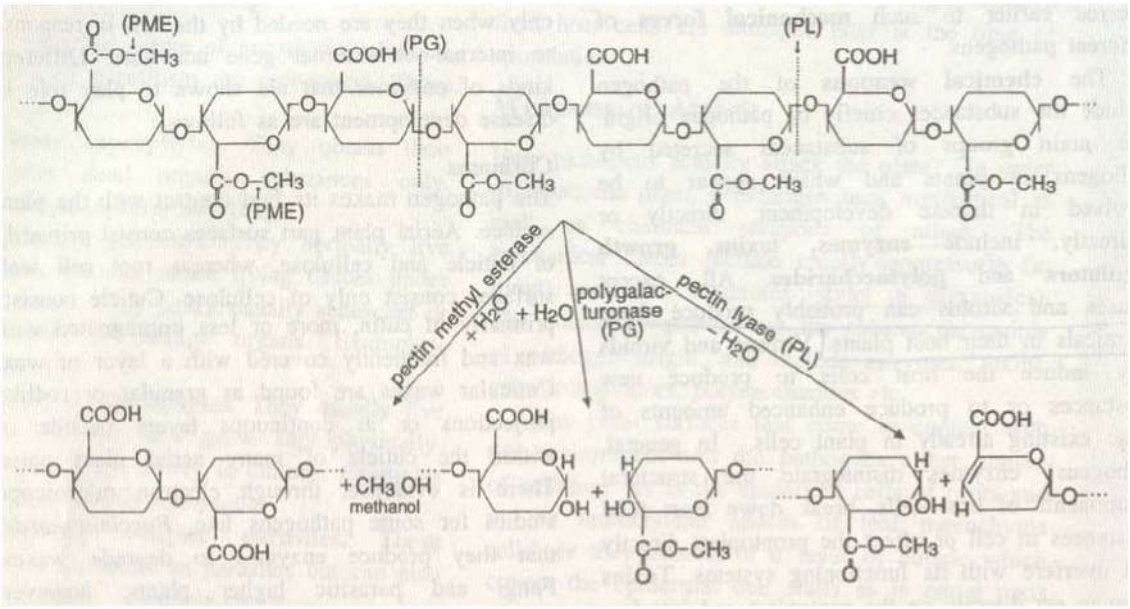
The epidermis of plants is covered by cuticle, whose major chemical substance is cutin in addition to cuticular wax.

Cuticular wax: Plant waxes are found as granular or rod like projections or as a continuous layer outside / within the cuticle. Wax formation is a continuous process and it is not a terminal phase in the development of leaf. Cuticular waxes are made up of long chain molecules of paraffin, hydrocarbons, alcohols, ketones and acids. Most of the fungi and parasitic higher plants penetrate wax layers by means of mechanical force alone.

Cutin: It is insoluble polyester of unbranched derivatives of **C16 and C18 hydroxy fatty acids**. Cutin is admixed with waxes on upper side and with pectin and cellulose on the lower side. **Cutinases** break cutin molecules and release monomers as well as oligomers from insoluble cutin polymer. Cutinases reaches its highest concentration at penetrating point of the germ tube and at infection peg of appressorium forming fungi Ex: *Colletotrichum gloeosporioides, Sphaerotheca pannosa, Venturia inaequalis,* and *Helminthosporium victoriae*.

Pectic substances: These are major components of middle lamella (intercellular cement that holds in place the cells of plant tissues). They also make up a large portion of primary cell wall in which they form an amorphous gel filling the spaces between cellulose microfibrils. Pectic substances are polysaccharides consisting mostly of **d-galactouronic acid** units with **α-1,4-glycosidic bonds**. These chains are esterified with **methyl** groups or linked with other carboxyl groups in calcium and magnesium salt bridges.

Pectic substances are of three types, namely, **pectic acid** (non methylated units), **pectinic acid** (<75% methylated galacturonan units) and **pectin** (>75% methylated units). Term **protopectin** is used to denote substances which are soluble in water and upon restricted hydrolysis yields pectinic acid.



The enzymes that degrade pectic substances are known as **pectinases** or **pectolytic** enzymes. Pectinases and pectolytic enzymes are pectin methyl esterases (PME's), polygalactouronases (PG's) and pectin lyases (PL's).

1. **Pectin methyl esterases (PME) or Pectinesterases (PE)**: Breaks ester bonds and removes methyl groups from pectin leading to the formation of **pectic acid** and **methanol** (CH3OH).

2. **Polygalacturonases**: Split pectin chain by adding a molecule of water and breaks the linkage between two galacturonan units. These enzymes catalyze reactions that break α-1,4-glycosidic bonds.

3. **Pectin lyases**: Split pectin chain by removing a molecule of water from the linkage, thereby breaking it and releasing products with unsaturated double bonds.

These pectin enzymes can be **exopectinases** (break only terminal linkage) or **endopectinases** (break pectin chain to random sites). Pectin degradation results in liquefaction of the pectic substances and weakening of cell walls, leading to tissue maceration.

Macerating enzymes: Are pectic enzymes which degrade (dissolve) middle lamella. Pectates to cause softening of tissues. Pectic enzymatic action result in the formation of phenols which cause browning in the plants. The cells of macerated tissues soon die. Eg. Pythium spp. causing damping off, Fusarium spp. causing wilt of castor, cotton, cumin, pigeonpea, Soft rot bacterium, *Erwinia caratovora* subsp. *caratovora* and other fungi like *Botrytis cinerea, Sclerotium rolfsii*, etc.

Cellulose: Cellulose is a polysaccharide, made of chains of **β-D-glucopyranose** units (where C1 is linked to C4). Glucose chains are held by **hydrogen** bonds. Cellulose occurs in all higher plants as the skeletal substance of cell walls in the form of microfibrils. Primary and secondary wall consists of a matrix in which a large number of microfibrils are embedded. These microfibrils are like bundles of iron bars in a reinforced concrete building. In some parts of microfibrils the chains are arranged in an orderly fashion attaining crystalline form, when arranged in less orderly fashion, it attains amorphous form. If the proportion of crystalline portion is more, the resistance of the host to pathogen is more. The space between microfibrils and between micelles or cellulose chains is filled with pectins, hemicelluloses and also lignin at maturity.

Cellulose is insoluble in **crystalline** form (native form), and soluble in **amorphous** form (modified cellulose). The enzymatic breakdown of cellulose results in final production of glucose molecules.

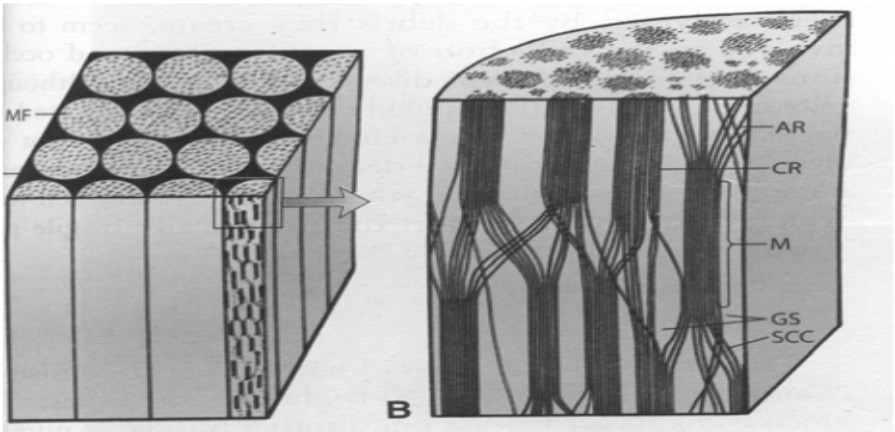
Cellulose is degraded by **cellulases**. Cellulase one (**C1**) attacks native cellulose by cleaving cross-linkages between chains. A second cellulase (**C2**) also attacks native cellulose and breaks into shorter chains. These shorter chains are then attacked by **Cx** enzyme, which degrade them into disaccharide, **cellobiose**. Finally cellobiose is degraded by the enzyme, **β-glucosidase** into glucose.

Cellulose degrading enzymes are produced by fungi, bacteria and nematodes and also by parasitic higher plants. They cause:

1. The break up of the cell wall entry and
2. Provide food in the form of glucose from cellulose on hydrolysis.

The cellulases (cellulolytic enzymes) are stimulated in the presence of cellulose. Many soft rot organisms produce cellulases as well as pectolytic enzymes. Cellulases play an

important role during pathogenesis in the Root rot, Soft rot and Leaf spot diseases and wood rots (Coversion of natural cellulose into glucose).



Schematic diagram of the gross struture of cellulose
l microfibrils (A) and of the arrangement of cellulose molecules
hin a microfibril (B). MF, microfibril; GS, ground substance (pectin,
nicelluloses, or lignin); AR, amorphous region of cellulose; CR,
stalline region; M, micelle; SCC, single cellulose chain (molecule).

Cellulase degrading enzymes play a role in softening and degradation of cell wall material and facilitate easy penetration and spread of pathogen in the host.
Ex: Basidiomycetes fungi

Hemicellulose: These are the major constituents of **primary cell wall** and also seen in middle lamella and secondary cell wall. The hemicellulose polymers include primarily **xyloglucan** but also glucomannans, galactomannans, arabinogalactans, etc. Hemicelluloses link the ends of pectic polysaccharides and various points of the cellulose microfibrils.
Hemicellulases degrade hemicelluloses and depending on the monomer released from polymer on which they act, they are termed as xylanase, galactanase, glucanase, arabinase, mannose, and so on. Ex: *Sclerotinia sclerotiorum, Sclerotinia fructigena.*

Lignin: Lignin is found in the **middle lamella**, as well as in the secondary cell wall of xylem vessels and the fibres that strengthen plants. It is an amorphous, three-dimensional polymer made up of basic structural unit, **phenylpropanoid**. Lignin forms by oxidative condensation (C-C and C-O bond formation) between phenylpropanoid units or substituted **cinnamyl alcohols** (p-coumaryl alcohol, coniferyl alcohol and sinapyl alcohol). **White rot**

fungi (Basidiomecetes) secrete one or more ligninases which enable them to utilize lignin. Ex: *Xylaria, Chaetomium, Alternaria, Cephalosporium*, etc.

Cell wall proteins: Cell wall proteins are similar to other proteins, except that they are rich in aminoacid, **hydroxy proline**. Five classes of structural proteins are found in cell walls: extensins, proline-rich proteins (PRP's), glycine-rich proteins (GRP's), Solanaceous lectins and arabinogalactan proteins (AGP's). Proteins are degraded by means of enzymes, **proteases** or **proteinases** or **peptidases**.

Proteolytic enzymes (Proteases): Pathogens are known to degrade cytoplasmic proteins and proteinaceous gel existing in the cell wall enzymatically. Eg. Blast of paddy casued by *Pyricularia oryzae*, Late blight of potato caused by *Phytophthora infestans*.

The proteases help the pathogen to invade the tissues easily and effectively.

Lipids: Various types of lipids occur in all plant cells. The most important ones are **phospholipids** and **glycolipids**. These lipids contain fatty acids, which may be saturated or unsaturated. Lipolytic enzymes, called **lipases** (phospholipases, glycolipases) hydrolyze lipids and release fatty acids.

Starch: Starch is the main reserve polysaccharide found in plant cells. It is a glucose polymer and exists in two forms: **amylose**, a linear molecule, and **amylopectin**, a highly branched molecule. Starch is degraded by enzyme, amylases.

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ROLE OF TOXINS IN PLANT PATHOGENESIS, ROLE OF GROWTH REGULATORS IN PLANT PATHOGENESIS AND ROLE OF POLYSACCHARIDES IN PATHOGENESIS

Def: Toxin can be defined as a microbial metabolite excreted (**exotoxin**) or released by lysed cells (**endotoxin**) which in very low concentration is directly toxic to the cells of the suscept (host).

The term toxin is used for a product of the pathogen, its host, or pathogen host interaction which even at very low concentration directly acts on living host protoplasm to influence disease development or symptom expression.

Toxins are different from enzymes in that they do not attack structural integrity of host tissues but affect the metabolism of the host because the toxins will act on protoplast of the cell.

Toxin hypothesis (Luke and Wheeler, 1955):

1. A toxin should produce all symptoms characteristic of the disease
2. Sensitivity to toxin will be correlated with susceptibility to pathogen
3. Toxin production by the pathogen will be directly related to its ability to cause disease.

Except, **victorin**, the toxic metabolite of *Cochliobolus victoriae*, the vast majority of toxins associated with plant diseases fail to exhibit all the above characters.

Classification of toxins (Wheeler and Luke, 1963) :

According to the **source of origin**, toxins are divided into 3 broad classes namely, ***pathotoxins, vivotoxins*** and ***phytotoxins.***

1. **Pathotoxins**: These are the toxins which play a major role in disease production and produce all or most of the symptoms characteristic of the disease in susceptible plants. Most of these toxins are produced by pathogens during pathogenesis.

Ex: **Victorin**: *Cochliobolus victoriae* (*Helminthosporium victoriae*), the causal agent of Victoria blight of oats. This is a host specific toxin.

Other examples:

a) *Selective*

T- toxin: *Helminthosporium maydis race T*

HC-toxin: *Helminthosporium carbonum*

HS- toxin: *Helminthosporium sacchari*

Phyto-alternarin: *Alternaria kikuchiana*

PC- toxin: *Periconia circinata*

b) *Non-selective*

Tentoxin: *Alternaria tenuis*

Tabtoxin or wild fire toxin: *Pseudomonas tabaci*

Phaseolotoxin: *Pseudomonas syringae pv. phaseolicola*

c) Produced by *plant or plant X pathogen interaction*

Amylovorin: *Erwinia amylovora* (Fire blight of apple and pears)

2) **Phytotoxins**: These are the substances produced in the host plant due to host-pathogen interactions for which a causal role in disease is merely suspected rather than established. These are the products of parasites which induce few or none of the symptoms caused by the living pathogen. They are non-specific and there is no relationship between toxin production and pathogenicity of disease causing agent.

Ex: Alternaric acid – *Alternaria solani*

3) **Vivotoxins**: These are the substances produced in the infected host by the pathogen and / or its host which functions in the production of the disease, but is not itself the initial inciting agent of the disease.

Fusaric acid – Wilt causing *Fusarium sp.*

Lycomarasmin – *Fusarium oxysporum f.sp. lycopersici*

Piricularin – *Pyricularia oryzae*

Classification based on specificity of toxins

1. **Host specific / Host selective toxins**: These are the metabolic products of the pathogens which are selectively toxic only to the susceptible host of the pathogen

Ex: Victorin, T-toxin, Phyto-alternarin, Amylovorin

2**. Non-specific / Non-selective toxins**

These are the metabolic products of the pathogen, but do not have host specificity and affect the protoplasm of many unrelated plant species that are normally not infected by the pathogen

Ex: Ten-toxin, Tab-toxin, Fusaric acid, Piricularin, Lycomarasmin and Alternaric acid

Differentiate between host – specific and non-host specific toxins

Host specific	**Non-host specific**
1. Selectively toxic only to susceptible host of the pathogen	1. No host specificity and can also affect the physiology of those plants that are normally not infected by the pathogen
2. Primary determinants of disease	2. Secondary determinants of disease
3. Produce all the essential symptoms of the disease	3. Produce few or none of the symptoms of the disease
4. Ex: Victorin, T- toxin	4. Ex: Tentoxin, Tabtoxin

Effect of toxins on host tissues :

A) *Changes in cell permeability*: Toxins kill plant cells by altering the permeability of plasma membrane, thus permitting loss of water and electrolytes and also unrestricted entry of substances including toxins. Cellular transport system, especially, H+ / K+ exchange at the cell membrane is affected.

B) *Disruption of normal metabolic processes*

- Increase in respiration due to disturbed salt balance
- Malfunctioning of enzyme system Ex: Piricularin inhibits polyphenol oxidase
- Uncoupling of oxidative phosphorylation

C) *Interfere* with the *growth regulatory system* of host plant Ex: Restricted development of roots induced by *Fusarium moniliforme.*

ROLE OF GROWTH REGULATORS IN PLANT PATHOGENESIS

Growth regulators

Growth regulators are of two types

1. Growth promoting substances and 2. Growth inhibiting substances:

Auxins, gibberellins and cytokinins are growth promoting substances, whereas, dormin, ethylene and abscissic acid are growth inhibiting substances. The imbalance in growth promoting and growth inhibiting substances causes **hypertrophy** (excessive increase in cell size) and **atrophy** (decrease in cell size). Symptoms may appear as tumors, galls, knots, witches broom, stunting, excessive root branching, defoliation and suppression of bud growth.

1. Growth promoting substances:

a) Auxins: *Indole-3-acetic acid (IAA)* is the naturally occurring auxin. It is continuously produced in young meristematic tissue and moves rapidly to older tissues. If auxin concentration is more, its concentration is reduced by the enzyme, **IAA oxidase**.

Functions: IAA regulates cell elongation and differentiation, also affects permeability of the membrane, increases respiration, and promotes synthesis of mRNA.

How disease is induced?

Increased IAA results in **hypertrophy** and decreased IAA results in **atrophy**. Increased IAA may be due to inhibition of IAA oxidase.

Ex: *Ralstonia solanacearum* (*Pseudomonas solanacearum*), the causal agent of wilt of Solanaceous plants, induces a 100 fold increase in IAA level in diseased plants. Increased plasticity of cell walls as a result of high IAA levels renders the pectin, cellulose and protein components of the cell wall more accessible to pathogen degradation. Increase in IAA levels may also inhibit lignifications of tissues.

Increased IAA levels have been reported in plants infected with the following pathogens *Phytophthora infestans* (Late blight of potato), *Ustilago maydis* (Maize smut), *Plasmodiophora brassicae* (Club root of crucifers), *Sclerospora graminicola* (Downy mildew of sorghum), *Agrobacterium tumefaciens* (Crown gall of apple), and *Meloidogyne* (Root knot nematode).

b) Gibberellins: First isolated from ***Gibberella fujikuroi*** (Conidial stage: *Fusarium moniliforme*), the causal agent of bakanae or foolish seedling disease of rice. Infected seedlings show abnormal elongation due to excessive elongation of internodes. Best known gibberellin is **Gibberellic acid**.

Functions: Cell elongation, stem and root elongation, promote flowering and growth of fruits. It also induces IAA synthesis. IAA and GA act synergistically. Ex: *Sclerospora sacchari*, the causal agent of downy mildew of sugarcane induces GA production.

c) Cytokinins: Kinetin was the first compound isolated from herring sperm DNA and does not occur naturally in plants. Cytokinins, such as **zeatin** and **isopentenyl adenosine** (IPA) have been isolated from plants

Functions: Cytokinins are necessary for cell growth and differentiation. It inhibits breakdown of proteins and aminoacids and thereby inhibit senescence and they have the capacity to direct the flow of aminoacids and other nutrients towards high cytokinin concentration. Cytokinin activity increases in club root, in crown galls and in rust infected bean leaves. Ex: Green islands are formed around infection in bean (*Phaseolus vulgaris*) leaves infected by *Uromyces phaseoli*.

2. Growth inhibiting substances

a) Ethylene (CH2=CH2): Ethylene exerts a variety of effects on plants, viz., chlorosis, leaf abscission, epinasty, stimulation of adventitious roots, fruit ripening and increased permeability of cell membranes.

Ex: Ethylene is involved in premature ripening of fingers in banana infected by *Pseudomonas solanacearum,* the causal agent of moko disease of banana. Ethylene was

also detected in leaf epinasty symptom of the vascular wilt syndrome. Ex: *Fusarium oxysporum f.sp. lycopersici* (Wilt in tomato).

b) Abscissic acid: It exerts dormancy in seeds, closure of stomata, inhibition of seed germination and growth and stimulated germination of fungal spores. It is one of the factors involved in stunting of plants.

c) Dormin / Abscissin II: Dormin induces dormancy by converting developing leaf primordia of a bud into bud scales. It acts as an antagonist of gibberellins and masks the effect of IAA. However, the exact role of dormin is not known.

ROLE OF POLYSACCHARIDES IN PATHOGENESIS

Polysaccharides: Fungi, bacteria and nematodes release varying amounts of mucilaginous substances that coat their bodies and provide interface between the outer surface of the micro-organism and its environment. The role of slimy polysaccharides is of utmost importance in wilt diseases. In the vascular wilts, large polysaccharide molecules released by the pathogen in the xylem causes mechanical blockage of vascular bundles and initiate wilting.

Ex: *Ralstonia solanacearum* (Bacterial wilt of Solanaceous plants)

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DEFENSE MECHANISM IN PLANTS

In general plants defend themselves against pathogens by two ways: structural or morphological characteristics that act as physical barriers and biochemical reactions that take place in cells and tissues that are either toxic to the pathogen or create conditions that inhibit the growth of the pathogen in the plant.

I. Structural defense mechanisms: These may be pre-existing, which exist in the plant even before the pathogen comes in contact with the plant or induced, *i.e*, even after the pathogen has penetrated the preformed defense structures, one or more type of structures are formed to protect the plant from further pathogen invasion.

A) Pre-existing structural defense structures

These include the amount and quality of wax and cuticle that cover the epidermal cells and the size, location and shapes of natural openings (stomata and lenticels) and presence of thick walled cells in the tissues of the plant that hinder the advance of the pathogen.

i) Waxes: Waxes on leaf and fruit surfaces form a hydrophobic or water repellent surface preventing the germination of fungi and multiplication of bacteria.

ii) Cuticle and epidermal cells: A thick cuticle and tough outer wall of epidermal cells may increase resistance to infection in diseases in which the pathogen enters its host only through direct penetration. Ex: Disease resistance in Barbery species infected with *Puccinia graminis tritici* has been attributed to the tough outer epidermal cells with a thick cuticle. In linseed, cuticle acts as a barrier against *Melampsora lini*.

The silicification and lignifications of epidermal cells offers protection against *Pyricularia oryzae* and *Streptomyces scabies* in paddy and potato, respectively.

iii) Sclerenchyma cells: The sclerenchyma cells in stems and leaf veins effectively blocks the spread of some fungal and bacterial pathogens that cause angular leaf spots.

iv) Structure of natural openings:

a) *Stomata*: Most of the pathogens enter plants through natural openings. Some pathogens like stem rust of wheat can enter its host only when the stomata are open. The wheat varieties (Cultivar, **Hope**) in which stomata open late in the day are resistant as the germ

tubes of the spores germinating in the night dew desiccate owing to evaporation of the dew before stomata begin to open. This can also be called as functional resistance. The structure of stomata provides resistance to penetration by certain plant pathogenic bacteria.

Ex: The citrus variety, **szinkum**, is resistant to citrus canker because it posses a broad cuticular ridge projecting over the stomata and a narrow slit leading to the stomatal cavity thus preventing the entry of bacterial and fungal spores into the interior of the leaf.

b) Lenticels: The shape and internal structure of lenticels can increase or decrease the incidence of fruit diseases. Small and suberised lenticels will offer resistance to potato scab pathogen, *Streptomyces scabies.*

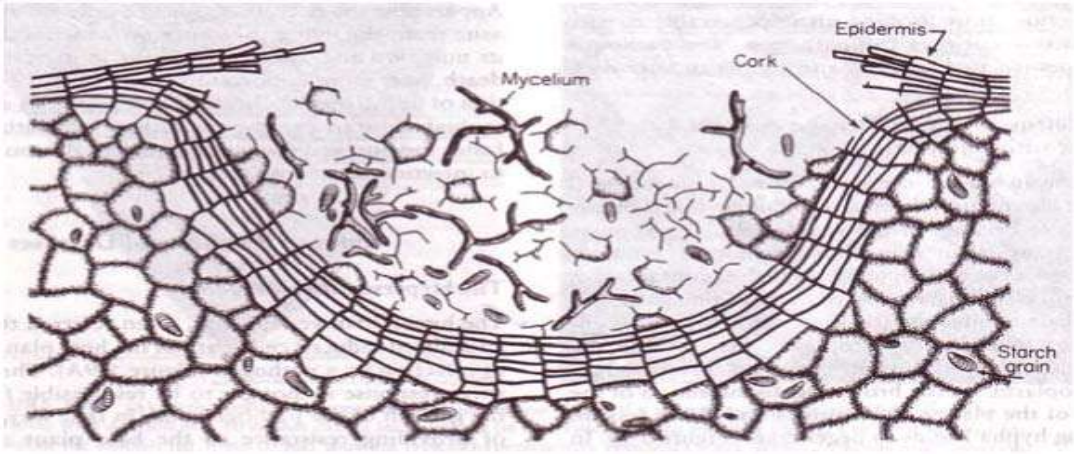
B) Post-infectional structural defense mechanisms/Induced structural barriers:

These may be regarded as histological defense barriers (cork layer, abscission layers and tyloses) and cellular defense structures (hyphal sheathing).

i) Histological defense structures

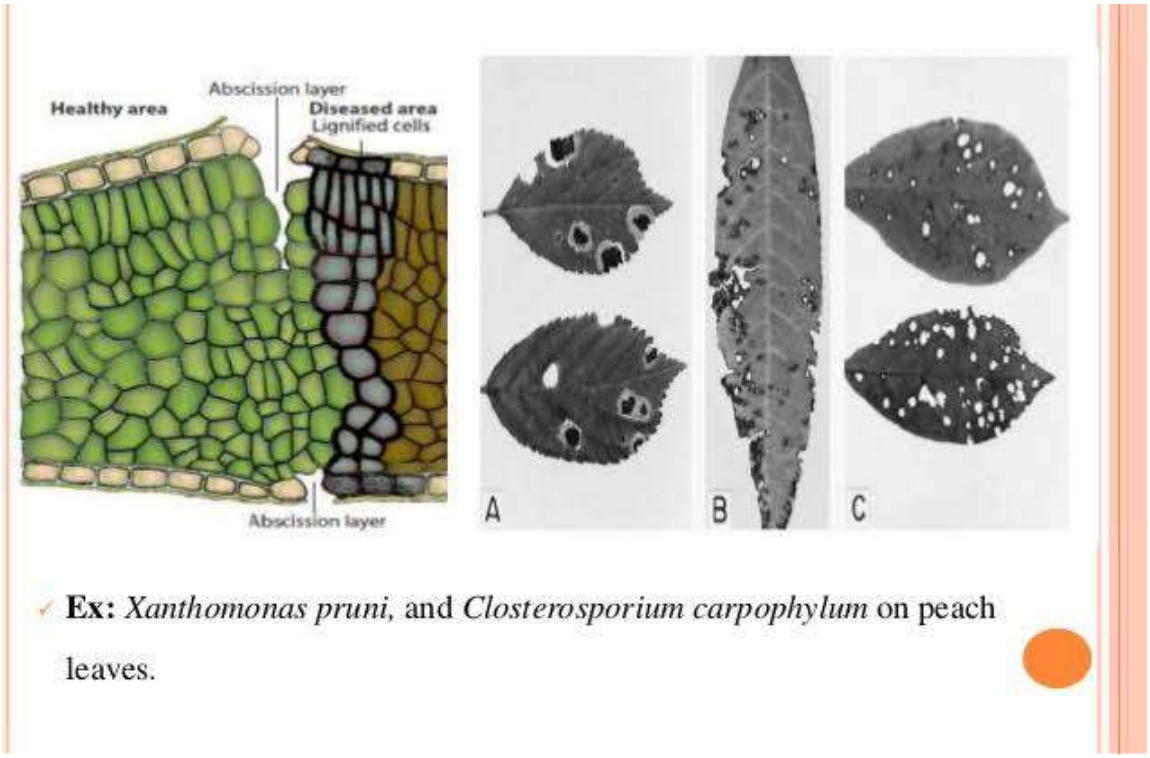
a) Cork layer: Infection by fungi, bacteria, some viruses and nematodes induce plants to form several layers of cork cells beyond the point of infection and inhibits the further invasion by the pathogen beyond the initial lesion and also blocks the spread of toxin substances secreted by the pathogen. Furthermore, cork layers stop the flow of nutrients and water from the healthy to the infected area and deprive the pathogen of nourishment.

Ex: Potato tubers infected by *Rhizoctonia*; *Prunus domestica* leaves attacked by *Coccomyces pruniphorae*.



b) Abscission layers

An abscission layer consists of **a gap formed between infected and healthy cells** of a leaf surrounding the locus of infection due to the disintegration of the middle lamella of parenchymatous tissue.



Gradually, infected area shrivels, dies, and sloughs off, carrying with it the pathogen.

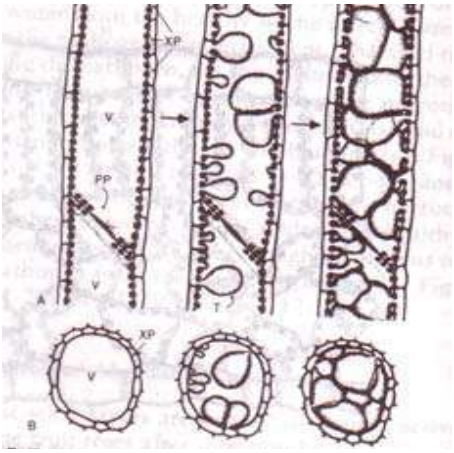
Abscission layers are formed on young active leaves of stone fruits infected by fungi, bacteria or viruses.

Ex: *Xanthomonas pruni* and *Closterosporium carpophylum* on peach leaves

c) Tyloses

Tyloses are the overgrowths of **the protoplast of adjacent living parenchymatous cells, which protrude into xylem vessels through pits**. Tyloses have cellulosic walls and are formed quickly ahead of the pathogen and may clog the xylem vessels completely blocking the further advance of the pathogen in resistant varieties. In susceptible varieties, few or no tyloses are formed ahead of pathogen invasion.

Ex: Tyloses form in xylem vessels of most plants under invasion by most of the **vascular wilt** pathogens.



ii) Cellular defense structures:

Hyphal sheathing: The hyphae penetrating the cell wall and growing into the cell lumen are enveloped by a cellulosic sheath (callose) formed by extension of cell wall, which become infused with phenolic substances and prevents further spread of the pathogen. Ex: Hyphal sheathing is observed in flax infected with *Fusarium oxysporum f.sp. lini*.

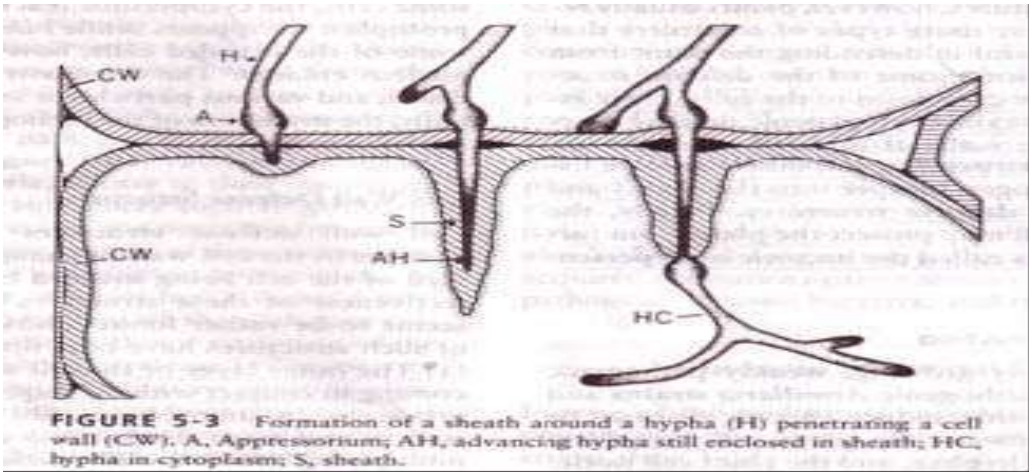


FIGURE 5-3 Formation of a sheath around a hypha (H) penetrating a cell wall (CW). A, Appressorium; AH, advancing hypha still enclosed in sheath; HC, hypha in cytoplasm; S, sheath.

II) Biochemical defense mechanisms: These can be classified as pre-existing and induced biochemical defenses.

1) Pre-existing chemical defenses:

***a) Inhibitors released by the plant in its environment*:**

Plants exude a variety of leaf and root exudates which contain aminoacids, sugars, glycosides, organic acids, enzymes, alkaloids, flavones, toxic materials, inorganic ions and

also certain growth factors. The inhibitory substances directly affect micro-organisms or encourage certain groups to dominate the environment which may act as antagonists to pathogen.

- Ex 1: Tomato leaves secrete exudates which are inhibitory to *Botrytis cinerea*
- Ex 2: Red scales of red onion contain the phenolic compounds, **protocatechuic acid** and **catechol**, which diffuse out to the surface and inhibits the conidial germination of **onion smudge** fungus, ***Colletotrichum circinans***. However, these fungitoxic phenolic compounds are missing in white scaled onions.
- Ex 3: Resistant varieties of apple secrete waxes on the leaf surface which prevents the germination of *Podosphaera leucotricha* (powdery mildew of apples).
- Ex 4: In *Cicer arietinum* (chickpea), the *Ascochyta* blight resistant varieties have more glandular hairs which have **maleic acid** which inhibit spore germination.
- Ex 5: Resistant varieties of linseed secrete HCN in roots which are inhibitory to linseed wilt pathogen, *Fusarium oxysporum f.sp. lini*.
- Ex 6: Root exudates of marigold contain α-terthinyl which is inhibitory to nematodes.
- Ex 7: **Chlorogenic acid** present in sweet potato, potato and carrot inhibits *Ceratocystis fimbriata.* Similarly, **caffeic acid** and **phloretin** are present in sweet potato and apple, respectively.

b) Inhibitors present in plant cells before infection:

- Antimicrobial substances pre-existing in plant cells include unsaturated lactones, cyanogenic glycosides, Sulphur containing compounds, phenols, phenolic glycosides and saponins
- Several phenolic compounds, **tannins**, and some fatty acid like compounds such as **dienes**, which are present in high concentrations in cells of young fruits, leaves or seeds are responsible for the resistance of young tissues to *Botrytis*. These compounds are potent inhibitors of many hydrolytic enzymes.

  Ex: Chlorogenic acid in potato inhibits common scab bacteria, *Streptomyces scabies,* and to wilt pathogen, *Verticillium alboatrum*
- **Saponins** have antifungal membranolytic activity which excludes fungal pathogens that lack saponinases. Ex: **Tomatine** in tomato and **Avenacin** in oats

- Similarly, **lectins**, which are proteins that bind specifically to certain sugars and occur in large concentrations in many types of seeds, cause lysis and growth inhibition of many fungi.
- Plant surface cells also contain variable amounts of **hydrolytic enzymes** such as **glucanases** and **chitinases** which may cause breakdown of pathogen cell wall.

2) Post inflectional or induced defense mechanisms:

a) *Phytoalexins* (*Phyton* = plant; *alexin* = to ward off)

- **Muller** and **Borger** (1940) first used the term phytoalexins for fungistatic compounds produced by plants in response to injury (mechanical or chemical) or infection.
- Phytoalexins are toxic antimicrobial substances produced in appreciable amounts in plants only after stimulation by phytopathogenic micro-organisms or by chemical or mechanical injury.
- Phytoalexins are not produced by uninfected healthy plants, but produced by healthy cells adjacent to localized damaged or necrotic cells in response to materials diffusing from the infected cells. These are not produced during compatible biotrophic infections.
- Phytoalexins accumulate around both resistant and susceptible necrotic tissues. However, resistance occurs when one or more phytoalexins reach a concentration sufficient to restrict pathogen development.

Characteristics of phytoalexins

1. Fungitoxic and bacteriostatic at low concentrations.
2. Produced in host plants in response to stimulus (elicitors) and metabolic products.
3. Absent in healthy plants
4. Remain close to the site of infection.
5. Produced in quantities proportionate to the size of inoculum.
6. Produced in response to the weak or non-pathogens than pathogens
7. Produced within 12-14 hours reaching peak around 24 hours after inoculation.
8. Host specific rather than pathogen specific.

Synthesis and accumulation of phytoalexins are shown in diversified families, viz., Leguminosae, Solanaceae, Malvaceae, Chenopodiaceae, Convolvulaceae, Compositae and Graminaceae.

Sr. No.	Phtoalexin	Host	Pathogen
1	Pisatin	Pea	*Monilinia fructicola*
2	Phaseolin	French bean	*Sclerotinia fructigena*
3	Rishitin	Potato	*Phytophthora infestans*
4	Gossypol	Cotton	*Verticillium alboatrum*
5	Cicerin	Bengalgram	*Ascochyta rabiei*
6	Ipomeamarone	Sweet potato	*Ceratocystis fimbriata*
7	Capsidol	Pepper	*Colletotrichum capsici*

b) Hypersensitive response (HR)

Hypersensitivity: Excessive sensitivity of plant tissue to certain pathogens. Affected cells are killed quickly blocking the advancement of obligate parasite.

- The term hypersensitivity was first used by **Stakman** (1915) in wheat infected by rust fungus, *Puccinia graminis*.
- The hypersensitive response is a **localized induced cell death** in the host plant at the site of infection by a pathogen, thus limiting the growth of pathogen. In the infected plant part, HR is seen as water soaked large sectors which subsequently become necrotic and collapsed.
- HR occurs only in **incompatible** host-pathogen combinations. HR may occur whenever virulent strains or races of pathogens are injected into non-host plants or into resistant varieties, and when avirulent strains or races of pathogens are injected into susceptible cultivars.
- HR is initiated by the recognition of specific pathogen-produced signal molecules, known as **elicitors**. Recognition of the elicitors by the host results in altered cell functions leading to the production of defense related compounds.

The most common **new cell functions** and compounds include:

- A rapid burst of oxidative reactions
- Increased ion movement, especially of K+ and H+ through cell membrane
- Disruption of membranes and loss of cell compartmentalization
- Cross-linking of phenolics with cell wall components and strengthening of plant cell wall
- Production of antimicrobial substances such as phytoalexins and pathogenesis-related proteins (such as chitinases)

Cellular responses during HR

- In many host-pathogen combinations, as soon as the pathogen establishes contact with the cell, the nucleus moves toward the invading pathogen and soon disintegrates.
- Brown resin like granules form in the cytoplasm, first around the point of penetration of pathogen and then throughout the cytoplasm
- As the browning discolouration of the cytoplasm continues and death sets in, the invading hypha begins to degenerate and further invasion is stopped.

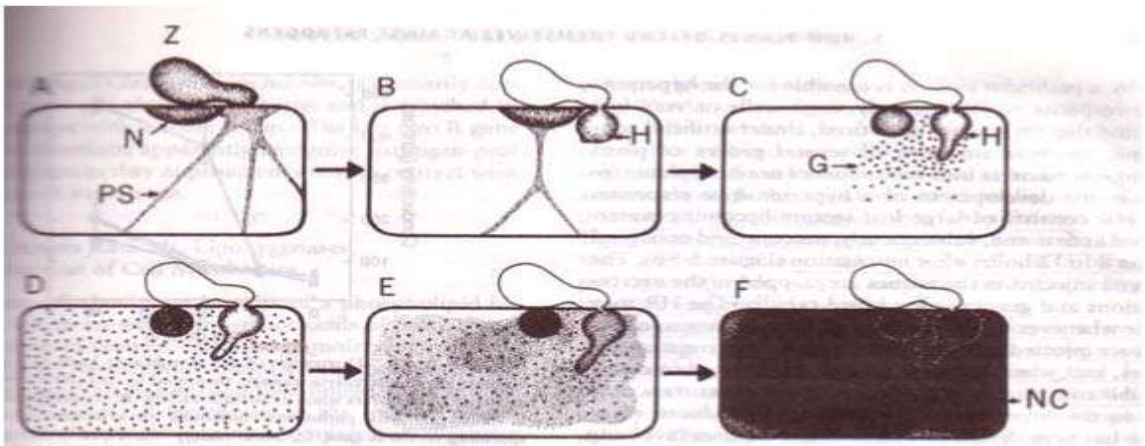


FIGURE 5-8 Stages in the development of the necrotic defense reaction in a cell of a very resistant potato variety infected by *Phytophthora infestans*. N, Nucleus; PS, protoplasmic strands; Z, zoospore; H, hypha; G, granular material; NC, necrotic cell. [After K. Tomiyama (1956), *Ann. Phytopathol. Soc. Jpn.* 21, 54–62.]

c) Plantibodies: Transgenic plants have been produced which are genetically engineered to incorporate into their genome, and to express foreign genes, such as mouse genes that produce antibodies against certain plant pathogens. Such **antibodies, encoded by animal genes, but produced in and by the plant**, are called plantibodies. Ex: Transgenic plants producing plantibodies against coat protein of viruses, such as, **artichoke mottle crinkle virus** have been produced.

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PLANT DISEASE EPIDEMIOLOGY AND REMOTE SENSING

Epiphytology or Epidemiology of plant diseases is essentially a study of the rate of multiplication of a pathogen and spread of the disease caused by it in a plant population. Epidemiology deals with outbreaks and spread of diseases in a population.

Importance of epidemiology:

Knowledge of epidemiology is useful in forecasting of a disease and also for the management of a disease

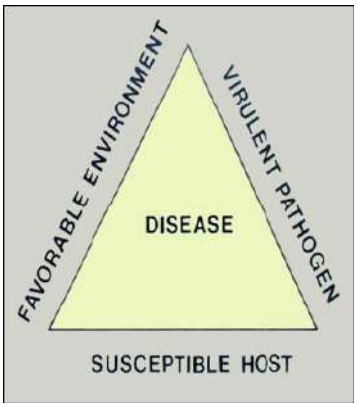
Terms compound interest and simple interest diseases were given by **Vanderplank** (1963) in his book "Plant Disease Epidemics and control"

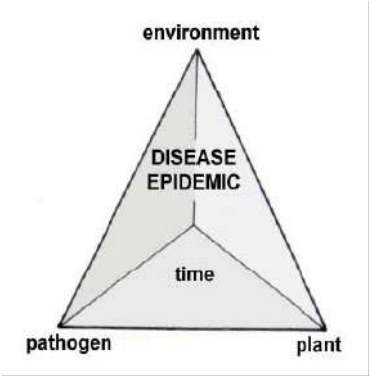
Differentiate between compound interest disease and simple interest disease:

Sr.No.	Compound interest disease/ polycyclic		Simple interest disease/Monocyclic
1	Rate of increase of disease is mathematically analogous to compound interest in money (Interest is added periodically to the capital; interest gets interest)		Rate of increase of disease is mathematically analogous to simple interest in money (Interest is added only at the end; interest does not get interest)
2	The primary inoculum is air borne		The primary inoculum is seed borne or soil borne
3	Pathogen produces spores at rapid rate (i.e. very rapid multiplication of pathogen)		Pathogen produces spores at very slow rate (i.e. very slow multiplication of pathogen)
4	Incubation period and sporulation period is short		Incubation period and sporulation period is long
5	There are several generations of the pathogen in the life of a crop		There is only one generation of the pathogen in the life of a crop
6	Polycyclic disease		Monocyclic disease
7	The infection is generally localized in nature		The infection is generally systemic in nature
8	The disease may spread in large		The diseases are restricted to small areas.

	areas in epidemic form		
9	Ex: Rusts of cereals, Late blight of potato & tomato, Cumin blight, Powdery mildews, Downy mildews		Ex: Loose smuts of wheat & barley, grain smut of sorghum, wilt, root rot,

Disease Triangle: The interactions of three components of disease, i.e., the host, pathogen and environment, can be visualized as a disease triangle. The length of each side is proportional to the sum total of the characteristics of each component that favour disease.





The interaction of susceptible host plant, virulent pathogen and favourable environmental conditions leads to the development of the disease.

Disease Pyramid: The disease triangle can be expanded to include two more components, time and humans. The amount of each of the three components of disease and their interaction in the development of the disease are affected by fourth component, time. Thus addition of time component to the disease triangle results into a **tetrahedron** or **disease pyramid**. The effect of time on disease development becomes apparent when we consider the importance of time of year, the duration and frequency of favourable temperature and rain, the time of appearance of the vector, the duration of the cycle of a particular disease. If the four components of disease pyramid could be quantified, its volume would be proportional to the amount of disease on a plant or in plant population. Humans affect disease development in various ways. They affect the type of plants grown in an area, their level of resistance, time of planting, density of planting, etc.

Essential components/conditions for an Epiphytotic:

1. Host factors 2. Pathogen factors 3. Environmental factors

1. Host factors

i) Distance of susceptible plants from the source of primary inoculum: Longer the distance from the source of survival of the pathogen, longer will be the time required for the buildup of an Epiphytotic in a susceptible crop.

ii) Abundance and distribution of susceptible hosts: Continuous cultivation of a susceptible variety over a large contiguous area helps in the buildup of the inoculum and improves the chances of epiphytotics.

iii) Disease proneness in the host due to environment: Susceptibility is genetically controlled but the disease proneness in the plant to get infected can be induced by environment and other factors (Host nutrition, excessive application of nitrogenous fertilizers, etc).

iv) Presence of suitable alternate or collateral hosts: These host plants help in the survival of inoculum of different pathogens in off season. Presence of Barbery which is an alternate host to *Puccinia graminis tritici* helps in the heterogenous infection chain. Presence of grass hosts helps in the survival of *Pyricularia oryzae* in the off-season.

2. Pathogen factors:

i) Presence of virulent/aggressive isolate of a pathogen: For any epiphytotic, rapid cycle of infection is essential, and successful infection can be caused only by virulent isolates of the pathogen.

ii) High birth rate: The fungi that assume epiphytotic form invariably have the capacity to produce enormous quantity of spores that are adapted to long distance dissemination in a short time.

iii) Low death rate of the pathogen: Epiphytotics is attributed to low death rate of the pathogens in those in which the causal agent is systemic and protected by the plant tissues.

iv) Easy and rapid dispersal of the pathogen: The ability of a pathogen to cause epiphytotics is much more dependent on its dispersal rate. The units of propagation need to be dispersed by external agencies, if epiphytotics are to develop.

Ex: Fungal spores disseminated by wind, water, etc

Viruses disseminated by insect vectors.

Bacteria dispersed through rain splashes and water.

v) Adaptability of the pathogen: Most of the pathogens causing epiphytotics adapt themselves to various adverse conditions.

3. Weather factors: Assuming that a particular fungus meets all the above requirements for causing an epidemic, the infection, invasion and development of epidemic may not occur if weather is not favourable for the germination of spores. Congenial environmental conditions, viz., optimum weather conditions for sporulation, dispersal, infection and survival of pathogen, are very important.

Weather conditions such as, optimum temperature, moisture, light, etc., are very essential for the development of an epidemics.

Science which deals with the relationship between weather and epiphytotics is called **metereopathology**.

REMOTE SENSING

Remote sensing is estimating an object/phenomenon without being in physical contact with it. Remote sensing is a science/art that permits us to obtain information about an object/a phenomenon through analysis of data obtained through sensory devices without being in physical contact with that object.

Objectives of remote sensing in plant Pathology:

1. Assessment of disease over a vast area
2. To know the relationship of diseases and environment
3. To know the origin and development of epidemics
4. Quantitative assessment of the disease.

Remote sensing techniques of importance to Plant Pathology:

1. Aerial photography and 2. Satellite remote sensing

1. Aerial photography: Aerial photography can detect objects on land over a larger area. **Colwell** (1956) first used remote sensing technique for monitoring stem rust of wheat. He showed that panchromatic colour and especially infrared aerial photography could be used to detect rusts and viral diseases of small grains and certain diseases of citrus. Later, infrared photography was used in England for late blight of potato.

The key to distinguish diseased and healthy parts of a crop is to use appropriate film or filter combinations. The main film types used are panchromatic, infrared, normal colour and colour infrared. The **infrared** films are preferred because of their superior sensitivity to visible light and to near infrared wavelengths of radiation (700-900 mμ). The colour infrared or Ektachrome Aero Infrared (Camouflage Detection Film) is superior as it can show the difference between diseased and healthy patches of plants in colour. The healthy foliage is highly reflective to the infrared wavelengths and appears red on this film whereas blighted or diseased foliage has low infrared reflectance and does not appear red in the photograph.

2. Satellite Imaging

Weather satellites

Often cyclones create heavy clouds with rains and an anti-cyclone creates a cloudless sky. All these can be effectively monitored by weather satellites. Sequential pictures show the movement of these systems before they arrive in an area. Therefore by monitoring epidemic favouring systems using a satellite, the disease occurrence on the field can be monitored. Ex: The spread and deposition of stem rust pathogen of wheat is influenced by definite synoptic weather conditions called **Indian stem rust rules**.

Earth resources technology satellites (LANDSAT, 1972, USA)

LANDSAT covers the entire globe every 18 days scanning the same area at a fixed time. The scanned data is compared for any major differences happened within 18 days. Nagarajan utilized LANDSAT infrared spectral bands 6 (0.7-0.8μm) and 7 (0.8-1.1μm) to differentiate healthy wheat crop of India and severe yellow rust affected crop of Pakistan.

Examples: Coconut root rot and wilt, black stem rust of wheat, citrus canker.

Advantages of Remote sensing

1. Reveals pattern of disease incidence, intensity and development over large area
2. Data generated by remote sensing is amenable to multidisciplinary approach
3. Gives synoptic view of large areas
4. Data generated is on a permanent scale and is unbiased
5. Data acquisition is fast compared to traditional methods and data analyzed is effectively utilized
6. Satellite data (ERTS) obtains information of an area periodically so that the information can be updated.
7. It frequently poses questions for ground investigators which cannot be generated by ground parties

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PRINCIPLES OF PLANT DISEASE MANAGEMENT

The epidemics of potato late blight, brown rot of rice, red rot of sugarcane, southern corn blight diseases illustrate that the diseases can be devastating on large scale. The population pressure necessiates that crop losses due to diseases be lessened.

Modern agriculture, necessary for large scale production, invites disease problems through:

1. Continuous monoculture of an annual crop and/or genetically similar plants by crowding hosts together in both area and time. This may lead to the development of aerial and soil born diseases and
2. Use of plants susceptible to pathogens.

Therefore, disease management is necessary. "Disease management is an essential integral component of crop production; it employs a logical system of technologies and requires accurate understanding of the destructive potential of disease. Thus, disease management is the selection and use of appropriate techniques to suppress disease to a tolerable level". Therefore 'plant disease management' is a better expression than 'plant disease control' as:

i. Word 'control evokes the notion (an idea/view) of finality.' that is permanent settlement of problem.
ii. No further operation to word –off diseases is necessary than, and
iii. It is an absolute term (100% control).

In reality this is not so, no plant diseases was been permanently controlled.

The word 'MANAGEMENT' evokes:

i. Process of continuous efforts.
ii. This involves the principles of maintaining the damage or loss below an economic level of injury or at least minimizing the occurrence of disease above that level.
iii. This involves continuous adjustments in crop cultivation system.
iv. This is relatively term – near 100% but not absolute.

Management: It conveys a concept of continuous process which is based not only on the principle of eradication of the pathogen but mainly on the principle of minimizing the damage or loss below economic injury level.

Importance: Plant diseases are important because of the losses (qualitative and quantitative) they cause. Loss may occur at any time between sowing of the crop and consumption of the produce. Measures taken to prevent the incidence of the disease, reduce the amount of inoculum that initiates and spreads the disease and finally minimize the loss caused by the disease are called as management practices.

Essential considerations in plant disease Management:

1. Benefit-cost ratio : Cost benefit ration 'is a very important consideration in application of management measures. It is known that plant disease always reduce economic gains. If control measures fail to increase economic gain even if the disease incidence is lowered, the growers or farmer do not accept the recommendation to combat plant diseases. The aim of the disease management is to check disease with economic gain.

2. Procedures for disease control should fit into general schedule of operations of crop production

3. Control measures should be adopted on a **co-operative basis** over large adjoining areas. This reduces frequency of applications (treatments), reduced cost of control, reduced chances of disease outbreak and increases chances of success of control measures

4. The knowledge regarding disease cycle or disease development on the following aspects is essential for effective and economic control:

   a. Cause of a disease
   b. Mode of survival and dissemination of the pathogen
   c. Host parasite relationship
   d. Effect of environment on pathogenesis in the plant or spread in plant population

5. Prevention of disease depends on management of primary inoculum

6. Integration of different approaches of disease management is always recommended.

GENERAL PRINCIPLES OF PLANT DISEASE MANAGEMENT :

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 pathogen / inoculum**: Preventing the inoculum from entering or establishing in the field or area where it does not exist.

3. **Eradication of pathogen**: 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 (Protective measures)**: Preventing infection by creating a chemical toxic barrier between the plant surface and the pathogen

5. **Disease resistance (Immunization) OR Development of plant resistance in disease management**: Preventing infection or reducing effect of infection by managing the host through improvement of resistance in it by genetic manipulation or by chemical therapy.

6. **Therapy of diseased plants (curative measures)**

I. <u>AVOIDANCE OF THE PATHOGEN</u>: These methods aim at avoiding the contact between the pathogen and susceptible stage of the crop. This is achieved by

a. Proper selection of geographical area

b. Proper selection of the field

c. Adjusting time of sowing

d. Disease escaping varieties

e. Proper selection of seed and planting material

a) Proper selection of geographical area: Many fungal and bacterial diseases are more severe in wet areas than in dry areas. Cultivation of bajra in wet areas is not profitable due to the diseases, smut (*Tolyposporium penicillariae*) and ergot (*Claviceps microcephala*).

b) Proper selection of the field: Proper selection of field will help in the management of many diseases, especially the soil borne diseases. If a pathogen causing a soil borne disease is present in field and if raising of a particular crop year after year in the same field makes the soil sick, where disease incidence and severity may be more. It is advisable to avoid that field (wherein the pathogen is present) for some years, i.e. suitably through rotation of crops.

Ex: Wilt of redgram, late blight of potato (*Phytophthora infestans*), green ear of bajra (*Sclerospora graminicola*), red rot of sugarcane (*Colletotrichum falcatum*), root knot diseases are the best examples.

c) Adjusting time of sowing: Generally pathogens are able to infect the susceptible plants under certain environmental conditions that means upon coinciding susceptible stage of crop and favourable conditions for pathogen, the disease incidence is most severe. Alteration of date of sowing (early or late sowing) can help in avoidance of favourable conditions for pathogen.

Ex: *Rhizoctonia* root rot of redgram is more severe in the crop sown immediately after the rains. Delayed sowing will help in reducing the incidence of disease.

Ex: Infection of black stem rust of wheat (*Puccinia graminis tritici*) is more in late sowing, hence, early sowing wheat crops helps in reduction of stem rust incidence.

Ex. Late sown Isabgul crop harbours less downy mildew disease.

d) Disease escaping varieties: Certain varieties of crops escape the disease damage because of their growth characteristics. Ex: Early maturing varieties of wheat or pea escape the damage due to *Puccinia graminis tritici* and *Erysiphe polygoni* (powdery mildew-pea), respectively. The disease escaping is simply a carry over (overtake) of a variety with the pathogen in such a way that its growth and maturity periods do not clash with activity of pathogen. In this way susceptible crop plants escape from the disease even though the pathogen is present in the field.

e) Proper selection of seed and planting material: Generally, the plant diseases are introduced through the seeds or propagating materials (cuttings, grafts, bulbs, tubers, rhizome, setts). So, selection of seed and seedling material from healthy sources will effectively manage the diseases such as loose smut of wheat (*Ustilago nuda tritici*), bunchy top of banana (*Banana virus-1*), Panama wilt of banana (*Fusarium oxysporum f.sp. cubense*), red rot of sugarcane (*Colletotrichum falcatum*), black scurf of potato, brown rot of potato and whip smut of sugarcane (*Ustilago scitaminae*). Potato seed certification or tuber indexing is followed for obtaining virus free seed tubers. Citrus bud wood certification programme will help in obtaining virus free planting material.

f) Modification of cultural practices:

If the following cultural practices are suitably modified, the disease incidence and crop losses may be greatly minimized;

Cultural practices (suitably modified)

a. **Distance:** Groundnut rosette is minimized, if plant to plant distance is reduced.
b. **Seed rate:** Tobacco damping off disease is less when seed rate is low.
c. **Amount of irrigation:** Powdery mildew of peas is more under heavy irrigation.
d. **Quantity and quality of fertilizer**: Bacterial blight of paddy increases by increased quality of nitrogenous fertilizer.
e. **Organic manures:** Soil borne diseases are reduced by organic manures.
f. **Time and method of planting:** Downy mildew of isabgul is less when the crop is sown late.
g. **Mixed cropping:** Tur (arhar) wilt is less when jowar is sown as a mixed crop.
h. **Depth of sowing:** Decay of groundnut seeds is more when seeds are sown more deep.

II. EXCLUSION OF THE PATHOGEN / INOCULUM: The aim of this principle is to prevent the contact between the pathogen and the seed materials in the filed, i.e., to prevent spread of the disease. These measures aim at preventing the inoculum from entering or establishing in the field or area where it does not exist. The disease free seed materials can be obtained through, OR Different methods of exclusion are seed treatment, ceed inspection & certification, and plant quarantine regulation.

a) Seed treatment:

This method is used for exclusion of pathogen by eradication. To exclude pathogen present in or upon seed materials (seeds; tubers; grafts; bulbs; cuttings; setts;) heat, or gas, or chemical treatments are given. The seed treatment increases germination count and lowers disease incidence in the field (as the quantity of pathogen, i.e. inoculum load of the seed materials is very much reduced). Treated seed materials help in preventing entry of the pathogen(s) in newer areas.

b) Crop inspection and certification: Crops grown for seed purpose are inspected periodically for the presence of diseases that are disseminated by seed. Necessary precautions are to be taken to remove the such diseased plants in early stages, and then the crop is certified as disease free. This practice will help in the prevention of inter and intra regional spread of seed borne diseases.

c) Plant quarantine regulation / Quarantine procedure: Plant quarantine is defined as "a legal restriction on the movement of agricultural commodities for the purpose of exclusion, prevention or delaying the spread of the plant pests and diseases in uninfected areas".

Plant quarantine laws were first enacted in **France** (1660), followed by Denmark (1903) and USA (1912). These rules were aimed at the rapid destruction or eradication of barbery bush which is an alternate host of *Puccinia graminis tritici* (black rust of wheat).

In India, plant quarantine rules and regulations were issued under **Destructive Insects and Pests Act (DIPA)** in 1914. In India, 16 plant quarantine stations are in operation by the "Directorate of plant protection and quarantine" under the ministry of food and agriculture, government of India.

The quarantine is a legal procedure adopted to prevent the spread of diseases/pests upon import or export of plants on plant parts. Plants quarantine aims to preventing entry of pathogens from infested areas to non-infested areas. The Government passes necessary regulations to stop entry of such materials from the infested areas. The quarantine may fail or does not work when:

a. The pathogen is air borne.
b. Smuggling of seeds from country to country.
c. Some pathogens may get transported by clothes and aircrafts, and
d. Sea water, birds also carry pathogens from one region to another.

Besides advantages of, there are certain limitations. There are:

a. Obstructs trades (delay due to procedures),
b. Obstructs import of food grains (when urgently required), and
c. Certain food materials/planting materials cannot be treated by toxic materials (become limit for consumption).

Plant quarantine measures are of 3 types.

1. Domestic quarantine: Rules and regulations issued prohibiting the movement of insects and diseases and their hosts from one state to another state in India is called domestic quarantine. Domestic quarantine in India exists for two pests (Rooted scale and Sanjose scale) and three diseases (Bunchy top of banana, banana mosaic and wart of potato).

Bunchy top of banana: It is present in Kerala, Assam, Bihar, West Begal and Orissa. Transport of any part of *Musa* species excluding the fruit is prohibited from these states to other states in India.

Banana mosaic: It is present in Maharashtra and Gujarat. Transport of any part of *Musa* species excluding the fruit is prohibited from these states to other states in India.
Wart of potato: It is endemic in Darjeeling area of West Bengal, therefore seed tubers are not to be imported from West Bengal to other states.

2. Foreign quarantine: Rules and regulations issued prohibiting the import of plants, plant materials, insects and fungi into India from foreign countries by air, sea and land. Foreign quarantine rules may be general or specific. General rules aim at prevention of introduction of pests and diseases into a country, where as the specific rules aim at specific diseases and insect pests. The plant materials are to be imported only through the prescribed ports of entry.

1. **Airports**: Bombay (Santacruz), Calcutta (Dum Dum), Madras (Meenambakam), New delhi (Palam, Safdarjung) and Tiruchurapally.

2. **Sea ports**: Bombay, Calcutta, Vishakapatnam, Trivandrum, Madras, Tuticorin, Cochin and Dhanushkoti.

3. **Land frontiers**: Hussainiwala (Ferozpur district of Punjab), Kharla (Amritsar district of Punjab) and Sukhiapokri (Darjeeling district of West Bengal)
3. Total embargoes: Total restriction on import and export of agricultural commodities.
Phytosanitary certificate: It is an official certificate from the country of origin, which should accompany the consignment without which the material may be refused from entry.

Plant diseases introduced into India before/after enforcement of plant quarantine laws:

S.No.	Disease	Year	Introduced into	From
1	Late blight of potato	1883	India	Europe
2	Coffee rust	1879	India	Srilanka
3	Flag smut of wheat	1906	India	Australia
4	Downy mildew of grapes	1910	India	Europe
5	Bacterial blight of rice	1964	India	Phillippines

6	Rice blast	1918	India (Madras)	South East Asia
7	Downy mildew of maize	1912	India (Madras)	Java
8	Ergot of bajra	1957	India (Bombay)	Africa
9	Panama wilt of banana	1920	India	Panama canal
10	Bunchy top of banana	1940	India	Srilanka
11	Wart of potato	1953	India	Netherlands
12	Golden cyst nematode of potato	1961	India	Europe

Diseases not entered into India: Swollen shoot of cocoa, leaf blight of rubber and many viral diseases.

III. ERADICATION OF PATHOGEN: These methods aim at breaking the infection chain by removing the foci of infection and starvation of the pathogen (i.e., elimination of the pathogen from the area by destruction of sources of primary and secondary inoculum). It is achieved by :

a) Rouging : The presence of diseased plants in the fields or orchards is a source of continuous release of inoculums for recurrence of diseases. Therefore, as far as practicable, such plants or their affected organs should be removed and destroyed carefully to reduce the amount of inoculum and spread of disease. This is called 'ROUGING'. Destruction of alternate and collateral hosts is essential in disease management. The examples are: loose smut of wheat, covered and loose smut of barley, whip smut of sugarcane, red rot of sugarcane, ergot of bajra, yellow vein mosaic of bhendi, wilt of arhar are effectively manageable by 'ROUGING'.

During 1927- 1935, to eradicate citus canker bacterium in USA, 3 million trees were cut down and burnt.

b) Eradication of alternate and collateral hosts: Careful removal and by destruction of alternate and collateral hosts, the life cycle of pathogens with continuous infection chain through such hosts is broken. Thus, the chances of initiation of infection are greatly minimized, e.g. wheat rust disease.

Eradication of alternate hosts will help in management of many plant diseases.
Ex: Barbery eradication programme in France and USA reduced the severity of black stem rust of wheat.
Ex: Eradication of *Thalictrum* species in USA to manage leaf rust of wheat caused by *Puccinia recondita*.
Eradication of collateral hosts, such as *Panicum repens*, *Digitaria marginata* will help in the management of rice blast disease (*Pyricularia oryzae*).

c) Crop rotation: Continuous cultivation of the same crop in the same field helps in the perpetuation of the pathogen in the soil. Soils which are saturated by the pathogen are often referred as **sick soils**. To reduce the incidence and severity of many soil borne diseases, crop rotation is adopted. Crop rotation is applicable to only root inhabitants and facultative saprophytes, and may not work with soil inhabitants (means survive for a long period of time). eg. Cumin crop is difficult to be raised in North Gujarat fileds due to wilt disease and prevalence of wilt disease in South Gujarat fields prevents Tur (arhar) cultivation.
Ex: Panama wilt of banana (long crop rotation), wheat soil borne mosaic (6 yrs) and club root of cabbage (6-10 yrs), etc.

d) Crop sanitation: Collection and destruction of plant debris from soil will help in the management of soil borne facultative saprophytes as most of these survive in plant debris. Collection and destruction of plant debris is an important method to reduce the primary inoculum.

e) Manures and fertilizers: The deficiency or excess of a nutrient may predispose a plant to some diseases. Excessive nitrogen application aggravates diseases like stem rot, bacterial leaf blight and blast of rice. Nitrate form of nitrogen increases many diseases, whereas, phosphorous and potash application increases the resistance of the host. Addition of farm yard manure or organic manures such as green manure, 60-100 t/ha, helps to manage the diseases like cotton wilt, Ganoderma root rot of citrus, coconut, etc.

f) Mixed cropping: Root rot of cotton (*Phymatotrichum omnivorum*) is reduced when cotton is grown along with sorghum. Intercropping sorghum in cluster bean reduces the incidence of root rot and wilt (*Rhizoctonia solani*)

g) Summer ploughing: Ploughing the soil during summer months expose soil to hot weather which will eradicate heat sensitive soil borne pathogens.

h) Soil amendments: Application of organic amendments like saw dust, straw, oil cake, etc., will effectively manage the diseases caused by *Pythium, Phytophthora, Verticillium, Macrophomina, Phymatotrichum* and *Aphanomyces*. Beneficial micro-organisms increases in soil and helps in suppression of pathogenic microbes.

Ex: Application of lime (2500 Kg/ha) reduces the club root of cabbage by increasing soil pH to 8.5.

Ex: Application of Sulphur (900 Kg/ha) to soil brings the soil pH to 5.2 and reduces the incidence of common scab of potato (*Streptomyces scabies*).

i) Changing time of sowing: Pathogens are able to infect susceptible plants under certain environmental conditions. Alternation in date of sowing can help avoidance of favourable conditions for the pathogens.

Ex: Rice blast can be managed by changing planting season from June to September/October.

j) Seed rate and plant density: Close spacing raises atmospheric humidity and favours sporulation by many pathogenic fungi. A spacing of 8'X8' instead of 7'X7' reduces sigatoka disease of banana due to better ventilation and reduced humidity. High density planting in chillies leads to high incidence of damping off in nurseries.

k) Irrigation and drainage: The amount, frequency and method of irrigation may affect the dissemination of certain plant pathogens. Many pathogens, including, *Pseudomonas solanacearum, X. campestris pv. oryzae* and *Colletotrichum falcatum* are readily disseminated through irrigation water. High soil moisture favours root knot and other nematodes and the root rots caused by species of *Sclerotium, Rhizoctonia, Pythium, Phytophthora, Phymatotrichum*, etc.

IV. PROTECTIVE MEASURES:

The inocula (singular: inoculum) of many fast spreading infectious diseases is shifted/transported by various agencies from field to field irrespective of distance. To destroy or inactivate such inocula different adopted protective measures include:

1. **Chemicals in suppressing disease development:**

   Relatively few chemicals are used extensively in practical disease management. Most are applied to foliage of vegetables, fruits, or ornamentals. None of the existing chemicals satisfies all criteria of the "ideal" chemical. Some are phytotoxic (sulphur). Some are selective and others are broad range. Some cause mutation and development of resistance in the pathogens through sprays and dustings which form a protective toxic layer on the host surface to avoid contact by the pathogen. The chemicals providing such protective covering are called **protective chemicals**. On the other hand eradicant chemicals, upon their application, destroy already established parasites on the host surface. The same chemical may be protective as well as eradicant.

2. **Chemical control of insect vectors:**

   Various insects act as virus-vectors. Destruction of such insect-vectors in 'seconds' through chemicals stop transmission of virus diseases is of almost importance.

3. **Modification of environments:**

   The environments in the field (especially the micronutrients) and storage conditions, if modified suitably as under, the losses due to diseases can be greatly minimized by:

Field conditions:

a. Improvement of aeration,
b. Reducing number of irrigations, and
c. Mixed cropping (cotton and moth bean, if mixed, cotton root rot incidence is reduced).

Storage condition:

d. Storage under cool and dry rooms reduces fruit rotting diseases.
e. Cold storage (at 4^0 C) protects potatoes from various diseases.

4. Modification of host nutrition:

Deficiencies or excesses of certain vital macro and micro – elements in crops lead to the development of various plants diseases in the field. The examples are:

a. Potash: if less, it renders leaves susceptible to water soaking
b. Nitrogen: if high, it favours many leaf diseases.
c. Calcium: if high, it imparts resistance against wilt diseases (pectic substances in the cell walls of host tissues impart resistance).

V. DEVELOPMENT OF PLANT RESISTANCE IN DISEASE DEVELOPMENT:

Use of resistance plants is one of the most effective approaches to suppressing plant disease.

The resistant plants do not require:

a. any attention by the grower during growing period
b. not disruptive to the environment,
c. Compatible with other disease management techniques and
d. Help sufficiently to suppress disease to tolerable levels.

Resistance is the characteristics of a plant, which suppresses pathogen and disease development. The magnitude of resistance can range from very small (in which case, the pathogen development is suppressed very slightly) to very large (in which case, the pathogen does not complete pathogenesis).

a. Resistance in cabbage yellows (induced by *Fusarium oxysporium f. sp. conglutinans*). In this case, the highest level of resistance was that conditioned by a 'single gene' effective for a long period of time.

b. Resistance in potato late blight (induced by *Phytophthora infestans*).

At no extra expenditure, an economical, practical and effective method of plant disease control is the use of resistant varieties developed through selection and hybridization. The use of such varieties insures farmers safely.

The resistance in the host is developed through the following various techniques:

a. Selection and hybridization for resistance:

Selection and hybridization techniques are purely based on physiological, structural and functional behavior of the host.

b. Resistance through Chemotherapy:

The pathogens fall to invade host tissues of foliages which were fed fungicides and antibiotics through roots. This is a temporary sort of physiological resistance.

c. Resistance through host nutrition:

Vigorously growing plants have the capacity to form new roots and shoots to partake functions of damaged roots and develop the capacity to resist many plant diseases.

VII. THERAPY OF DISEASED PLANTS (curative measures):

In most herbaceous crops once damaged by diseases cure is not possible. In certain crops and fruits trees, the pathogens could be eradicated as under by application of chemicals and physical therapy.

1. **Chemotherapy:**

   Chemicals like systemic fungicides and antibiotics translocate through tissues of diseased plants and there by curing (eradicating the pathogen) such plant tissues.

2. **Heat therapy:**

   These treatments are especially used for seeds, tubers, bulbs, grafts, setts, cuttings. Through heat therapy (air and water) the pathogen is destroyed by heat when thermal inactivation point is achieved in the plants materials under treatments i.e. Ratoon stunting and various virus diseases.

3. **Tree therapy:**

   After removing all damaged on affected parts of a tree, the prunned areas (wounds) are covered by fungicidal paste (e.g. Bordeaux paste) to protect them from infection. Examples are pink disease of apple, gummosis of citrus.

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PHYSICAL METHODS, ROLE AND MECHANISMS OF BIOLOGICAL CONTROL AND PLANT GROWTH PROMOTING RHIZOBACTERIA (PGPR)

PHYSICAL METHODS: Physical methods include soil solarization and hot water treatments.

i. Soil solarization: Soil solarization or slow soil pasteurization is the hydro/thermal soil heating accomplished by covering moist soil with polyethylene sheets as soil mulch during summer months for 4-6 weeks. Soil solarization was developed for the first time in Israel (Egley and Katan) for the management of plant pathogenic pests, diseases and weeds.

ii. Soil sterilization: Soil can be sterilized in green houses and sometimes in seed beds by aerated steam or hot water. At about 50^0C, nematodes, some oomycetous fungi and other water molds are killed. At about 60 and 72^0C, most of the plant pathogenic fungi and bacteria are killed. At about 82^0C, most weeds, plant pathogenic bacteria and insects are killed. Heat tolerant weed seeds and some plant viruses, such as TMV are killed at or near the boiling point (95-100^0C).

iii. Hot water or Hot air treatment: Hot water treatment or hot air treatment will prevent the seed borne and sett borne infectious diseases. Hot water treatment of certain seeds, bulbs and nursery stock is done to kill many pathogens present in or on the seed and other propagating materials. Hot water treatment is used for controlling sett borne diseases of sugarcane [whip smut, grassy shoot and red rot of sugarcane (52^0C for 30 min)] and loose smut of wheat (52^0C for 10 min).

Biological methods:

Def: Biological control of plant disease is a condition or practice whereby survival or activity of a pathogen is reduced through the agency of any other living organism (except human beings), with the result that there is reduction in incidence of the disease caused by the pathogen (Garett, 1965).

Def: Biological control is the reduction of inoculum density or disease producing activity of a pathogen or a parasite in its active or dormant state by one or more organisms

accomplished naturally or through manipulation of the environment of host or antagonist by mass introduction of one or more antagonists (Baker and Cook, 1974)

Mechanisms of biological control :

1. Competition: Most of the biocontrol agents are fast growing and they compete with plant pathogens for space, organic nutrients and minerals. Most aerobic and facultative anaerobic micro-organisms respond to low iron stress by producing extracellular, low molecular weight (500-1000 daltons) iron transport agents, designated as **Siderophores**, which selectively make complex with iron (Fe^{3+}) with very high affinity. Siderophore producing strains are able to utilize Fe^{3+} - Siderophore complex and restrict the growth of deleterious micro-organisms mostly at the plant roots. Iron starvation prevents the germination of spores of fungal pathogens in rhizosphere as well as rhizoplane. Siderophores produced by *Pseudomonas fluorescens* (known as **pseudobactins** or **pyoveridins**) helps in the control of soft rot bacterium, *Erwinia caratovora.*

2. Antibiosis: Antagonism mediated by specific or non-specific metabolites of microbial origin, by lytic agents, enzymes, volatile compounds or other toxic substances is known as antibiosis.

a. **Antibiotics**: Antibiotics are generally considered to be organic compounds of low molecular weight produced by microbes. At low concentrations, antibiotics are deleterious to the growth or metabolic activities of other micro-organisms.
Ex: *Gliocladium virens* produces **gliotoxin** that was responsible for the death of *Rhizoctonia solani* on potato tubers.
Ex: Colonization of pea seeds by *Trichoderma viride* resulted in the accumulation of significant amount of the antibiotic **viridin** in the seeds, thus controlling *Pythium ultimum*.
Ex: Some strains of *Pseudomonas fluorescens* produce a range of compounds, viz., 2,4-diacetyl phloroglucinol (DAPG), phenazines, pyocyanin, which have broad spectrum activity against many plant pathogenic bacteria and fungi

b. **Bacteriocins**: These are antibiotic like compounds with bactericidal specificity closely related to the bacteriocin producer. Ex: The control of crown gall (caused by

Agrobacterium tumefaciens) by the related *Agrobacterium radiobacter* strain K 84 is by the production of bacteriocin, **Agrocin K84**.

c. **Volatile compounds**: Antibiosis mediated by volatile compounds has been observed in the management of soil borne pathogens, viz., *Pythium ultimum, Rhizoctonia solani* and *Verticillium dahlia*, by *Enterobacter cloacae*. The volatile fraction responsible for inhibition was identified as ammonia.

3. Hyperparasitism: Direct parasitism or lysis and death of the pathogen by another micro-organism when the pathogen is in parasitic phase is known as hyperparasitism.
Ex: *T. harzianum* parasitize and lyse the mycelia of *Rhizoctonia* and *Sclerotium*.

Biocontrol agents for the management of plant pathogens :

Biocontrol agent Pathogen/disease

Biocontrol agent		**Pathogen/disease**
1. *Ampelomyces quisqualis*		Powdery mildew fungi
2. *Darluca filum, Verticillium lecanii*		Rust fungi
3. *Pichia gulliermondii*		*Botrytis, Penicillium*

Biocontrol agent		**Nematode**
1. *Pasteuria penetrans* (Bacteria)		Juvenile parasite of root knot nematode
2. *Paecilomyces lilacinus* (Fungus)		Egg parasite of *Meloidogyne incognita*

Important fungal biocontrol agents:

Most of the species of Trichoderma, viz., *T. harzianum, T. viride, T. virens* (*Gliocladium virens*) are used as biocontrol agents against soil borne diseases, such as, root rots, seedling rots, collar rots, damping off and wilts caused by the species of Pythium, Fusarium, Rhizoctonia, Macrophomina, Sclerotium, Verticillium, etc.

Formulations of biocontrol agents available: *T. viride* (**Ecofit**, **Bioderma** in India), *G. virens* (**GlioGard** in USA), *T. harzianum* (**F-Stop** in USA) and *T. polysporum* (**BINABT**)

Important bacterial biocontrol agents:

1. *Pseudomonas fluorescens* (**Dagger-G** against damping off of cotton seedlings in USA)
2. *Bacillus subtilis* (**Kodiak** against damping off and soft rot in USA)
3. Agrobacterium radiobacter K-84 (**Gallex** or **Galltrol** against crown gall of stone fruits caused by *Agrobacterium tumefaciens*)

Plant growth promoting Rhizobacteria (PGPR):

Rhizosphere bacteria that favourably affect plant growth and yield of commercially important crops are designated as plant growth promoting rhizobacteria. The growth promoting ability of PGPR is due to their ability to produce phytohormones, Siderophores, Hydrogen cyanide (HCN), chitinases, volatile compounds or antibiotics which will reduce infection of host through phyto-pathogenic micro-organisms.

Many bacterial species, viz., *Bacillus subtilis, Pseudomonas fluorescens*, etc., are usually used for the management of plant pathogenic microbes. *Bacillus* has ecological advantages as it produces endospores that are tolerant to extreme environmental conditions. *Pseudomonas fluorescens* have been extensively used to manage soil borne plant pathogenic fungi due to their ability to use many carbon sources that exude from the roots and to compete with microflora by the production of antibiotics, HCN and Siderophores that suppress plant root pathogens.

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CHEMICAL METHODS AND HOST PLANT RESISTANCE (IMMUNIZATION)

PROTECTION: Use of chemicals for the control of plant diseases is generally referred to as protection or therapy.

Protection: The prevention of the pathogen from entering the host or checking the further development in already infected plants by the application of chemicals is called protection and the chemicals used are called **protectants**.

Therapy means cure of a disease, in which fungicide is applied after the pathogen is in contact with the host. Chemicals used are called **therapeutants**.

Fungicide: Any agent (chemical) that kills the fungus

Fungistat: Some chemicals which do not kill fungi, but simply inhibit the fungus growth temporarily.

Antisporulant: The chemical which inhibits spore production without affecting vegetative growth of the fungus.

Fungicides are classified into three categories: Protectants, eradicants and therapeutants. ***1. Protectants***: These are the chemicals which are effective only when used before infection (prophylactic in behavior). Contact fungicides which kill the pathogen present on the host surface when it comes in contact with the host are called protectants. These are applied to seeds, plant surfaces or soil. These are non-systemic in action (i.e, they cannot penetrate plant tissues). Ex: Zineb,sulphur, captan, Thiram, etc.

2. Eradicants: Those chemicals which eradicate the dormant or active pathogen from the host. They can remain on/in the host for some time. Ex: Lime sulphur, Dodine.

3. Therapeutants: These are the agents that inhibit the development of a disease syndrome in a plant when applied after infection by a pathogen. Therapy can be by physical means

(solar and hot water treatment) and chemical means (by use of systemic fungicides, i.e., chemotherapy).

CLASSIFICATION OF FUNGICIDES BASED ON CHEMICAL NATURE :

Many fungicides have been developed for purpose of managing crop diseases which may be used as sprays, dusts, paints, pastes, fumigants, etc. The discovery of Bordeaux mixture in 1882 by Professor Millardet, University of Bordeaux, France led to the development of fungicides. Major group of fungicides used include salts of toxic metals and organic acids, organic compounds of sulphur and mercury, quinones and heterocyclic nitrogenous compounds. Copper, mercury, zinc, tin and nickel are some of the metals used as base for inorganic and organic fungicides. The non metal substances include, sulphur, chlorine, phosphorous etc. The fungicides have been classified based on their chemical nature as follows:

COPPER FUNGICIDES: Copper fungicides can be classified as preparatory and proprietory copper compounds.

PREPARATORY COPPER FUNGICIDES

Common name	Chemical composition	Diseases managed
1. Bordeaux mixture	It is prepared by suspending 5 Kg of copper sulphate and 5 Kg of lime in 500 liters of water (1%)	Downy mildew of grapes, Coffee rust, Tikka leaf spot of groundnut, citrus canker, citrus scab, etc.
2.Bordeaux paste	It is prepared by mixing 1 Kg of copper sulphate and 1 Kg of lime in 10 liters of water	It is a wound dressing fungicide and an be applied to the pruned parts of the host plants such as fruit crops and ornamentals. Ex: Citrus gummosis, Stem bleeding of coconut, Bud rot of coconut, etc.
3.Burgundy mixture	Sodium carbonate is used in place of lime. It is prepared	Downy mildew of grapes, Coffee rust, Tikka leaf spot of groundnut, citrus

	by mixing 1 Kg of copper sulphate and 1 Kg of sodium carbonate in 100 liters of water	canker, citrus scab
4.Cheshunt compound	It is a compound prepared by mixing 2 parts of copper sulphate and 11 parts of ammonium carbonate	It is used for soil drenching only. Sclerotial wilt diseases of chilli, tomato and groundnut. Fusarial wilt diseases. Damping-off diseases of solanaceous crops.
5.Chaubattia paste	It is a compound prepared by mixing 800g of copper sulphate and 800g of red lead in 1 liter of lanolin or linseed oil	Pink disease of citrus, stem canker and collar rot of apple and pears

Proprietary copper fungicides or Fixed or insoluble copper fungicides: In the fixed or insoluble copper compounds, the copper ion is less soluble than in Bordeaux mixture. So, these are less phytotoxic than Bordeaux mixture but are effective as fungicides.

Common name	Trade name	Dosage	Disease managed
1. Copper oxy chloride	Blitox-50, Blue copper- 50, Cupramar- 50	0.3 to 0.5% for foliar application, 25 to 35 Kg/ha for dusting	Anthracnose of grapevine, Tikka leaf spot of groundnut, Sigatoka leaf spot of banana, citrus canker, black arm of cotton
2. Cuprous oxide	Fungimar and Perenox	0.3% for foliar spray	Anthracnose of grapevine, Tikka leaf spot of groundnut, Sigatoka leaf spot of

			banana, citrus canker, black arm of cotton
3. Copper hydroxide	Kocide	0.3% for foliar spray	Blister blight of tea, False smut of rice, Tikka leaf spot of groundnut

SULPHUR FUNGICIDES

Sulphur is probably the oldest chemical used in plant disease management for the control of powdery mildews and can be classified as inorganic sulphur and organic sulphur. Inorganic sulphur fungicides include lime sulphur and elemental sulphur fungicides. Organic sulphur fungicides, also called as carbamate fungicides, are the derivatives of dithiocarbamic acid.

Common name	Trade name	Dosage	Disease managed
Preparatory sulphur compounds			
1. Lime sulphur	It is prepared by mixing 20 Kg of rock lime and 15 Kg of sulphur in 500 liters of water	10-15 liters in 500 liters of water	Powdery mildew of apple, Apple scab, bean rust
2. Sulphur dust	Kolo dust, Mico- 999	4-5g/Kg seed for ST, 10-30 Kg/ha for dusting on crops, 100 Kg /ha for soil application in tobacco, 500 Kg/ha for furrow application in potato	Common scab of potato, Grain smut of jowar, Powdery mildew of tobacco, chilli, rose, mango, grapes, etc.
3. Wettable sulphur	Sulfex, Thiovit, Cosan	0.2-0.4 % for foliar spray	Powdery mildews of various crops

ORGANIC SULPHUR COMPOUNDS

Organic sulphur compounds are derived from dithiocarbamic acid and are widely used as spray fungicides. In 1931, Tisdale and Williams were the first to describe the fungicidal nature of Dithiocarbamates. Dithiocarbamates can be categorized into two groups, viz., dialkyl dithiocarbamates (ziram, ferbam and thiram) and monoalkyl dithiocarbamates (nabam, zineb, vapam and maneb).

Common name	Trade name	Dosage	Disease managed
Dialkyl Dithiocarbamates			
1. Ziram	Ziride, Hexazir, Milbam, Zerlate	0.15 to 0.25% for foliar spray	Anthracnose of pulses, tomato, beans, tobacco, etc., bean rust
2. Ferbam	Coromet, Ferbam, Fermate, Fermocide, Hexaferb, Karbam Black	0.15 to 0.25% for foliar spray	Fungal pathogens of fruits and vegetables, leaf curl of peaches, apple scab, downy mildew of tobacco
3. Thiram	Arasan, Hexathir, Tersan, Thiram, Thiride	0.15 to 0.2% as foliar spray, 0.2- 0.3% as dry seed treatment, 15- 25Kg/ha as soil application	Soil borne diseases caused by *Pythium, Rhizoctonia solani, Fusarium*, etc. Rust of ornamental crops, Scab on pears and *Botrytis* spp. on lettuce
Monoalkyl dithyiocarbamates			
1. Nabam	Chembam, Dithane D-14, Dithane A-40 and Parzate liquid	0.2% as foliar spray	Used as foliar spray against leaf spot diseases of fruits and vegetables. Also used against soil borne pathogens, *Fusarium, Pythium* and *Phytophthora*

2. Zineb	Dithane Z-78, Hexathana, Lanocol and Parzate	0.1 to 0.3% for foliar application	Chilli die-back and fruit rot, Apple scab, Maize leaf blight, early blight of potato
3.Vapam or Metham sodium	Chem-vape, vapam, vitafume, VPM	1.5 to 2.5 liters per 10 m^2 area	Fungicide with fungicidal, nematicidal and insecticidal properties. Soil fungal pathogens like *Fusarium, Puthium, Sclerotium* and *Rhizoctonia*.
4. Maneb	Dithane M22, Manzate and MEB. Mancozeb (78% Maneb + 2% zinc ion): Dithane M 45, Indofil M 45	0.2% to 0.3% as foliar application	Early and late blight of potato and tomato, rust diseases of field and fruit crops

HETROCYCLIC NITROGENOUS COMPOUNDS

The group of heterogeneous fungicides includes some of the best fungicides like captan, folpet, captafol, vinclozoline and Iprodione. Captan, folpet and captafol belong to dicarboximides and are known as pthalamide fungicides. The new members of dicarboximide group are Iprodione, vinclozolin, etc.

Common name	Trade name	Dosage	Disease managed
1.Captan (Kittleson's killer)	Captan 50W, Captan 75 W, Esso fungicide, Orthocide 406, Hexacap, Vancide 89	0.2 to 0.3% for dry seed treatment, 0.2 to 0.3% for foliar spray, 25 to 30 Kg/ha for furrow	Onion smut, Chilli die-back and fruit rot, Damping off of beans, chilli and tomato, seed rots and seedling blights

		application	of maize
2. Folpet	Phaltan	0.1 to 0.2% for spraying	Apple scab, tobacco brown spot, rose black spot
3.Captafol	Difosan, Difolaton, Sanspor, Foltaf	0.15 to 0.2% for spraying, 0.25% for seed treatment, 0.15% for soil drenching	Sorghum anthracnose, cotton seedling diseases, seed rot and seedling diseases of rice, downy mildew of crucifers, apple scab
4. Iprodione	Rovral, Glycophene	0.1 to 0.2% for foliar application	Diseases caused by Botrytis, *Monilinia, Alternaria, Sclerotinia, Helminthosporium* and *Rhizoctonia*
5. Vinclozolin	Ornalin, Ronilan, Vorlan	0.1 to 0.2% for foliar application	Effective against sclerotia forming fungi like *Botrytis, Monilinia* and *Sclerotinia*

MISCELLANEOUS FUNGICIDES

Common name	Trade name	Dosage	Disease managed
1.Chlorothalonil	Bravo, Daconil, Kavach, Thermil, Exotherm, Safegaurd	0.2 to 0.3% for foliar application	A broad spectrum contact fungicide often used in greenhouses for control of *Botrytis* on ornamentals and for several molds and blights of tomato. Also used for the control of sigatoka leaf spot of

			banana, onion purple blotch, tikka leaf spot and rust of groundnut
2. Dinocap	Karathane, Arathane, Capryl, Mildex, Mildont and crotothane	0.1 to 0.2% for spraying	It is a good acaricide and contact fungicide and it controls powdery mildews of fruits and ornamentals effectively. This can be safely used on sulphur sensitive crops like cucurbits and apple varieties for control of powdery mildews
3. Dodine	Cyprex, Melprex, Guanidol and Syllit	0.075% for spraying	Apple scab, black spot of roses and cherry leaf spot

SYSTEMIC FUNGICIDES

The systemic fungicides were first introduced by Von Schelming and Marshall Kulka in 1966. The discovery of Oxathiin fungicides was soon followed by confirmation of systemic activity of pyrimidines and benzimidazoles. A systemic fungicide is capable of managing a pathogen remote from the point of application. On the basis of chemical nature these fungicides are classified as follows

Common name	Trade name	Dosage	Disease managed
ACYLALANINES			
1. Metalaxyl	Ridomil 25 % WP, Apron 35 SD, Subdue, Ridomil	3-6 g/Kg seed for seed treatment, 1 to 1.5 Kg a.i/ha for	It is highly effective against *Pythium, Phytophthora* and many

	MZ-72WP	soil application, 0.1 to 0.2% for foliar spray	downy mildew fungi
2. Benalaxyl	Galben 25% WP and 5% G	0.1 to 0.2% for foliar spray, 1 to 1.5 Kg a.i/ha for soil application	Blue mold of tobacco, late blight of potato and tomato, downy mildew of grapevine
AROMATIC HYDROCARBONS			
1. Chloroneb	Demosan	0.2% for seed treatment	Seedling diseases of cotton , peanut, peas and cucurbits caused by species of *Pythium, Phytophthora, Rhizoctonia* and *Sclerotium*
BENZIMIDAZOLES			
1. Carbendazim	Bavistin 50WP, MBC, Derosol 60WP, Agrozim, Zoom	0.1% for foliar spray, 0.1% for soil drench, 0.25% for ST, 500-1000ppm for post-harvest dip of fruits	Effectively controls anthracnose, powdery mildews and rusts caused by various fungi. It is also used as a soil drench against wilt diseases and for post harvest treatment of fruit
2. Benomyl	Benlate 50WP	0.1 to 0.2% for ST, 50-60g/100 L for foliar spray, 50-200ppm for soil drenching, 12-45 Kg a.i/ha for soil broadcast, 100- 500	Effective against powdery mildews of cucurbits, cereals and legumes. It is highly effective against diseases caused by the species of *Rhizoctonia,*

		ppm for post harvest fruit dip	*Theilaviopsis* and *Cephalosporium*. Benomyl has no effect against Oomycetes and some dark coloured fungi such as *Alternaria* and *Helminthosporium*
3.Thiabendazole	Mertect 60WP, Mycozol, Arbotect, Tecto and Storite	0.2 to 0.3% for spraying, 1000 ppm for fruit dip	Blue and green molds of citrus, loose smut of wheat, Tikka leaf spot of groundnut
ALIPHATICS			
1. Prothiocarb	Previcur, Dynone	5.6 Kg a.i/ha for soil application	Highly active against soil borne Oomycetes like *Pythium* and *Phytophthora*
2. Propamocarb	Previcur-N, Dynone-N, Prevex, Benol	3.4 and 4.8 Kg a.i/ha for soil application	Effective against against soil borne Oomycetes like *Pythium* and *Phytophthora*
OXATHINS or CARBOXIMIDES			
1. Carboxin	Vitavax 75WP, Vitaflow	0.15 to 0.2% for seed treatment, 0.5% for spraying	Highly effective against smut diseases. Commonly used for the control of loose smut of wheat, onion smut, grain smut of sorghum. As a soil drench it is used for the control of diseases caused by *Rhizoctonia solani* and *Macrophomina*

			phaseolina.
2. Oxycarboxin	Plantavax 75 WP, Plantavax 20EC, Plantavax 5% liquid	0.1 to 0.2% for foliar spray, 0.2 to 0.5% for ST	Highly effective against rust diseases. Commonly used for the control of rusts of wheat, sorghum, safflower, legumes, etc.
IMIDAZOLES			
1. Imazalil	Fungaflor, Bromazil and Nuzone	0.1 % as post harvest dip	Blue and green molds of citrus
2. Fanapanil	Sistane 25 EC	0.05% foliar spray	Spot blotch of barley, loose and covered smut of barley
MORPHOLINES			
1. Tridemorph	Calixin 75EC, Bardew, Beacon	0.1% for foliar spray	Powdery mildew of cereals, vegetables and ornamentals. Rusts of pulses, groundnut and coffee, Sigatoka leaf spot of banana, pink disease of rubber, Ganoderma root rot & wilt
ORGANOPHOSPHATES			
1. Iprobenphos	Kitazin 48EC, Kitazin 17G, Kitazin 2% D	30-45 Kg of granules/ha, 1 to 1.5 liters of 48% EC in 1000 ml of water for foliar spray	Fungicide with insecticidal properties. Highly specific against rice blast, stem rot and sheath blight of rice
2. Ediphenphos	Hinosan 30 and 50% EC, Hinosan 2%D	400 to 500 ppm for spraying, 30 to 40	Highly specific against rice blast, stem rot and

		Kg/ha	sheath blight of rice
ALKYL PHOSPHONATES			
1. Fosetyl-Al or Aluminium Tris	Aliette 80WP	0.15% for foliar spray, 0.2% for soil drench	Ambimobile fungicide. Specific against Oomycetes fungi
PYRIMIDINES			
1. Fenarimol	Rubigan 50% WP, 12%EC	2g/Kg seed as ST, 20 to 40 ml/100 liters of water for spraying	Powdery mildew of cucurbits, apple, mango, roses, grapes and ornamental crops
THIOPHANATES			
1. Thiophanate	Topsin 50WP, Cercobin 50WP	0.1 to 0.2% for spraying	Powdery mildew of cuurbits and apple, club root of crucifers, rice blast
2.Thiophanate methyl	Topsin M 70WP, Cercobin M 70WP	0.1% for spraying	Blast and sheath blight of rice, sigatoka leaf spot of banana, powdery mildew of beans, chilli, peas and cucurbits
TRIAZOLES			
1. Triadimefon	Bayleton, Amiral	0.1 to 0.2% for spraying, 0.1% for seed treatment	Highly effective against powdery mildews and rusts of several crops. Effective against diseases caused by species of *Erysiphe, Sphaerotheca, Puccinia, Uromyces, Phakopsora, Hemileia* and *Gymnosporangium*
2. Tricyclazole	Beam 75WP, Baan	2g/Kg seed for ST,	Highly effective against

	75WP, Trooper 75WP	0.06% for spraying	blast of rice
3. Bitertanol	Baycor and Sibutol	0.05 to 0.1% for foliar spray	Powdery mildews and rusts of various crops, apple scab, *Monilinia* on fruit crops, late leaf spot of groundnut and sigatoka leaf spot of banana
4.Hexaconazole	Contaf 5%EC, Anvil	0.2% for spraying	Sheath blight of rice, powdery mildew and rust of apple, rust and tikka leaf spot of groundnut
5.Propiconazole	Tilt 25% EC, Desmel	0.1% for foliar application	Sheath blight of rice, Sigatoka leaf spot of banana, brown rust of wheat
6. Myclobutanil	Systhane 10WP	0.1 to 0.2% for spraying	Apple scab, cedar apple rust and powdery mildew of apple
STROBILURINS			
1. Azoxystrobin	Amistar, Quadris	0.1% for spraying	Broad spectrum fungicide
2.Kresoxim methyl	Ergon, Discus, Stroby	0.1% for spraying	Commonly used for control of ornamental diseases

Differentiate between Systemic fungicides and Non-systemic fungicides:

	Systemic fungicides	**Non-systemic fungicides**
1	Any compound capable of being translocated after penetrating the plant &	Do not get absorbed and translocated into the plant.

	reaches to the point of infection.	
2	Movement of fungicide may be **apoplastic** (Upward movement) or **symplastic** (Downward movement)	No movement of fungicides (contact in nature)
3	Monosite specification	Multisite specification
4	Long persistence	short persistence
5	More chances of development of fungicidal resistance	Minimum chances of development of fungicidal resistance
6	Used in low concentration i.e. 0.025 to 0.05%	Used in higher concentration i.e. 0.2 to 0.3%
7	Eg. Carbendazim, Hexaconazole, Thiophanate methyl and all Traizole fungicides etc.	Eg. Thiram, Captan, Mancozeb, MEMC, Copper oxychloride etc.

CLASSIFICATION OF FUNGICIDES BASED ON METHOD OF APPLICATION

The fungicides can also be classified based on the nature of their use in managing the diseases.

1. Seed protectants: Ex. Captan, thiram, carbendazim, carboxin etc.
2. Soil fungicides (preplant): Ex. Bordeaux mixture, copper oxy chloride, Chloropicrin, Formaldehyde, Vapam, etc.
3. Soil fungicides: Ex. Bordeaux mixture, copper oxy chloride, Captan, PCNB, thiram etc.
4. Foliage and blossom: Ex. Capton, ferbam, zineb, mancozeb, chlorothalonil etc.
5. Fruit protectants: Eg. Captan, maneb, carbendazim, mancozeb etc.
6. Eradicants: EX. Lime sulphur
7. Tree wound dressers: Ex. Boreaux paste, chaubattia paste, etc.
8. General purpose sprays and dust formulations.

Differentiate between Spraying of fungicides and Dusting of fungicides:

	Spraying of fungicides	**Dusting of fungicides**
1	Formulation used in liquid form either wettable powder (WP) or EC formulation	Formulation is used in powder form
2	Can be used in high wind velocity	Not used in high wind velocity
3	More area covered	Less area covered

4	Material needs preparation	Materials is ready to use
5	More effective	Less effective
6	Particle size biger	Particle size smaller
7	Sprayer is used	Duster is used
8	Water is required	Water not required
9	Eg. Mancozeb, Carbendazim, Thiophanate methyl, Chlorothalonil, Traizole fungicides etc.	Eg. Sulphur dust

HOST PLANT RESISTANCE (IMMUNIZATION)

Disease resistance: It is the ability of a plant to overcome completely or in some degree the effect of a pathogen or damaging factor.

Susceptibility: The inability of a plant to resist the effect of a pathogen or other damaging factor.

Advantages of resistant varieties:

1. Resistant varieties can be the most simple, practical, effective and economical method of plant disease management.
2. They not only ensure protection against plant diseases but also save the time, energy and money spent on other measures of control
3. Resistant varieties, if evolved can be the only practical method of control of diseases such as wilts, viral diseases, rusts, etc.
4. They are non-toxic to human beings, animals and wild life and do not pollute the environment
5. They are effective only against the target organisms, whereas, chemical methods are not only effective against target organisms but also effective against non-target organisms.
6. The resistance gene, once introduced, is inherited and therefore permanent at no extra cost.

Disadvantages:

1. Breeding of resistant varieties is a slow and expensive process

2. Resistance of the cultivar may be broken down with the evolution of the pathogen

Types of resistance:

1. **Vertical resistance**: When a variety is more resistant to some races of the pathogen than others, the resistance is called vertical resistance (race-specific resistance, qualitative resistance, discriminatory resistance). Vertical resistance is usually governed by single gene and is unstable.

2. **Horizontal resistance**: When the resistance is uniformly spread against all the races of a pathogen, then it is called horizontal/generalized/non-specific/field/qualitative resistance. Horizontal resistance is usually governed by several genes and is more stable.

3. **Monogenic resistance**: When the defense mechanism is controlled by a **single gene pair**, it is called monogenic resistance.

4. **Oligogenic resistance**: when the defense mechanism is governed by a **few gene pairs**, it is called oligogenic resistance.

5. **Polygenic resistance**: When the defense mechanism is controlled by **many genes** or more groups of supplementary genes, it is called polygenic resistance.

Cross protection: The phenomenon in which plant tissues infected with mild strain of a virus are protected from infection by other severe strains of the same virus. This strategy is used in the management of severe strains of *Citrus Tristeza* ***virus***

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INTEGRATED PLANT DISEASE MANAGEMENT (IPDM)

IPDM involve management systems which utilize compatible combinations of all the available techniques to keep the pathogen population below the economic threshold level (ETL) which would not result in economically unacceptable damage to the crop. IPDM is based on five principles of plant disease management and integrates multidisciplinary approaches for the management of plant diseases.

Main components of IPDM:

1. Cultural practices
2. Regulatory measures (quarantine)
3. Chemical methods
4. Biological methods
5. Physical methods
6. Genetic engineering

Main strategies of IPDM:

1. Need based application of pesticides
2. Encouragement and enhancement of biocontrol agents
3. Use of resistant or tolerant cultivars of plants
4. Modification of cultural practices
5. Use of any other strategies that interrupts host-pathogen interactions

Advantages of IPDM:

1. Avoids chemical pollution of soil, water, air and food products
2. Avoids development of resistance in the plant pathogens against fungicides
3. It is an eco-friendly strategy for management of plant diseases
4. It is an economically feasible approach
5. It is a multipronged strategy for efficient management of plant diseases

Therefore, IPDM utilizes all suitable strategies in a compatible manner to reduce and maintain pathogen populations at levels below those causing economic losses.

Rice diseases and IPDM:

Fungal diseases

1. Blast: Foliar disease and the pathogen survives on collateral hosts
2. Brown spot of rice – Seed borne and a foliar disease
3. Sheath rot, sheath blight, foot rot and stem rot – Soil borne diseases
4. False smut – seed borne disease

Bacterial diseases: Bacterial leaf blight and bacterial leaf streak – Seed borne and survives on collateral hosts and weeds

Viral or Phytoplasmal diseases – Rice tungro virus, Rice yellow dwarf – Survives on weeds and dissemination is by insect vectors

IPDM strategy in rice:

1. Selection of healthy seed
2. Selection of resistant cultivars
3. Removal and destruction of collateral hosts
4. Balanced fertilization
5. Rouging of diseased plants
6. Seed treatment with carbendazim or tricyclazole at 2g/Kg seed
7. Need based foliar application of carbendazim@0.1% or Tricyclazole@0.06% for the management of blast.
8. Need based foliar application of validamycin for the management of sheath blight and sheath rot.
9. Soil application of carbofuran granules or foliar spray of any systemic fungicide is followed to manage insect vectors, thereby decreasing the spread of viral diseases.

Sugarcane diseases and IPDM

1. Red rot – sett borne disease which spreads through irrigation water
2. Whip smut - sett borne and disseminate through wind borne sporidia
3. Pine apple disease, sett rot – Sett borne disease
4. Grassy shoot – Vector borne Phytoplasmal disease
5. Ratoon stunting – Sett borne (*Clavibacter xyli*)

6. Sugarcane mosaic – Survives on weeds and disseminated by insect vectors

IPDM in sugarcane:

1. Collection and destruction of infected crop debris
2. Hot water treatment of setts (52^0C for 30 min)
3. Hot air treatment of setts (54^0C for 2-3 hrs)
4. Balanced irrigation and fertilization
5. Avoid selection of seed material from Ratoon crop
6. Need based spray of systemic insecticides to minimize the spread of viral and Phytoplasmal diseases
7. Selection of disease resistant or tolerant cultivars

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APPLICATION OF BIOTECHNOLOGY IN PLANT DISEASE MANAGEMENT

Biotechnology: It is defined as genetic modification and manipulation of living organisms through the novel technologies such as tissue culture and genetic engineering resulting in production of improved or new organisms that can be used in variety of ways.

Application of biotechnology in plant disease management:

1. Diagnosis of plant diseases

a) Diagnostic kits helps in identification of plant diseases, viz., bacterial canker of tomato, soybean root rot, viral diseases of potato, etc., at an early stage of development and helps in devising suitable management practices.

b) Polymerase Chain Reaction (PCR): Detection of very small amount of pathogen in a sample by amplifying the pathogen sequences to a detectable level. PCR is especially used in plant quarantine.

2. Strain improvement of biocontrol agents: It has the following advantages

a) Expanding the range of target species

b) Restricting the range of non-target species

c) To improve the survival ability or rhizosphere competence

d) Expanding the bio-agents environmental range beyond its congenial habitat

e) Development of fungicide tolerant strains

3. Transgenics for plant disease management

a) Coat protein mediated resistance for papaya ring spot virus in Hawaii islands

b) Cloning of resistance genes, viz., *Xa 21*, bacterial blight resistance gene isolated from frican rice, *Oryza longistaminata* was introduced into cultivable rice, *Oryza sativa*

4. Determination of biochemical nature and the signals involved in plants reaction to pathogen invasion and disease development. Ex: Host-pathogen interaction has been studied in rice blast disease incited by *Magnaporthe grisea*.

5. Manipulation of resistance of host by expression of PR-proteins, antifungal peptides, etc. Ex: Expression of multiple PR-proteins (Chitinases and β-1,3 glucanases) in rice enhanced disease resistance to rice sheath blight pathogen*, Rhizoctonia solani.*

PLANT TISSUE CULTURE: *In vitro* culture of plant cells, tissues as well as organs. **Totipotency** is the ability of a plant cell to perform all the functions of development which are characteristic of zygote, i.e., its ability to develop in to a complete plant.

IMPORTANT TISSUE CULTURE TECHNIQUES OF IMPORTANCE TO PLANT PATHOLOGY:

1. Meristem tip culture
2. Protoplast culture

A. Production of virus free plants through plant tissue culture:

Meristem tip culture: Cultivation of axillary or apical meristems, particularly of shoot apical meristem, is known as meristem culture.

1. Explant: the explant must consist of the meristematic dome of cells together with atleast one leaf primordial. Meristem tips varying in size from 0.1 to 2.0 mm in diameter (usually 0.3-1.5 mm) can be used for meristem tip culture. The infected parent plant or organ of the plant from which explant is excised is generally subjected to thermotherapy in a temperature controlled cabinet at 30^0C to 40^0C for six to twelve weeks to inactivate the virus.

2. Culture initiation on suitable medium: In general Murashige and Skoog medium has been found satisfactory for most plant species. But for some species, a much lower salt concentration may be adequate or even necessary since the high salt concentration of MS medium may be deleterious or even toxic. Culture initiation consists of surface sterilization of explants and establishing them *in vitro* on culture medium. Culture initiation often involves anti-metabolite chemicals such as ribavirin (virazole) in the tissue culture medium.

3. Shoot multiplication: After 2-3 weeks, the cultures are transferred to a shoot multiplication medium designed to promote axillary branching. This medium generally

contains cytokinins, either alone or in combination with an auxin. Higher concentration of cytokinins induces adventitious buds. During culture initiation and shoot multiplication phases, the cultures are generally kept at 25^0C.

4. Rooting of shoots: In general, the rooting medium has low salt (1/2 or even ¼ salts of S medium) and reduced sugar levels. But in most species, 0.1-1 mg/l Naphthalene Acetic Acid (NAA) or Indole-3-Butyric acid (IBA) is required for rooting. Rooting takes about 10-15 days depending on species.

5. Transfer of plantlets to soil: Rooted shoots are removed from the medium, agar sticking to roots is washed with tap water, and they are transplanted into plastic cups containing a suitable potting mix. Plants are kept in high (>90%) humidity and initially low light intensities. The humidity is generally decreased to the ambient level after about 7-15 days, and the light intensity is increased. The plants are finally exposed to greenhouse conditions (**hardening**).

6. Indexing, clone selection and stock maintenance: Virus indexing is done several times during first year and the virus free plantlet is used as a nuclear stock material for commercial multiplication. Virus indexing is generally made by Enzyme Linked Immuno-Sorbent Assay (ELISA) or Immuno Sorbent Electron Microscopy (ISEM).

B. Protoplast culture: Fungal protoplasts are important tools in physiological and genetic research. Interspecific, intraspecific and intrageneric hybridization could be done by this technique for strain improvement of biocontrol agents to enhance the biocontrol potential for the management of pathogenic fungi. Isolation and self-fusion of protoplasts were achieved in *Trichoderma harzianum* and *T. viride*.

Steps in protoplast fusion:

1. Isolation of protoplasts is achieved by treating cells with a suitable mixture of cell wall degrading enzymes.
2. The pH of enzyme solution is adjusted between 4.7 and 6.0 and temperature is kept around 25-30^0C. The osmotic concentration of enzyme mixture and of subsequent media is elevated to stabilize the protoplasts and to prevent them from bursting. Usually, 50-100 m mol/l CaCl2 is added to the osmoticum as it improves plasma membrane stability.

3. The protoplasts of different strains are treated with 28-50% **Poly Ethylene Glycol** (**fusogen**) for 15-30 min followed by gradual washing of the protoplasts to remove PEG. The washing medium may be alkaline and contain high calcium ion concentration (50 m mol/l). Protoplast fusion occurs during washing step.
4. Selection of hybrid cells and culturing on suitable medium.

Gene cloning/ Recombinant DNA technology / Genetic engineering:
Integration of specific fragment of foreign DNA into a cell through a suitable vector in such a way that the inserted DNA replicate independently and transferred to progenies as a result of cell division.

Recombinant DNA molecule is a vector into which the desired DNA fragment has been inserted to enable its cloning in an appropriate host. Recombinant DNA molecule is produced by joining together two or more DNA segments usually originated from different organisms.

Steps in gene cloning:
1. Identification and isolation of the desired gene or DNA fragment to be cloned (Restriction digestion and electrophoresis)
2. Insertion of the isolated gene in a suitable vector (ligation)
3. Introduction of this vector into a suitable organism or cell called host (transformation)
4. Selection of transformed host cells (selectable markers)
5. Multiplication / integration followed by expression of the introduced gene in the host

Enzymes involved: Restriction endonucleases, DNA ligases, DNA polymerases, RNA polymerases and reverse transcriptases.

Vectors used in gene cloning: A vector is a DNA molecule that has the ability to replicate in an appropriate host cell, and into which the DNA fragment to tbe cloned (called DNA insert) is integrated for cloning. Ex: Tumor inducing (Ti) plasmid of *Agrobacterium tumefaciens*, pBR322, Bacteriophages, cosmid vectors (derived from phage λ).

Ti plasmid of *Agrobacterium tumefaciens*:

- Ti plasmid is a large conjugative plasmid or megaplasmid of about 200 kb.
- Ti plasmid has a T-DNA region (15-24 kb) which is bounded by a pair of 24 bp repeats. T-DNA carries genes for **auxin, cytokinins** and **opine** synthesis which are responsible for tumor formation (tumorigenesis).
- Transfer of T-DNA depends on 35 kb **virulence (vir) region** of the Ti plasmid. This region has 7 operons ranging from *vir* A to *vir* H (*vir* A, *vir* B, *vir* C, *vir* D, *vir* E, *vir* G and *vir* H). The protein products of these genes respond to phenolics to generate a copy of T-DNA and mediate its transfer into the cell.
- The T-DNA when transferred from the *Agrobacterium* to the plant cell integrates with the chromosome, and the plant cells which are affected begin to synthesize opines, auxins and cytokinins.

Ori (origin of replication)

- Opines are tumor specific compounds formed by the condensation of aminoacid, ketoacid and sugar. The **opines** (**octopine**, **nopaline**, succinamopine or leucinopine) can be metabolized only by Agrobacteria
- The IAA (auxin) and Isopentenyl-AMP (cytokinins) are phytohormones which cause the proliferation of plant cells and induction of the gall.
- Plant wound exudates contain phenolics, which attract *Agrobacterium* and induce *vir* genes. The strong *vir* gene inducers are syringic acid, ferulic acid, **acetosyringone** and sinapinic acid. Only Agrobacterium with Ti plasmid are attracted by these compounds.
- The exogenous DNA is inserted into the T-DNA region of the Ti plasmid by homologous recombination using an intermediate vector system or directly using
- binary vectors.

DEVELOPMENT OF DISEASE RESISTANT TRANSGENIC PLANTS THROUGH TI PLASMID MEDIATED GENE TRANSFER:

1. The appropriate gene construct is inserted within the T-DNA of a disarmed Ti plasmid; either a co-integrate or binary vector is used. The recombinant vector is placed in

Agrobacterium, which is co-cultured with the plant cells or tissues to be transformed for about 2 days.

2. In case of many plant species, small (a few mm diameter) leaf discs are excised from surface sterilized leaves and used for co-cultivation. In general the transgene construct involves a selectable reporter gene (Bacterial ***neo*** gene), the presence of which confers resistance to kanamycin.

3. During the leaf disc-Agrobacterium co-culture, **acetosyringone** released by plant cells induces the *vir* genes which bring about the transfer of recombinant T-DNA into many of the plant cells. The **T-DNA** would become integrated into the plant genome, and the transgene would be expressed. As a result, the transformed plant cells would become resistant to kanamycin.

4. After 2 days, the leaf discs are transferred onto a regeneration medium containing appropriate concentrations of kanamycin and carbenicillin. **Kanamycin** allows only transformed plant cells to divide and regenerate shoots in about 3-4 weeks, while **carbenicilin** kills *Agrobacterium* cells. The shoots are separated, rooted and finally transferred into soil.

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GENERAL INTRODUCTION

What are the **nematodes**?

Nematodes are belonging to **animal kingdom**, they are **triploblastic**, **unsegmented**, **bilateral symmetrical**, **pseudocoelomate** and having well developed **reproductive, nervous, excretory** and **digestive system** where as the **circulatory** and **respiratory systems** are absent but govern by the pseudo coelomic fluid**.**

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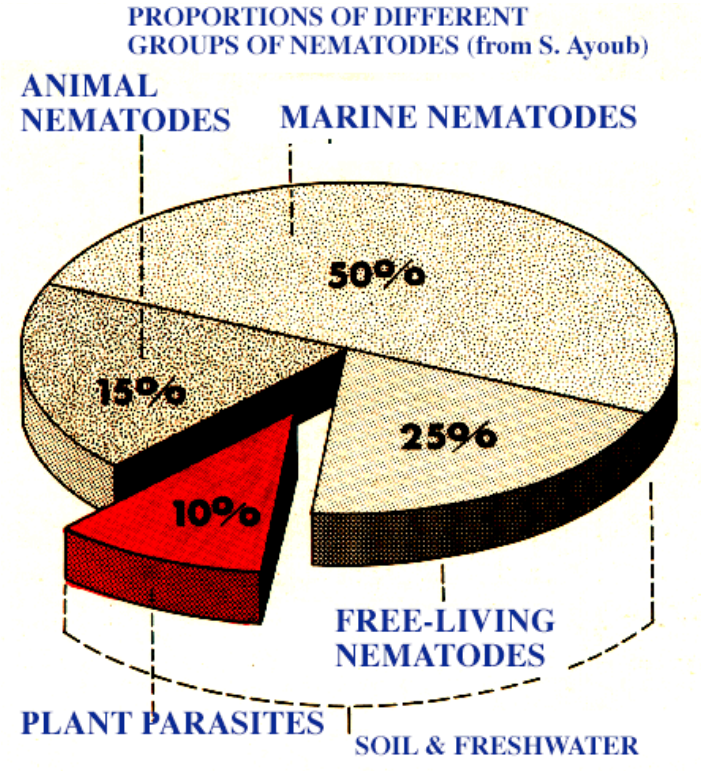
Plant Nematology: Nematology is a science deals with the study of morphology, taxonomy, classification, biology, symptomatology and management of {plant pathogenic} nematode (PPN).
The word nematode is made up of two Greek words, *Nema means* thread like and *eidos* means form.

The words Nematodes is derived from Greek words **'Nema + oides'** meaning 'Thread + form' (**thread like organism**) therefore, they also called threadworms. They are also known as roundworms because nematode body tubular is shape. The movement (serpentine) of nematodes like eel (marine fish), so also called them **eelworm in U.K. and Nema in U.S.A. Roundworms by Zoologist**

Nematodes are a diverse group of organisms, which are found in many different environments. Approximately **50%** of known nematode species are marine, **25%** are free-living species found in soil or freshwater, **15%** are parasites of animals, and **10%** of known nematode species are parasites of plants (see figure at left).



The study of nematodes has traditionally been viewed as three separate disciplines:

(1) **Helminthology** dealing with the study of nematodes and other worms parasitic in vertebrates (mainly those of importance to human and veterinary medicine).

(2) **Marine nematology** dealing with the study of marine nematode.

(3) **Nematology** dealing with the study of nematode parasites of plants and invertebrates and "free-living" nematodes (those living in soil or water and feeding on bacteria, fungi, decaying organic matter, other nematodes, microscopic invertebrates, etc.).

Nematology is an important branch of biological science, which deals with a complex, diverse group of round worms known as Nematodes that occur worldwide in essentially all environments. Nematodes are also known as eelworms in Europe, nemas in the United States and round worms by zoologists. Many species are important parasites of plants and animals, whereas others are beneficial to agriculture and the environment.

On the basis of nematode habitats and diversity, they can be grouped into 2 categories.

Parasitic	**Free living**
Human: *Ascaris, Enterobius, Wucheraria, Onchocerca, Ancylostoma, Dracunculus*	**Marine:***Chaetostoma, Chromodora, Deontostoma*
Other vertebrates : *Trichuris, Dioctophyma,*	**Freshwater and Soil** *Plectus,Dorylaimus, Mononchus*
Invertebrates : *Mermis*(Grasshoppers) *Romanomermisculicivorax*(Mosquitoes) EPNs (*Steinernema*and*Heterorhabditis*)	**Bacterial feeder soil nematodes:** *Caenorhabditis elegans, C. briggsae*
Plants: *Anguina, Meloidogyne, Heterodera etc*	*Panagrellusredivivus*(biological indicators)

General characteristics of the Plant Parasitic Nematodes

• Nearly microscopic

• Unsegmented

- Posses a **stylet**, Colourless

• Wormlike in appearance (pseudoworm)

• Highly developed pseudo-coelomates

• Bilaterally symmetrical invertebrates; with a body cavity or vacuoles (pseudocoelom?)

- Triptoblstic, tapering at both end, cylenderical

• Only plant parasites (other than Protozoa) belonging to the **animal kingdom**

- Obligate parasite

• The basic difference between PPN and other are that the PPN have well developed Stylet for the penetration of the host and feeding sap of the plant.

• Parasites of animals (humans, vertebrates, invertebrates) and plants.

They Posses

- Stylet or spear bearing

• a complete digestive system (mouth, intestine, anus)

• A tri-radiate pharynx

• A circumpharyngeal nerve ring and a well developed nervous system

• Four sets of longitudinal muscles separated by epidermal (hypodermal?) cords

• One or two tubular gonads, which open through vulva in female and into the rectum in male

Movement

 They are moved by Sinusoidal movements facilitated by contraction of the longitudinal muscles against the elastic cuticle made rigid by the high hydrostatic pressure of the body fluids - a hydrostatic skeleton .

Significance

• They can cause significant plant damage ranging from negligible injury to total destruction of plant material

• Majority of these parasites feed on underground parts of plants

They lack

• Specialized **respiratory** and **circulatory** systems are absent.

Economic Importance of Plant Parasitic Nematodes

- ✓ Nematodes are ubiquitous, present in all moist to watery ecological niches in diverse situations like cold oceans, hot springs, mountain peaks, soil ecosystem, plants as well as animals.
- ✓ Phytoparasitic forms comprise about 10% of all the forms of nematodes that exist in various habitats and have different feeding behaviours.
- ✓ Phyto-nematodes parasitize all types of plants - from algae to plants and trees and damage to world food crops as well as tree crops, turf and ornamentals.
- ✓ There are about 6000 known species of phyto-parasitic nematodes belonging to 197 genera.
- ✓ Being obligate parasites, they must draw their nutrition from plant hosts and infect the plants.
- ✓ A handful of soil from around the roots of any plant would yield hundreds of plant parasitic nematodes belonging to at least 4-5 genera.
- ✓ One gram of coconut root may contain about 4000 eggs, juveniles and adults of burrowing nematode or about 5000 individuals of coconut ring nematode.
- ✓ Single acre of arable land contain 3×10^9 nematodes
- ✓ Marine beach sand contain 15×10^8 nematodes/acre
- ✓ Single wheat gall: Max. 1,00,000 nematodes
- ✓ Besides this direct damage, they also associate with bacteria, fungi and viruses to cause complex diseases.
- ✓ In the world **12.3 %** yield losses was observed due to different nematodes, in developing countries it was **14.6 %,** whereas in developed countries it was **8.8 %.**
- ✓ The losses due to cereal cyst nematode cause M**olya disease of wheat and barley** was 32 million rupees in Rajasthan, Punjab, Hariyana and Himachal Pradesh, whereas 25 million rupees losses was reported only in Rajasthan state alone.
- ✓ Seed gall nematode, *Anguina tritici*(causing 'Ear cockle of wheat when alone and 'Tundu disease' when in combination with bacterium) was estimated to be Rs. 70 million in wheat only in North India.
- ✓ *Pratylenchus coffeae*in an area of about 1,000 ha in Karnataka was estimated to be Rs. 25 million is assessed in coffee due to.
- ✓ **Beneficial nematodes**
- ✓ In addition to the harmful plant parasitic forms, there are groups of nematodes which are beneficial as they parasitize the harmful insects and thus can be exploited as biological control agents against these pests.The entomopathogenic nematodes ***Steinernema*** and ***Heterorhabditis***spp.and slug parasitic nematode *Phasmarhabditis hermaphrodita*in a mutual association with bacteria have proved to be successful biocontrol agents and are now being commercially produced in many countries.

NO.	CROP	NEMATODE	YIELD LOSS, %
1	Bidi Tobacco	Root-knot nematodes.*M. incognita*&*M. javanica*	51.0,50.0
2	Brinjal	Root-knot nematodes.*M. incognita*	26.2--50.0
3	Castor	Reniform nematode, *Rotylenchulusreniformis*	33.0
4	Chickpea	Root-knot nematodes,*Meloidogyne spp*.	84.0
5	Chilly	Root-knot nematodes.*M. incognita*	28.0
6	Cotton	Reniform nematode, *Rotylenchulusreniformis*	8.0-15.0
7	Cowpea	Root-knot nematodes,*Meloidogyne spp*.	30.3-71.3
8	Fennel	Root-knot nematodes,*Meloidogyne javanica patho.-2*	28.1
9	Ginger	Root-knot nematodes,*Meloidogyne spp.*	51.2
10	Groundnut	Root-knot nematodes,*Meloidogyne javanica* patho.-2	35.0, 59.4
11	Mung bean	Root-knot nematodes, *Meloidogyne spp.* Reniform nematode, *Rotylenchulus reniformis*	7.5-65.0 18.0-21.0
12	Mushroom	*Aphelenchoides spp*	40.6-100
13	Pigeon pea	Pigeon pea cyst nematode, (*H.cajani)* Root-knot nematodes,*Meloidogyne* spp.	46.0 25-31
14	Rice	Rice root nematode, (*H. oryzae*) Rice cyst nematode, (*H. oryzicola*)	4.3-53.5 7.9-26.8
15	Sunflower	Root-knot nematodes. *M. incognita*	38-84.0
16	Tomato	Root-knot nematodes. *M. incognita*	28-47
17	Turmeric	Root-knot nematodes. *M. incognita*	31.3
18	Wheat	Root-knot nematodes. *M. incognita*	32.9
19	Citrus	Root-knot nematodes, *Meloidogyne* spp.	14.2
20	Banana	Root-knot nematodes, *Meloidogyne* spp.	19.2

Range of size

• From <100 μm (*Greeffiella minutum* - marine) to >8 m (*Placentonema gigatissima* - parasite of the spermaceti whale).

Fossil evidence

• The fossil record of nematodes is at the least sparse. There are traces of what may be nematodes in Chinese Precambrian layers (>670 Myr), but few are older than 50 million years and none are old enough to give information regarding the origin of specific nematode groups.

Types of nematodes on the basis of feeding habit and motility

– **Ectoparasitic nematodes**

• Migratory Ectoparasite

• Sedentary Ectoparasite

– **Semi endoparasitic nematodes**

– **Endoparasitic nematodes**

• Migratory Endoparasite

• Sedentary Endoparasite

1. Ectoparasitic nematodes: These nematode live freely in the soil and move closely or on the root surface, feed intermittently on the epidermis and root hairs near the root tip. Generally does not pose much problem. Some ectoparasite transmit plant viruses dagger (*Xiphinema* spp.,), Needle (*Longiderous* spp.) and stunt nematode (*Trichodorous* and *Paratrichodorous* spp)

A. Migraotory ectoparasite: These nematodes spend their entire life cycle free in the soil . When the roots are disturbed they detach themselves. Examples: *Criconemoides* spp. *Paratylenchus* spp., and *Trichodorus* sp., etc.

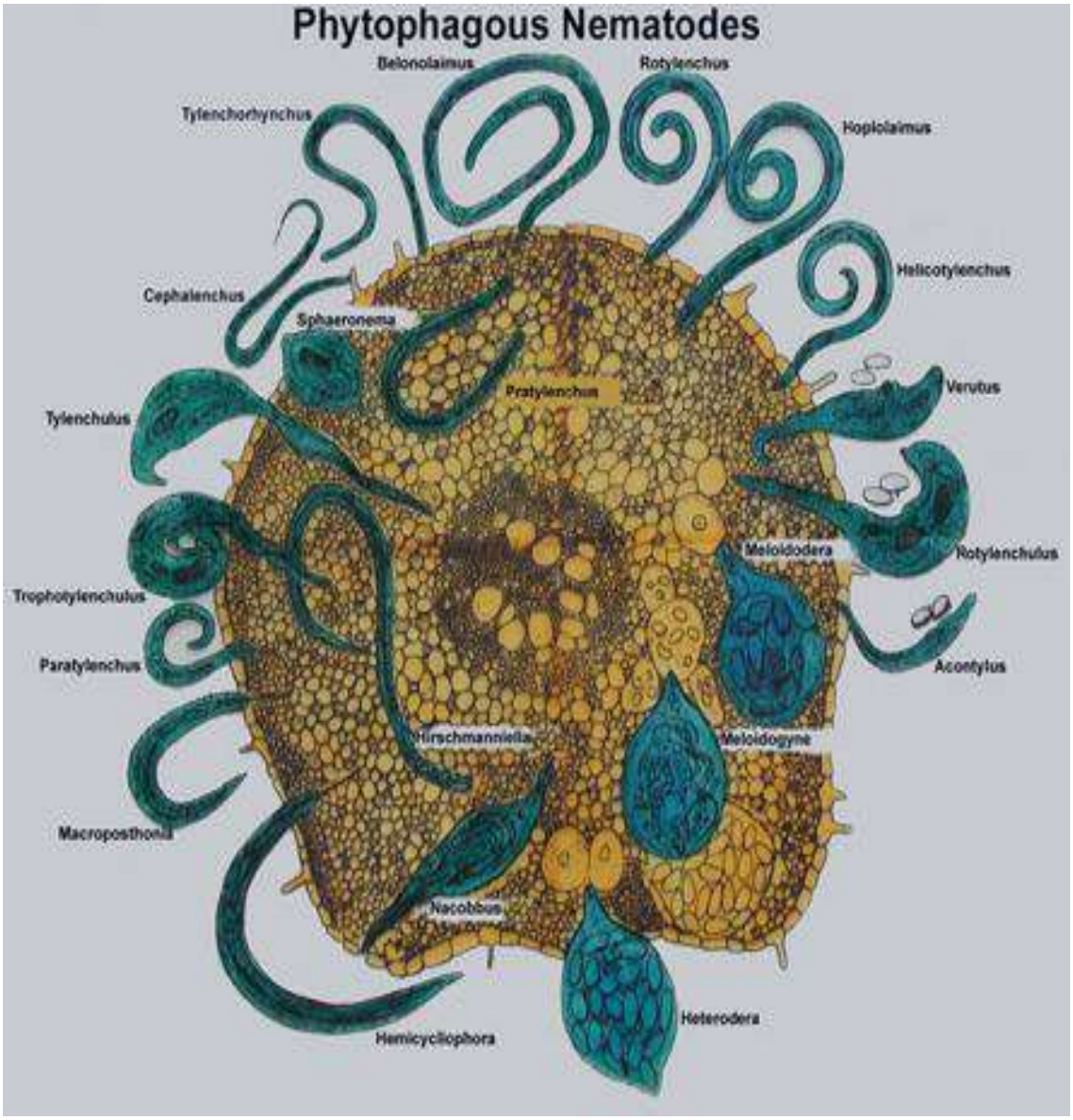
B. Sedentary ectoparasites: In this type of parasitism, the attachment of nematode to the root system is permanent otherwise they spent are like migratory ectoparasite. Examples are *Hemicycliphora arenaria* and *Cacopaurus pestis* etc.

2. Semi – endoparasitic nematodes: The anterior part of the nematode, head and neck being permanently fixed in the cortex and the posterior part extents free into the soil. Example : *Rotylenchulus renifomis* and *Tylenchulus semipenetrans*.

3. Endoparasitic nematodes: The entire nematode or major portion of their body is found inside the root.

A. Migratory endoparasite: These nematodes move in the cortical parenchyma of host root. While migrating they feed on cells, multiply and cause necrotic lesions. Examples arae *Hirschmanniella* spp., *Pratylenchus* spp and *Radopholus similes*

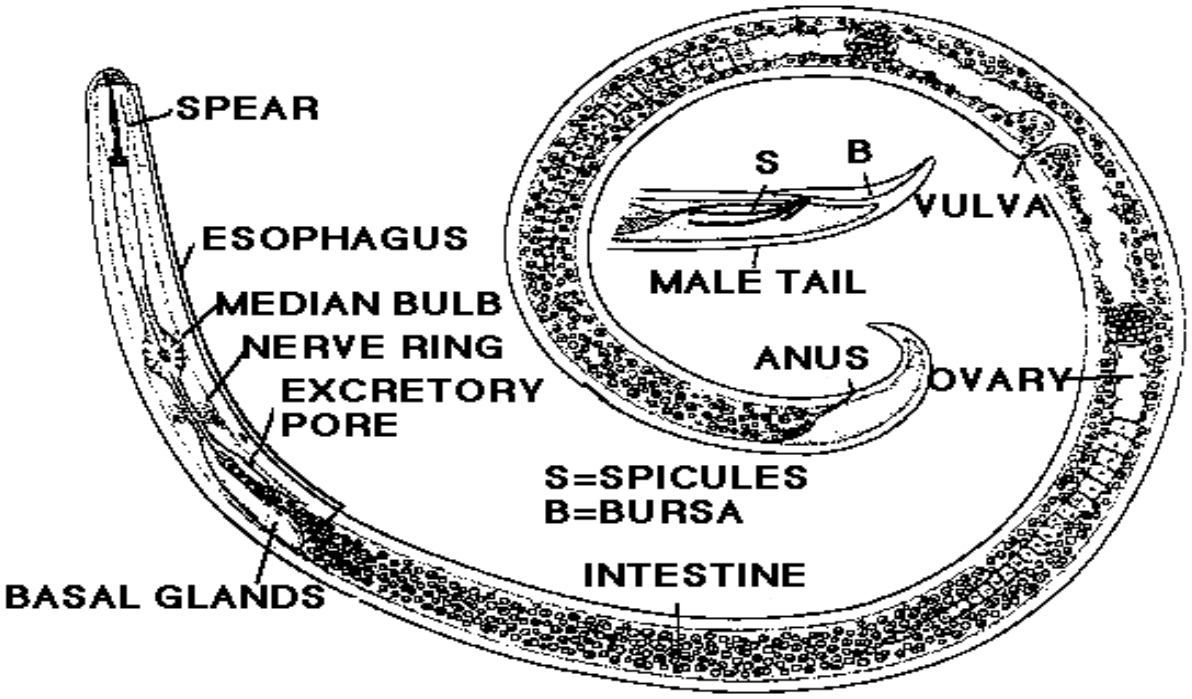
B. Sedentary endoparasite: The second stage larvae penetrate the rootlets and become sedentary throughout the life cycle, inside the root cortex. Examples: *Heterodera* spp and *Meloidogyne* spp.



Genera of the most common PPN (Common and Scientific name)

Awl nematode	*Dolichodorus spp*
Cyst nematode	*Globodera spp*. and *Heterodera spp*.
Dagger nematode	*Xiphinema* spp.
Foliar nematode	*Aphelechoides* spp.
Lance nematode	*Hoplolaimus* spp
Lesion nematode	*Pratylenchus* spp.
Needle nematode	*Longidorus* spp
Pin nematode	*Paratylenchus* spp.
Reniform nematode	*Rotylenchulus renifomis*
Ring nematode	*Criconemella* spp.
Root – knot nematode	*Meloidogyne* spp.
Sheath nematode	*Hemicycliophora* spp.
Spiral nematode	*Helicotylenchus* spp
Sting nematode	*Belonolaimus* spp.
Stubby – root nematode	*Paratrichodorus* spp and *Trichodorus* spp.
Stunt nematode	*Tylenchorhynchus* spp.
Stem and bulb nematode	*Ditylenchus* sp.
White tip nematode	*Aphelenchoid besseyi*
Seed gall nematode	*Anguina tritici*
False root gall nematode	*Nacobbus batatiformis*
Potato Golden cyst nematode	*Globodera rostochinensis*
Red ring disease on coconut -	*Rhadinaphelenchus cocophilus.*
Chrysanthemum foliar nematodes	*A. ritzembasoi.*
Pigeon pea cyst nematode -	*Heterodera cajani*
Wheat cyst by	*Heterodera avenae*
Citrus nematode	*Tylenchulus semipenetrans*
Burrowing nematode	*Radopholus similis*
Rice root nematode	*Hirschmaniella sp*
Ufra disease of rice	*D. angustus*

TYPICAL STRUCTURE OF PLANT PARASITIC NEMATODE



Body Shape

- Nematode body shape is typically vermiform, thread-like and tubular, tapering towards both anterior as well as posterior ends and having maximum diameter near mid body.
- The plant parasitic nematodes are slender, spindle shaped or fusiform organisms and circular in cross section.
- Sexual dimorphism occurs in few species.

Some exceptions in shape:
1. Filiform – More elongated towards the body extremities e.g. *Xiphinema, Longidorus, Paralongidorus*

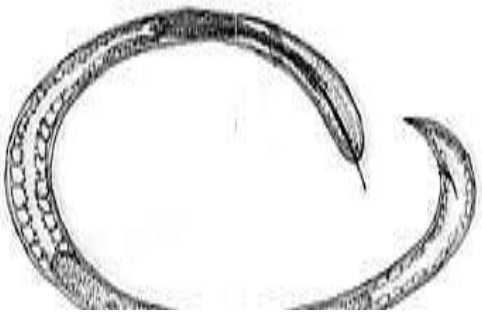


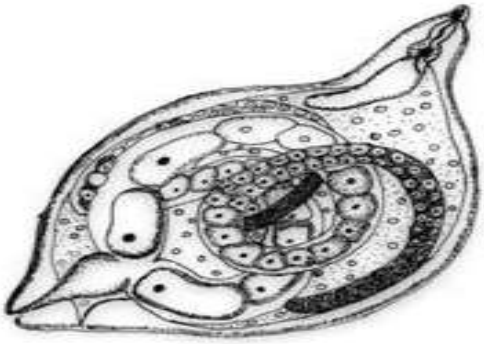
Fig.*Xiphinema* SP.

2. Sausage shaped or plump- When body length is reduced but breadth remains same giving a plump look. e.g. *Criconematids*group.

Sausage shaped

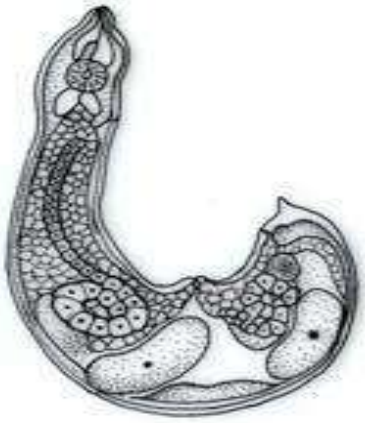
3. Pyriform or flask shaped - Females of certain Genera swell to acquire saccate, pear shape or flask like structure. e.g. Females of *Meloidogyne*spp.and*Heterodera* cysts.

Meloidogyne



Heterodera

4. Kidney shaped or Reniform- eg. *Rotylenchulus reniformis*

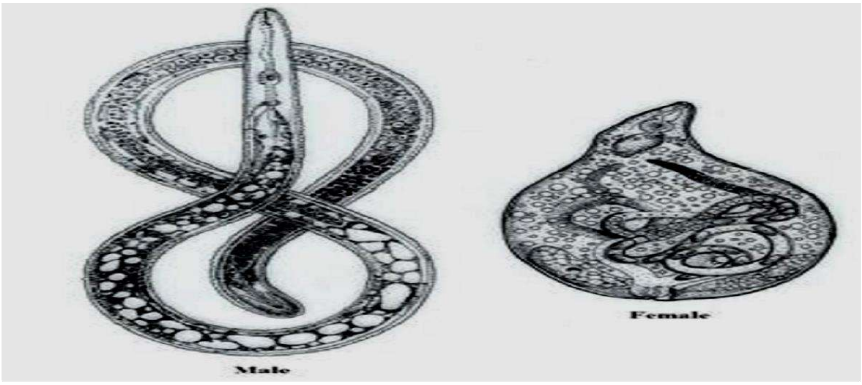


Rotylenchulus reniformis

Tylenchulus semipenetrans

- *Morphological differentiation between the genders of same species* is referred as **sexual dimorphism.**
- Male and females look alike (similar) in most of the nematode species. Males being smaller than females.
- In certain cases (sedentary endoparasites/ semi-endoparasites), the females may deviate from the normal cylindrical form and acquire a typical shapes (swollen, saccate, lemon-shaped, reniform) upon maturity, where as males remain vermiform.
- Such a morphological dissimilarity in the general shapes of males and females within a species is known as **Sexual dimorphism.**
- Sexual dimorphism has been observed in number of genera of order *Tylenchida*.

- The females on maturity assume pear shape in *Meloidogyne*, globular shape in *Globodera*, reniform/kidney shape in *Rotylenchulus reniformis*or saccate in *Tylenchulus semipenetrans*.
- The swelling of female nematodes increases the reproductive potential of the organism.



Male and female of *Meloidogyne* sp. Sexual dimorphism in PPN

History and Development of Nematology

Pre-1850

- Vedas are the earliest religious scriptures in the human history. There are occasional references to nematode parasites of human beings by the name Krmin or Krmi in Sanskrit (meaning worms) in Rig, Yajur and Atharvavedas written 6000-4000 BC. The vedic people were aware about the krimis, their symptoms, vectors, and their cure. Later, when indigenous medical science Ayurveda developed following Atharvveda (3000 BC and later),

- Charak recognized 20 different organsims as krimis in his Samhita, which included nematodes besides arthropods and ticks.

- The earliest written account of a nematode "sighting," as it were, may be found in the Old Testament in the Bible: "And the Lord sent fiery serpents among the people, and they bit the people; and much people of Israel died". Many nematologists assume and the circumstantial evidence suggests the "fiery serpents" to be the Guinea worm, *Dracunculus medinensis*, as this nematode is known to inhabit the region near the Red Sea.

- Before 1750, a large number of nematode observations were recorded; many by the notable great minds of ancient civilization.

- **Hippocrates** (ca. 420 B.C.), Aristotle (ca. 350 B.C.), Celsus (ca 10 B.C.), Galen (ca. 180 A.D.) and Redi (1684) described nematodes parasitizing humans, other large animals and birds.

- **Petrus Borellus** (1656) was the first to describe the first free-living nematode, which he dubbed the "vinegar eel;" scientifically referred as *Turbatrixaceti*.

- **Turbevil Needham** (1743) accidently recorded first plant parasitic nematode when he crushed one of the shrunken and blackened wheat grains and observed "Aquatic worms, Eels, or Serpents, which they very much resemble." He named it *Vibrio tritici*. Later this nematode was rightly named as *Anguina tritici* by Steinbuch.

19th century History of Nematology (Events of significance):

- **Berkley** (1855) discovered first root-knot nematode from galled roots of green-house cucumbers in England.
- **Kuhn** (1857) found that *Dictylechus dipsaci* infecting the Heads of Teasel
- **Schacht** (1859) ascribed the decline in sugar beet to a cyst nematode later named as *Heterodera schachtii*by Schmidt in 1871.
- **Kuhn** (1871) was the first to use soil fumigation to control *Heterodera schachtii*, applying carbon disulfide (CS2) treatments in sugar beet fields in Germany.
- **RitzemaBoss** (1884) Biological Race of *Dictylenchus dipsaci.*
- **Goeldi** (1887) described first *Meloidogyne exigua*causing galls on the roots of coffee in Brazil.
- **RitzemaBoss** (1891) discovered for the first time a foliar nematode when he discovered *Aphelenchoides fragariae*on strawberry.
- **Atkinson** (1892) described for the first time the interactive role of nematodes in producing disease complexes. He reported that the cotton wilt caused by *Fusarium oxysporum* increased manifold in presence of root-knot nematode.
- **Leibscher** (1892) reported pea cyst nematode *Heterodera goettingiana*on pea.
- **Filipjev (**1889-1937) Manual of Agril Helminthology.
- **K. Rudolphi (1771-1832, from Germany).** Often considered as "Father of Helminthology":He gave the name Nematoidea and produced a publication "Entozoorum Synopsis" with 350 species belonging to 11 genera (1819).
- **H. C. Bastian (1837-1915, from England)**"Monograph of the Anguillulidae". He described 100 species of 30 genera in which 23 genera were new. Illustrations were fairly good. He also reported different ways to collect nematode, from soil, plant tissue, fresh and salt water (1866).
- **O. Butschli (1848-1920, from Germany)**
- The first detailed descriptions of the morphological characters used in taxonomy (1873). Perhaps, credit for founding the science of nematology should belong to him

<u>Modern History</u>

- Although 18th and 19th century scientists yielded a considerable amount of important fundamental and applied knowledge about nematode biology, real qualitative and quantitative research in nematology began only near the turn of the 20th century.
- In 1907, **Nathan Augustus Cobb**, an extremely productive scientist, a keen observer, an artist and a prolific writer later called The **Father of Nematology**, joined Sugar Plant Research Centre in Hawaii (USA). His important contributions are:

Nathan Augustus Cobb (1859-1932)

- He described detailed morphology of plant parasitic nematodes and described minute sensory organs of nematodes like amphids, deirids, cephalids, papillae and phasmids.
  - Devised nematode extraction techniques from soil, methods for sampling, nematode sectioning and mounting. Many of his techniques are still unsurpassed.
  - He devised "Cobb's slides" in which nematode specimens are mounted between two coverslips. The slides allow observation of the specimen from both sides and minimize refractive interference of the image. Even now, important nematode specimens are mounted on Cobb's slides.
  - Invented a new camera lucida device for drawing and measurements of nematodes.
  - Discovered many new species of plant parasitic nematodes.
  - Published his first paper on nematology in the United States (1913).
  - He separated free living nematodes and plant parasitic nematodes. He advised to shift plant parasitic nematodes from Helminthology to Nematology and coined the term Nema (1914).
  - Cobb (1918) published “Contributions to a Science of Nematology” and his lab manual “Estimating the Nema Population of Soil” for the benefit of new workers in this field.
  - There are 568 publications to his credit.

N.A.Cobb

Cobb’s Slide

- In 1933 Tom Goodey published his book named “Plant Parasitic Nematodes and the Diseases they cause”.
- In 1934 I N Filipjev in Russia produced his book “Nematodes that are of Importance for Agriculture”.
- In 1937 B G Chitwood and M B Chitwood wrote a book "An Introduction to Nematology"
- In 1951 Tom Goodey published a book "Soil and Fresh Water Nematodes" which was revised by his son, J B Goodey, in 1963.
- 1961: Society of Nematologists (SON) was formed in USA.
- Stainer : "Plant parasitic nematodes the growers should know" (1949)

Milestones/ Landmarks in History of Nematology

- 1941: Discovery of the golden nematode, *Globodera rostochiensis* in the potato fields of Long Island led U.S. quarantine officials on a trip to the potato fields of Europe, where the devastating effects of this parasite had been known for many years earlier.
- 1943: The introduction of the soil fumigants, D-D (Dichloropropane –Dichloropropene) by **Carter** for nematode control
- 1945: **Christie** discovered another fumigant petroleum byproduct EDB (ethylene dibromide) for nematode control on a field scale.
- 1951: **Christie and Perry** demonstrated several ectoparasitic nematodes like *Xiphinema, Longidorus, Trichodorus, Dolichodorus, Hemicycliophora* etc. to act as primary plant pathogens.
- 1948 **– Allen** – Taught the World's first formal university course in Nematology at the University of California, Berkeley.
- 1950 – **Oostenbrink** – Wrote a Book of on "The Potato Nematode, A dangerous parasite to Potato Monoculture".
- 1953: **Suit and Ducharme** discovered that the burrowing nematode, *Radopholus similis* was responsible for causing 'Spreading decline of citrus' in Florida, USA. Serious problem of 'Pepper Yellows' in Bangka islands of Indonesia that shattered the economy of the country was also discovered to be caused by this nematode.
- 1955: Society of European Nematologists (SON) was formed. In 1956, this society started publishing first ever exclusive journal in the field of Nematology by the name of Nematologica (name now changed to 'Nematology').
- 1958: First report of virus transmission by plant parasitic nematodes was given by **Hewitt, Raski and Goheen** when they demonstrated *Xiphinema index* to transmit fan leaf grapevine virus in grape vines.
- 1971: *Bursaphelenchus xylophilus*was found to be responsible for serious 'Pine wilt' disease in Japan that devastated vast pine forests.
- 1978 – Revue de Nematologie published from France.
- **Gerald Thorne -** Many contributions to the science of nematology, especially on nematodes in the order Tylenchida, Dorylaimida and Rhabditida. His book "Principles of Nematology" (1961) is an outstanding contribution.
- 1930s – 1990s – **Barron, Duddington, Mankau, Linford, Sayre and Zuckerman** – they provided an insite on the Biological control of plant – parasitic nematodes. Enhanced understanding of antagonists and related biology enhancing the potential for practical biocontrol.
- 1940-s – 1990s – **Triantaphyllou** – Provided advancement in Cytogenetics, modes of reproduction/sexually – and information data base for genetics/molecular research. Enhanced understanding of evolution and taxa interrelationships.

- 1950s – 1990s – **Caveness, Jones, Oostenbrink, Sasser and Seinhorst** – International programme such as International *Meloidogyne*project – They expanded educational base of nematologistsworld wide and provided ecological – taxonomic data base.

- 1960s- 1990s **– Nickle, Poinar and Steiner** – Biological control of insects with nematodes.

- 1960s- 1990s – Brenner, Dougherty and Nicholas – Caenorhabditis elegans developmental biology and genetics – model system – provided fundamental information on cell lineage, behavior, gene function ageing and overall genome for this model biological system.

History of Nematology in India

- 1901: **Barber** reported root knot nematode infecting tea in South India – first ever report of plant parasitic nematodes from India.

- 1919: **Butler** reported that Ufra disease of rice was caused by *Ditylenchus angustus*.

- 1934: **Ayyar** reported root knot nematode infesting vegetables and other crops in South India.

- 1936: White tip disease of rice caused by *Aphelenchoides besseyi*was reported by **Dastur.**

- 1958: **Molya disease of wheat and barley** (caused by ***Heterodera avenae)*** recorded in Rajasthan by **Vasudeva.**

- 1959: **Siddiqi** discovered **citrus nematode** and other plant parasitic nematodes from Aligarh soil in UP.
- 1961: **Jones** reported **golden nematode of potato** from Nilgiri Hills in Tamil Nadu.
- 1961: Nematology unit was established at the Central Potato Research Institute, Shimla.
- 1964: International Nematology Course at IARI, New Delhi participated by FGW Jones, J B Goodey and D J Raski.
- 1966: **Nair, Das and Menon** reported burrowing nematode, ***Radopholus similis*** **on banana** in Kerala for the first time.
- 1966: Division of Nematology was started at IARI, New Delhi.

- 1967-68: First South East Asia Nematology course held at AMU Aligarh and IARI, New Delhi.

- 1969: Nematological Society of India was founded and first All India Symposium was held at IARI, New Delhi.

- 1971: Nematological Society of India commenced publication of its biannual journal "Indian Journal of Nematology"

- 1977: All India Coordinated Research Project (AICRP) on nematode pests of crops and their control was started at 14 centers with the headquarters of project coordinator at IARI, New Delhi.

- M.Sc. (Ag.) Plant Nematology course started 1979- Tamil Nadu Agricultural University, Coimbatore.

ECOLOGICAL CLASSIFICATION OR

CLASSIFICATION OF PLANT PARASITIC NEMATODES ON THE BASIS OF FEEDING HABIT

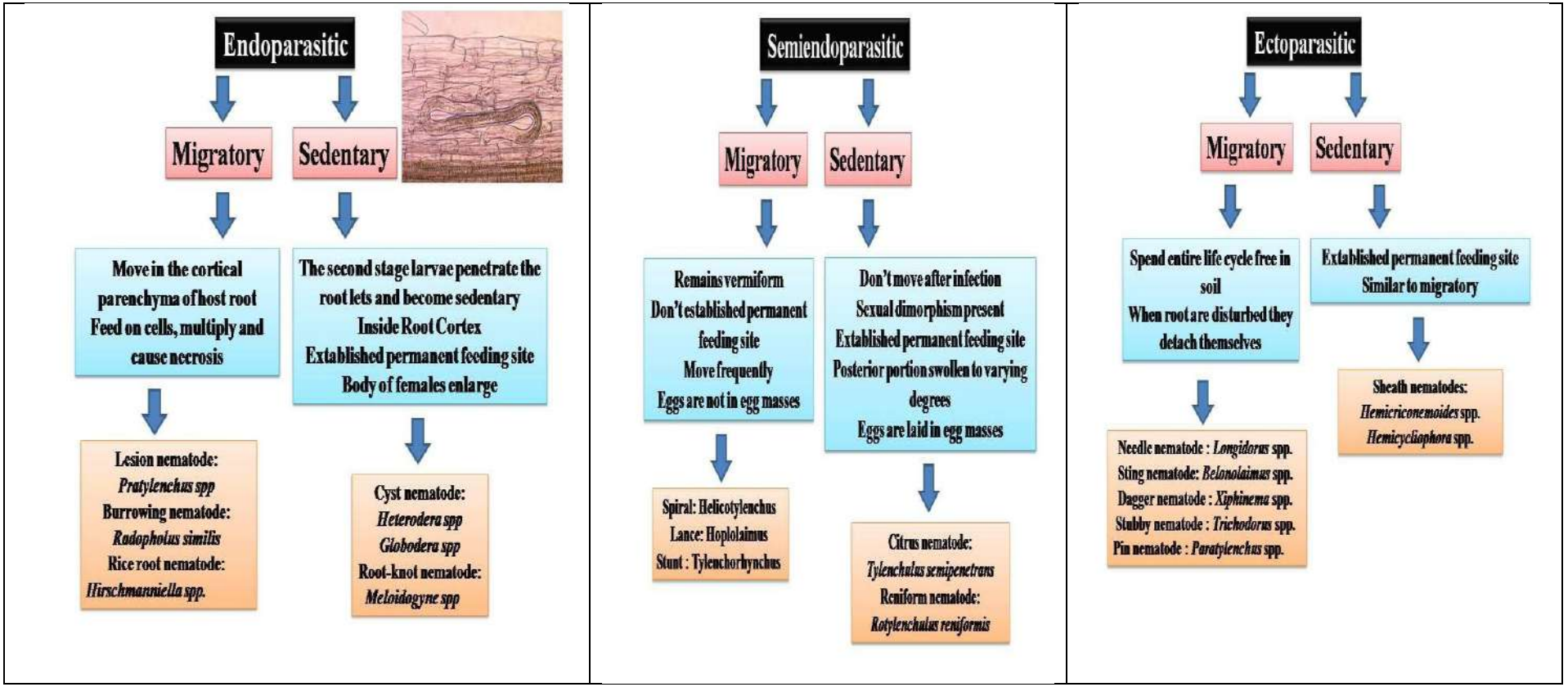
Above ground feeder/Arial nematode

Seed gall nematode , *Anguina tritici ***Stem and bulb , *Ditylenchus spp***

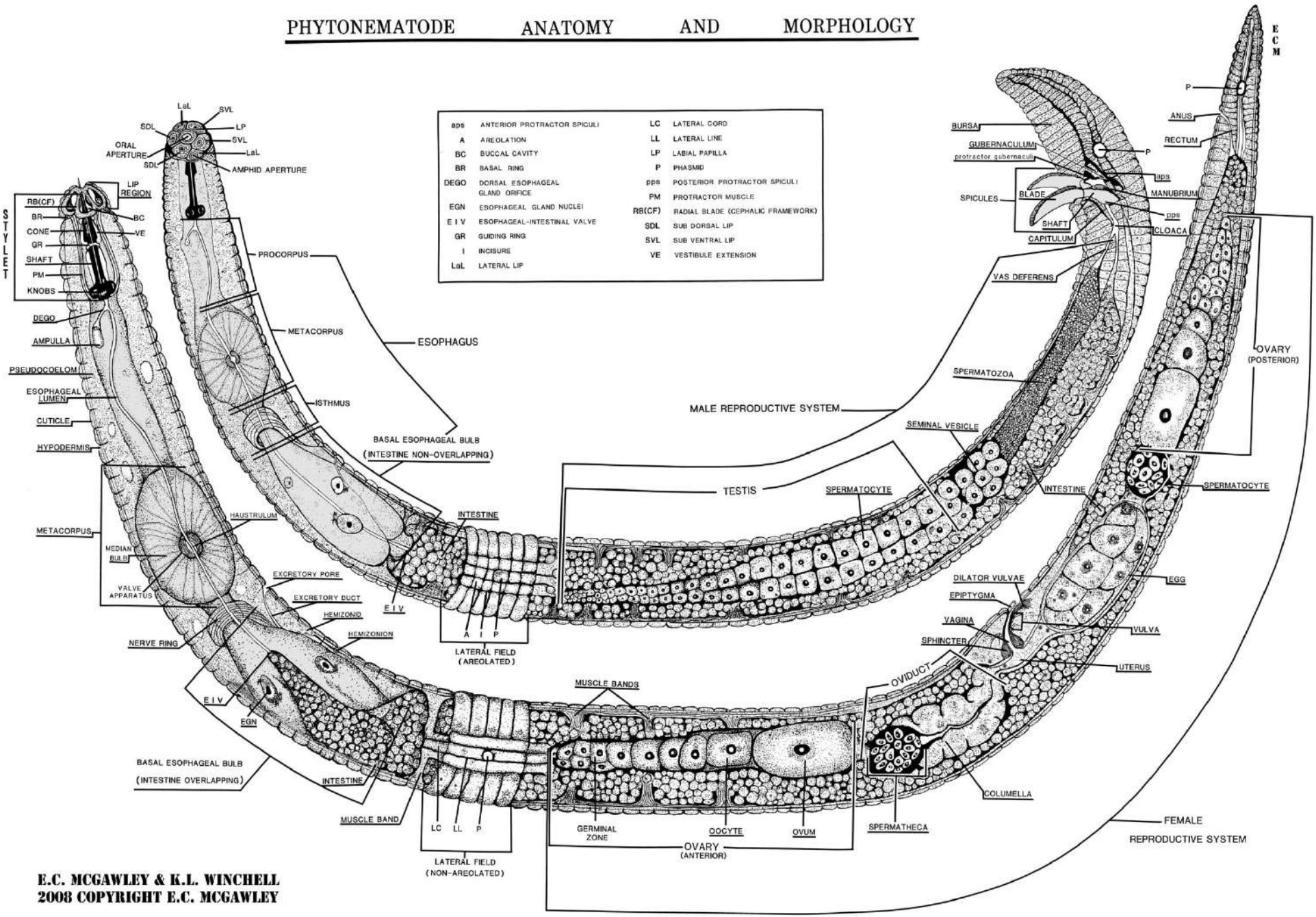
Red ring nematode,*Rhadinaphelenchus cocophilus ***Leaf and bud nema, *Aphelenchoides spp***

***Pine wilt nema *Bursaphelenchus* sp**

Below ground feeder



PHYTONEMATODE ANATOMY AND MORPHOLOGY



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Biology and Ecology of nematodes-types of parasitism-complex diseases caused by Plant parasitic nematodes:

Biology :

The plant parasitic nematodes have 6 stages and four moulting in their life cycle. They are

1. Egg ,

2. First stage larva/juvenile (J1)

3. Second stage larva/juvenile (J2),

4. Third stage larva/juvenile (J3)

5. Fourth stage larva/juvenile (J4) and

6. Adult

The first stage juvenile undergo molting with in the egg after hatching and second stage juveniles will be released into the soil, which is the infective stage. Larva or juvenile undergo moulting thrice and then develops into adult.

The female lays eggs in the soil or plant tissue either singly or in masses that hatch into larvae which are almost similar to adults in appearance. The first moulting usually occurs before hatching when the larvae is within the egg shell. The larva undergoes series of four moults. The period of growth between moults are called juvenile stages.The first larval stage is ended by the first moult, the 2nd stage by second moult. After 4th moult the adult is formed. In case of *Xiphinema index* the larvae emerge from the egg before the first moult. The larval cuticle is shed after each moult. The average number of eggs laid by nematode may be 200-500. The total lifecycle will be completed in 20-40 days.

NATURE OF DAMAGE:

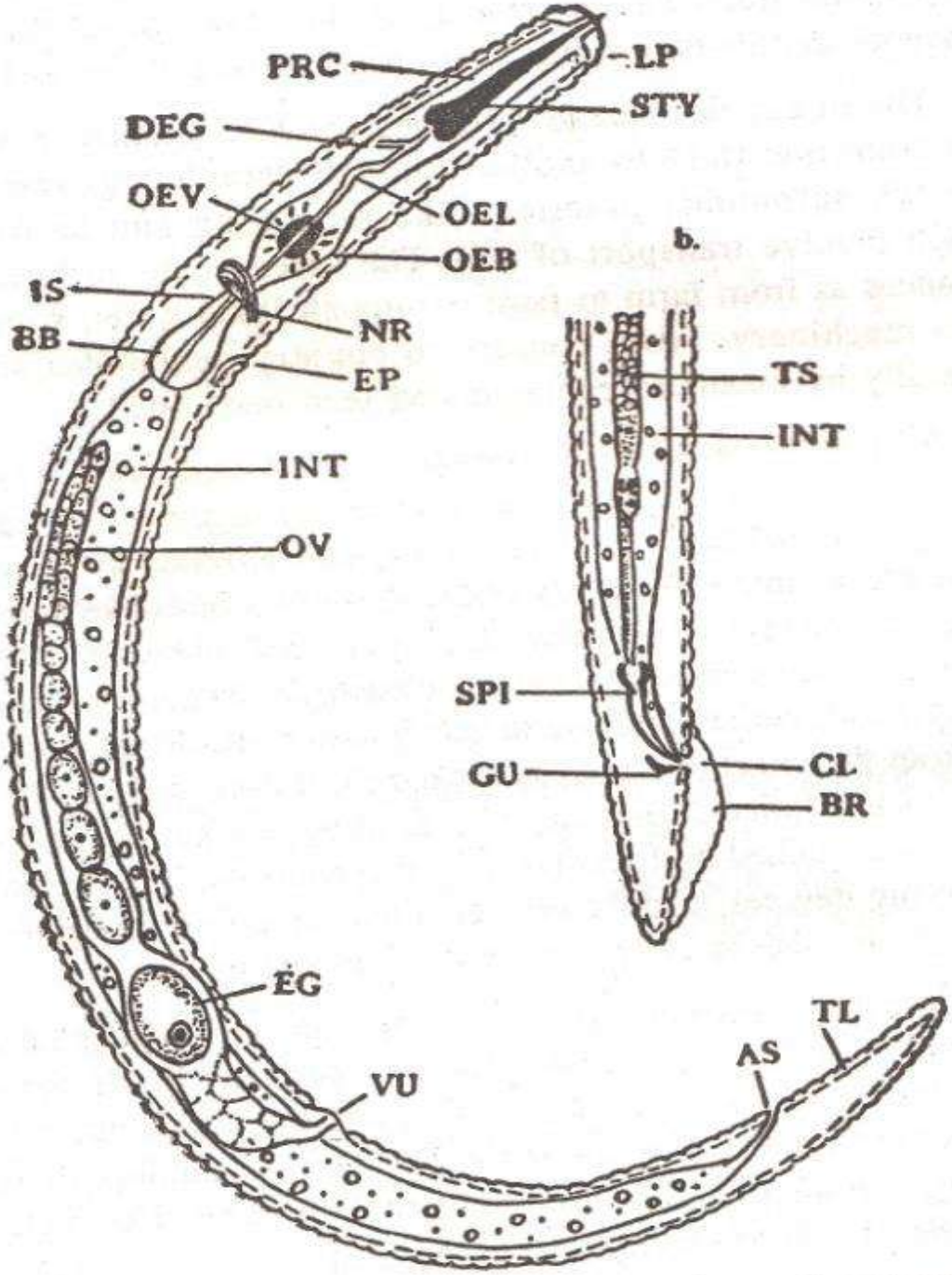
Majority of phytophagous nematodes are polyphagous infecting wide range of

hosts among which a few may be more favourable than others. These nematodes feed on plant tissues by sucking sap from the host cell with the oral stylet that is used as a hypodermic needle to puncture the host cell. Nematodes usually cause damage by Mechanical injury – by penetration and movement through plant tissues causing cellular changes like necrosis – death of cells and causes some Physiological changes in the host by interruption in uptake of nutrients and water from roots as vascular bundles get damaged.

Tail:

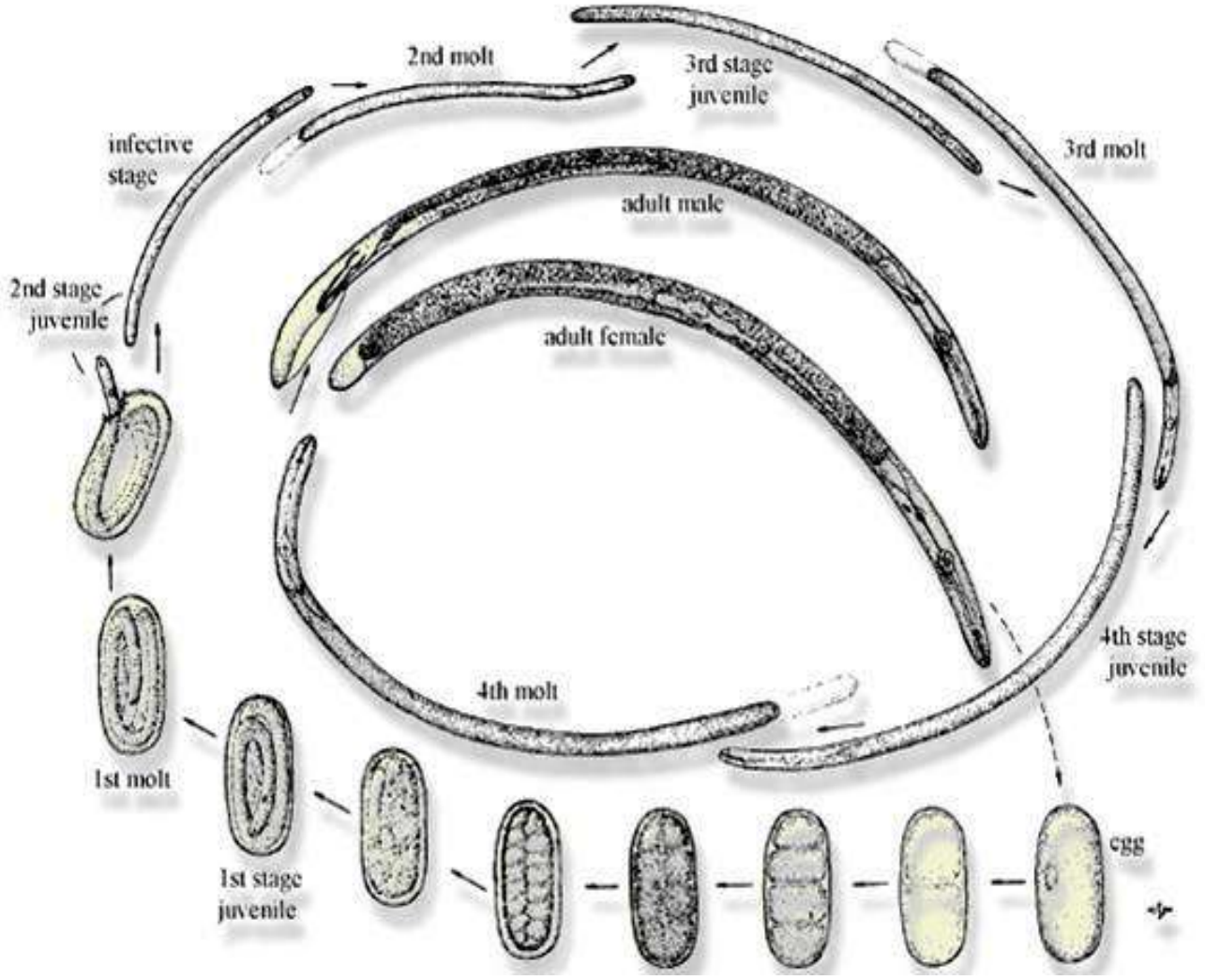
Present in male, female and larva. Tail vary in shape and size. Sometimes it may be different in larva and adult or it may be present in larva but reduced or absent in adult as in *Heterodera* and *Globodera.*

Tail is a locomotory organ useful for swimming in aquatic nematodes. Other structures present at tail region include caudal alae, bursa and genital papillae that help in reproduction. Tail tip may be filiform, conoid, hooked short bluntly conoid, hemispheroid or clavate type.



A typical female nematode, b. Posterior portion of a male nematode, AS, Anus; BB, basal bulb; BR, Bursa; CL, Cloaca; DEG, dorsal oesophageal gland opening; EG, egg; EP, excretory pore; GV, Gubernaculum; INT, intestine; IS, isthmus; LP,lip; NR,nerve ring; OEB,median oesophageal bulb; OEL, oesophageal lumen; OEV, oesophageal valve, OV, ovary, PRC, procorpus, SPI, spicule, STY, Stylet, TL,Tail; TS, testis, VU, vulva.

GENERAL LIFE CYCLE OF PLANT PARASITIC NEMATODE



$$J_1 - M_1 - J_2 - M_2 - J_3 - M_3 - J_4 - M_4 - \text{Adult}$$

Ecological classification of plant parasitic nematodes or Feeding habit

I. Above ground feeders

a. Feeding on flower buds, leaves and bulbs.

i. Seed gall nematode : *Anguina tritici*

ii. Leaf and bud neamtode : *Aphelenchoides*

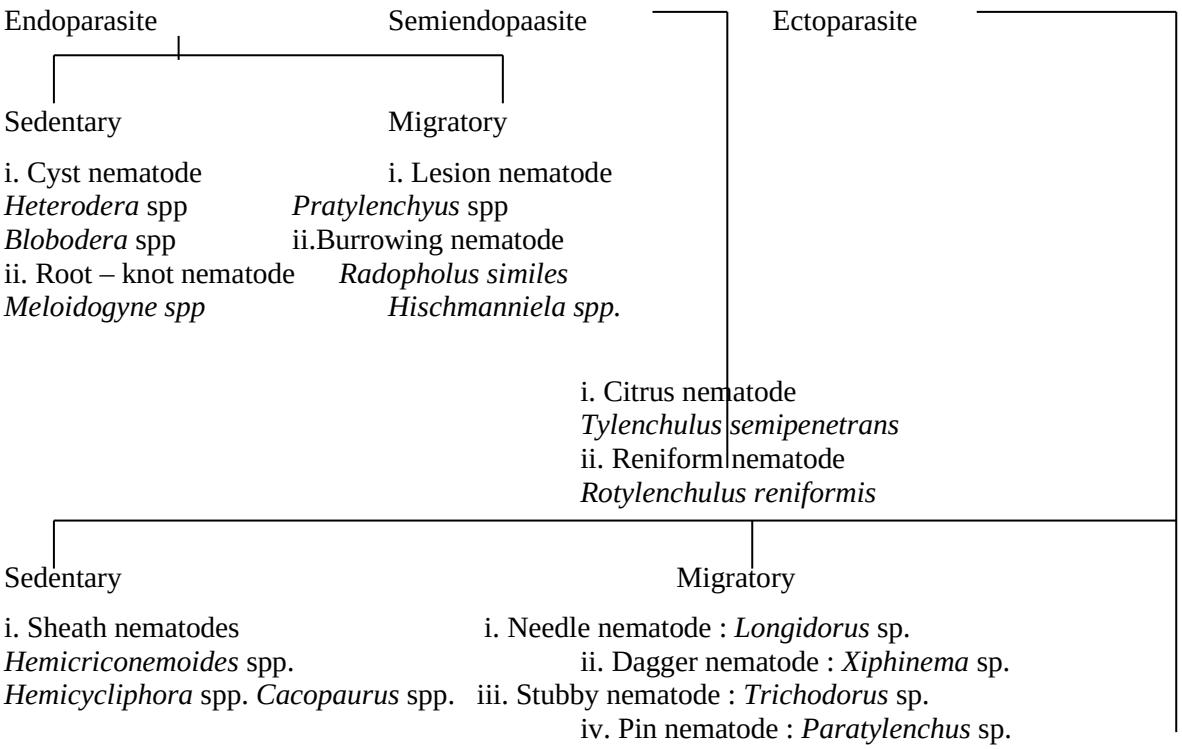
iii. Stem and bulb nematode **:** *Ditylenchus*

b. Feeding on tree trunk

i. Red ring nematode : *Rhadinaphelenchus cocphilus*

ii. Pine wilt nematode : *Bursaphelenchus xylophilus*

II. Below ground feeders



According to feeding habits, the nematodes can be divide into

1. Ectoparasitic nematodes,
2. Semi endoparasitic nematodes and
3. Endoparasitic nematodes

1. Ectoparasitic nematodes : These nematode live freely in the soil and move closely or on the root surface, feed intermittently on the epidermis and root hairs near the root tip.

A. Migraotory ectoparasite : (e.g.) *Criconemoides* spp. *Paratylenchus* spp., and *Trichodorus* sp., etc., These nematodes spend their entire life cycle free in the soil . When the roots are disturbed they detach themselves.

B. Sedentary ectoparasites : (eg.) *Hemicycliphora arenaria* and *Cacopaurus pestis* etc., In this type of parasitism the attachment of nematode to the root system is permanent but for this, it is similar to the previous one.

2. Semi – endoparasitic nematodes : (e.g.) *Rotylenchulus renifomis* and *Tylenchulus semipenetrans*. The anterior part of the nematode, head and neck being permanently fixed in the cortex and the posterior part extents free into the soil.

3. Endoparasitic nematodes : The entire nematode is found inside the root and the major portion of nematode body found inside the plant tissue.

a. Migratory endoparasite : (eg.) *Hirschmanniella* spp., *Pratylenchus* spp and *Radopholus similes* etc., These nematodes move in the cortial parenchyma of host root. While migrating they feed on cells, multiply and cause necrotic lesions.

b. Sedentary endoparasite : (eg.) *Heterodera* spp and *Meloidogyne* spp. The second stage larvae penetrate the root lets and become sedentary through out the life cycle, inside the root cortex.

Nematode Disease symptoms on crop plants

Most of the plant parasitic nematodes affect the root portion of plants except *Anguina spp., Aphelenchus spp., Aphelenchoides spp.,Ditylenchus spp, Rhadinaphelenchus cocophilus* and *Bursaphelenchus xylophilus.* Nematodes suck the sap of the plants with the help of stylet and causes leaf discolouration, stunted growth, reduced leaf size and fruits and lesions on roots, galls, reduced root system and finally wilting.

Symptoms of nematode diseases can be classified as

I. Symptoms produced by above ground feeding nematodes

II. Symptoms produced by below ground feeding nematodes

I. Symptoms produced by above ground feeding nematodes

1. Leaf discolouration : The leaf tip become white in rice due to rice white tip nematode *Aphelenchoides besseyi*, yellowing of leaves on Chrysanthemum due to Chrysanthemum foliar nematodes, *A. ritzemabosi*.

2. Dead or devitalized buds : In case of straw berry plants infected with *A. fragariae*, the nematodes affect the growing point and kill the plants and result in blind plant.

3. Seed galls : In wheat, *Anguina tritici* larva enter into the flower primordium and develops into a gall. The nematodes can survive for longer period (even upto 28 years) inside the cockled wheat grain.

4. Twisting of leaves and stem : In onion, the basal leaves become twisted when infested with *D. angustus*.

5. Crinkled or distorted stem and foliage: The wheat seed gall nematode. *A tritici* infects the growing point as a result distortions in stem and leaves take place.

6. Necrosis and discolouration : The red ring disease on coconut caused by *Rhadinaphelenchus cocophilus*. Due to the infestation, red coloured circular area appears in the trunk of the infested palm.

7. Lesions on leaves and stem: Small yellowish spots are produced on onion stem and leaves due *D. dipsaci* and the leaf lesion caused by *A. ritzemabosi* on Chrysanthemum.

II. **Symptoms produced by below ground feeding nematodes:** The nematodes infest and feed on the root portion and exhibit symptoms on below ground plant parts as well as on the above ground plants parts and they are classified as

A. Above ground symptoms

B. Below ground symptoms

A. Above ground symptoms

1. Stunting : Reduced plant growth, and the plants can not able to withstand adverse conditions. Patches of stunted plants appears in the field. (eg.) in potato due to *Globodera rostochiensis*, in maize, due to *Heterodera zeae & Pratylenchus zeae* and in wheat by *Heterodera avenae*.

2. **Discolouration of foliage** : Patchy yellow appearance in coffee due to *Pratylenchus coffeae, G. rostochiensis* infested potato plants show light green foliage. *Tylenchulus semipenetrans* induce fine mottling on the leaves of orange and lemon trees.

3. **Wilting :** Day wilting due to *Meloidogyne* spp. *i.e*. in hot weather the root-knot nematodes infected plants tend to droop or wilt even in the presence of enough moisture in the soil. Severe damage to the root system due to nematode infestation leads to day wilting of plants.

B. Below ground symptoms

1. Root galls or knots : The characteristic root galls are produced by root-knot nematode, *Meloidogyne* spp. False root galls are produced by *Nacobbus batatiformis* on sugar beet and tomato. Small galls are produced by *Hemicycliophora arenaria* on lemon roots. *Ditylenchus radicicola* cause root galls on wheat and oats. *Xiphinema diversicaudatum* cause galls on rose roots.

2. Root lesion : The penetration and movement of nematodes in the root causes typical root lesions eg. Necrotic lesions induced by *Pratylenchus* spp on crossandra; the burrowing nematode, *Radopholus similes* in banana. Similarly *Pratylenchus coffeae* and *Helicotylenchus multicinctus* cause reddish brown lesion on banana root and corn. The rice root nematode also causes brown lesions on rice root.

3. Reduced root system : Due to nematode feeding the root tip growth is arrested and the root produce branches. This may be of various kinds such as coarse root, stubby root and curly tip.

4. **Coarse root :** If the growth of lateral roots is stopped just as most of them are breaking through the cortex or while they are very short , an open system made up mostly of the main roots largely devoid of small branch rootlets may result. This type of symptom is produced by *Belonolaimus longicaudatus* on corn. *Paratrichodorus* spp. infestation arrests the growth of lateral roots, and leads to open root system with only main roots without lateral roots.

5. Stubby roots : The lateral roots produce excessive rootless. If the branch rootlets manage to attain a moderate length before their growth is stopped, the resulting root system may be composed of numerous short, stubby branches often arranged in clusters. This type of symptoms is produced by *Trichodorus christie* on corn.

6. **Curly tip** : In the injury caused by *Xiphinema* spp. the nematode retard the elongation of roots and cause curling of roots known as "Fish book' symptom.

7. Root proliferation : Increase in the root growth or excessive branching due to nematode infestation. The infested plant root produced excessive root hair at the point of nematode infestation. (eg.) *Trichodorus christei, Nacobbus* spp., *Heterodera* spp. *Meloidogyne hapla* and *Pratylenchus* spp. etc.

8. Root – rot : The nematodes feeds on the fleshly structure and resulting in rotting of tissues (eg.) Yam nematode *Scutellonema bradys* and in potato *Ditylenchus destructor* cause root rot.

9. Root surface necrosis : The severe injury caused by *T. semipenetrans* on citrus leads to complete decortications of roots and results in root necrosis.

10. Cluster of sprouts on tubers : On the tubers, clusters of short and swollen sprouts are formed due to *D. dipsaci* infestation in many tuber plants.

11. Pearly disease of pigeonpea:- Immature whitish females of cyst nematode are found attached with the roots of pigeonpea . It is known as pearly disease of pigeonpea. Eg, *H. cajani.*

12. Dirty root:- In banana, castor, pigeonpea, due to infection of *Rotylenchulus reniformis,* soil particles adhere to the gelatinous matrix of eggmasses, which are not removed by washing the roots, thus giving dirty appearance of roots.

13. Beard shape:- The root tip, after attacked by stunt nematodes comes out large number of branches from point of infection , these branches in turn again infected and branches again .This condition is known as beard shape At feeding point root is cut off, so stop the nutrient movement , so many secondary & tertiary roots comes out from cut end eg. *Tylenchorhynchus vulgaris*

Difference between root galls and root nodules

Root galls / Root knot	Root nodules
Formed within root	Formed on the side of root
Irregular in shape	Regular in shape
Very hard	Soft
Harmful host-parasite relationship	Useful symbiotic relationship
Easily not detachable	Easily detachable
Nematodes found on dissection	Bacteria found on dissection
Pinkish colour fluid not seen	Pinkish colour fluid seen
Do not fix atmospheric N2	Fix atmospheric N2
e.g. *Meloidogyne* spp	e.g. *Rhizobium* spp.

INTERACTION OF NEMATODES WITH OTHER PLANT PATHOGENS

Plant parasitic nematodes favours the establishment of secondary pathogens viz., fungi, bacteria, virus etc. The nematodes alter the host in such a way that the host tissue becomes suitable for colonization by the secondary pathogens. Even though the nematodes themselves are capable of causing considerable damage to the crops, their association with other organisms aggravate the disease. The nematodes cause mechanical wound which favours the entry of micro organisms. In some cases, the association of nematode and pathogen breaks the disease resistance in resistant cultivators of crop plants.

Nematode – fungus Interaction

Nematode – fungus interaction was first observed by **Atkinson (1892) in** cotton. *Fusarium* wilt was more severe in the presence of *Meloidogyne* spp. Since then the nematode – fungus interaction had received considerable attention on important crop like banana, cotton, cowpea, brinjal, tobacco and tomato. Some examples of nematode – fungus interaction are given in the following table.

Crop	Name of the disease	Nematode	Fungus	Role of nematode
Cotton	Damping off	*Meloidogyne incognita acrita* *M. incognita acrita*	*Rhizoctonia solani* *Pythium debaryanum*	Assists *Pythium*
	Vascular wilt	*M. incognita acrita*	Fusarium *Osysporum* F. *vasingectum*	Assists
	Rotylenchulus Reniformis *Belonolaimus gracicilis* *B. longicaudatus*		*F. oxysporium* f.sp *Vasinfectum* *F.oxysporum* f.sp	Assists Assists
Blank shank (vascular wilt)	*M. incognita acrita*		*Pytophtora Parasitica var nicotianae*	Assists

Nematode – Bacterium interactions

Nematode – bacterium interactions are comparatively fewer than the nematode – fungal interactions. Some examples of nematode – bacteria association are presented.

Crop	Name of the disease	Nematode	Bacterium	Role of nematode
Wheat	Tundu/yellow slime/ear rot of wheat	*Anguina tritici*	*Corynebacterium tritici*	Essential Assists
	Vascular wilt	*M. incognita*	*Pseudomonas solanacearum*	

Nematode – Virus Interaction

In nematode – virus complex, nematode serves as a vector. Numerous virus – nematode complexes have been identified after the **pioneer work by Hewit, Raski and Goheen (1958)** who found that *Xiphinema index* was the vector of grapevine fan leaf virus. *Xiphinema* spp., *Longidorus* spp., and *Paralongodorus* spp. transmit the ring spot viruses which are called "NEPO" derived from **Nematode transmitted polyhedral shaped particles.** *Teichodorus* spp. and *Paratrichodorus* spp. transmit the rattle viruses and called "NETU" derived from **Nematode transmitted tubular shaped particles.** All these nematodes have modified bottle shaped oesophagus with glands connected by short ducts directly to the lumen of the oesophagus. This actually of nematodes. Certain examples of the viral diseases and the nematode vectors are given in the following table.

Viruses	Nematode
NEPO – virsus	
Arabis mosaic	*Xiphinema diversicaudatum*
Arabis mosaic, grapevine fan leaf Arabis mosaic, Grapevine	*X. paraelongatum*

NETU VIRUSES

Tabacco rattle	*Paratrichodorus pachydermus* *P.allius,P.nanus,* *P.porosus,P.teres* *Trichodorus christei* *T.Primitivus, T.cylindricus,* *T. hooperi*
Pea early browning	*P.amenous, P.pachydermus, P.teres,T.viruliferus.*

Nematodes acquire and transmit the virus by feeding, which requires as little as one day. Once acquired, the virus persists for longer period in the nematode body than in vitro. For example, the grapevine fan leaf virus will exist for as many as 60 days in X. index. Two types of mechanisms are observed in virus transmission (i) retention through close biological association between virus and vector as in *Xiphinema*; (ii) retention of virus mechanically as in *Logidours.* Virus is retained in the inner surface of the guiding sheath of *Longidorus,* cuticle lining of the lumen of *oesophagus* in *Trichodorus* and *Paratrichodrous*, cuticle lining of stylet extension and oesophagus in *Xiphinema.* The virus particles are released into plant cell with the help of oesophagus.

Different types of nematodes

1. Seed gall nematode: *Anguina tritici*

Host Range: Wheat

First plant parasitic nematode identified . Nematode larvae revived even after 28 years of storage.

• Obligate plant parasites with obese body.
• Female spiral in form and immobile.
• Males usually shorter than females.
• Infective stage: **juvenile stage (J2).**
• The juveniles moult thrice in quick succession within 3-5 days after invasion of flower primordia and change into adult males and females.

Symptoms:

• Nematode infected seedlings show slight enlargement of basal portion of the stem after about 20-25 days of germination.
• Leaves emerging from the seedling are twisted and crinkled, often folded with their tips held near the growing point.
• Infested plants generally show profuse tillering.
• Glumes may be loosely arranged and **galls** replace the seeds.
• Conversion of all or few grains in the ear into **cockles**.

2. Stem and bulb nematode: *Ditylenchus sp.*

Host Range: Paddy , potato, mushroom (*D.myceliophagus*) and Onion

Important pest during storage.

• Rarely striated head, lip region is without dovious annuls.
• Oesophageal glands may overlap intestine slightly.
• Female with monodelphic, prodelphic ovary.
• Male with one testis.
• Infective stage - **juvenile stage (J4)**

• **Symptoms:**
• The leaves may become slightly thinner and more flaccid and sometimes chlorosis may be observed.
• The chlorotic portion of leaves becomes brown to dark brown.

- Twisting of leaf and leaf sheath.
- • Panicles do not emerge and are enclosed completely within the flag leaf sheath.

3. Leaf and bud nematode: *Aphelenchoid sp.*

Host Range: Rice, mushroom ,strawberry and chrysanthemum

• Cuticle is marked by fine transverse striae.
• Lip region is not striated.
• Spear is with thickening at the base but without well developed knobs
• Female with single ovary out stretched. Vulva posterior,
• The nematode is carried beneath the hull of the rice kernel in quiescent, immature, usually preadult stage.
• These nematodes do not survive in the soil after harvesting and are not transmitted through seeds.
• Infective stage - **juvenile stage (J2)**

Symptoms:

• White tip of seedlings.
• Initially the seedling growth is stunted and germination is delayed.
• Turning of 3-5cm of leaf tip into pale yellow to white colour at the tillering stage.
• The emerged panicles are generally short and flower becomes sterile.
• Glumes of affected spikelets are white and do not change in shape and size.

4.Red ring nematode: *Rhadinaphelenchus cocophilus*

Host Range: Coconut
Very slender body, massive sclerotization of the labial arches, elongated medium bulb , wide vulvar flap, curved vagina, speculum form and sclerotized spade like extension of male tail.
• Transfer of nematodes is purely mechanical through palm weevil *Rhynchophorus Palmarum*

Symptoms:
• Yellowing and browning of the leaves begin at the base of the tree and progress upward till the leaves die, break and hang down.
• A cross section shows 1-2 inches **red ring** that extends from the base of thetrunk to a height of 4 feet or more.
• Infested roots show discoloured tissues.

5.Root knot nematode: *Meloidogyne sp.*

Polyphagous
Host Range:Tomato, brinjal, bhendi, chilli, cowpea, greengram and tobacco
Female:
• The cuticle is transcuscent and glistening giving the white , pearly appearance to the nematode.
• the posterior end of the females possess distinctive arrangements of striae about the vulva- anus areas(perineal area).
• The excretory pore is infront of the muscular bulb of the oesophagus and close behind the stylet base.
Male: • Vermiform, having blunt tail and slightly tapering neck region.

• Spicules simple, curved and gubernaculum is small.
• Caudal alae absent.

Symptoms:
• The infected plants usually exhibit yellowing of foliage, reduced unthrifty growth, smaller foliage, poor and fewer fruits and a tendency to wilt during warmer parts of the day.
• Poor emergence and death of young seedlings .
• Early poor fruiting for a relatively shorter period .
• Wilting and drying of crop in the field in patches.
• Presence of **galls or knots** on roots is the important diagnostic symptom

6. Cyst nematode: *Heterodera sp. and Globodera sp.*

Host Range: Wheat, Barley. Rie, Oats, Pulses and Potato etc
• **Heterodera:**
• the mature cyst is **lemon shaped** with a short neck and terminal cone
• Male vermiform, stylet and oesophagus well developed, tail end twisted, spicules robust with out bursa.
• **Infective stage :** juvenile stage(J2)

Globodera:

The mature cyst **spheroidal** (round) with a short projecting neck, terminal region not forming a cone.
- Vulval fenestra circumfenstrate, no anal fenestra.
Infective stage : juvenile stage(J2)

Symptoms of potato cyst nematode:
- The disease usually occurs in patches under severe infection
- Stunted growth, general necrosis, in severe infection
- The newly formed tubers are few in no. and considerably reduced in size.
Symptoms of *Heterodera avenae*:
The effected fields usually give patchy appearance during initial stages of infection.
- Stunted growth, general necrosis, in severe infection, there may not be any grain formation.
- Infected roots are short, with multiple branches giving a bunchy appearance.

7. Root lesion nematode(*Pratylenchus sp.)*

The head is relitively broad and continuos with body. Tail blunty rounded.

Symptoms:
In infected plants exhibit thin stem, leaves are small ,chlorotic ,crinkled and diseased plants are stunted ,become prematurely old. The roots of diseased plants show discreate elliptical lesions in the intial stage which are yellow, brown or black in colour. The infected plants can be easily pulled out from the ground. The wounds caused by nematode are invaded by secondary pathogens like fungi and bacteria.

8. Rice root nematode*(Hirschmanniella sp*.)

It is migratory, endoparasitic aquatic nematode. Spear shape well developed.Tail is abruptly conoid with a mucro at the terminous.

Sympotoms:
Damage to roots, retardation of growth ,stunting of plants chlorosis and reduction in tillers can be observed .
In case of severe infection, flowering is delayed by about 12 to 15 days resulting in late maturation of crop.
Infected root exhibits necrosis and browning and too much vaculation in the cortex.

9. Burrowing nematode(R*adopholus sp.)*

Host Range: Wheat, Barley. Rie, Oats, Pulses and Potato etc
Both adult female and male are vermiform .In female, the lip region rounded, marked by striae.
Tail is concoid to blunt with rounded terminus.in adult males, lip region is subspherical with or without striae.
Infective stage: Juvenile (J3) and adult Female

Symptoms:
Spreading decline of citrus: (*R.citrophilus)*
Infection results in stunted plants with sparce foliage, small fruits and retarded terminal growth.
Trees possess large number of drying branches. Typical lesions are found on new roots
Root and rhizome rot banana:
Leaf chlorosis, dwarfing, thin pseudostem, small bunches and premature lodging of plants occur due to nematode infection.
Yellow disease of pepper:
Infection results in increase in the number of yellow leaves , arrests growth of veins, leading to severe die back and death of the plants

10. False root knot nematode (*Nacobbus sp.)*

Female body is swollen and irregularly shaped . While male is typical filiform with
short tail enveloped by a narrow bursa. Large galls frequently contains no living female and only masses of darkened tissues are found . Cell wall collapses and enlarged cells are formed due to coalesion of adjacent cells .

11. Citrus nematode (*Tylenchulus semipenetrans*)

Females long with variable saccate body, stylet short , blunt tail. Male: Slender, vermiform, stylet and oesophagus degenerated
Symptoms:
Infection results in reduced terminal growth, chlorosis and shedding of leaves, dieback of branches, and reduction in size and number of fruits. Infected fibrous roots show irregular thickening and exhibit extensive necrosis which give them a rust brown colour.

12. Reniform nematode (*Rotylenchulus sp.)*

It is kidney shaped. Female irregular shaped, reniform, tail concoid with rounded terminus. In case of male tail is pointed; spicules are curved.
Infective Stage: Immature Female
Symptoms: Stunted growth of plants, premature decay and loss of secondary roots
Cotton: Browning and necrosis of epidermal cells are found on infected roots.
Castor: Infection results in reduction in growth, shedding of leaves, early flowers malformation and discolouration of seeds ultimately leading to dieback symptoms
Vegetables: Infection destroys the epidermal cells causing necrosis and browning of surrounding cells.

13. Lance nematode *(Hoplolaimus sp*.):

Yellowish in colour spear robust with prominent tulip shaped knobs. Vulva near middle of body, tail with rounded terminus

Symptoms:
H.indicus stunting of upper internodes ;drooping of leaves ;withering leaf tips the infected roots exhibit gallaries. young lateral roots developed reddish brown it is important in inducing the wilt fungi *fusarium monoliformae* .

14. Spiral nematodes *(Helicotylenchus sp.and Rotylenchus sp*.)

Spiral or helical shape, pore like tiny phasmids. *Rotylenchus* is migratory ectoparasite of roots
Helicotylenchus dihystera: Roots attacked by the nematode are blunt, malformed and much reduced in volume

15. Sheath nematode (H*emicycliophora sp.)*

Ovary is prodelphic and outstretched tail elongated, pointed and rounded. In males spicules are variously shaped, almoststright, slightlycurved, sicklelike. Due to feeding on root tips, galls are caused growth of hoots stop

16. Pin nematode (*Paratylenchus sp.):*

Usually they lie immobile in semi circularposition. Head is conical or rounded .tail isconical to various shaped point. Though the nematode have long styletsbut generally feed only upon epidermal tissues or roothairs.

17. Dagger nematode(X*iphinema sp*.)

Long, sword like stylet and given the common name "dagger nematodes" stylet very long hypodermal needle like. Tail short rounded to filiform.
X. index is a vector for NEPO virus transmitting the **fan leaf virus of grape**

18. Needle nematode*(Longidorus sp.):*

Longidorus are known to transmit polyhedral viruses.Eg*: L. elongates* transmits **raspberry ring spot viruses** and ***L. attenuates*** transmits **tobacco black ring viruses**.

19.Stubby root nematodes (*Trichodorus and Paratrichodorus*):

Stylet with hair like mural tooth. The nematodes with blunt rounded tails and thick cuticle. Transmit soil borne plant Viruses Feeding is confined to root tips causing ,stunted growth of roots hence, the
name "stubby root" It is a vector of NETU virus.

Common name	Scientific name	Infective stage
Cyst nematode	*Globodera spp*. and *Heterodera spp*.	***juvenile stage (J2)***
Foliar nematode	*Aphelechoides* spp.	***juvenile stage (J2)***
Lesion nematode	*Pratylenchus* spp.	***juvenile stage (J2)***
Reniform nematode	*Rotylenchulus renifomis*	*Immature Female*
Root – knot nematode	*Meloidogyne* spp.	***juvenile stage (J2)***
Stem and bulb nematode	*Ditylenchus* sp.	***juvenile stage (J4)***
Seed gall nematode	*Anguina tritici*	***juvenile stage (J2).***
Golden cyst nematode	*Globodera rostochinensis*	***juvenile stage (J2)***
Burrowing nematode	*Radopholus similis*	***Juvenile (J3) and adult Female***

Anatomy/ General structure of Nematode

Body is tubular and divided into three regions
1. Outer body tube or body wall.
2. Body cavity or Pseudocoelom in which excretory, nervous and reproductive system
3. Inner body tube or alimentary canal.
are present.

1. Outer body tube:

Consists of three layers; the cuticle, epidermis(hypodermis) and somatic muscle layer.
Cuticle: is the outermost layer non cellular secreted by epidermal cells derived from ectoderm serves as exoskeleton and protect the inner soft body tissues.

Cuticle has three layers
1. Outer cortical - has keratin and collagen disulphide group – give resistance nature.
2. Matrix - Consists of protein resembling collagen. It is not metabolically active layer.
3. Fibre Layer - protect nematode from environment.

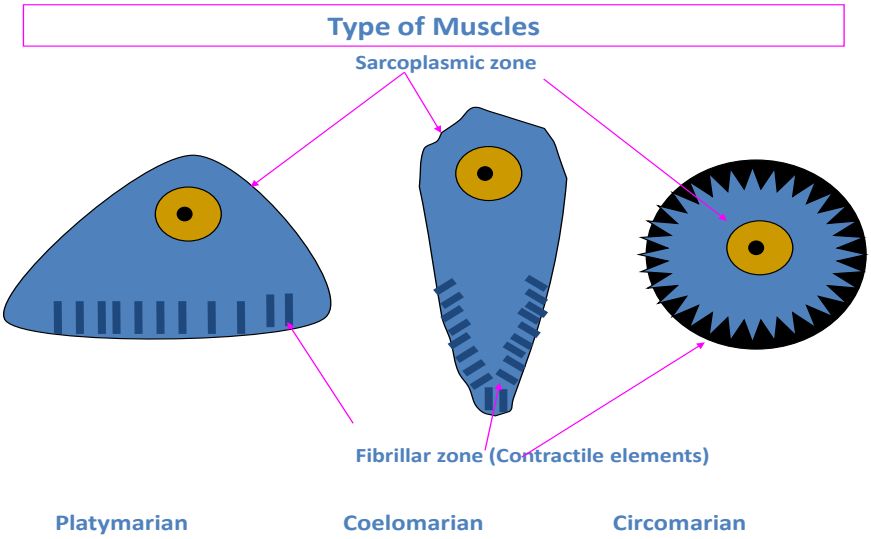
The functions of cuticle
- Cuticle gives definite shape and size to the body,
- acts as an exoskeleton,
- helps in movement,
- being semipermeable, it regulates permeability and provides important taxonomic characters for identification of nematodes.
- Protect the inner soft body tissues
- Being elastic, it maintains turgor pressure of the body.
- Cuticle is the seat of many sensory structures

Hypodermis / Epidermis : It lies between cuticle and somatic layer. It is responsible for formation of cuticle. In higher nematodes it is rich in lipids and glycogen which serve as source of reserve energy .

Somatic muscle layer:
These are arranged longitudinally beneath the hypodermis in the interchordal zones.

Based on arrangement the following types of cells are present
1. Holomyarian - Have 2 muscle cell in each zone.
2. Meromyarian - 2 or 5 muscle cells in each zone.
3. Polymyarian - More than 5 muscle cells per zone

Based shape of cells they are grouped in to –
1. Platymyarian - flat type of muscle cell
2. Coclomyarian - ‘U’ shaped cell muscle fibre are adjacent and perpendicular to epidermis
3. Circomyarian` - Muscle fibre completely surround the cytoplasm
Nematode muscles are unusual, in that muscle sends branches to nerves rather than nerves send branches to muscle.



2.Body Cavity:

Body cavity is devoid of **mesoderma**l lining,hence, it is called **pseudocoelom**.. Externally it has somatic muscle cells and internally cells of alimentary canal.Pseudocoelom is filled with fluid which is rich in proteins and bathes all internal organs and functions as part of the **turgor pressure** system on the exoskeleton. The pseudocoelom is also linked with a network of fenestrated membrane that support visceral organs.

(I) Reproductive System in Nematodes

The nematodes are generally dioecious. Majority of plant parasitic nematodes do not exhibit any differences as far as body shape. Both sexes are vermiform. However, sexual dimorphism is observed in some genera *viz.*, *Meloidogyne, Heterodera, Globodera, Rotylenchulus, Tylenchulus* and *Nacobbus*. The females of these genera become enlarged and assume different shapes after attaining maturity.

Sexually reproduced -Most of the cases sexual (Amphimictic) reproduction is reported -males and females are present and both are separately: Lesion, stunt and lance nematodes.
Some have automixis type reproduction – Generally the males are lesser in numbers then females or may be completely absent, which indicates a tendency towards hermaphroditism and parthenogenesis.
Hermaphroditism means individuals which function both as male and females. It may be of two types:
Syngonic hermaphroditism : where the gonad first produces sperms that are stored and fertilize the eggs developed by the same gonads later, eg. *Caeonorhabditis*spp.
Digonic hermaphroditism : where both sperms and ova are simultaneously produced by different parts of the same gonad, e.g. *Helicotylenchus*spp
Parthenogenesis: In which males are not found or lesser in number and the fertilization of ovum does not take place with sperm or spermatocytes, either produced by males or females but females lays fertilized eggs without fertilization e.g. *Meloidogyne, Heterodera, Tylenchulus*and *Ditylenchus*.
Sex reversal: In root-knot nematodes sometimes during scarcity of food or in unfavorable conditions the developing females get converted into males.
Inter sexes: In genera like *Meloidogyne*and *Ditylenchus*inter sexes are found. In such cases one reproductive system acts as male gonad and other one as female gonad.

Female Reproductive System

- ❑ The various parts of the female genital tract are ovary (gonad), oviduct, spermatheca, uterus, vagina and vulva.

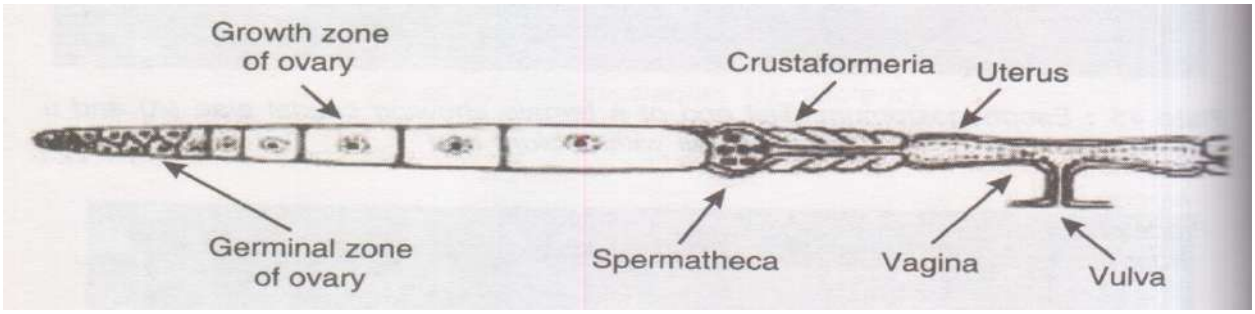
(1) The ovary

- ❑ It is hollow elongate tube.
- ❑ The apical end of ovary has a cap cell at the tip which is called as ***germinal zone***or zone of multiplication in which rapid cell division takes place to give rise germinal cells (oocytes).
- ❑ This region is followed by ***growth zone*** which constitutes the greater part of ovary.

- The oocytes or germ cells in this zone become big and ripe which are generally arranged in single rows.
- After maturity they are called ***oogonia***.

(2) Oviduct

- Next to growth zone of ovary, the gonad has oviduct.
- The oocytes when ripe they pass in to oviduct.
- Oviduct may serve as spermatheca in some nematode. However in *Paratylenchus*, the spermatheca is in the proximal part of uterus or in the post-vulvar sac at the distal end of gonad.



(4) The Spermatheca

- The upper part of uterus serves as spermatheca in some nematodes.
- After maturation, oocytes pass down to spermatheca via oviduct.
- Spermatheca stores the sperms (male sex cell) after mating and when the oocytes pass through it, they get fertilized.

(5) Crustaformeria or Quadricolumella

- It is the region of uterus consisting of specialized cells that secrete egg membrane.
- Layers on the egg (ovum) are laid, while it is passing through the glandular crustaformeria.

(6) The uterus

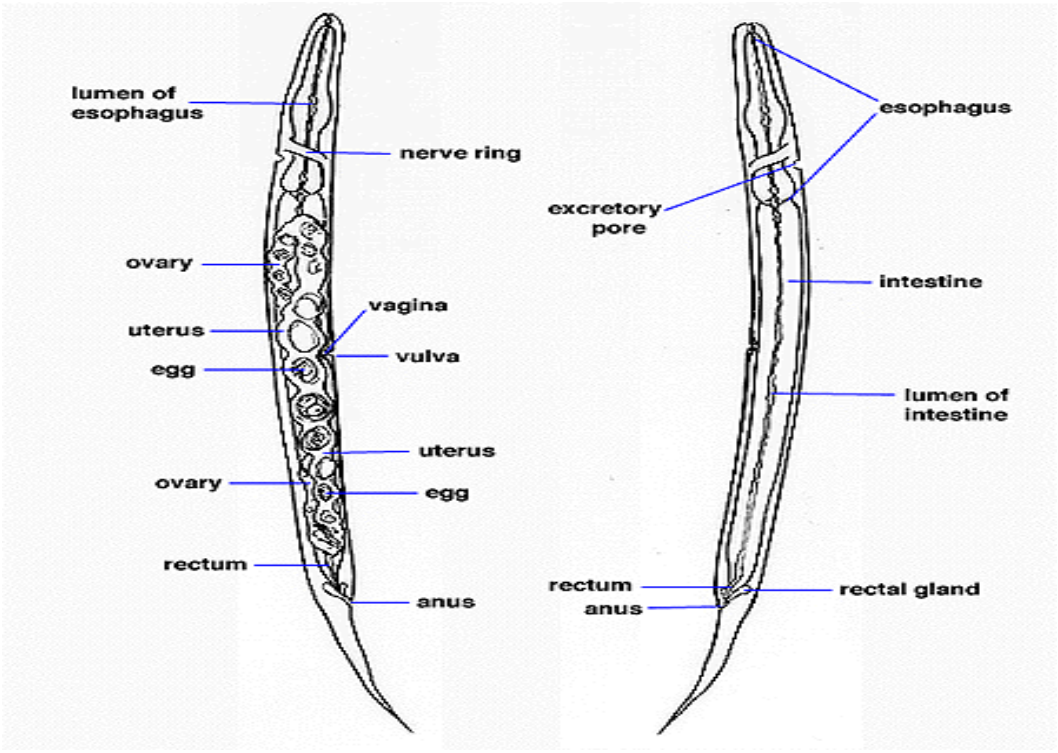
- It is a structure that contains developed eggs prior to egg laying.
- It is the largest and most complex part of the gonad.
- The functions of uterus are fertilization, egg shell formation and laying of eggs.
- As stated above, the upper part of uterus serves as spermatheca in some nematodes.

(7) Vagina

- The uterus entered in common vagina, which is a short, narrow and flattened tube lined with cuticle and provided with muscles near to vulval opening.
- It helps in the expulsion of egg to the outside and opens on the ventral side through a slit-like opening, the vulva.

(8) Vulva

- The vagina opens through female gonopore, the vulva.
- The eggs are expelled through a vulva which is normally situated middle of the body.

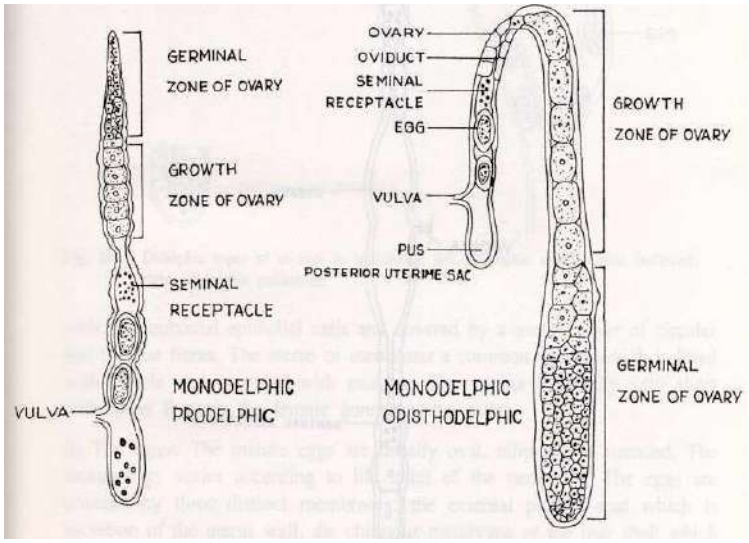


Types of Ovaries in female Nematodes

- On the basis of number and arrangements ovaries may vary in nematodes.
- **On the basis of number**

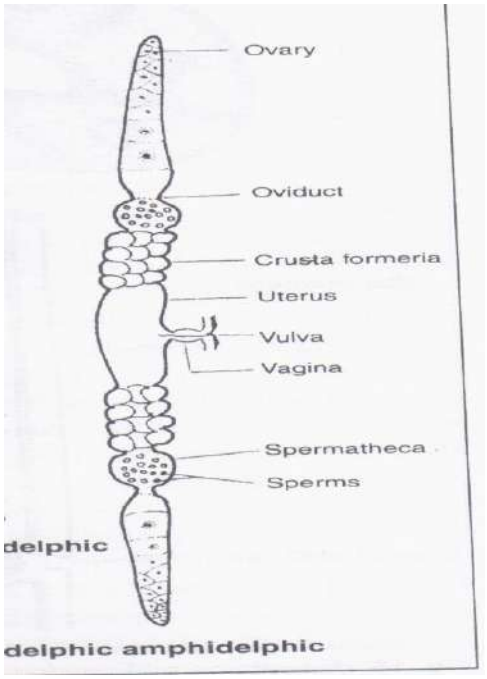
1. **Monodelphic or Monoovarial:** When the nematodes have **a single ovary** thenthe female is called as monodelphic. e.g.*Pratylenchus*
2. **Didelphic or Diovarial:** When the nematodes have **two ovaries** then the female is called as didelphic.

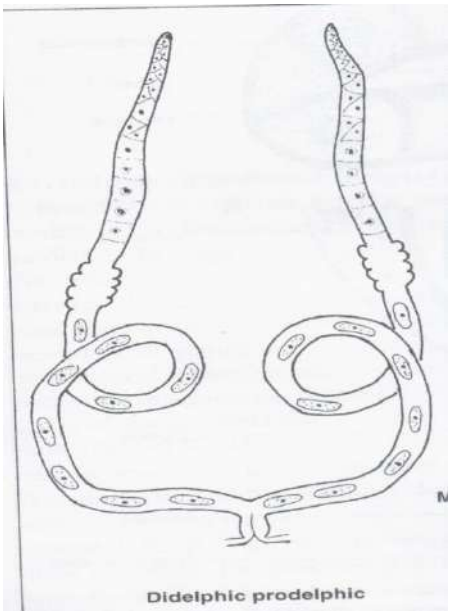
- **On the basis of arrangement**
- **Monodelphic Prodelphic:** When females possess only one gonoduct (monodelphic) which may be directed anteriorly (prodelphic) then female is called as Monodelphic prodelphic.
- **Monodelphic Ophisthodelphic :**When females may possess only one gonoduct (monodelphic) which may be directed posteriorly (ophisthodelphic) then female is called as Monodelphic Ophisthodelphic . e.g. *Xiphinema sp.*



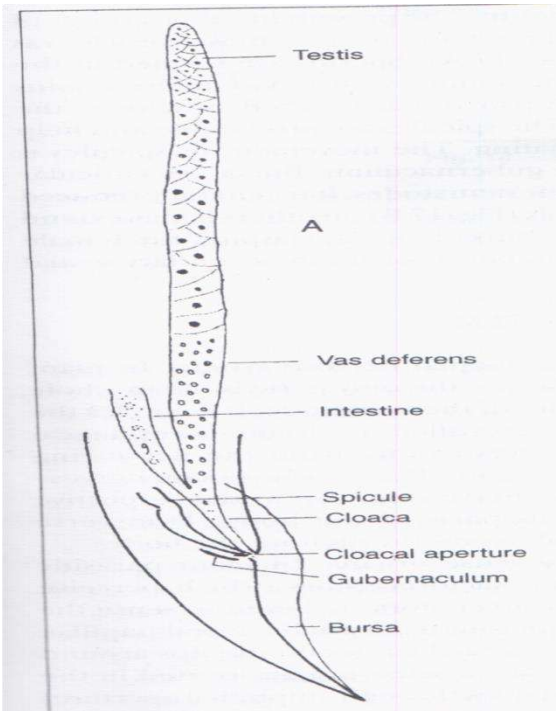
- **DidelphicProdelphic:** When females possess two ovaries which are directed anteriorly then female is called as DidelphicProdelphic. e.g. *Heterodera, Globodera, Meloidogyne*

- **Didelphic Ophisthodelphic :**When females possess two ovaries and both ovaries are directed posteriorly then female is called as MonodelphicOphisthodelphic. e.g. *Ditylenchus*
- **Didelphic Amphidelphic:** The two ovaries are opposite to one another, such as one is anteriorly directed and other posteriorly directed. e.g. *Helicotylenchus, Hoplolaimus, Tylenchorhynchus*





- **Some Terminology**
- **Vulval flap:** The vulval opening is a transverse slit and not covered with any flap, but in *Agelenchus*and *Coslenchus* vulva is covered with a membranous flap known as vulval flap.
- **Epiptygma:** The vaginal tube in *Hoplolaimus* is provided with a cuticular sclerotised structure encircling the tube known as epiptygma.
- Ovary in most of the plant parasitic nematodes is always straight and does not curve back. Such ovaries are called as **outstretched ovaries.** e.g. *Tylenchorhynchus, Radopholus* and *Hirschmaniella*etc.
- In *Dorylaimid* nematodes, the tip of the ovary is curved back. It is known as **reflexed ovary.**
- **Hologenic State:** Ovary is called hologenic, if it produced oocytes throughout it length.
- **Telogenic State:** Ovary is called telogenic, if it producing oocytes only at its distal end.



Male Reproductive System

- The male reproductive system generally consists of three primordial parts: the testis, seminal vesicle, and vas deference.

(1) The Testis

- The production of sperms takes place in testis. In the testis, the germinal and growth zone can be easily distinguished.
- In germinal zone spermatogonial division takes place, while in growth zone, spermatocytes increases in size. The spermatocytes are arranged in single or double rows.

(2) Seminal Vesicle

- At the end of the growth zone of testis, maturation of spermatocytes takes place and the spermatocytes are stored in seminal vesicle.

(3) Vas deference

- It consist of an anterior glandular region and posterior muscular region and containing the ejaculatory duct which opens into the cloaca at the posterior end.
-
- Vas deferens joins posteriorly with rectum to form cloaca.

(4) Ejaculatory glands

- Ejaculatory glands are associated with ejaculatory duct.
- The ejaculatory duct helps in the ejection of sperms during fertilization.
- It also secret the adhesive cement deposited during copulation with the vulva in some species of nematodes.
- It tapers gradually and opens ventrally into the cloaca.
- The cloaca is provided male copulatory structures such as spicules, gubernaculums etc.

Other major male reproductive organ

(5) Spicules

- A pair of hard curved structures, the spicules are located in a cloacal pouch.
- The spicules are protrusible and help in the insemination of females during mating by spreading the female vulva.

(6) Gubernaculum

- The movement of spicules is guided by a sclerotized plate-like structure, the gubernaculum. It is guiding structure for spicules.

(7) **Bursa or Caudal alae**

- A flap like structure called bursa is usually present covering the tail region. Bursa helps to clasp the female during mating.

Terminology

- **Monarchic:** The nematode may have one testis are called monarchic. e.g. *Tylenchids*
- **Diarchic:** The nematode may have two testis are called diarchic. e.g. *Trichodorus*, *Meloidogyne*

(II) Excretory system:

Different types of excretory systems are found in Nematodes, **Vonlinstow**, 1909 classified nematodes into **4** groups based on excretory system viz:

1. Secerentes 2. Resorbents 3. Pleuromyarli 4. Adenophori.Now secerentes are synonymous to resorbants; Pleuromyarli with Adenophori. Morphologically excretory system is of two types

(1) **Renette type** has large cell in the pseudo coelomic cavity known as renette or ventral gland, which is connected to the excretory pore by a duct Eg.

Adenophorea

(2) **Tubular** or **Canalicular** type : Eg. Secernentea

Tubular type that may be

(a) simple H shaped (Oxyuroid type)

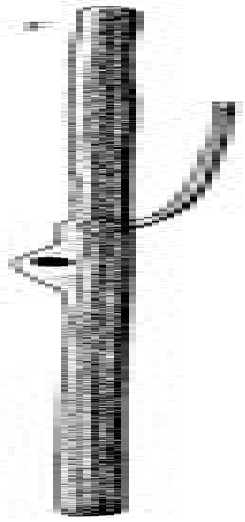
(b) Rhabditoid type

(c) Ascaridid type 3 Nuclei (Invented U shape)

(d) Asymmetrical type or uni nucleate system.

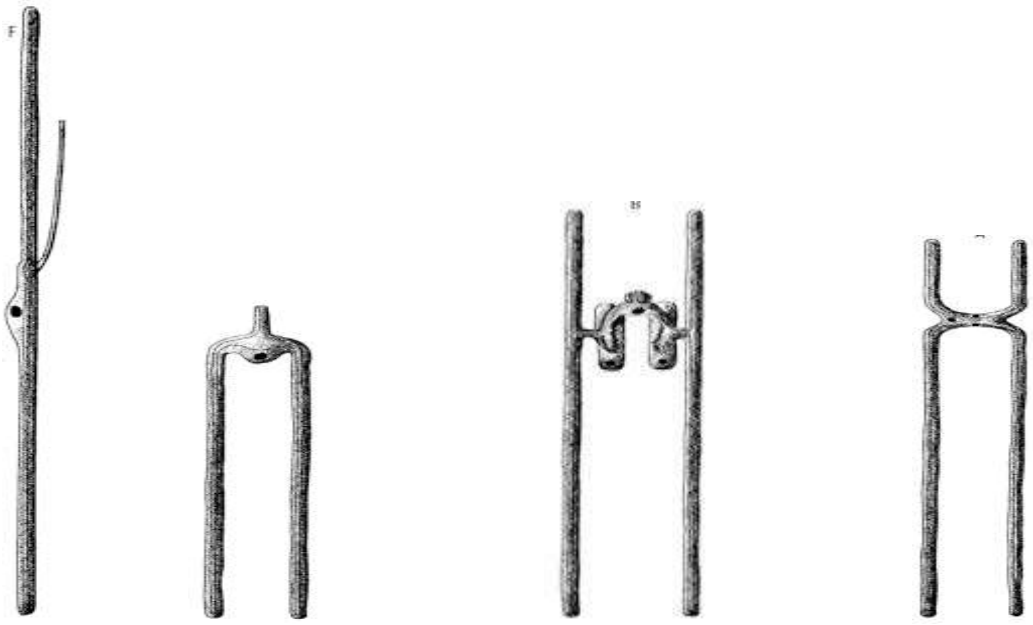
In tylenchida, the terminal duct points the lateral canal and there are (2) nuclei, The excretory system also plays important role in osmoregulation, in addition to excretion so it is called as excretory secretory system.

Glandular type



Tubular type:-

Tylenchid type



Inverted 'U' shaped type **Rhabditid type** **Simple 'H' shaped**

(III) Nervous system:

It is not well studied in nematodes particularly in Tylenchida due to its small size.

The central nervous system has nerve ring, associated ganglia and the ventral nerve.

The nerve ring is broad, flat present around the oesophagus isthamas.

In Tylenchida, it encircles the middle part of the oesophagm.

In Dorylaimida, it is present around the narrow anterior part of oesophagus.

Ventral nerve is a chain of ganglia. The first and largest ganglia is known as

Retro – vesicular ganglia

Sensory organs

Amphids : lateral pair of sensory organs or chemoreceptors present in the cephalic region of the nematode. They are larger in males than females.

Phasmids: Paired lateral sense organs usually one on each side of tail.

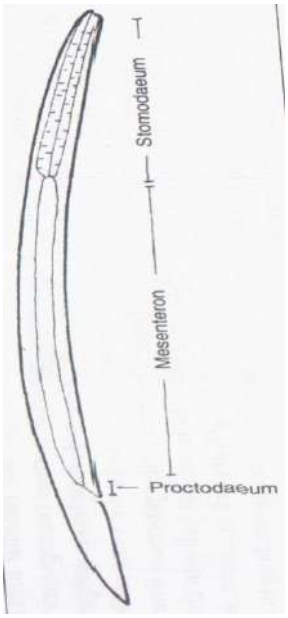
Deirids: paired papillae present in the oesophageal mid region of the body opposite to the excretory pore. Do not open outside Also called as Cervical papillae function as mechanoreceptors.

Hemizonids and Hemizonion : Highly refractive biconvex structure forming a semicircle on the ventral side of the body and ending at the lateral fields. Hemizonion is a small nerve commisure.

Cephalids: Highly refractive band like structure present in the cuticle dorsally**.**

Caudalids: small nerve commissure in caudal region slightly posterior to anus.

3.Inner body tube / Digestive system



- The inner body tube forms the alimentary canal or the gut of plant parasitic nematodes which can be divided into 3 zones;

A. **The stomodaeum** (fore-gut)

B. **The mesenteron**(mid-gut) and

C. **The proctodaeum**(hind-gut).

- *Stomodaeum and proctodaeum are internally lined with cuticle.*

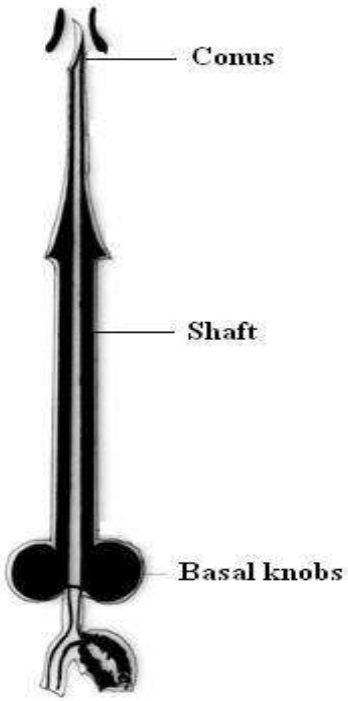
(A) Stomodaeum

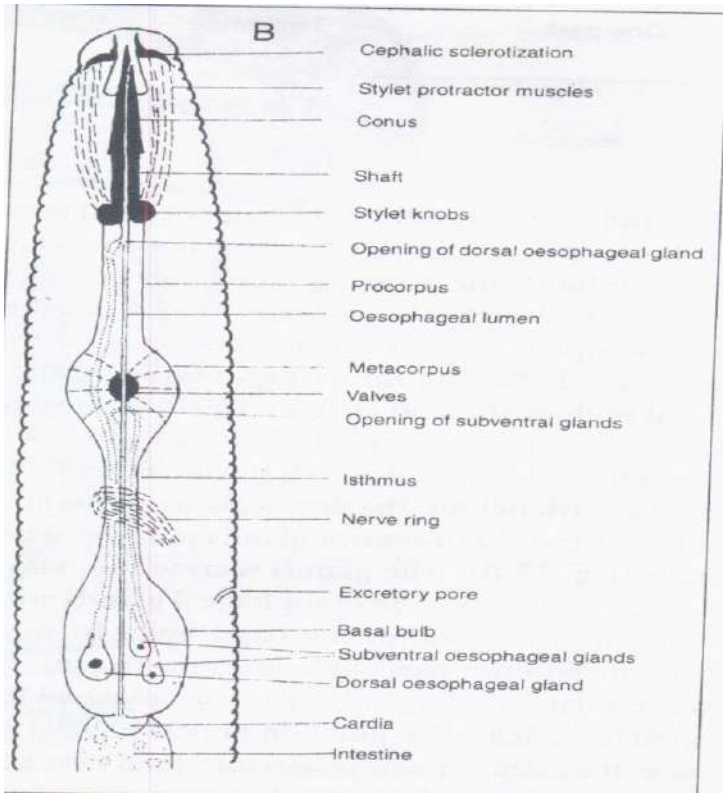
- It includes the mouth and lips, the stoma and the oesophagus.

- It starts at the oral aperture or the mouth which is located terminally and is surrounded by 6 lips (hexaradiate). (2 sub-dorsal, 2 sub-ventral and 2 lateral in position).
- **Stoma or Buccal cavity:** The stoma, which is also called as mouth cavity or buccal cavity forms the feeding apparatus of nematodes mouth and it is the portion of the inner body tube lies between the mouth and the oesophagus.

Stylet

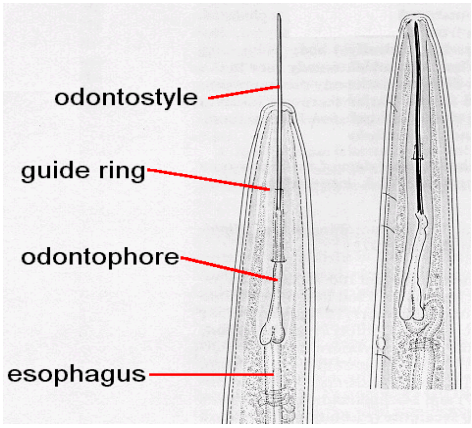
- Plant parasitic nematodes are armed with a protrusible **stylet** which is usually hollow and functions like a hypodermic needle.
- In, Secernentea, the stylet is thought to be derived from fusion of the stomatal lining and therefore called as **stomatostylet.**
- The stamatostylet consists of a anterior cone, a cylindrical shaft and three rounded basal knobs.
- In Adenophorea, the stylet is thought to be derived from a tooth and, therefore, it is called as **odontostylet.**
- The flanges that serve as points of attachment for the stylet protractor muscles(regulate the movement).
- In some plant parasitic nematodes like *Trichodorous*and *Paratrichodorus*the odontostylet is distinctly curved ventrally, lacks flanges and it is not hollow.
- In functions to pierce the cell wall of the root.
- The nematode secrets a hollow tube out of its stoma that connect it with the plant. This feeding tube serves as the interface between the nematode and the plant.





Types of Stylet

- **Stomatostlet**: Stylet emerging out from mouth part or stoma of nematode. eg. Root-knot, stunt, reniform nematodes. **Order : Tylenchida**
- **Odontostylet or Onchiostylet:** Stylet emerging out from Oesophagus of nematode eg. Dagger nematode, Needle nematodes. **Order: Dorylaimida.**



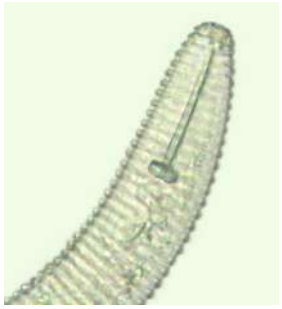
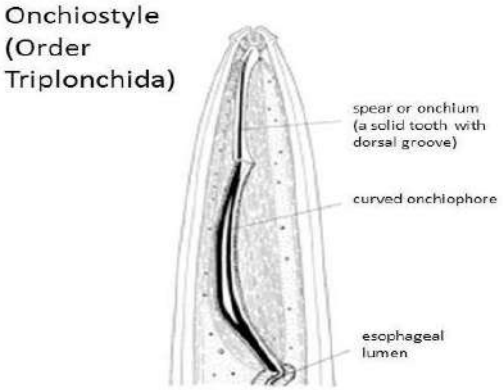


Fig.Odontostylet or Onchiostylet **Stomatostylet**

- **Solid or curved stylet**: Stubby root nematode. **Order: Dorylaimida**



Oesophagus or pharynx is basically a pumping organ which sucks in and pushes the ingested food material into the intestine.

- In Adenophorea, the **oesophagus is divided into a narrow anterior procorpus and a broad posterior corpus**. Three to five oesophageal gland cell empty into the lumen one dorsal and two to four subventral in position.

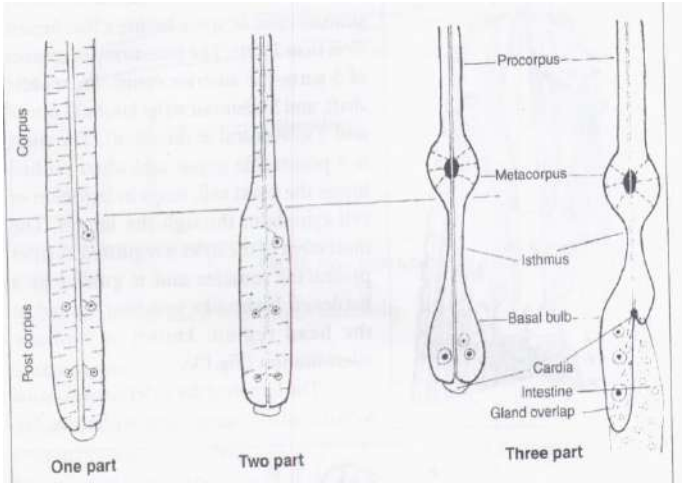
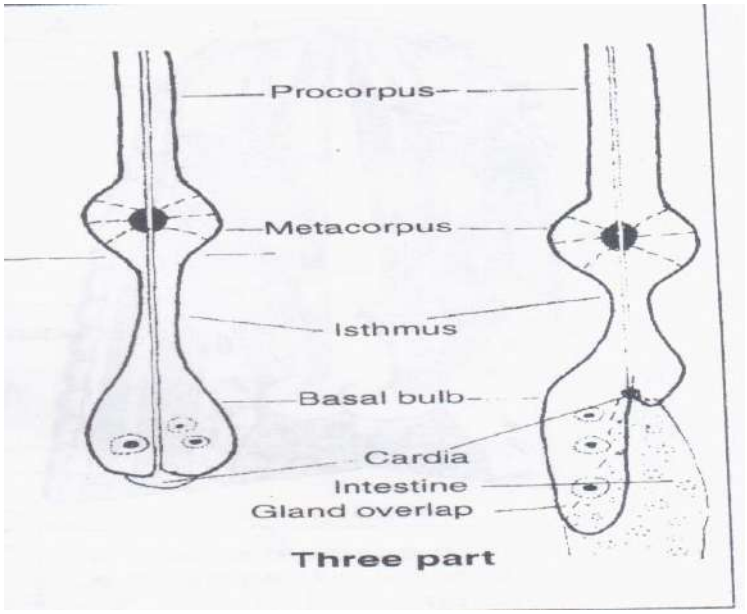


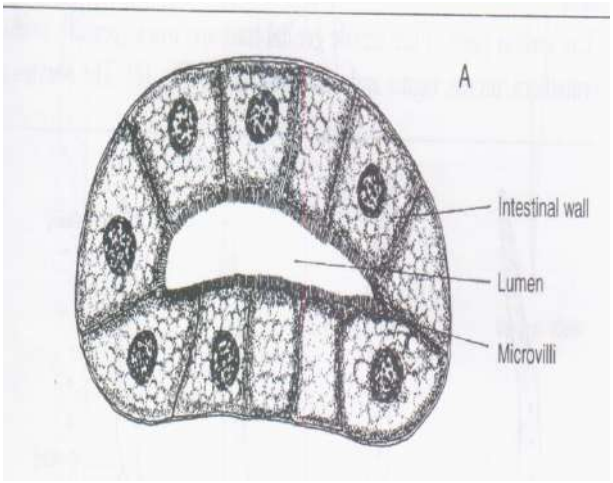
Fig. Types of Oesophagus

- In Secernentea the oesophagus is divided into three distinct regions(Tripertile), such as narrow **procorpus,** followed by a broad muscular **median bulb or pump,** a narrow **isthmus and gland lobe.**The gland lobe may overlap the intestine in some genera and contain three to six gland cells (One dorsal and tow dub – ventral).
- The oesophagus has valve (cordia) at the posterior end which prevents the regurgitation of food.



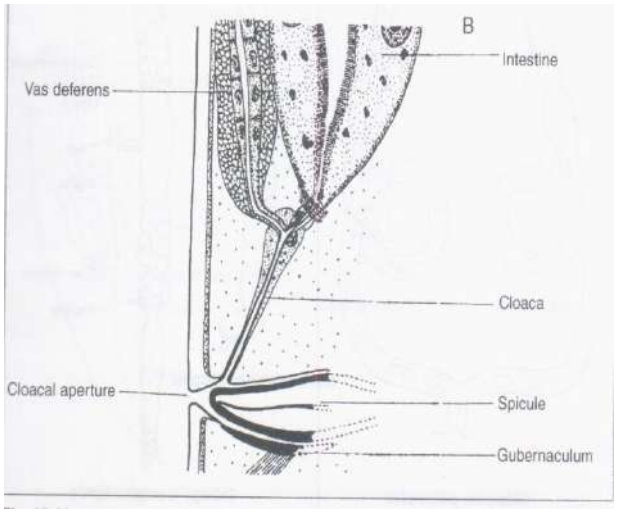
(B) Mesenteronor Intestine

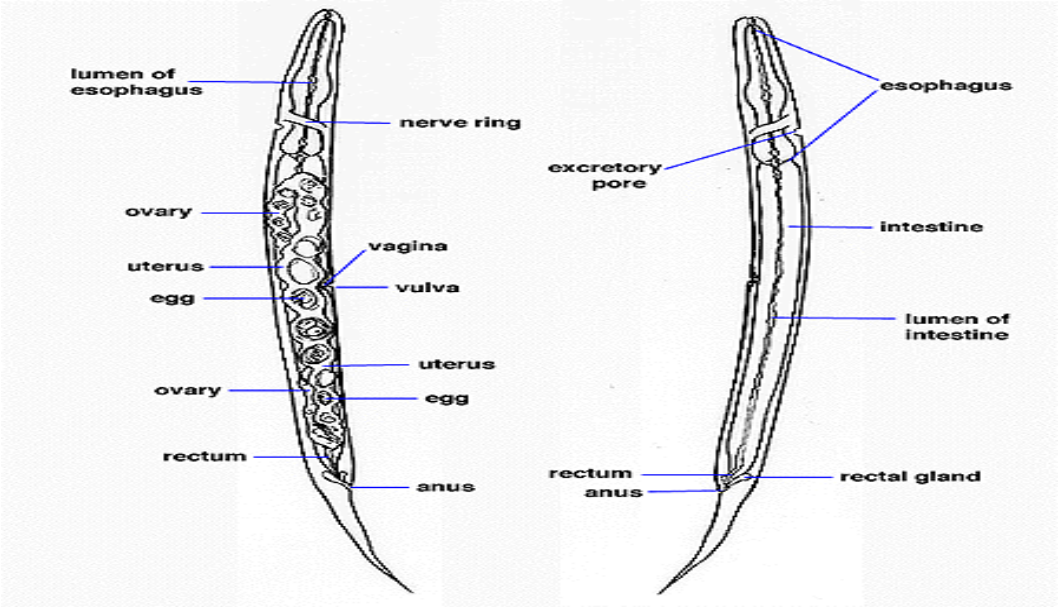
- It consists of a simple straight tube i.e. the **intestine** running most of the length of the body.
- The intestine is generally divided in to three region anterior or ventricular region, the mid intestinal region and posterior pre-rectal region.
- The **microvilli** are finger like projection of the plasma membrane projecting in to the intestinal regions. They increase the surface area of the intestine and are both secretary and absorptive in function. The whole intestine is separated from the **pseudocoelom by a basement membrane.** The food moves in the intestine by the ingestion of more food and also by locomotory activity of the nematode.



Proctodaeum

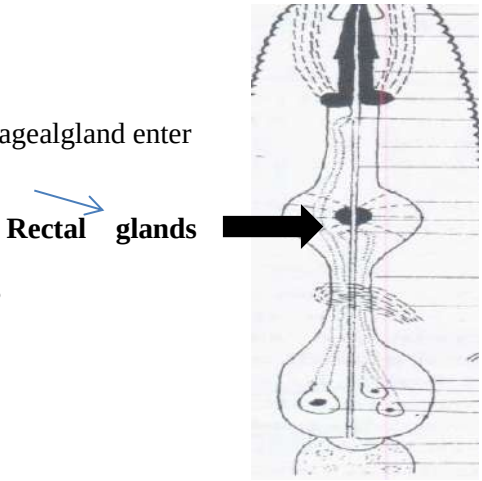
- Proctodeum comprises rectum and anus.
- The intestinal tube is connected with a narrow small tube at the posterior end, through a valve known as **rectum**. It regulates the flow of undigested food material which is to be passed outside the nematode body through a ventrally located aperture known as anus.
- In case of males, the reproductive system joins the rectum to form a common tube called **cloaca,** which opens to the outside through **cloacal aperture** on the ventral side and serve as common opening for digestive and reproductive systems**.**





Glands

- Oesophageal and rectal glands are present in nematodes. The oesophagealgland enter the stomodeum and rectal gland enter proctodeum.



- **Rectal glands** responsible for the copious production of gelatinuous mucopolysacccharide matrix in which eggs are deposited as a mass.
- It protects the eggs from adverse environmental conditions.

Function of Digestive system

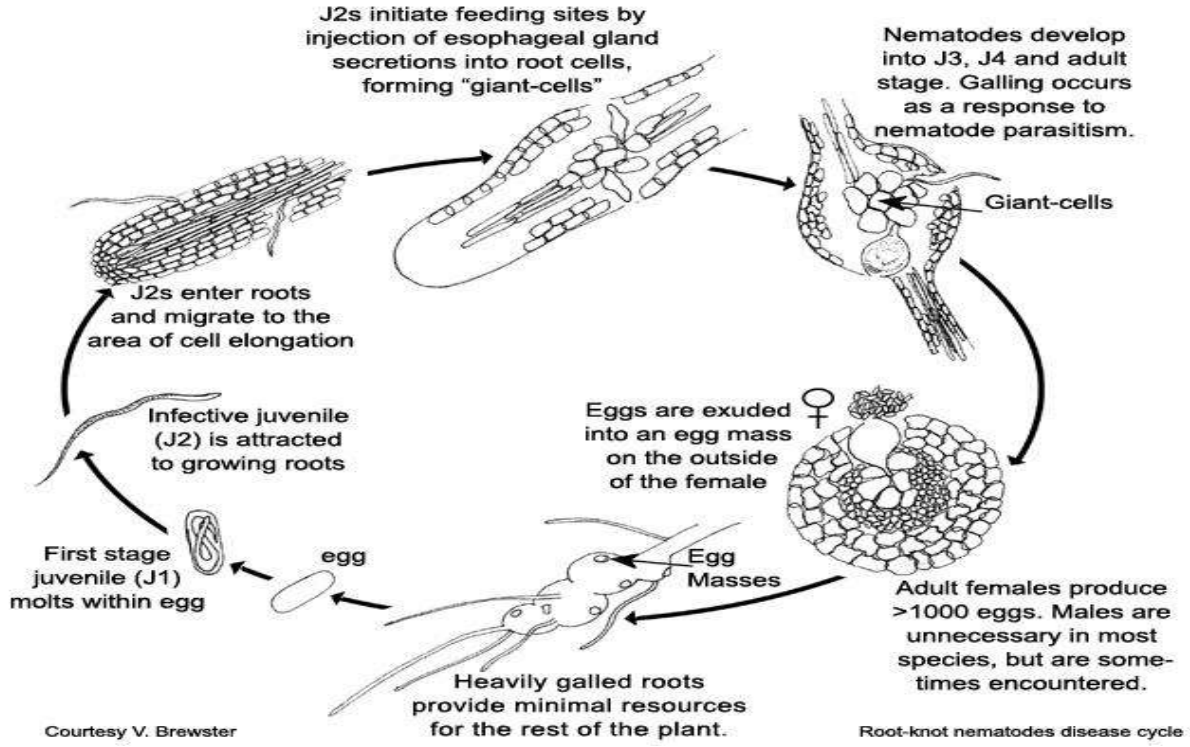
- Digestive juices which is secreted from dorsal oesophageal glands are injected into the host plant cell by means of the stylet. During feeding, a distinct zone develop around the feeding site in the host cell. There are two feeding phases- **1) Injection phase or salivation phase** and **2) Ingestion phase**.

1) **Injection phase or salivation phase:** During this phase, the flow of salivary juices into the host cell occurs due to contraction of lateral muscle of the median bulb.

2) **Ingestion phase:** During this phase, rhythmical contraction of the posterior part of oesophagus associated with the median bulb occurs

LIFE CYCLE OF ROOT KNOT NEMATODE

The life cycles of root-knot (*Meloidogyne* sp.) nematodes illustrates some of the possible modifications of the typical life cycle. These nematodes are sedentary endoparasites. The invasive or second stage juvenile (J2) hatches from the egg and seeks a feeding site within a root. The juvenile molts to the J3 and begins enlarging as the reproductive system develops. Nematodes which become females are no longer able to leave the root. They continue to enlarge as they go through the J3 and J4 stages. During this time, cells around the head of the nematode enlarge to form nurse cells or giant cells. For root-knot nematode, galls will typically develop on the root. Upon becoming adults, root-knot nematodes will begin to lay eggs (up to several hundred) which are contained in a gelatinous matrix at the posterior end of the body. The egg mass may be within the root or partly or wholly exposed on the root surface while the swollen body of the female remains within the root. Eggs in matrices often remain attached to root fragments in the soil after the female dies. Root-knot egg and juvenile populations decline by up to 70 to 90 percent during winter in the absence of reproduction. This poor survival is compensated by a very wide host range that enables overwintering juveniles and those hatching from eggs in spring to reproduce on crop or weed hosts that are present during spring an summer.



TAXONOMY AND CLASSIFICATION

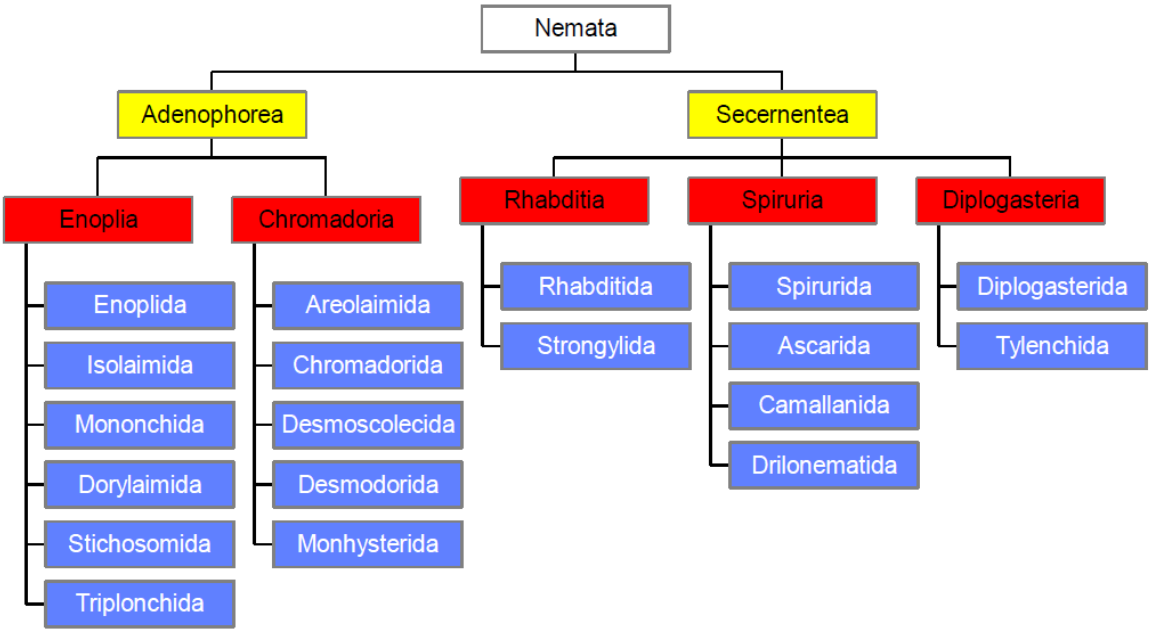
Taxonomy: Law of arrangement of living.

Taxonomy and systematics = these terms are largely synonymous. Taxonomy deals with identifying, naming and classifying organisms, whereas systematics has a broader definition and includes aspects of the phylogeny, evolution, biogeography, genetics and physiology of the organisms.

Classification: The arrangement of animals and plants in taxonomic groups according to their observed similarities (including at least kingdom and phylum in animals, division in plants, and class, order, family, genus, and species). Nematodes are placed in the group invertebrata,

Kingdon Animalia. Under separate phylum, Nemata/Nematoda, which consist of two classes, Secernentea and Adenophorea.

Taxonomic Classification of Nematodes



Difference/Diagnostic characters of class Secernentea and Adenophorea

Secernentea (Phasmida)	Adenophoreea (Aphasmida)
Amphidial opening is on the head near the lip region.	Amphids open behind the head i.e. post labial
Lateral canals open into the excretory duct	Lateral canals and excretory duct end in a cell
Oesophagus is divided into procorpous, median bulb, isthmus and basal bulb.	Oesophagus is cylindrical with an enlarges glandular base
Male tail with bursa (Caudal alae)	Male tail lacks bursa but posses genital papillae.
Glands are absent	Caudal glands are present
Phasmids are present	Phasmids are absent
The mesenterial tissues are less developed	The mesenterial tissues are well

TAXONOMIC CLASSIFICATION OF SOME IMPORTANT PLANT PARASITIC NEMATODES

KINGDOM ;Animalia PHYLUM; Nematoda

Genus	family	Super family	Sub order	Order	Class
Meloidogyne, Globodera, Heterodera	**Heteroderidae**	**Heteroderoidea**	**Tylenchina**	**Tylenchida**	**Scerernentea**
Rotylenchulus	**Nacobbidae**				
Ditylenchus, Anguina	**Tylenchidae**	**Tylenchoidea**			
Pratylenchus, Radopholus	**Pratylenchidae**				
Holplolaimus	**Hoplolaimidae**				
Aphelenchoides	**Aphelenchidae**	**Aphelenchoidea**			

Xiphinema, Longidorus,	**Longidoridae**	**Dorylaimoidea**	**Dorylaimina**	**Dorylaimida**	**Adenophorea**
Trichodorus, Paratrichodorus	**Trichodoridae**	**Trichodoroidea**			

Taxonomic/Systematic position of *Tylenchus filiformis parvus*.

Taxonomic category	**Suffix**	**Name**
Kingdom	variable	Animalia
Phylum	-a	Nematoda
Class	-ea	Secernentea
Subclass	-ia	Tylenchia
Order	-ida	Tylenchida
Suborder	-ina	Tylenchina
Superfamily	-oidea	Tylenchoidea
Family	-idae	Tylenchidae
Subfamily	-inae	Tylenchinae
Tribe	-ini	Tylenchini
Genus	variable	*Tylenchus*
Species	variable*	*filiformis*
Subspecies	variable*	*parvus*

NEMATODE MANAGEMENT

Plant parasitic nematodes can be controlled by several methods. The nematode control aims to improve growth, quality and yield by keeping the nematode population below the economical threshold level. The control measures to be adopted should be profitable and cost effective. It is essential to calculate the cost benefit ratio before adopting control measures.

The nematode management methods are

1. Regulatory (Legal) method

2. Physical method

3. Cultural method

4. Biological method

5. Chemical method

1. Regulatory Method

Regulatory control of pests and diseases is the legal enforcement of measures to prevent them from spreading or having spread, from multiplying sufficiently to become intolerably troublesome. The principle involved in enacting quarantine is exclusion of nematodes from entering into an area which is not infested, in order to avoid spread of the nematode Quarantine principles are traditionally employed to restrict the movement of infected plant materials and contaminated soil into a state or country. Many countries maintain elaborate organizations to intercept plant shipments containing nematodes and other pests. Diseased and contaminated plant material may be treated to kill the nematodes or their entry may be avoided. Quarantine also prevent the movement of infected plant and soil to move out to other nematodes free areas.

Plant Quarantine in India

The Destructive Insects and Pests Act, 1914 (DIP) was passed by the Government of India which restricts introduction of exotic pests and disease into the country from abroad. The agricultural pests and disease acts of the various states prevent interstate spread of pests within the country. The rules permit the plant protection advisor to the government of India or any authorizes officer to undertake inspection and treatments. Strict regulations have been made against *G. rostochiensis*, the potato cyst nematode and *Rhadinaphelenchus cocophilus*, the red ring nematode of coconut. Domestic quarantine regulations have also been imposed to restrict the movement of potato both for seed and table purposes in order to present the spread of potato cyst nematode from Tamil Nadu to other states in India.

2. Physical control

It is very easy to kill the nematodes in laboratory by exposing the nematodes to heat, irradiation and osmotic pressure etc., but it is extremely difficult to adopt these methods in field conditions. These physical treatments maybe hazardous to plant or the men working with the treatments and the radiation treatments may have residual effects

Heat treatment of soil

Sterilization of soil by allowing steam is a practice in soil used in green house, seed beds and also for small area cultivation. Insects, weed seeds, nematodes, bacteria and fungi are killed by steam sterilization. In such cases steam is introduced into the lower level of soil by means of perforated iron pipes buried in the soil. The soil surface needs to be covered during steaming operation. Plastic sheets are used for covering. In the laboratory and for pot culture experiments autoclaves are used to sterilize the soil.

Hot water treatment of planting material

Hot water treatment is commonly used for controlling nematodes. Prior to planting the seed materials such as banana corms, onion bulbs, tubers seeds and roots of seedlings can be dipped in hot water at **50 – 55 ˚C for 10 minutes** and then planted.

Irradiation

Irradiation also kills the nematode. Cysts of *G. rostochiensis* exposed to 20,000 Ɣ contained only dead eggs and at 40, 000 Ɣ exposure, the eggs lost their contents. *Ditylenchus myceliphagus* in mushroom compost exposed to Ɣ rays between 48,000 to 96,000 Ɣ inactivated the nematodes. UV light also kills the nematodes. But this irradiation is not practically feasible under field conditions.

Osmotic pressure

Feder (1960) reported 100% nematode mortality when sucrose or dextrose was added to nematode infested soil @ 1 to 5% by weight. But these methods are not practical and economical.

Washing process

Plant parasitic nematodes are often spread by soil adhering to potato tubers, bulbs and other planting materials. Careful washing of such planting material helps to avoid the nematodes in spreading in new planting field. Washing apparatus for cleaning potato and sugar beet tubers are commercially developed and are being used in many countries.

Seed cleaning

Modern mechanical seed cleaning methods have been developed remove the seed galls from normal healthy wheat seeds.

Ultrasonic

Ultrasonic have little effect on *Heterodera* spp. The use of this ultrasonic is not practically feasible.

3. Cultural methods

Cultural nematode control methods are agronomical practices employed in order to minimize nematode problem in the crops.

Selection of healthy seed material

In plants, propagated by vegetative means we can eliminate nematodes by selecting the vegetative part from healthy plants. The golden nematode of potato, the burrowing, spiral and lesion nematodes of banana can be eliminated by selecting nematode free plant materials. The wheat seed gall nematode and rice white tip nematode can be controlled by using nematode free seeds.

Adjusting the time of planting

Nematode life cycle depends on the climatic factors. Adjusting the time of planting helps to avoid nematode damage. In some cases, crops may be planted in winter when soil temperature is low at that time the nematodes cannot be active at low temperature. Early potatoes and sugar beets grow in soil during cold season and escapes cyst nematode damage since the nematodes are not that much active, to cause damage to the crop during cold season.

Fallowing

Leaving the field without cultivation, preferably after ploughing helps to expose the nematodes to sunlight and the nematodes die due to starvation without host plant. This method is not economical.

Deep summer ploughing

During the onset of summer, the infested field is ploughed with disc plough and exposed to hot sun light, which in turn enhances the soil temperature and kills the nematodes.

Solarisation of soil: Exposure of soil covered with polythene cover during April- June will effectively reduce nematode population in soil.
For raising small nursery beds for vegetable crops like tomato and brinjal seed beds can be prepared during summer, covered with polythene sheets (25-micron LLDPE) which enhance soil temperature by 5 to 10˚C which kills the nematodes in the seed bed. This method is very effective and nematode free seedling can be raised by soil solarization using polythene sheets.

Manuring: Raising green manure crops and addition of more amounts of farm yard manure, oil cakes of neem and castor, press mud and poultry manure etc enriches the soil and further encourages the development of predacious nematodes like *Mononchus* spp. and also other nematode antagonistic microbes in the soil which checks the parasitic nematodes in the field.

Flooding: Flooding can be adopted where there is an enormous availability of water. Under submerged conditions, anaerobic condition develops in the soil which kills the nematodes by asphyxiation. Chemicals lethal to nematodes such as hydrogen sulphide and ammonia are released in flooded condition which kills the nematodes.

Trap cropping: Two crops are grown in the field, out of which one crop is highly susceptible to the nematode. The nematode attacks the susceptible crop. By careful planning, the susceptible crop can be grown first and then removed and burnt. Thus, the main crop escapes from the nematode damage. Cowpea is highly susceptible crop can be grown first and then removed and burnt. Cowpea is highly susceptible to root-knot nematode and the crop can be destroyed before the nematodes mature.

Antagonistic crops: Certain crops like mustard, marigold etc have chemicals or alkaloids as root exudates which repel or suppress the plant parasitic nematodes. In marigold (*Tagetes* spp.) plants the **α – terthinyl and bithinyl** compounds are present throughout the plant from root to shoot tips. This chemical kills the nematodes. In mustard **allyl isothiocyanate** and in pangola grase **pyrocatechol** are present which kills the nematodes. Such enemy plants can be grown along with main crop or included in crop rotation.

Removal and destruction of infected plants / Sanitation: Early detection of infested plants and removal helps to reduce nematode spread. After harvest the stubbles of infested plants are to be removed. In tobacco, the root system is left in the field after harvest. This will serve as an inoculum or the next season crops. Similarly, in *D. angstus* the nematode remains in the left-out stubbles in the field after harvest of rice grains. Such stubbles are to be removed and destroyed and land needs to be ploughed to expose the soil.

Use of resistant varieties: Nematode resistant varieties have been reported from time to time in different crops. Use of resistant varieties is a very effective method to avoid nematode damage.

Crop	**Resistant variety**	**Against nematodes**
Barley	C-164, Rajkiran, BH-75	*Heterodera avenae*
Citrus	Trifoliate orange	*Tylenchulus semipenetrans*
Coffee	Robusta root stock	*Pratylenchus coffeae*
Potato	Kufri swarna	*Globodera rostochiensis*
Tomato	SL-120, PNR-7, Hisar lalit, Nemared, Nematex, Atkinson	*Meloidogyne spp.*
Tobacco	GT-5, ABT-10	*Meloidogyne incognita, M. javanica*
Cow pea	GC-1	*Meloidogyne sp.*

4.BIOLOGICAL CONTROL

Biological control aims to manipulate the parasites, predators and pathogens of nematodes in the rhizosphere in order to control the plant parasitic nematodes. Addition of organic amendments such as farm yard manure, oil cakes, green manure and press mud etc encourages the multiplication of nematode antagonistic microbes which intern checks the plant parasitic nematodes.

The addition of organic amendments acts in several ways against the plant parasitic nematodes. Organic acid such as formic, acetic propionic and butyric acids are released in soil during microbial decomposition of organic amendments. **Ammonia and hydrogen sulphide gases** are also released in soil during decomposition. These organic acids and gases are toxic to nematodes.

Nematode antagonistic microbes multiply rapidly due to addition of organic matter.

Organic amendments improve soil conditions and help the plants to grow. The organic matter also provides nutrition for the crop plants.

Predacious fungi: Most of the predacious fungi come under the order Moniliales and phycomycetes. There are two types of predacious activities among these fungi. They are nematode trapping fungi and endozoic fungi.

Non-constricting rings: The trap is formed similar to the constricting ring. It is a non – adhesive trap. The ring becomes an infective structure and kills the nematode e.g. *Dactylaria candida*. In addition to formation of traps and adhesive secretions, the predacious fungi may also produce toxin which kills the nematodes.

Endozoic fungi: The endozoic fungi usually enter the nematode by a germ tuber that penetrates the cuticle from a sticky spore. The fungal hyphae ramify the nematode body, absorb the contents and multiply. The hyphae then emerge from dead nematode. *Catenaria vermicola* often attacks sugarcane nematodes. *Pasteuria penetrans* was found to be very effective against the root-knot nematodes in many crops. The *P. penetrans* infested J2 of root knot nematodes can be seen attached with spores throughout the cuticle.

Oppurtunistic fungi - Colonize nematodes
E.g.: *Paecilomyces lilacinus (Trade name Yorker)*

Bacteria:
E.g.: - *Pseudomonas fluorescens*
Parasitic protozoa - *Duboscquia penetrans*
Predaceous nematodes - *Mononchus, Diplogaster vorax.*

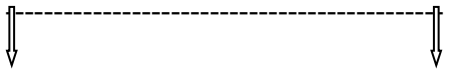
5.Chemical control

History

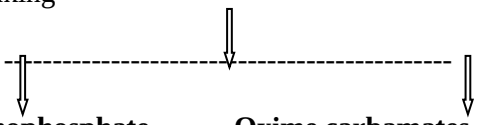
Kuhn (1881) first tested CS2 to control sugar beet nematode in Germany and he could not get encouraging results. In South Carolina State, U.S.A. Bessey (1911) treated CS2 or the control of root-knot nematodes but the method proved impractical. Later on the chemicals like formaldehyde, cyanide and quick line were observed to have nematicidal properties, but all these chemical were found to be highly expensive. Mathews (1919) observed the effect of chloropicrin (tear gas) against plant parasitic nematodes in England. Carter (1943) an entomologist of Hawaii, Pineapple Research Institute, reported the efficacy of 1,3 di-chloropropene 1,2 di-chloropropane (DD) mixture@ 250 1b/acre, against the plant parasitic nematodes. In 1944, scientists from California and Florida states of USA reported the efficacy of ethylene dibromide (EDB). In the same year the Dow Chemical company, USA introduced the chemical as a soil fumigant for the management of nematodes. The introduction of these two nematicides viz., DD and EDB paved way for the chemical control of nematodes.

Classification of Nematicides

FUMIGANTS	**NON-FUMIGANTS**
o Non-systemic	o Systemic
o Highly volatile	o Non volatile
o Mostly available in gaseous forms	o Available in granular forms
o Less persistent	o More persistent
o Phytotoxic, cannot be applied in standing crops	o Less phytotoxic, can be applied in standing crops
o Surface seal required soon after application	o Do not require
o Hazardous to human beings	



o Can be applied as spot, strip, seedling dip, nursery bed, seed coating & seed soaking



Halogenated hydrocarbons
1. D.D. (1.3-dicloropropene 1-2 di-chloropropane)
2. EDB (Ethene di-bromide)
3. D.B.C.P. (Di-bromo cloropropane)
4. M.Br.(Methyl bromide)
5. CS2(Carbodi-sulphide)
6. Chloropicrin

Methyl iso-thiocyanate
Liberators (MIT)
1.Dazomet
2.Metham sodium

Organophosphate
1. Phenamiphos
2. Fensulphothion
3. Ethoprop
4. Phorate
5. Thionazin
6. Dichlorofenthion

Oxime carbamates
1. Carbofuran
2. Methomyl
3. Oxamul
4. Aldicarb
5. Aldicarb sulphone
5. Benfuracarb

Description of some important nematicides

Ethylene dibromide (EDB) : 1.2 Dibromonehane. It is a colourless liquid and the gas in non- inflammable. It is available ad 83% liquid formulation containing 1.2 kg active ingredient per litre and as 35% granules. It is injected or dibbled into the soil for the control of nematodes at 60 to 120 1 or 200 kg ai/ha but it is not very effective against cyst nematodes *Heterodera* spp. and soil fungi. Crops like onion, garlic and other bulbs should not be planted after soil treatment with EDB. It is available as Bromofume and Dowfume.

Dibromochloropropane (DBCP): 1,2 – dipromo – 3 – chloropropane. It is a straw coloured liquid, a litre of it weighing 1.7 kg. It can be used s soil treatment before planting, at the time of planting or as post when the soil temperature is above 20˚C. It is applied as a sprinkle depending upon the crop and stage. Certain crops like tobacco and potato are sensitive due to high bromine content in the chemical. It functions more efficiently than other fumigant at high soil temperature due to its high boiling point (195.6˚C). trade names are Nemagon and fumazone.

DD mixture: It is the trade name of the mixture of compounds, chief of them contain thecis and the trans isomers in equal quantities of 1,3 – dichloropropene 30.35%, and a few other chlorinated compounds up to about 5%. Of these, dichloropropene is the most toxic component and among its two isomers, the transisomer is twice as toxic as the cis-isomer. It is a black liquid of 100% formulation and a litre of it contains approximately 1kg of technical compounds. It is used in the control of soil insects and nematodes and injected into the soil at a depth of 15 -20 cm at 25 x 30 cm spacing. It is a fungicide a very high diseases. Since it is highly phytotoxic, it is used for preplant soil application at least 2 -3 weeks before planting. It is used for preplant soil application at least 2 -3 weeks before planting. It is used as such at 225 – 280 1/ha, but in clay and peaty soils a higher dosage is required. It taints potato tubers and carrots grown in treated soil. Dichloropropence is available under the trade name Telone and in mixture with dibromoetane under the name Dorlane.

Methyl bromide or Bromomethane : It boils at 4.5˚C. At ordinary temperatures it is a gas and therefore, confined in containers under pressure as a liquid. The gas is 1.5 times as heavy as air. Its insecticidal properties were described by Le Goupil in 19-32. Its power of penetration into packed foodstuffs such as flour is remarkable. As it kills insects slowly a longer period of exposure to gas may be required. For control of stored grain pests it is used at 24 – 32 g.m3, exposure period being 48 h. In tent fumigation for the control of termites and powder post beetles, the dosage recommended is 32-64 g/ m3. For fumigating live plants, the dosage is 16-32 g/ m3 some plants are likely to be injured. In soil application for the control of nematodes.

Phorate : 0,0–diethyl S–(ethylthiomethyl) phosphorodithioate.

Trade name is Thimet. It is systemic insecticide cum nematicide, available as 10% granule. It has got both contact and fumigant action. It does not persist for a longer period and gets metabolically oxidized yielding for rat: oral 16 – 3.7; dermal 2.5 to 6.2.

Aldicarb : 2 – methyl – 2 (methilthio) propinaldehyde 0 (methylcarbomy) oxime.

Trade name is Temik. The sulphur atom in the molecule is oxidized to sulfoxide and then to sulfone. It is a systemic 10% granule. The residues remain in plants for 30 -35 days as a lethal dose. It also acts as repellent, contact nematicide and interferes with reproduction of the nematodes by way of sex reversal.

Carbofuran: C12 H15 No3. It is 2,3 – dihydro – 2, 2, dimethyl 7 benzofuranyl methyl carbamate.

Trade name Furadon. It is a systemic insecticide cum nematicide. It is formulated as 3% granule and also as 40F. The residual effect last for 30 – 60 days. It has also got phtotonic effect. This systemic chemical has got acropetal action and applied @ 1 -2 kg ai. /ha.

Integrated Nematode Management

Definition

INM can be defined as a systems approach to reduce nematode to tolerable levels through a variety of techniques including predators and parasites, genetically resistant hosts, natural environmental modifications and when necessary and appropriate chemical nematicides.

Integrated Nematode Management

The grower combines several measures to prevent the growth of damaging nematode populations. Where there is an advisory service equipped to monitor field populations, these control measures can be utilized with minimum amounts of nematicides. Without advisory services, integrated control of nematode is hardly possible and relatively heavy dosages of chemical nematicides are employed. Decisions to implement nematode management programmes are based on the perceived value of the potential crop loss and the cost of the management procedure. Theoretically this involves determining an economic threshold. Management procedures should usually be implemented when the marginal revenue derived from the management input is equal to or excess the marginal cost. The design development and implementation of INM systems for plant protection requires extensive interaction of nematologists with scientists in complementary disciplines. INM includes following components.

a. Exclusion

Crop losses caused by plant parasitic nematodes can be avoided through preventing the introduction of specific nematodes or nematode problems in areas where the species do not exist. The focal point of exclusion is the target nematode species. Exclusion procedures should be used as first order defences to prevent dissemination and establishment. Exclusion procedures include sanitation, certified plant material, nematode free soil or planting media, population reduction or eradication procedures and regulatory activities. Quarantines are used to prevent or slow the spread of plant parasitic nematodes. Certified plant material and nematode free planting media or equipment are used for nematode exclusion. All available exclusion procedures should be considered in the system evaluation and design of the INM programme.

b. Population Reduction

Crop losses caused by plant parasitic nematodes can be alleviated by procedures designed to decrease population densities of the target species to an acceptable level. Whenever possible population reduction strategies should stabilize nematode population densities below the damage threshold. The focal point of population reduction is the target species. It is assumed that the species of concern is present and eradication is not feasible. Established nematode populations are usually managed through population reduction by cultural, physical, biological and chemical procedures. Cultural procedures include the use of fallowing, flooding, cover crops, crop rotation, planting date,

Roguing, trap crops or weed management for population reduction. Heat is the physical factor most widely used in nematode population reduction. Nematode biocontrol resources include prokaryons (viruses, rickettsia and bacteria), nematophagus fungi and predacious, parasitic or environmental modifying invertebrates (nematodes, protozoa, mites etc) and antagonistic plants. Nematicides can directly or indirectly reduce populations associated with nematode infested materials and can be used for soil application, root tips, foliar applications or seed treatments.

c. Tolerance

Crop losses caused by plant parasitic nematodes can be reduced by manipulating the host in relation to its environment. The objective of tolerance is to elevate the damage threshold. The focal point is the host crop. Procedures that protect or increase plant tolerance have excellent potential for INM. These include cultural manipulation, chemical application, mycorrhizal colonization and resistant cultivars.

ENTOMOPATHOGENIC NEMATODES

(Nematoda: Rhabditida families: Steinernematidae and Heterorhabditidae)

Entomopathogenic nematodes are a group of nematodes causing death to insects. It's a beneficial group of nematodes or we can say farmers friendly nematodes. The term entomo-pathogenic has a Greek origin entomon, refers to insect, and pathogenic, which denotes causing disease. The entomopathogenic forms are often referred to as entomogenous (Greek *entomon* = insect, *genés* = born), entomophagous (Greek *phagein* = to eat) or entomophilic (Greek *philein* = to love) nematodes.

Although nematode species in at least 20 families are primary or facultative parasites of insects, those in the order Rhabditida have been most exploited as biological control agents. Entomopathogenic nematodes are soft bodied, non-segmented roundworms that are obligate or sometimes facultative parasites of insects. They occur naturally in soil environments and locate their host in response to carbon dioxide, vibration and other chemical cues.

Entomopathogenic nematodes enter the host via natural body openings or through the cuticle. Some species utilize an anterior stylet or a tooth to rasp the cuticle and gain entrance into the Insect. Others ingress by oviposition on the host food source and the eggs hatch in the host midgut. Species in two families (Heterorhabditidae and Steinernematidae) have been effectively used as biological control agents in pest management programs. Entomopathogenic nematodes fit nicely into integrated pest management or IPM programs because they are considered non-toxic to humans, relatively specific to their target pest, and can be applied with standard pesticide equipment.

Life Cycle

• The infective juvenile stage (J1) is the only free-living stage of entomopathogenic nematodes.

• The juvenile stage penetrates the host insect via the spiracles, mouth, anus, or in some species through intersegmental membranes of the cuticle and then enters into the Insect.

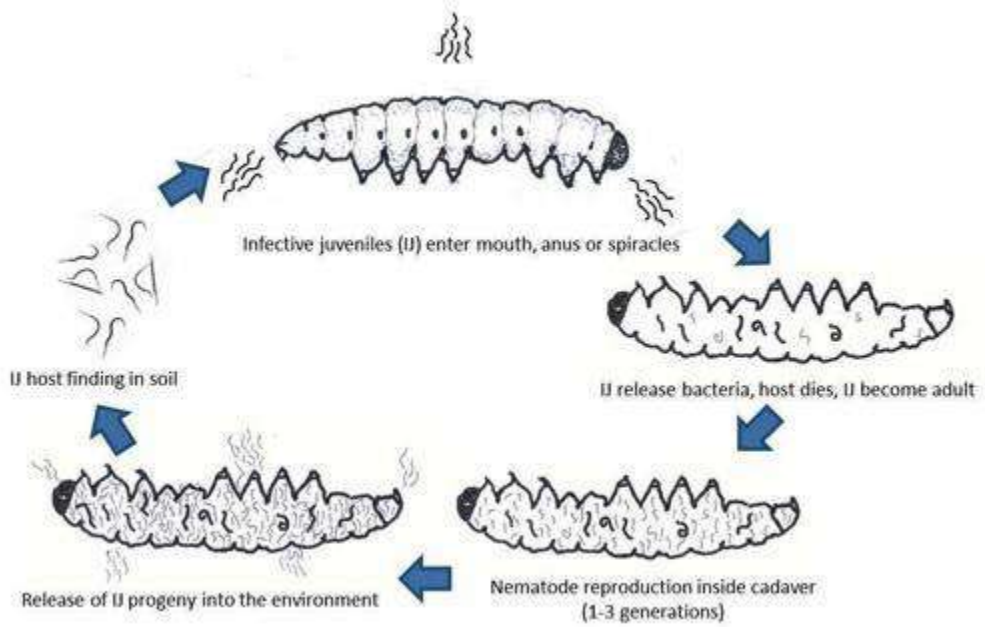
• The juvenile stage release cells of their symbiotic bacteria from their intestines into the Insect.

• The bacteria multiply in the insect haemolymph and the infected host usually dies within 24 to 48 hours.

• After the death of the host, nematodes continue to feed on the host tissue, mature and reproduce.

• The progeny nematodes develop through four juvenile stages to the adult.

• Depending on the available resources one or more generations may occur within the host cadaver and a large number of infective juveniles are eventually released into environment to infect other hosts and continue their life cycle.



INTEGRATED NEMATODE MANAGEMENT (INM) STRATEGIES

For Nursery (Transplanted crops): -

- Keep nursery area fallow (2-3 months)
- Deep summer ploughing (2-3) April-May
- Grow non-host or antagonistic crops
- Soil solarization- 100-gauge LLDPE for 20 days
- Follow rabbing at 7 kg(husk)/m3
- Use resistant varieties
- Green manuring with sunhemp
- Use bioagents like *P. lilacinus, T. viride, T. plus, P. fluorescens at 10 to 20 g/m2*
- Use effective nematicides like carbofuran 3 G or phorate 10 G at 1 to 2 kg a.i. /ha.

For Field Crops: -

- Keep the field fallow (2-3 months)
- Deep summer ploughing (2-3) April-May
- Flooding (2-3 months)
- Crop rotation

- Field sanitation
- Healthy planting material
- Resistant varieties
- Green manuring with sunhemp
- Grow trap crop, non-host or antagonistic crops
- Inter or mixed crops with marigold
- Use FYM or compost at 20-25 t/ha
- Use non-edible oil cakes at 1 to 2 t/ha
- Use bioagents like *P. lilacinus, P. fluoreascens, T. plus, T. viride at 5 kg/ha*
- Seed treatment with carbosulfan 25 DS at 3 % w/w
- Seedling root dip with carbosulfan 25 EC at 0.05 % for 4 to 6 hours.
- Seed soaking with carbosulfan 25 EC at 0.05 % for 12 hours.
- Soil application of carbofuran 3G, phorate 10 G at 2 to 4 kg a.i./ha.

UNIT 1: CLASSIFICATION OF PLANT DISEASES

I. On the basis of extent to which plant diseases associated with plant:

1. Localized Disease: A disease may be localized affecting only the special organ or parts of the plant. It cause lesser damage than systemic infection in the host. e.g. Leaf spot of ground nut

2. Systemic Disease: A disease may be systemic affecting the entire plant, i.e. the pathogen moves through the entire plant. It causes severe damage to the host plant and entire plant may kill off. e.g. white rust of mustard. All infections caused by fastidious vascular bacteria, mollicutes (phytoplasma and Spiroplasmas) and protozoa and all natural infections caused by viruses and viroids are systemic

II. **On the basis of natural perpetuation and mode of infection:**

1. **Soil borne:** When disease causing agent survive and spread through soil it is known as soil borne pathogen and disease known as soil born disease.
2. **Air borne:** When pathogen or disease disperse through the agency of air, it is known as air borne disease.
3. **Seed borne:** Diseases or pathogens perpetuated through seed are known as seed borne disease or seed borne pathogen.

(a) Internally Seed Borne Disease : Pathogen remains in internal parts of the seed viz. cotyledon, embryo, etc. Loose smut of wheat, Ascochyta blight of chick pea.

(b) Externally Seed Borne Disease : Pathogen remains on external parts viz. seed coat. Covered smut of barley, Grain smut of sorghum, Bunt of Wheat.

- *Phomopsis blight of egg plant is both externally and internally seed borne.*

III. On the basis of symptoms: Rusts, Smuts, Rots, Blight, Leaf spot, Canker, Wilt, Downy mildews, Powdery mildew, Anthracnose etc

IV. On the basis of cause (Pathogen identity)

1. **Infectious/ parasitic (Biotic)**

a) Diseases incited by attack of parasitic organisms or virus or viroids under a set of suitable environment are infectious.
b) Micro organism responsible for this types of diseases are Fungi, bacteria, virus, viroids, phytoplasma, RLBs, nematodes, higher parasitic plants etc.
c) This types of diseases also called as animate or biotic diseases.
d) This types of diseases is contagious in nature i.e transmitted to diseases to healthy plant through various agencies.

1	Fungal diseases	Powdery mildew of grapes, Late blight of Potato, Rust

2	Slime mold diseases	Club root of crucifers
3	Bacterial diseases	Bacterial blight of rice, Black arm of cotton
4	Phytoplasma diseases	Sesame phyllody, Little leaf of Brinjal
5	Spiroplasma diseases	Corn stunt disease, Citrus stub borne, Sandal spike disease
6	Fastidious vascular bacteria	Citrus greening, Pierce's disease of grapevine (It is also known as Rickettsia-like organism (RLOs)) diseases)
7	Algal diseases (Chlorophycean algae)	Red rust of mango and tea
8	Protozoan diseases	Hart rot of coconut and oil palm (Fatal wilt), Phloem necrosis of coffee
9	Viral diseases	Tobacco mosaic virus, Tomato leaf curl, Yellow vein mosaic of okra
10	Viroid diseases	Potato spindle tuber disease, Coconut cadangcadangdisease
11	Nematode diseases	Root knot of vegetables, Ear cockle of wheat Molya disease of wheat and barley
12	Phanerogamic higher plant parasites diseases	a) Total stem parasite of berseem :Cuscuta (Dodder, Amarbel, Love vine) b) Total root parasite of tobacco : Orobanche (Broom rape, Vakumba) c) Partial stem parasite of mango: Loranthus (Dendrophthae, Vrikshabhaksha, Giant mistletoe, Vando) d) Partial root parasite of sorghum: Striga (Witchweed)

2) Non infectious (Abiotic)

a) These are diseases with which no animate or virus pathogen is associated.

b) This types of diseases also called as in-animate or abiotic diseases or non parasitic diseases.

c) This types of diseases is not contagious in nature i.e not transmitted to diseases to healthy.

d) These types diseases are due to disturbances in plant body caused by lack of proper environmental condition or nutrional deficiencies etc.

e) e.g. Nutrional deficiencies e.g. khaira disease of rice due to Zn deficiency, unfavorable environment e.g. frost injury, physiological wilt etc

Abiotic (Non living) factors

a) **Temperature**: High temperature-Root rot of cotton and castor
b) **Moisture**: Low-Root rot, High- Damping off,
c) **Wind**: Foliar-Powdery mildew, Rust, Blight
d) **Light**: Etiolation (elongation and yellowing of shoot due to lack of
    a. light)
e) **Soil pH:** Acidic pH -Club root rot of cabbage,
    i. Alkaline pH-Common scab of potato
f) **Soil structures** –Sandy-Root knot nematode,
    i. Black soil -wilt cotton
g) **Nutrition and toxic substance**:
h) **Deficiency of nutrients**:
    i. Khaira of rice (Zn),
    ii. Mottle leaf of citrus(Zn),
    iii. Chlorosis of groundnut (Fe)
i) **Toxic gas:** Sulphur dioxide-Mango tip rot or fruit necrosis
j) **Lack of oxygen:** Hollow and black heart of potato
k) **Mineral toxicity:** Leaf crinkle of cotton due to manganese toxicity, Akiochi in Paddy due to H_2S (Hydrogen sulphide) toxicity.

Nutrional deficiency

a) Khaira disease of rice due to Zinc (Zn) deficiency.
b) Mottle leaf of citrus due to Zinc (Zn) deficiency.
c) Chlorosis of groundnut due to Iron (Fe) deficiency.
d) Blossom end rot of tomato due to Calcium (Ca) deficiency
e) Whip tail of cauliflower due to Molybdenum (Mo) deficiency
f) Wither tip of apple due to Copper (Cu) deficiency

(V) Improper cultural practices or Iatrogenic diseases:

a) It is a disease produced by appropriate therapy for another disease, usually occurring as a side effect of pharmacological agents. "***An iatrogenic disease of a crop is one that is induced or worsened by a plant pathologist's prescription for that crop***". **OR**
b) ***Diseases which result from or increased in severity by the use of a specific crop protection chemical may be referred to as iatrogenic disease.***
c) Zineb fungicides utilized for the controls of downy mildew disease in grapes but it enhance the incidence of powdery mildew disease in grapes.

(VI) On the basis of occurrence and distribution (Spread & Severity of Infection)

a) Endemic : The word Endemic means *"prevalent in"* or *"confined to"*. When a diseases more or less constantly prevalent/present from year to year in a moderate to severe form in a particular area or country. *E.g., Wart disease of potato is endemic to Darjeeling*

b) Epidemic or Epiphytotic: The term *"Epidemic"* is derived from *Greek* word which means "*among the people*". An Epiphytotic disease is one which occurs widely but periodically. It may be present constantly in the locality but assume severe form only on occasions. This is because the environment or conditions favorable for rapid development of the diseases occur only periodically. e.g. Cereal Rust, Late blight, Mildews

c) Sporadic: Diseases which occur at very irregular interval and location in a moderate to severe form. e.g., Angular leaf spot of cotton or blotch disease of cucumber

d) Pandemic: When diseases occurs throughout the country or continent or sub-continent or the world in a severe form and resulting in mass mortality is known as pandemic diseases. e.g., Late blight of potato

(VII) Based on pathogen generations

1. Simple interest diseases/ Monocyclic diseases

a) In simple interest disease the rate of increase in disease is mathematically analogous to simple interest in money.
b) There is only one generation of the pathogen in life of the affected crop.
c) In these diseases, primary inoculums is seed or soil borne and secondary infection rarely occurs during the season.
d) Some examples are the wilt diseases caused by *Fusarium oxysporum*, loose smut of wheat caused by *Ustilago tritici*, covered smut of barley caused by *U. hordei* and head smut of jowar caused by *Sphacelotheca reiliana*.

(2) Compound interest diseases/ polycyclic diseases

a) In compound interest disease the rate of increase in disease is mathematically analogous to compound interest in money.
b) There are several generation of the pathogen in life of the affected crop.
c) The pathogen produce spore at vary rapid rate. Incubation and sporulation period is short.
d) Some examples are late blight of potato caused by *Phytophthora infestans*, stem rust of wheat, powdery mildews, downy mildews, leaf spots, blights, root-knot nematodes in vegetable crops.

(3) Polyetic diseases

a) These are also polycyclic diseases but they complete their disease cycle in more than one year over years e.g. Cedar Rust of Apple

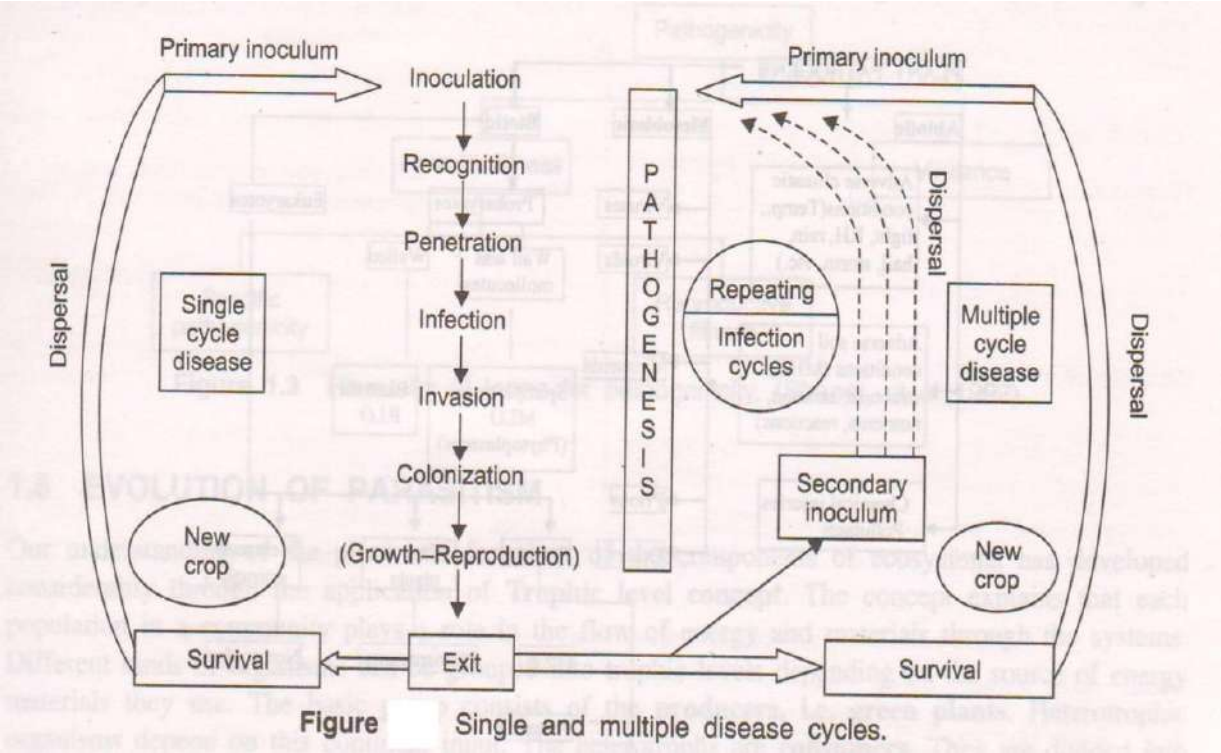


Figure Single and multiple disease cycles.

(VIII) Based on host-pathogen dominance system

Kommedahl and Windels (1979), classify the plant disease based on host-pathogen dominance system, have divided diseases as ***'Pathogen Dominant Diseases'*** **(PDD)** or ***'Host Dominant Diseases'*** **(HDD)**.

1. **Pathogen Dominant Diseases**
   a) In the pathogen-dominant group, the pathogen is dominant over the host, but the relationship is transitory because the resistance of the host is less initially than it becomes eventually.
   b) Such pathogens are **tissue-nonspecific** and attack young, immature root tissues or senescent tissues of mature plant roots. They seldom damage a rapidly growing, maturing root, so the ***period of disease development is short***.
   c) Sometimes such pathogens are macerative in that they degrade host tissues; sometimes they are toxicogenic and kill tissues by toxins or sometimes both.
   d) In any of these situations, pathogenesis is due primarily to the virulence of the pathogen and the host has little influence on the course of disease.
   e) Physiological specialization is relatively uncommon.

2. **Host Dominant Diseases**
   a) In the host-dominant diseases are included those in which the host is dominant and the pathogen is successful only when factors favor the pathogen over the host.

b) The resistance of the host or the factors favoring host growth are strong enough so that the pathogen can not develop disease. While damage is most severe in plants in the reproductive and senescent phases.
c) In this group, some pathogens which are tissue-nonspecific but ***most are tissue-specific***. This represents a degree of specialization within the group of host-dominant diseases. The pathogen may be macerative, toxicogenic, or both.

Introduction:

Plant disease cause enormous economic losses, both quantitatively and qualitatively. According to conservative estimate, pre and post harvest diseases result in more than 30% losses in the developing countries. Consequently, man has developed variety of control devices to combat plant diseases so that he can obtain increased quantity and improved quality of plant products. But, protecting the crop from diseases is encountering many problems, such as *emergence of new virulent strains of pathogens causing dramatic failure in chemical application, environmental hazards* etc. These problems mainly result in due to a 'quick fix' mentality in which there is continuous and indiscriminate application of fungicides and the ignorance of cultural and biological means. Moreover, the complete destruction of pathogens under field conditions within a growing season using different control devices separately is not only impossible but also economically impractical. One has to repeat the control operation in each crop season toward of the disease.

What is required is a more lasting solution, using a combination of different disease management practices based on a sound understanding of the whole crop ecosystem. This approach is called **integrated disease management (IDM)**.

IDM progress

- Much of the early progress towards an integrated approach was made by entomologists attempting to manage insect- pests.
- The concept arose largely from problems with insecticides, including the rapid development of resistance and environmental hazards associated with their use. It was also realized that chemicals used to control pests often affected non target species, including natural enemies of the pest.
- However there was less emphasis given on integrated disease management for the control plant pathogens for a number of reasons.
- Currently more emphasis is given on integrated disease management due to reduce input costs and environmental impacts, as from the shortcomings of any individual control strategy.
- There are comparatively few examples of fully integrated systems, which combine cultural, chemical, biological, and host genetic factors to control plant pathogens.
- Nevertheless, progress is being made by putting together packages of measures, which offer significant advantages to growers as well as reducing reliance on chemical agents.

Evolution of IPM/IDM:

- Green revolution has attained self sufficiency in food through introduction of hybrids and high yielding varieties.

- Intensive cultivation of high yielding variety invited or demanded more of inputs in the form of fertilizers especially inorganic which in turn attracted more of pest and diseases.

- This necessitated intensive control measures to restrict the damage caused to the crops and the control was achieved mainly through chemical pesticides.

After World War II the use of pesticides mushroomed, but with all the benefits of the use pesticides, it has adverse side effects not just on humans but also in animals.

During the massive use of pesticides, Rachel Carson, an American biologist, warned the people about the side effects of the use of pesticides through her book entitled, Silent Spring. Through her book, she raised a lot of questions about the real benefits of the use of pesticides as well as the risks of pesticides rendered in the environment and public health. An over-reliance on chemical pesticides led to development of pesticide resistance, development of multiple resistance, emergence of secondary pest as major pests, resurgence of pests, elimination of natural enemies of pests, hazards to non target species, hazards to agricultural workmen and deleterious effects on the environment, increase of production costs, etc.

Presence of residues in foods, feed and organisms caused widespread concern about contamination of environment.

The importance of integrated approaches to pest control was then felt and the concept of IPM/IDM evolved.

In integrated control, it is aimed to give the control measures to the optimum and not to the maximum.

Definition of IDM:

Modern concept of pest management is based on ecological principles and integration of different control tactics into a pest management system

Integrated control was defined by Stern *et al.*, (1959) as applied pest control which combines and integrates the biological and chemical control.

According to FAO (1967), IPM was defined as "a pest management system in the context of associated environment and population dynamics in pest species, utilizes all suitable techniques and methods in as compatible manner as possible and maintains the pest population at levels below those cause economic injury".

In 1972 the term IPM was accepted by CEQ (Council of Environmental Quality) where IPM includes
I - Integration that is harmonious use of multiple methods to control the impact of single pest as well as multiple pests.
P- Pest - any organism that is detrimental to humans including vertebrates and invertebrate or weed or pathogens.
M- Management refers to a set of decisions or rules based on ecological principles, economic and social consideration.

Basic concepts of IDM

1. Maintaining pest population below levels that cause economic damage.
2. The use of multiple tactics to manage insect population.
3. Conservation of environmental quality

IPM seeks to minimize the disadvantages associated with use of pesticides and maximizing socio, economic and ecological advantages.

1. **Understanding the agricultural ecosystem:** An agro ecosystem contains a lesser diversity of animal and plant species than natural ecosystem like forests. An agro ecosystem is intensively manipulated by man. Agro ecosystem is a complex of food chains and food webs that interact together to produce a stable unit.
2. **Cost benefit ratio:** Based on the possibility of pest damage by predicting the pest problem and by defining ETL, emphasis should be given to cost benefit ratio.
3. **Tolerance of pest damage:** The pest free crop is neither necessary in most cases for high yields nor appropriate for insect pest management. Exceptions occur in case of plant disease transmission by vectors. The relationship between density of pest population and profitability of control measures is expressed through threshold values.
4. **Leaving a pest residue:** Natural enemy population is gradually eliminated not only in the absence of their respective insect hosts because of the indiscriminate use of broad spectrum insecticides, which in turn also eliminate natural enemies. Therefore, it is an important concept of pest management, to leave a permanent pest residue below economic threshold level, so that natural enemies will survive.
5. **Timing of treatments:** Treatment in terms of pesticide spray should be need based, with minimum number of sprays, timely scheduled, combined with improved techniques of pest monitoring and crop development.
6. **Public understanding and acceptance:** In order to deal with various pest problems special effort should be made for effective communication to the people for better understanding and acceptance of pest management practices. The IPM practices followed should be economical and sustainable.

Objective:

1. To reduce disease status below economic injury level. Complete elimination of pest is not the objective.
2. To manage insects by not only killing them but by preventing feeding, multiplication and dispersal.
3. To use eco-friendly methods, which will maintain quality of environment (air, water, wild life and plants)
4. To make maximum use of natural mortality factors, apply control measures only when needed.
5. To use component in sustainable crop production.

Different components or tools of IPM

1) Pest surveillance
2) Cultural methods
3) Mechanical methods
4) Physical methods

5) Biological methods (Use of parasites, predators & host plant resistance)
6) Regulatory / Legislative methods and
7) Chemical methods
8) Behavioural methods
9) Genetic / Biotechnology methods

Central idea of IDM:

- Integrated disease management (IDM) aims at prevention or reduction in the incidence or severity of the disease incited by the plant pathogen.
- Hence the central idea revolves around the tactical management of the **host, the pathogen, and the environment** (the three factors of a disease triangle) so as to keep the damage or loss below the economic threshold level.
- The strategy utilizes host plant resistance together with other possible tactics in harmony with the natural regulatory factors of the ecosystem to keep the disease inciting pathogens below the economic threshold level.
- **Management of the host** involves the practices directed to improve plant vigour and host resistance through nutrition, induction of genetic resistance through conventional plant breeding and through genetic engineering, and providing protection against attack by Chemical means.
- **Management of the pathogen** involves avoiding contact between the pathogen and the host, *eradication, and protective application of chemicals to prevent infection*.
- **Management of the environment** involves water management, soil management, and crop management.

Criteria for priority:

- The Food and Agriculture Organization of the United Nations has been interested in developing integrated pest/disease management since 1963 and has developed and implemented several field projects in different parts of the world.
- This organization recommended for global programme to be undertaken or the basis of programme priority.
- Criteria of programme priority have been ascertained that point out the necessity of IPM/IDM. These criteria are the following.

1. Crop must be of very important for the nation and regional importance.
2. Serious losses are caused by pests/pathogens.
3. Inadequate control by use of organic pesticides.

4. Use of pesticide is generating more problems

5. An integrated approach can be developed, which will yield desirable results and be acceptable to growers.

- ❖ Emphasis has been laid on major crops namely rice, maize, sorghum, cotton, etc.
- ❖ Potato, sugarcane, grain legumes, tapioca, coconut, etc., have also been considered as second order of priority.

Phases of IDM:

Integrated disease management is a system approach and consists of following important phases, which significantly contribute the development of a complete IDM system.

1. **Single tactic phase** - In the initial stage, the best method for tackling or managing a disease is found out.

2. **Multiple tactic phase** - In this phase, all the available methods - cultural, biological, chemical, and use of resistant varieties are worked out in relation to ecology, inoculum density and damages. This phase is the most critical one and needs careful work out.

3. **Biological monitoring phase** - After the multiple tactics have been decided, they are to be carefully monitored, particularly biological ones, in relation to beneficial organisms and host plants so that management measures may be suitably timed and phased out.

4. **Modeling phase** - Data obtained from phases 1, 2, and 3 require to be very carefully worked out so that a model can be developed pointing out the major steps that need to be taken regarding the application of management measures and the critical stages in the host plant. The entire approach has to be systematized through critical analysis of data.

5. **Management phase** - After the different models of disease management have been worked out in an integrated manner, the entire process has to be incorporated into the overall production techniques of the crop concerned.

6. **Acceptance phase** - The most critical stage is the development of system approach acceptable to growers. The approach has to be technologically sound, economically viable, and not two cumbersome for adaptation by growers.

Strategies adopted for IDM:

Integrated disease management (IDM) systems are being developed with their main goals to:

(1) Eliminate or reduce the initial inoculums,
(2) Reduce the effectiveness of initial inoculums,
(3) Increase the resistance of the host,
(4) delay the onset of disease and
(5) slow the secondary cycles of the disease.

These systems therefore combine cultural, chemical, biological and host genetic factors to reduce plant diseases.

1. Growers are advised to grow healthy, certified, and treated seeds. They should periodically remove and monitor diseased seedlings so that initial inoculum could be checked without further spread in main field.
2. Crop rotation of cereals, paddy, sugarcane, and coarse millets with vegetable crops substantially reduces the soil-borne disease.
3. Crop residues, rotted fruits, infected plant parts, and weeds should be periodically removed. This reduces pathogen perpetuation and also pathogen inoculums.
4. In vegetable crops, sowing time and transplanting Pattern particularly ridge sowing reduces water stagnation, root rot, and wilt incidence. Plant spacing should be adjusted keeping plant canopy in view to avoid easy disease spread in dense crop.
5. Disinfection of agricultural implements, tools, plant support materials are advised particularly for viral disease management.
6. Lower, older, and Senescent leaves should be removed. This reduces infection site by various facultative pathogens.
7. Maximum use of organic supplement, alternate year green manuring for augmentation of antagonistic microbial population and biological control.
8. Integration of fungicides with host resistance and integration of cultural and biological measures should be followed.
9. There should be strict legislation for pesticide dealers so that they do not sale spurious chemicals and cannot misguide the growers by giving so many pesticide, growth regulators, etc. for use at a time without its real need.
10. The use of trap crop, border/barrier crop, disease escape varieties, etc. proves to be effective in reduction of disease.
11. In vegetables, allelopathic crop, which produces toxic root exudates for pathogen, must be encountered.
12. Postharvest practices should be used to avoid any rotting and seed infection during storage and transportation.
13. Growers should be essentially trained for correct diagnosis of the disease to apply different measures of integrated disease management at critical period.

Integrated management of disease in an annual crop:

An integrated management programme of an annual crop, such as potato crop is taken here for instance. In Indian subcontinent and elsewhere, late and early blight diseases, black scurf/stem canker, bacterial wilt and brown rot, root knot, and mosaic and leaf curl are quite common diseases of this crop. Due to nature of propagation of the crop all the diseases are mainly seed (tuber) borne. Some of them are soil borne also. Spread by soil, insects, water, and wind is common. Therefore, in this crop the improvement of quality and quantity of the yield through disease control can be brought about by adopting a schedule prepared on the following lines.

1) The first step is the proper selection of the field for potato. Fields in which there has been serious incidence of soil-borne diseases of potato during last some growing seasons should be avoided. Also, the selected field must have proper drainage and high level of fertility.
2) The second step in the selection of resistant varieties for those areas where late blight regularly occurs every year even if they do not yield as much as a susceptible but high yielding variety.
3) The third step in the selection of seed. Potato tuber seed may carry variety of pathogens (e.g., several viruses, bacteria, fungi, nematodes) on or within it and may introduce the pathogen in the field. Therefore, use of clean and disease-free seed is of paramount importance. Such seeds should be obtained from sources, which guarantee seed certification.
4) The fourth step is the additional precautions in the form of chemical treatment of seeds. Additional precautions should be taken by treating the seed tubers with fungicides which helps eradicating residual inoculum and protects it during germination and emergence.
5) The fifth step is the date of planting. The latter should be decided according to prevailing soil temperature and moisture. High soil temperature and moisture should be avoided.
6) The sixth step is the protection of plants from infection. If the diseases occur regularly year after year in that area, the grower should start spraying with fungicides such as Zineb, Mancozeb, Copper oxychloride, etc.
7) The seventh step is the destruction of infected plant debris before harvesting. Infected plant debris must be destroyed before harvesting so that the pathogen inoculums could not come in contact with the tubers when they are dug up.
8) The eighth step is to protect tubers from being wounded. Tubers must be harvested carefully to avoid wounding that would allow storage rot fungi such as *Fusarium* and *Pythium,* and bacteria such as *Erwinia caratovora*, to gain entrance into the tuber. Wounded tubers, if any, must be sorted out and discarded.
9) The ninth step is the proper storage. Healthy tubers should be stored at about 15^0c for healing of wounds and then at about 10^0 c to prevent development of fungus rot in storage.

Integrated management of diseases in perennial crop:

1) Removal through pruning and destruction of dead twigs, branches, and fruits. This ensures reduced amount of initial inoculums of the pathogens that initiate infection.
2) Pruning shears and saws should be disinfected before moving to new trees so that the spread of the inoculums should be avoided.
3) A dormant spray containing a fungicide-bactericide (e.g., Bordeaux mixture) or a plain fungicide plus acaricide-insecticide (e.g., Superior oil), should be applied before bud break.
4) After the buds open, blossoms and leaves must be protected with fungicide and/or a bactericide sprays and possibly, an insecticide and/or acaricide that does not harm bees.
5) Young fruits appear when blossoming is over. If the young fruits arc infected, appropriate chemical sprays should be undertaken.
6) Usually the fruits become susceptible to several fruit-rotting fungi at early maturity through harvest and storage. Therefore, fruits must be sprayed at 15 day intervals with chemicals that

will control these pathogens till harvest. Most fruit rots begin at wounds made by insects and therefore, insect control must continue.

7) Also, wounding of fruits during harvesting and handling must be avoided so that any fungus infection should be avoided.
8) Harvested fruits should be washed in a water solution containing a fungicide to further protect the fruits during storage and transit. During packing, infected fruits must be sorted out and discarded.

Limitations of IPM:

1) An IPM programme requires a higher degree of management: Making the decision not to use pesticides on a routine or regular basis requires advanced planning and therefore a higher degree of management.
2) This planning includes attention to field histories to anticipate what the pest problems might be, selecting crop varieties which are resistant or tolerant to pest damage, choosing tillage systems that will suppress anticipated pest damage while giving the crop the greatest yield potential.
3) IPM can be more labour intensive, consistent, timely and accurate field scouting takes time.
4) Without this information, intelligent management decision cannot make.
5) Success of IPM programmes can be weather dependant. Therefore good IPM planners will have an alternate plan for when these problems arise.
6) May be not suitable for different regions.

UNIT 3: ECONOMIC IMPORTANCE OF DISEASES

Plant diseases have been associated with many important events in the history of mankind on earth**.**

World scenario

1) **Irish Potato Famine**:Great Irish Famine, or Famine of 1845–49, famine that occurred in Ireland in 1845–49 when the potatocrop failed in successive years. The crop failures werecaused by late blight, a disease that destroys both the leaves and the edible roots, or tubers, of the potato plant. The causative agent of late blight is *Phytophthora infestans*. The Irish famine was the worst to occur in Europe in the 19th century (**Fig.1**).

   The Irish Famine of 1846-50 took as many as one million lives from hunger and disease, and changed the social and cultural structure of Ireland in profound ways. The Famine also spurred new waves of immigration, thus shaping the histories of the United States and Britain as well. The combined forces of famine, disease and emigration depopulated the island; Ireland's population dropped from 8 million before the Famine to 5 million years after. If Irish nationalism was dormant for the first half of the nineteenth-century, the Famine convinced Irish citizens and Irish-Americans of the urgent need for political change. The Famine also changed centuries-old agricultural practices, hastening the end of the division of family estates into tiny lots capable of sustaining life only with a potato crop.
2) Coffee rust in Sri Lanka (1869) took less than three decades to anhilate the entire coffee production of the island.
3) *Downy mildew*occurred in epiphytotic way during 1878-1882 in France.
4) Powdery mildew of grape - France (1851 and 1878).
5) Panama wilt of banana damaged the crop during 1910 in Panama and Surinam.
6) Sigatoka leaf spot of banana damaged the crop during 1930 in Central and South America.
7) Bunchy top of banana durin 1940 in Sri Lanka

Indian scenario

1) Bengal famine: In 1943, Bengal had to face a serious famine. One of the reasons to which this famine has been attributed is the loss of yield of rice due to attack of *Helminthosporium oryzae* causing leaf spot disease . At least, 2 million people died from starvation and malnutrition during a famine in the Indian province of Bengal.
2) In 1946-47 wheat rust epidemic caused food shortage.
3) In 1963 bacterial leaf blight of paddy in Bihar wiped the crop completely.
4) Tungro virus in 1985-87 and blast during 1985-88 caused havoc to rice crop in Tamil Nadu.
5) In 1938-39 red rot of sugarcane occurred in large scale in Bihar, Punjab and U. P.
6) Bunchy top of banana wiped out the whole banana plantation in Palani hills of Tamil Nadu.

Important famines/epidemics in world and India and their importance

1. IRISH FAMINE

Country	Ireland
Location	Ireland
Period	1845 – 1852
Total population	8 million
Total deaths	1.5 million
Migration	1.6 million
Reason	Policy failure, potato blight
Impact on demographics	Population fell by 20-25% due to mortality and emigration
Consequences	Permanent change in the island's demographic, political and cultural landscape
Scientific Advancement	Birth of Plant Pathology as a science **Anto De Bary and Speer Schneider** (1861) proved Fungi as causal agent (*Phytophthora infestans*)

2. COFFE RUST

Country	Ceylon (Srilanka)
Period	1867 -1893
Cause	Cofffee Rust by *Hemileia vastatrix*
Impact	Economy ruined
Consequences	1. Yield reduced from 228 kg berries /acre to 101 kg/ acre 2. Export decline by 93 per cent by 1893 3. Coffee replaced by tea plantations

	4. Drinking habit changed from coffee to tea
Scientific Advancement	**M.J Berkly** –Idetified the cause as *Hemileia vastatrix*
	H.M.Ward – Elucidated the Role of Environment and Monocropping in disease epidemic

4. DOWNY MILDEW

Country	France (Europe)
Period	1878
Reason/ Cause	Downy mildew caused by *Plasmopara viticola*
Consequences	1. Reduced grape yields and quality greatly 2. Killed young vines in many vineyreds 3. Wine industry affected and Economy ruined
Scientific Advancement	Discovery of **Bordeaux mixture (1885) by P.A.Millardet**

5. BENGAL FAMINE

Country	India
Location	Bengal state
Period	1943
Cause	Brown spot of paddy
Pathogen	*Drechslera oryzae*
Impact	1. Shortage of food 2. Raise in the price of rice 3. Death of 2 million people due to hunger 4. Migration of people to other states. 5. Change in the Agriculture policy.

Significance of Plant Disease/ Plant Pathogens

Of the 30,000 plant diseases recorded from different countries about 5,000 diseases are present in India. It is estimated that the total annual production for all agricultural crops worldwide is about 12000-13000 billion $. Of this, about 500 billion $ worth of produce is lost annually to diseases, insects and weeds.

Following are the significance of Plant diseases

1. **Famine:** Irish famine – Late blight of potato, Bengal famine – Brown spot of paddy
2. **LOSSES**

a) Quantity (Yield loss): During Wheat rust epidemic in Bihar (1956-57) yield reduced from 900kg/ha to 50 kg/ha.

b) Crop quality losses: Example: Citrus canker, Chiku rough storage, Apple scab, Groundnut – aflatoxin, Wheat – bunt, ear cockle.

3. **Human/ animal hazard /food poisoning:** Consumption of Ergot infected rye – causes gangrene, loss of fingers, limbs and death. (Known as Saint Antony's fire). Death of one Lakh turkeys in England due to consumption of Peanut meal contaminated with Aflatoxin. Aflatoxin is immune suppressive and causes Liver cancer in Human beings.
4. **Cropping pattern changed:** In Srilanka Coffee was replaced by Tea plantation due to Coffee Rust (1867).
5. **Limit the cultivation of variety:** Due to Downy mildew of Bajra, cultivation of GJ 104 variety was discouraged in Gujarat.Due to Wilt & red rotcultivation of co. 671, co. 8603 sugarcane variety was discouraged in south Gujarat.
6. **Diet change/ food habit change:** InSri Lanka drinking habit changed from coffee to tea due to coffee rust. In Southern U.S.A food habit changed *i.e.* from wheat to corn due to wheat rust
7. **Agro- industries affected:** Due toWilt and redrot diseases Sugar Industries in Gujarat affected. Wine industry in France was severly affected due to Downy mildew disease.
8. **People's migration:**Due to Irish Famine in more than 1.6 million people migrated from Ireland to North American continent.
9. **Sociological effect :**. Increase in Poverty due to close of Agro industries
10. **High/rise market price:** Shortage of food due to heavy crop loss leads to Rise in price of Agril. Commodities
11. **High cost of production:**Extra Expenditure to be incurred on pesticides, labour etc. for control of diseases
12. **Political effect:** Due to Irish famine, the free trade was allowed between England and Ireland
13. **Destroy the beauty of environment:** By damaging plants around houses, along street, in gardens & in forest.
14. **Ecological implications:**Environmental pollution due to fungicidal use or due to disease.

UNIT 4: DETECTION AND DIAGNOSIS OF VARIOUS PLANT DISEASES

The word **diagnosis** is derived from ***Greek*** word ***diagignoskein*** which means **to distinguish**. Detection and identification of plant pathogens and the diagnosis of plant diseases caused by them are essential requirements of plant pathological studies. It is necessary to first determinewhether the disease is caused by a pathogen or an environmental factor.

- **Detection:** It is used for identifyingmicroorganism.
- **Diagnosis:** It is used to draw conclusion from symptoms that a particular pathogen is associated.

Purpose of Disease Diagnosis

1. Determine the presence and quantity of thepathogen(s).
2. Assess the effectiveness of protectiontechnologies.
3. Certify planting materials for plant quarantine andcertification.
4. Determine the extent of disease incidence and consequent yieldloss.
5. Assess pathogen infection in plant materials in breedingprograms.
6. Detect and identify new pathogens rapidly, to prevent furtherspread.
7. Study taxonomic and evolutionary relationships of plantpathogens.
8. Resolve the complex diseases incited by two or morepathogens.
9. Study pathogenesis and genefunctions.

There are variety of techniques used to detect plant pathogen and diagnose the concerned, plant disease, which can be grouped under two broad category.

1. **TraditionalApproaches**
2. **MolecularDiagnostics**

1. Traditional Approaches

In plant pathology, certain traditional approaches are used to detect and identify the plant pathogens. These approachesare

- Symptomatology

- Hostrange
- Morphology of thepathogen
- Isolation of pathogen onto selective culturemedia.

If there still remains an uncertainty, further the ***Koch's postulates*** are applied to identify the pathogen and to get its pathogenicity test. For viruses and phytoplasmas, electron microscopy or inoculation of indicator plants may be used.

Koch's postulates

Association of microorganisms with the diseased symptoms of an infected tissue may not conclude their association as the real cause of the disease. Hence the real causal organism for particular plant disease has to be verified by Koch's Postulates. **Robert Koch (1884)** laid down the **three** postulates or ***"rules of proof"***which must be satisfied to definitively establish causal relationship of a micro-organism with a disease; **a fourth one** was appended by **E.F. Smith (1905).** These rules for experimental proof of the pathogenicity (Koch's postulates)are:

(1) The suspected causal micro-organism must be constantly associated with thediseases.
(2) The microorganism should be separated (isolated) from the diseased host and grown in artificial (pure)culture.
(3) The pathogen from the artificial culture should be able to reproduce the disease, when inoculated on a healthy plant of the same kind from which it was isolated. Symptoms produced should be identical with those seen on the plant from which isolation was made.
(4) The microorganism should be re-isolated from the inoculated infected host and identified as being identical to the original specific causative agent (as obtain in step 2).

(A) Symptomatology (VisualObservation)

Plant pathogen induce various reactions in the body of their host plant. This result in creation of abnormalities which become visible on the plant.

- **Symptom:** A visible or detectable abnormality in structure or function observed on the plant as a result of disease or disorder is called***symptom.***
- **Sign:** Pathogen or its parts or products seen on the affected parts of a host plant is called***sign***.
- **Syndrome:** It may defined as sequential appearance of disease symptoms on a plant during the development of the disease **or** Sum of total symptoms exhibited by a disease. These are the visible expression of host-pathogen interaction.

1. Symptoms Induced by ParasiticFungi

Diseases caused by fungi are identified primarily by symptoms and signs produced on host. Major symptoms of plant diseases caused by fungal pathogen are summarized below.

Sr. No.	Symptoms	Fungus	Diseases
1	Pathogen seen as white, gray, brownish or purple growth on lower side of the leaves surface	Downy mildew fungi	Downy mildews
2	Enormous numbers of spores formed on superficial growth giving host surface a dusty or powdery appearance; black fruiting bodies (***Cleistothecia***) mayalso develop.	Powdery mildew fungi	Powdery mildews
3	Pustules of spores, usually breaking through host epidermis, dusty or compact. Red, brown, yellow or black in colorspore found on the host surface	Rust fungi	Rust diseases
4	Charcoal-like and black or purplish-black dust like masses developed on the affected plant parts, mostly on floral organs and inflorescence are called smut	Smut fungi	Smut diseases
5	On leaves of *cruciferous* and other plants numerous white coloured blister-like pustules are produce on the host epidermis which break open and expose powdery masses ofspores.	*Albugo*	White blister or White rust
6	Excessive growth of host tissues; abnormal increase in size due to abnormally increased cell size (**hypertrophy**) or increased cell divisions (**hyperplasia**)	*Albugo*, Downy mildew fungi,	Galls, curl, knots, witches, broom, wart
7	**Atrophy** known as **hypoplasia** or **dwarfing** which is resulted from the inhibition of growth dueto reduction in cell division or cell size.	Several fungi	Stunting, dwarfing, curling, puckering

8	Localized lesions on host leaves consisting of dead and collapsed cells. (Necrosis) **Necrosis:** It indicates degeneration of protoplast followed by the death of cells, tissues and organs resulting from infection by pathogen. Necrotic symptoms include spots, blights, burn, canker, streaks, stripes, damping-off,rot etc.	Several fungi	Leaf spots
9	Uniform, general and very rapid browning and death of foliage (leaves. branches, twigs, floral organs).	Several fungi	Blights
10	Necrosis and sunken ulcer like lesions on stem, leaf, flower or fruits. It typically gives *shot hole symptoms* on leaves.	*Colletotrichum* spp. *Glomerella* spp.	Anthracnose diseases
11	Necrotic dead area in the bark or cortex of the stem and root especially in woody plants.	Several fungi	Cankers
12	The softening, discoloration and often decay or disintegration of a succulent plant tissue as a result of fungal or bacterial infection.	Many fungi	Rots
13	Loss of turgidity, flaccid, dropping of leaves or shoot due to disturbance in the vascular system of root or the stem. Wilting may be result of injury to the root system, to partial plugging of water conducting vessels or toxic substances secreted bythe pathogen.	*Fusarium* spp. *Verticillium* spp.	Wilts
14	It is the collapse and death of seedlings due to extensive necrosis of stem tissues before or after they emerge from the soil.	*Pythium*, *Rhizoctonia*	Damping off of seedlings

15	Drying of plant organs such as stem or branches which starts from the tip and progressesgradually towards the main stem or trunk.	Several fungi	Die-back or Wither tip
16	Final stage of certain fruit rot in which the dried, shriveled and wrinkled fruit is called a "mummy". The stage is brought about by loss of moisture due to permeation of the flesh byfungus hyphe.	Several fungi	Mummification
17	*Roughened or crust-like lesion* or to a freckled appearance of the diseased organ. Appearanceof crust like lesions on the host as observed in some diseases	Several fungi and Bacteria	Scab
18	Dark and hard structures of various shaped composed of dormant mycelia of some fungi called sclerotia. Sometimes, sclerotia are developed on the affected parts of the plant. Presence of sclerotia on the host surface is specifically called a sign of disease rather thansymptom.	*Sclerotium* spp.	Sclerotial Development
19	Burning of leaf margins as a result of unfavorable environmental conditions *like as;* high temperature.	-	Scorch

2. Symptoms induced by bacterial pathogens

Formation of encrustations or bacterial ooze from infected tissues is unique feature associated with plant bacterial diseases. The various types of symptoms *like as;* leaf spots, blights, scabs, cankers, tumors, wilts, soft rots produce due to bacterial infection in plant. Sometimes masses of bacterial cells (ooze) comes out form the surface of affected plant parts and form some drops or smear, it is called **exudation**. This exudation forms a crust on the host surface afterdrying.

3. Symptoms induced by nematodes

Nematodes infect both root and above ground portion of plants. The various types of root symptoms *like as;* root knots, root galls, root lesions, excessive root branching, injured root tips and root rots produce due to nematode infection in plant. Certainspecies

of nematodes infect the above ground portions of plants. They cause galls, necrotic lesions and rots, twisting and distortion of stem and leaves and abnormal development of floral parts.

4. Symptoms induced by viruses and viroids

Plant viruses cause a variety of symptoms, depending on the host plant species. The viruses induce primary symptoms on leaves, which exhibit chlorotic or necrotic local lesion or vein clearing and colour changes. Various kinds of changes in size and shape of plant part may be seen as leaf roll, leaf curl, enation, leaf crinkle, galls and tumors. General stunting of branches or entire plant, rosette nature of leaves, and bunching or crowding of leaves at the apex leading to bunchy top are some of the symptoms associated with plant virusdisease.

Colour change

- **Etiolation**: When the yellowing of the leaves is due to lack of light or prolonged exposure to darkness, the condition is known as***etiolation***.
- **Chlorosis**: When the yellowing of the leaves is due to influence of low temperature, lack of iron, excess of lime or alkali, presence of certain virus diseases or fungal or bacterial diseases is known as***chlorosis***.
- **Albinism**: When the leaves are devoid of any pigment and looked bleached or white. The colorless condition is known as***albinism***.
- **Chromosis:** It is a condition in which the green pigment is replaced by red, purple or orangepigment.

5. Symptoms induced by phytoplasmas(MLO)

The MLOs cause general stunting or dwarfing of affected plant parts or whole plants. Chlorosis and reduced leaves are also frequently observed. **Phyllody or virecence** i.e. floral organ converted in to vegetative structure (greening of petals and conversion of petals to leafy structure observed) due to its infection. Partial or total sterility of infected plants may be commonly noted. These symptoms are observed in various plant diseases such as; aster yellows, little leaf of brinjal and sesame phyllody.

(B) Host Range

Host range is also a valuable traditional approach for detection and identification of a pathogen, particularly of its physiological races. Some fungal and bacterial pathogens are given trinomials according to their host. For example, the fungal pathogen *Puccinia graminis* is classified into many physiological races according to the hosts that they infect. *Puccinia graminis* f. sp. *tritici* infects wheat and *Puccinia graminis* f. sp. avenae infects oats causing rust diseases. Physiological races show variation in their virulence towards a range of genotypes of the host plant, resulting in severe symptoms on some host plants but not produce any symptoms on others. For that, host rang is utilized for detection of a pathogen anddiseases.

(C) Morphology of PlantPathogen

The morphological features of fungi such as spores, fruiting bodies or sporophores, which differ from one fungus species to another. It is important for diagnostic of many diseases. Different features such as; cell shape, flagellation, and gram reaction are essential for diagnosis of bacterial diseases. Later, the pathogen can checked in an appropriate book of plant pathology to see whether it reported to be pathogenic, especially on the plant on which it was found. If the symptoms of the plant as well as the morphology of the pathogen correspond those listed in the book, then the diagnosis of the disease is considered complete. The characteristics of some bacterial genera that are used invariably for diagnosis are summarized inTable.

(D) Isolation of Pathogen on to selectivemedia

Some plant pathogens grow on specific culture media. For these, placing piece of infected plant part on a suitable culture medium and inspecting the resulting colonies of the pathogen may be sufficient for a positive identification of plant disease. e.g. Nash and Snyder medium (NS) used for isolation of *Fusarium* spp.

Table: Morphological characteristics of some Plant Pathogenic bacterial

Bacterial Genera	Gram Reaction	Shape	Flagellation	Colony colour	Diffusible pigments	Diseases symptoms
Clavibacter (Corynebacterium)	Gram Positive	Rod, straight or slightly curved	None	Variable orange, yellow or blue	Usually absent	Wilt or leaf spot
Erwinia	Gram Negative	Rod	Peritrichous	White or yellow	Usually absent Pink or bluein somespecies	Soft rot, vascular wilt, leaf spot
Pseudomonas	Gram Negative	Rod	One or many polar flagella	White or yellow	Fluorescent	Soft rot, vascular wilt, leaf spot
Xanthomonas	Gram Negative	Rod	Polar	Yellow (rarely white)	Usually absent	Canker, leaf spot, vascular wilt
Agrobacterium	Gram Negative	Rod		White (rarely yellow)	Usually absent	Crown gall, hairy root, soil saprobes
Streptomyces	Gram Positive	Mycelium	None	White	Usually absent	Scab, corky tissue
Xylella	Gram Negative	Straight Rod	None	Small, Smooth Non-Pigmented	Usually absent	Infect xylem of plant tissue

2. Molecular diagnostics

All organisms have unique molecular characteristics (biochemical characteristics), which can be used for identification of plant pathogen and corresponding plant disease diagnosis.

- **Characteristics of ideal molecular diagnostictechnique**

(1) Accuracy (high specificity for targetpathogen)
(2) Sensitivity (ability to detect small amounts ofpathogen)
(3) Simplicity (does not require complex laboratoryfacilities)
(4) Rapidity (early detection ofpathogen)
(5) Cheapness (easily affordable anddisposable)
(6) Safety (non-hazardous foroperator)

- **Methods for molecular diagnosis ofdisease**

(1) Chemo diagnostic tests
(2) Serologicaltechniques
(3) Fatty acidprofiles
(4) Proteinanalysis
(5) Techniques of nucleic acidanalysis

1. Chemo-diagnostic tests

Chemo-diagnostic tests depend on the reaction between reagents and certain chemical compounds present in the infected plants which gives visible colour reaction. Detection of viruses, phytoplasma (MLOs), fungi and bacteria by using such tests involve use of dyes, chemicals, selective media and fluorescent markers.

The presence and viability of fungal spores can be determined by **fluorescein diacetate assay (FDA)**. Lipase activity is detected in the extracts of viable teliospores of *Tilletia controversa* by using fluorescein diacetate assay. Similarly, Wheat seed embryos are stained with **trypan blue** to detect loose smut disease. If the fungal mycelium is present in seed embryos, it looks blue.

In some cases, the pathogens can be identified by their production of unusual metabolites. For example, strains of the fungus *Aspergillus flavus,* which produce aflatoxin. This metabolite is not produced by non-toxigenic strains of this fungus.

The bacterial pathogens can be detected by use of selective media and several biochemical tests *like as;* reduction of nitrates, hydrogen sulfide production, production of indole acetic acid, utilization of carbon and nitrogen compounds, starchhydrolysis,

ammonia production etc. **Fluorescent marker** substances have been used for indexing plant materials for the diagnosis of citrus greening and exocortis disease.

Different kind of chemical test *like as;* protein test, carbohydrate test, enzyme test, polyphenol test, nucleic acid test and fluorescent test has been used for detection of plant viral diseases. Tetrazolium chloride test is used for detection of banana bunchy top virus and cassava mosaic virus. If infected plants retain brick red color, it indicates viral infection. Similarly, iodine test is used for detection of rice tugro virus infection. Several histo-chemical methods have been used for the detection of infection of phytoplasma (MLOs). **Dienes's stain** has been used to know the presence of phytoplasma (MLOs) in rice yellow dwarf, little leaf of brinjal, sandal spike and coconut root wilt. Dark blue colour is retained in the phloem cells of phytoplasma infected plants, indicating the positive reaction.

(2) Serologicaltechniques

The study of relation between an antigen and an antibody is called **serology**. Antigen and antibody reaction results in the formation of visible substrate (precipitation), which is perhaps due to the binding between the two, which is utilized for plant disease diagnosis. This techniques is mostly used for detection and diagnosis of plant viral disease.

- **Antigens:** Any substance which induces the production of antibodies is called an antigens. It includes proteins, polysaccharides, lipids, carbohydrates, nucleic acids enzymes, toxinsetc.
- **Antibody:** A specific protein formed in the blood of warm-blooded animals in response to injection of an antigen (protein or polysaccharide). It is responsible for specific recognition and elimination (neutralization) of antigens. Antibodies are proteins termed as γ globulin or immunoglobulin (Ig). Each antibody is specific to a particular antigen and will bind toit.
- **Types of SerologicalTest**

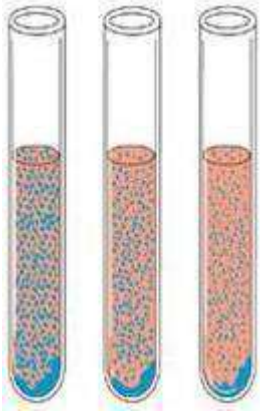
(A) Direct test	**(B) Indirect test**
• **Precipitationtest** 1. Tubeprecipitation 2. Ring interfaceprecipitation • **Micro Precipitationtest** • **Agglutinationtests** 1. Chloroplastagglutination 2. Latexagglutination ✓ **Gel diffusiontest** ✓ **Immuno-electrophoresis**	• **ELISAtest** 1. Direct ELISA (DASELISA) 2. Indirect (DACELISA) 3. DIBAELISA • **Immunofluorescence** • **Immuno Sorbent Electron Microscopy(ISEM)** • **Lateral flowtechniques**

(A) Direct test

- **Precipitationtest**

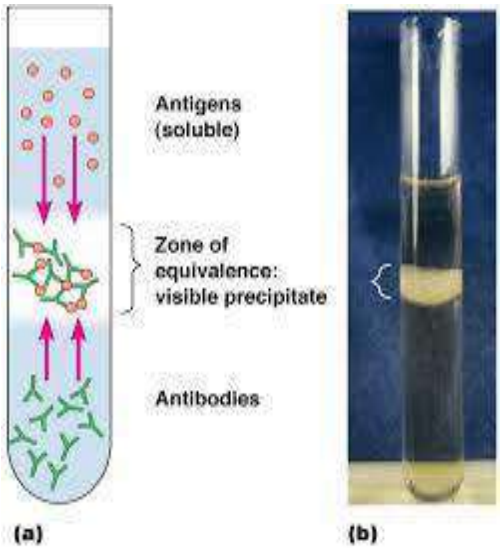
1. Tube precipitation

This test is widely used. Various dilutions of clarified virus suspension and antibody are mixed in narrow thin walled glass tubes and incubated at 37°C in a water bath. The precipitation is observed in the mixture. The type of precipitate formed depends on the shape of the antigen (virus particles).



2. Ring interface precipitation

If the virus-containing suspension is carefully layered on top of the viscous, undiluted antibody suspension (antibody to which glycerol or sucrose had been added) in a small glass tube, at interface a ring or band rapidly becomes visible. With time, anti- body diffuses into the virus solution and virus diffuses into the antiserum and near the boundary a specific precipitate ring is formed.



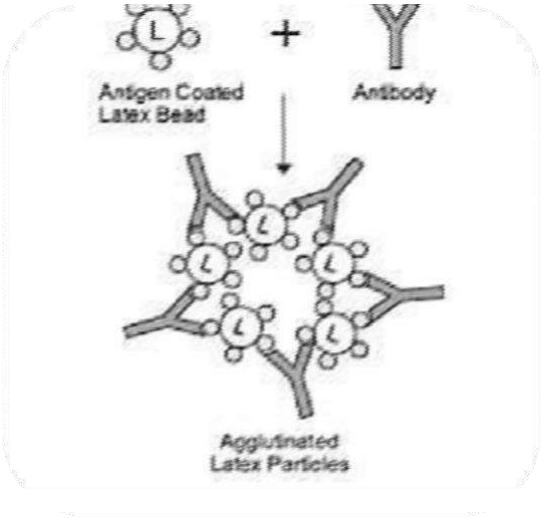
- **Micro Precipitationtest**

This test is done on a micro-scale to reduce the wastage of antibody (antiserum). Drop of dilution mixture (antiserum & virus suspension) are mixed at bottom of a Petri plate. The precipitates produced are observed with a microscope with dark-ground illumination. The precipitation varies, depending on the ratio of concentrations of antigen and antibodies.

- **Agglutinationtests**

Chloroplast agglutination test used for diagnosis and detection of plant viruses. It is most effective for viruses with elongate particles and which usually occur in high concentrations in the plant. In this test, drops of antiserum against the virus are added to drops of crude plant extract on a microscope slide. If the plant is infected, the antibodies will bind to virus particles adsorbed to the chloroplasts. If enough antibodies are present for extensive linking of chloroplasts, these organelles will be agglutinated. This is visible with naked eye, as a clumping of the chloroplasts in thedrop.

In **latex agglutination test** an antibody is adsorbed onto **polystyrene latex** (a larger object) beads. The antibody linked sensitized latex is mixed with antigen in round- bottom microtiter plates. Flocculation of the latex indicates antigen-antibody combination.



✓ **Gel diffusiontest**

This test is done in petri-dishes and precipitation in the form of a visible band or spur is seen in solid agar media. The reactants, antiserum and virus solutions are placed in wells cut in the agar (l % agar containing 0.85% NaCI and 0.02% sodium azide) on the bottom of a petri-dish. In the **Ouchterlony test** the antibodies and virus diffuse into the agar towards each other from the adjacent wells and where they meet precipitation zones in the form of white bands are formed. Bands can be recorded by direct observation with appropriatelighting.Thevirussuspensionkeepinperipheralwellsaroundacentralwell

containing the antiserum. There is no spur or band formation with healthy sap. Since both the reactants diffuse in the test, it is also known as the ***double-diffusion test***.

✓ **Immuno-electrophoresis**

In this test components of the antigen mixtures are first separated in an electric field in an agar gel containing an appropriate buffer. Thereafter, antiserum is put into a long trough at one side of the well parallel to the direction of the electrophoretic migration and an immunodiffusion test is carried out. Antigenic components and antibodies then diffuse together and produce bands, which can be observed. Closely related strains of virus may then bedistinguished.

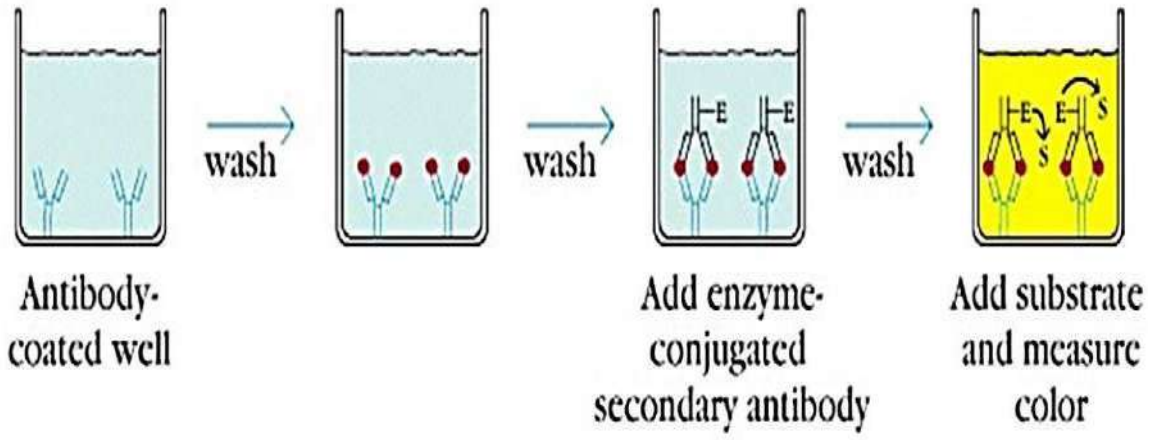
(B) Indirecttest

✓ **ELISA Test (Enzyme Linked Immunosorbent Assay)**

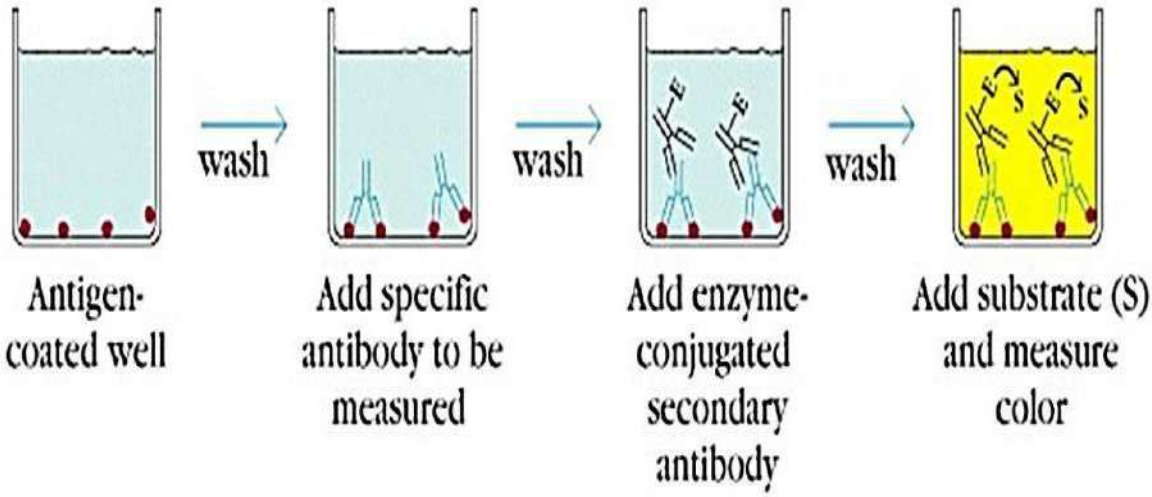
Most commonly used serological method is ELISA (Enzyme Linked Immunosorbent Assay) which was developed by **Clark & Adam (1977)** for the detection of plant viruses. The ELISA procedure has been standardized for the detection of many plant viruses, fungi, bacteria and mycoplasma-like organisms. The tests are usually carried out in multi well plastic plates or polystyrene microtiter plate. The basic principle of ELISA is the detection of solid phase bound antigen and antibody complex by a colour reaction using specific enzyme andsubstrate.

Steps for ELISA Test

- ✓ Firstly, antibodies specific to a virus are fixed to the base of thewells.
- ✓ Then the sap, extracted from plant tissue, is placed in the wells. If the sap contains virus particles matching the antibodies, the virus will bind to theseantibodies.
- ✓ Further antibodies bound to enzyme such as ***alkaline phosphatase***, are added to the wells and these antibodies will also bind to thevirus.
- ✓ A suitable colorless substrate (p-nitrophenyl phosphate) is added in the well. The substrate is degraded and a bright yellow colour is produced due to production of p-nitrophenol. This indicates a positive reaction. The coloration can be measured quantitatively in a spectrophotometer at 405nm.



DAS ELISA (Double antibody sandwich ELISA)



DAC ELISA (Direct antigen coating ELISA)

- **Types of ELISATest**

1. DAS - ELISA (Double antibody sandwichELISA)
2. DAC - ELISA (Direct antigen coatingELISA)
3. DOT - ELISA (Dot immunobinding assay)(DIBA)

- **Advantage ofELISA**

1. It is comparatively fast method 96 samples tested in 2-3hr.
2. It is very sensitive for detection of pathogen (1-10ng/ml).
3. It can able to detect specific pathogen fromsample.
4. Small sample sizerequired
5. Application against large number of sample
6. No radiation hazards, easy to learn and simpleprocedure

Features	ELISA	DIBA
Full form	Enzyme linked immune sorbent assay	Dot immunobinding assay
Discovered by	Clark and Adams	Bantarri and Goodwin
Plate/membrane used	Polystyrene	Nitrocellulose membrane
Final product	Soluble	Insoluble
Volume of antigen (sample) required	More	Less
Substrate	Paranitro phenol phosphate	BCIP (5 bromo-4 chloro, 3-indolyl phosphate) and NBT (Nitro blue tetrazolium)
Intensity of color observed in	Spectrophotometer	Densiometer
Application in field diagnosis	Not used for field diagnosis	Easy to transport to the field
Complexity and time consuming	More complex and time consuming	Less complex, simple and rapid than ELISA

- **Immuno Sorbernt electron microscope (ISEM)**

This method is combination of electron microscopy and serology. It was first time described by Derrick in1973. The film on electric magnetic grid is first coated with specific antibody for the virus being studied. After that virus and antiserum are reacted together. Antigen are trapped onto grid coated with specific antiserum which is negatively stained with uranyl acetate and the result viewed in the electron microscope. This methods generally useful for detection of virus and for study of itsmorphology.

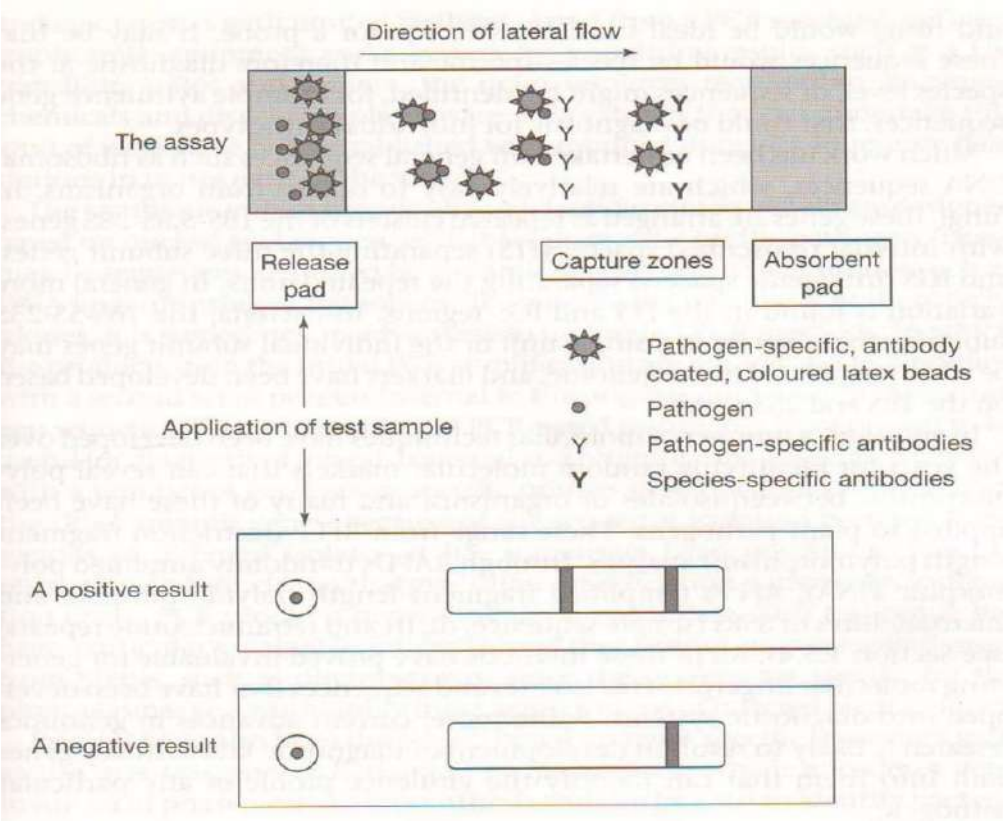
- **Immunofluorescence**

This method is utilized for detection of intercellular location & distribution of viruses. Globulins mixed with a fluorescent dye (Fluorescein isothiocyanate and Rhodamine B) introduced into the infected cells/tissue, with antigen and antibody reaction, fluorescence takes place. The technique is particularly suited to detecting viral antigen in insect vector. Cells to which fluorescent antibody bind emit a bright fluorescent colour which may be green, red,yellow.

- **Lateral flowtechniques**

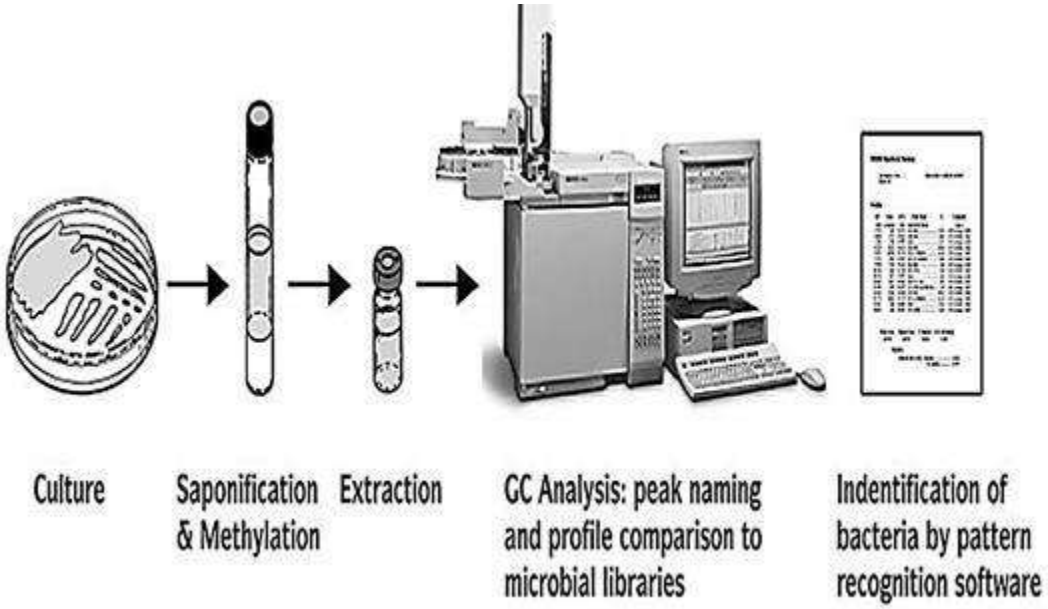
Dipstick methods suitable for field use by crop inspectors and extension workers without using any sophisticated equipment for detection of some plant pathogens such as the potato viruses PYX and PVY. This method provide results within a few minutes. The

pathogen-specific antibodies are bound to coloured latex beads or gold particles preimmobilised onto membranes in the release pad. When the sample (diluted sap extract from plant tissue) is applied to the lateral flow strip, any antigen within it will form a complex with the tagged antibodies as the solution moves through the membrane by capillary action, The bound antigen/antibody complex will continue to move by lateral flow through the membrane until it reaches the capture zones. The first preprinted capture line (containing pathogen-specific antibodies) is designed to trap the bound antigen/antibody complex, and as these are arrested, the line becomes visible as the coloured latex or gold accumulates. The second capture line is the control line, containing species-specific antibodies designed to capture all the unbound antibodies and confirm that the assay has run correctly.



3. Fatty acid profiles (FAMEanalysis)

Application of fatty acid profiles to identify phytopathogenic bacteria has proven very accurate. In fatty acid methyl ester analysis (FAME analysis) about 40 mg of wet bacterial cells are saponified and methylated, and fatty acid methyl esters (FAME) are extracted in a mixture of ether-hexane. The latter is analyzed by gas chromatography and the zones of the resulting peaks on the chromatograms are calculated and compared with profiles of known reference strains by computer programmes.



4. Nucleic Acid Based Methods

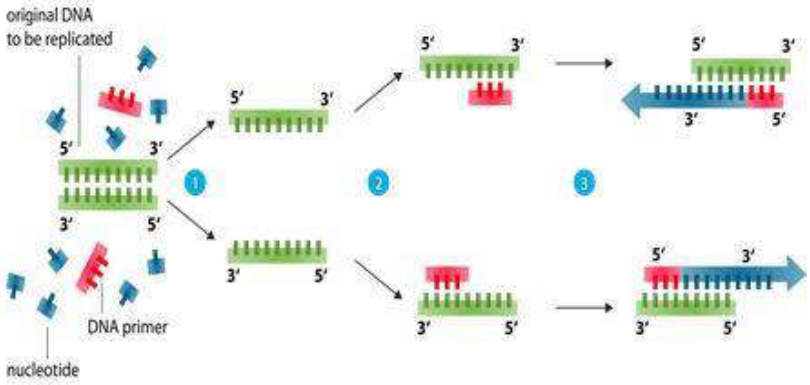
❑ **Polymerase chain reaction(PCR)**

Polymerase chain reaction (PCR) is a method widely used in molecular biology to make many copies of a specific DNA segment. Using PCR, a single copy (or more) of a DNA sequence is exponentially amplified to generate thousands to millions of more copies of that particular DNA segment. Based on the DNA hybridization and replication, PCR is initially used for highly specific detection of diseases caused by plant pathogens. This technique was developed by **Kary mullis** in 1983. The primary materials or reagents used in PCR are DNA nucleotides, template DNA, primers, DNA polymerase*etc.*

❑ **Steps ofPCR**

1. **Denaturation:** Two strand of DNA separates (melt down) to form single stranded DNA. This step is generally carried out at 94^0-96^0 C for 2minutes.
2. **Annealing: The two primers bind the suitable complementary strand.** This step is carried out at 45^0-55^0C.
3. **Extension:** In this step, the DNA polymerase synthesizes a new DNA strand complementary to the DNA template strand by adding free dNTPs (Deoxynucleoside triphosphates) from the reaction mixture that are complementary to the template in the 5'-to-3' direction. It is carried out at temperature of 72^0C.

These three steps are repeated 20-30 times in an automated thermocycler that canheat and cool the reaction mixture in tube within very short time. This results in exponential accumulation of specific DNA fragments.



- **Gelelectrophoresis**

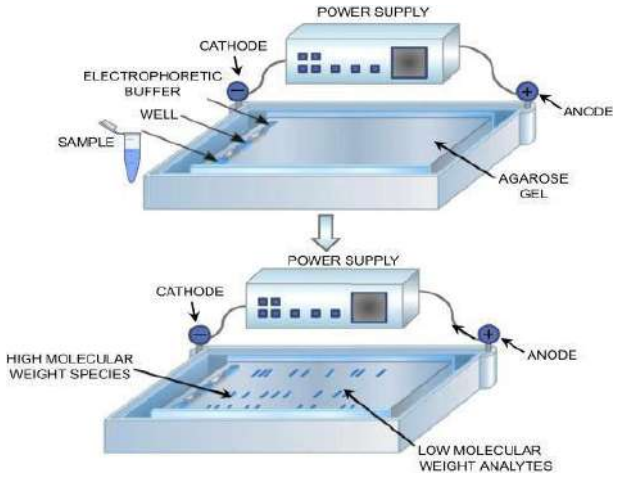
✓ It is a technique used to separate DNA fragments according to their size.DNA samples are loaded into wells (indentations) at one end of the agarose gel, and an electric current is applied to pull them through thegel.

✓ DNA fragments are negatively charged, so they move towards the positive electrode. When a gel is stained with a DNA-binding dye, the DNA fragments can be seen as bands, each representing a group of same-sized DNAfragments.

✓ Nucleic acids extracted from plant pathogens is amplified by polymerase chain reaction. These amplified fragments are separated according to their size by gel electrophoresis.

✓ In gel electrophoresis, agarose gels are used to separate nucleic acid fragments of longer length. Polyacrylamide gels are used for separate fragments of shorterlength.

✓ The bands differentiated on gels are stained with ethidium bromide to make them visible under ultravioletlight.



❑ **Different types ofPCR**

Types of PCR	Use
RT- PCR (Reverse Transcription – PCR)	For detection of RNA containing viruses
IC -PCR (Immuno Capture PCR)	Used for detection of insect vectors of pathogen
M – PCR (Multiplex - PCR)	For simultaneous detection of two or more pathogens in a single sample.
Real Time -PCR	Quantifying the accumulation of pathogen
Bio - PCR	Used to detect the plant pathogenic bacteria present in extremely low level, in seeds and other propagative materials

Molecular markers in detection of Plant Pathogens

1. PCR Basedapproaches

(A) RAPD (Random Amplification of PolymorphicDNA)

- ✓ It is a type of PCR, but the segments of DNA that are amplified arerandom.
- ✓ No knowledge of the DNA sequence of the targeted genome is required, asthe primers will bind somewhere in the sequence, but it is not certain exactly where. Only one type of primer utilized in thismethod.
- ✓ The resultant PCR products are resolved on agarose gel following the application of ethidium bromide or fluorescently labeledprimers.

(B) Amplified Fragment LengthPolymorphism

- ✓ This technique is a combination of RFLP and RAPD. The technique was described by Vos and Zabeau in1993.
- ✓ This method is technically more sensitive and highlyreproducible.
- ✓ AFLP uses restriction enzymes to digest genomic DNA, followed by ligation of adaptors to the sticky ends of the restrictionfragments.
- ✓ A subset of the restriction fragments is then selected to be amplified. This selection is achieved by using primers complementary to the adaptor sequence, the restriction site sequence and a few nucleotides inside the restriction sitefragments.
- ✓ The amplified fragments are separated and visualized on denaturing on agarose gel electrophoresis.

2. Non- PCR Based approaches

(A) Restriction Fragment Length Polymorphism (RFLP)

- ✓ The technique involves fragmenting DNA sample by a restriction enzyme. Restriction enzyme can recognize a specific short sequence of DNA and cut it. This process known as a restriction digest.
- ✓ The resulting DNA fragments are then separated by length through a process known as agarose gel electrophoresis and transferred to a membrane via the Southern blotprocedure.
- ✓ Hybridization of the membrane to a labeled DNA probe then determines the length of the fragments which are complementary to theprobe.

Features	RAPD	RFLP	AFPL
Full form	Random amplified polymorphic DNA	Restriction fragment length polymorphism	Amplified Fragment Length Polymorphism
Discovered by	William	Bolstein	Vos and Zabeau
Principle	Random amplification of DNA using randomprimer	Restriction of DNA using restriction enzymes and then run on a gel	Cutting of DNA with restriction enzymes, ligation of adapters and amplification of sets of restriction fragments
Based on	PCR	Restriction enzyme	Restriction enzyme, Ligation and PCR
Primer/Probe used	Random primer	Probe not primer	Selective primers
Reproducibility	Intermediate	High	High
It involve	Amplification and Detection	Cutting and Detection	Cutting, Ligation and Detection
DNA required quantity	Low	High	Medium
Prior information of targeted genomeneeded	Yes	No	No
Recurring cost	Low	High	Medium
Detection	DNA staining	Southern blotting	DNA staining

❑ BlottingTechniques

- ✓ It is a method of transferring proteins, DNA or RNA, onto a carrier (nitrocellulose or nylon membrane) which is done after a gelelectrophoresis.
- ✓ After the blotting, the transferred proteins, DNA or RNA are then visualized by colorant staining which are then used as target for hybridization with labeledprobes.
- ✓ There are three types of blottingtechniques.

1. Southern blotting

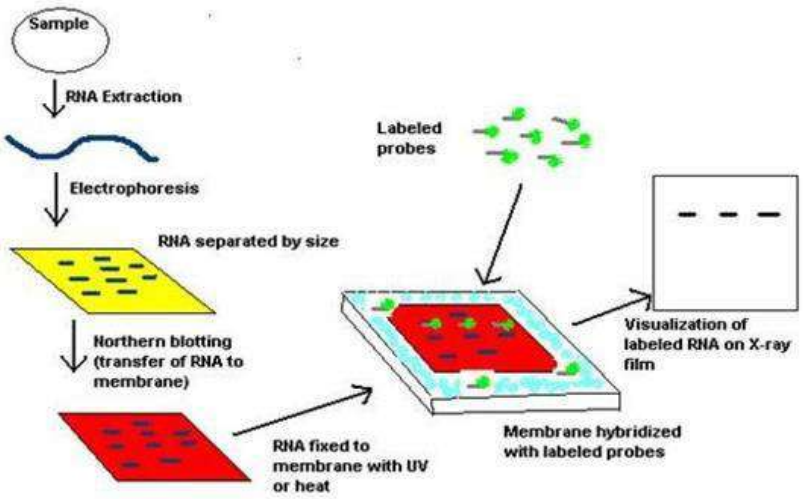
- ➢ This method was developed by Sir Edwin Southern in 1975. It is a method used in molecular biology for detection of a specific DNA sequence in DNA samples. This method involves three steps *like as;* separation, transfer andhybridization.
- ➢ Southern blotting combines with electrophoresis which separated DNA fragments. Later separated DNA fragments transfer to a filter membrane and subsequent fragment detection by probehybridization.

2. Northern Blotting

- ➢ Northern blotting was developed by James Alwine and GeorgeStark.
- ➢ The northern blot is used for the detection of specific RNA sequences in complex samples. In northern blotting techniques, first separates samples by size *via* gel electrophoresis before they are transferred to a blotting matrix and detected with labeled RNAprobes.

3. WesternBlotting

- ➢ A Western blot is used for the detection of specific proteins in complexsamples.
- ➢ In this method, proteins are first separated by size using electrophoresis on SDS- PAGE (Sodium dodecyl sulphate) before being transferred to an appropriate blotting matrix (Polyvinylidene fluoride (PVDF) or nitrocellulose) and subsequent detection byantibodies.



Features	PCR	Southern	Northern
Discovery	Kary Mullis	Edwin Southern	Alwine &Stark
Purpose	DNA amplification	Detection of specific DNAsequence	Detection of specific RNAsequence
Stages	Denaturation Annealing Extension	Separation Transfer Hybridization	- - -
Hybridization	-	DNA	RNA
Radioactive detection	-	Yes	Yes

Features	Northern/Southern blotting	Western blotting
Use	Southern blotting is employed for DNA Northern blotting is employed for RNA	Western blotting is employed forProtein
Discovered by	Southern blotting – E. M. Southern Northern blotting – Alwine & Stark	Name given by: Burnette Procedure given by: Towbin
Gel used	Agarose gel	Polyacryl amide gel
Transfer from gel to membrane	Passive transfer	Active transfer
Rate of transfer and efficiency	Transfer is slow and less efficient	Transfer is fast and more efficient

Chapter: 5
Calculation and Dynamic of Economic Injury Level and Importance of Economic Threshold Level

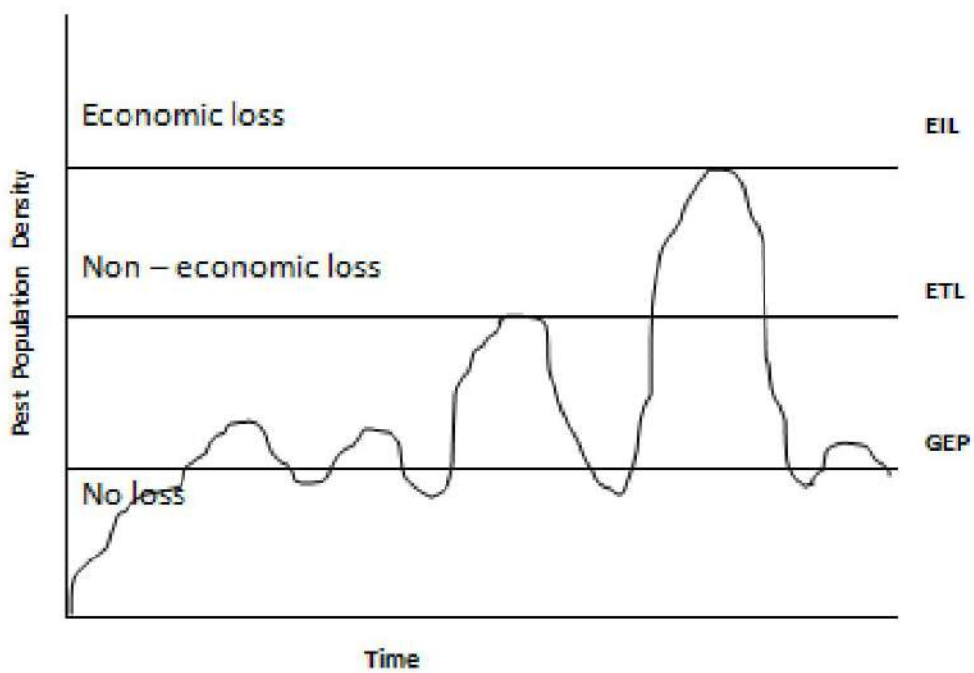
Concept of Injury Levels

The concept of injury levels helps us to identify the population level at which an insect/pathogen can cause damage to the crop. Most popular terms used in this connection are the economic injury level (EIL) and the economic threshold level (ETL).

- **Economic Injury Level (EIL):** Lowest pest population at which the pest will cause economic damage

or

It is the pest level at which the damage can not be tolerated by crops and therefore it is the level at or before which the control measures are initiated.



- **Economic Threshold Level (ETL)**

✓ It is the index for making pest management decisions.

✓ **ETL is defined as** the pest population density at which control measures should be initiated against an increasing pest population to prevent economic damage.

✓ Relationship between EIL and ETL can be expressed as when no action is taken at ETL the population reaches or exceeds EIL.

- ✓ In economic term, ETL is defined as the level to which a given pest population should be reduced to achieve the point where marginal revenue just exceeds marginal costs.
- ✓ The relationship between EIL and ETL is shown in chart.
- ✓ When no action is taken at ETL, the population reaches to the EIL; while when management steps for pest suppression are taken as the population crosses ETL, the population is forced down before it could reach EIL.

- ❑ **General equilibrium position (GEP):** It is the average population density of pest over a long period of time, unaffected by temporary interventions of pest the control. However the economic injury level may be at any level well above or below general equilibrium.

Calculation of Economic Injury Level

- ✓ If management action (pest suppression) can be taken quickly and loss avoided completely, the EIL can calculated as….

$$EIL = \frac{C}{V \times I \times D \times K}$$

EIL = No. of injury equivalents per production unit (pest/ha).
C = Cost of management activity per production unit (Rs/ha)
V = Market value per unit of product (Rs/tonne)
I = Crop injury per pest density
D = Damage per unit injury (tonne reduction/ha)
K = Proportional reduction in injury with management

Sometimes two variables D+I combine in to a single variable,

$$EIL = \frac{C}{V \times D' \times K}$$

D' = Percent yield loss per pest.

Example:
If a seasonal average of one pest per plant causes a 10% reduction in yield, the **market value of the crop** is Rs. 40 per kg fruit and you expect 5 kg fruit/plant yield, the **cost of control** is Rs. 450/plant, and you can count on a 75% reduction in damage with the control tactic used, then calculate Economic injury level for this pest.

$$EIL = \frac{C}{V \times D' \times K}$$

$$\text{EIL} = \frac{\text{Rs. 450 cost per plant}}{\text{(Rs. 40 x 0.5 kg/pest x 0.75)}}$$

$$= 30 \text{ pest/plant}$$

Factors Influencing ETL and EIL

❑ **Primary factors**

a. Market value of crop: When crop value increases, EIL decreases and vice-versa.

b. Management costs: When pest management costs increase, EIL also increases.

❑ **Secondary factors**

c. Degree of injury per insect:

- ✓ Insects damaging leaves or reproductive parts have different EIL (Lower EIL for reproductive part damages).
- ✓ If insects are vectors of disease, EIL is very low, even 1 or 2 insects if found management to be taken.
- ✓ If insects found on fruits - Marketability reduced - EIL very low

d. Crop susceptibility to injury: If crop can tolerate the injury and give good yield. EIL can be fixed at a higher value. When crop is older, it can withstand high pest population - EIL can be high.

❑ **Importance of Economic Threshold Level**

- ✓ It helps in decision making process for scheduling various control/management methods against different pest.
- ✓ In ETL optimal control of pest occurs, which can be used to minimize risk of economic damage and environmental hazards.

Factors Important For Determining ETL

1. Life cycle of pest/pathogen
2. Time required for control
3. Environmental conditions

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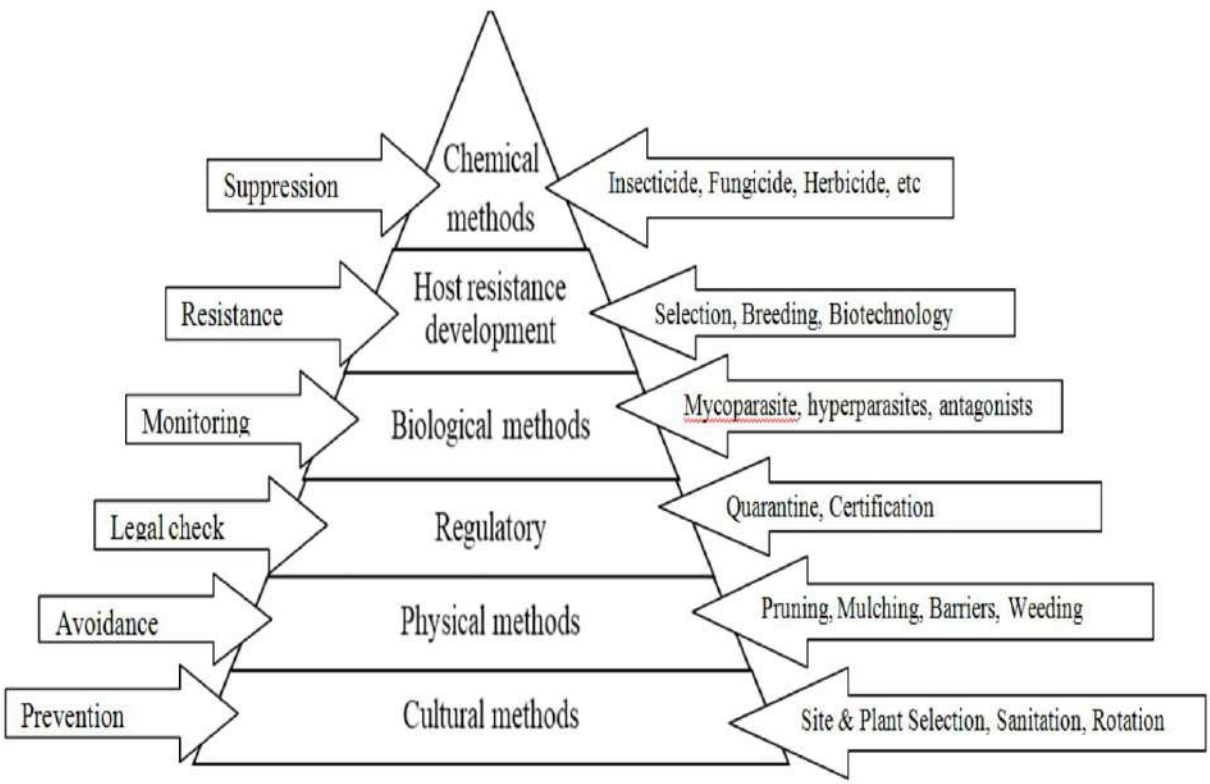
Chapter: 7
METHODS OF PLANT DISEASES CONTROL

The main objective of plant pathology is the economic control of plant disease. Different methods are utilized for the management of plant diseases *such as;*

1. Cultural methods
2. Physical methods
3. Legal methods
4. Biological methods
5. Host resistance development
6. Chemical methods



I. Cultural Control

The term ***cultural control*** describes the activities of humans for controlling disease through the cultural manipulation of plants. Cultural practices involve all the activities carried out on the farm before, during and after planting of crops. They are grouped into: pre-planting, planting and post-planting operation.

Pre Planting Operations

1. Crop rotation

Crop rotation is essentially a preventive measure and has its effect mainly on the succeeding crop. Crop rotation is the oldest and cheapest method adopted in agriculture for eradication of certain types of pathogens from infested soil. Continuous cropping or mono-culturing provides opportunity for perpetuation of pathogenic organisms in the soil when the same crop is raised year after year in the same field. After sometime, the soil becomes so heavily infested that it becomes unfit for cultivation of the particular crop. Virus diseases of crop plants and their vectors are found to increase after every crop if a crop is cultivated continuously in a field.

On the other hand, when ***immune, resistant or non-host crops*** are grown for a definite duration after a susceptible crop in the field, the pathogen will be starved off and the population of such pathogens consequently decreases due to absence of nutrition. It is also possible that different crops release some biochemical substances from their root which either directly kill the pathogen or encourage development of antagonistic microorganisms in the soil. In this way, crop rotation is one of the most effective methods of plant disease control. This method is more effective against pathogens which have limited host range and restricted survival ability in soil.

Examples:

- ✓ Bean anthracnose fungus and cabbage black rot bacterium lives in soil for 1 to 2 years. They can be eliminated from soil by adopting 3 or 4 year rotation with non-host crops.
- ✓ Crop rotation is effective in the control of brown stem rot of soybean by rotating soybean with corn.
- ✓ Crop rotation with sugarcane or paddy is effective in the control of 'Panama wilt' of banana. Similarly, crop rotation with paddy is effective in the control of red rot of sugarcane.
- ✓ Rotation of cereal crops like pearl millet, finger millet or fox-tail millet is recommended for the control of root rot of pulse crops.
- ✓ Two year crop rotation with lucerne is recommended in the control of *Verticillium* wilt of cotton.
- ✓ Many diseases such as wilt of pigeon pea, foot rot of betel vine, bacterial blight of rice and cotton are controlled by this method.

2. Fallowing

Keeping the land fallow for some time starves the pathogen and helps in reduction of the inoculum by elimination of the host. This method is not acceptable because of the economic consideration. Root rot disease in different crop plants is controlled by this

method. Flood fallowing has been found effective against wilt disease in banana and root knot nematodes in many crops.

3. Application of organic manures

Addition of organic manures like farm yard manure or green manures or oil cakes to the soil increases the antagonistic microorganisms in the soil. These antagonistic microorganisms reduce the population of soil borne plant pathogens and the diseases caused by them. Application of farm yard manure reduced the root rot of cotton. Application of 5 kg of neem cake/tree three times in a year reduces the basal stem rot of coconut.

Soil amendment

Common scab of potato is reduce by green manuring with soybean. Soil amendment material like as; agriculture waste, rice husk, powdered oyster shells, silicon dioxide, calcium oxide, ferrous oxide *etc* found effective against many soil borne pathogen.

4. Summer ploughing

Deep ploughing during summer periods expose inoculum of pathogen present in the soil. Pathogen propagules destroyed when exposed to sunlight due to the higher temperature prevailing during the summer. During the ploughing operation; infected self sown plants, volunteer hosts plants, weed hosts, alternate and collateral hosts are also destroyed. Here, further spread of the disease is avoided. Groundnut stem root is managed by deep ploughing during summer.

5. Levelling of field and provision of drainage facilities

Water stagnation in different patches of field favours the fungi like *Pythium, Phytophthora, Rhizoctonia* etc. for which proper levelling of the field before sowing or planting is very essential. Further proper drainage facility is necessary in the control of sheath blight of rice. In the control of damping-off diseases of vegetable and other crops, raising seedling in the raised beds method is followed.

6. Burning of stubbles and crop residues

Burning of plant wastes, crop residues, stubbles in the areas selected for raising nurseries for vegetable crops heats the soil and kills the inoculum of the pathogens present in the top layer of the soil. When nurseries are raised in these areas incidence of damping off disease is highly reduced. This practice is also followed in pits made for planting coconut, banana and other fruit trees. Burning of wheat plant every second or third year is suggested for eradication of pathogen from the field.

7. Solar heating (Soil solarization)

When the soil is covered with white polythene sheets during hot seasons, soil temperature increases. Due to soil solarization, soil temperature increase as a result wilt

pathogens eliminated from tomato field. High soil temperature also favors antagonistic fungi.

During Crop Growing Seasons

1. Adjustment of sowing time

Diseases incidence is more severe, when the susceptible stage of the plant growth and favourable conditions for the pathogens coincides. While choosing the time of sowing, it should be taken into consideration that susceptible stage of the crop growth and favourable condition for pathogen does not fall at the same time.

Late planted wheat crops have less infection than wheat planted on normal dates. Avoiding cool and cloudy days for planting will help to reduce red rot of sugarcane. Late sowing of winter wheat and barley is considered to be the most effective measures in reducing take all disease of wheat. 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. Leaf spot diseases incidence is less in early sowing kharif ground nut.

2. Selection of seeds and seed materials

Seeds and seed materials carry many pathogens on it and along with them pathogen may introduce into the field. Seeds and seed materials act as a primary source of infection for disease. Therefore seed and seed materials should be disease free and have a high germinating capacity. The absence of an initial inoculum in seeds is definitely helpful in delaying or suppressing the incidence of the disease. It is a preventive method.

The seed borne diseases *like as;* loose smut of wheat, bacterial blight of rice, bacterial blight of cotton, panama wilt of banana, red rot of sugarcane are control by selecting healthy seed or planting material.

3. Seed rate

Use of higher seed rate in the nursery creates favourable microclimate for the pathogens causing damping-off in vegetables, tobacco, chillies and forest nurseries. Hence, use of optimum seed rate should be used in such crops.

4. Depth of sowing

Shallow planting in wet soils protects wheat plants from flag smut disease of wheat. Deep planting may cause delay in the emergence of seedlings, which may be vulnerable to pre-emergence damping off. Early emergence results in early lignification of tissues which become resistant to attack of soil-borne pathogens. Collar rot incidence in groundnut is more when sowing at higher depth.

5. Spacing

Closer spacing invariably alters the microclimate underneath the canopy of the crop which may provide favourable environment for development of diseases. Boll rot in cotton is quite common in crowded crop. Closer spacing favours many air borne diseases because of high humidity in the crop canopy. Early and late blight of groundnut and blister blight of tea are more in dense canopy. Early spread of black rot of cabbage takes place in closer spacing.

In certain virus diseases like groundnut rosette the incidence is observed to be less when wider spacing is adopted. Closer spacing of rice reduces rice tungro virus infection particularly when vector population is less. Late blight of potato and downy mildew of grapevine spread fast in closer spaced crops.

6. Mixed cropping/ Intercropping

Mixed cropping materially helps in checking certain diseases. Blight of pulse crop has been successfully overcome by growing pulses as a mixed crop with cereals like sorghum and pearl millet. Mixed cropping of pigeon pea and sorghum reduce wilt disease of pigeon pea. Intercropping with moth bean in a cotton field reduced the root rot incidence.

7. Decoy crop, trap crop and antagonistic or enemy crop

Decoy crops (cover crops) are non-host crops sown with the purpose of making soil-borne pathogens waste their infection potential before susceptible main crop grown. This is affected by activating dormant propagules of fungi, larva of nematodes, seeds of parasitic plants *etc.* in absence of the host. Sudan grass used as decoy crop for the management of Striga infection in sorghum.

Trap crops are highly susceptible host crops of the pathogen, sown to attract pathogens but destined to be harvested or destroyed before they complete their life cycle. Such crop is effective in checking the population of root knot and cyst nematodes. Oat is used as trap crop for cereal cyst nematode. Fodder sorghum can be raised as a trap crop to reduce downy mildew of sorghum.

Antagonistic or enemy crop produce some toxic compounds that directly destroy the pathogen present in soil. They are non-host of the pathogen. Marigold is an enemy crop for the control of root knot nematode.

Post planting operations

1. Management of top soil (Mulching)

Mulching or covering of top soil with organic residues often helps in reducing plant diseases. Mulches of non-host origin should be used in the field. These mulches are known to release inhibitory substances in the underlying soil and also promote development of

parasites and predators of nematodes. Mulches laid on the soil around the crop plant, have been found to be highly effective in controlling aphid vectors.

2. Management of irrigation water

The aim of irrigation is to wet the soil only to the extent that roots easily get water and mineral nutrients. If excess water is added to soil, it may directly affect activity of pathogens and/or it may affect disease incidence through the effect on the host.

Common scab attack on potato tubers is prevented by maintaining soil moisture near field capacity during tuber formation. Because bacterial flora antagonistic to *Streptomyces scabies* increases under high moisture conditions.

The root rot pathogen attack on cotton crop, when the soil temperature rises and there is water stress. By irrigating the field, soil temperature is brought down, stress is removed and the disease is suppressed. Supply of frequent but low quantity of irrigation water is recommended for reducing chances of damping off in nurseries.

3. Roguing

Roguing consists of completely removing or uprooting the diseased plants to prevent further spread of the disease. This method is widely adopted in the control of virus diseases which is spread by insects (mosaic of black gram, citrus tristeza, katte disease of cardamom, bunchy top of banana). The whip smut of sugarcane checked by roguing infected plant from field. In Jamaica, a country-wide campaign of destroying infected plants has succeeded in the control of panama wilt of banana.

4. Management of plant nutrients

The plant nutrients in general when applied in excess may increase or reduce the resistance in plants against diseases. Application of excess nitrogenous fertilizers increase vegetative growth of plant and make the tissue more succulent which become highly susceptible to the attack of diseases like rust, powdery mildew, blast and some bacterial diseases.

Late application of nitrogenous fertilizers increases wheat leaf blotch and powdery mildew. Some diseases are favoured by ammoniacal form of nitrogen while others are favoured by nitrate form of nitrogen. In rice, blast disease is favoured by ammonical nitrogen, while brown spot is favoured by nitrate nitrogen. In maize, northern corn leaf blight is favoured by ammonical nitrogen; while stalk rot is favoured by nitrate nitrogen.

6. Avoiding injury

Injury of plant parts should be avoided in order to check the entry of pathogens. Clipping of tips of tall rice seedlings provide way for entry of bacterial blight pathogen and hence incidence of the disease increase. Hence clipping should be avoided at the time of transplanting of rice. Injury to the fruits during harvesting from tree will provide way for

the entry of pathogen and causing fruit rot. It also reduces the storage life of fruits. Hence much care should be taken to avoid wounds during the harvest time.

7. Time of harvesting

Time of harvesting affects the cleanliness of the seeds. Delayed harvesting of grain crops in temperate climatic conditions enables the pathogen more time to contaminate the seeds. Stem end rot disease of mango incidence is increase when mango is harvested in cloudy and humid weather.

8. Ratooning

Ratooning is a general practice in sugarcane when the incidence of grassy shoot disease and red rot are very high. Hence ratooning should be avoided.

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II. PHYSICAL METHODS

Introduction

Physical methods for disease control act as alternative to fungicides for the management for seed and soil-borne pathogens. They usually do not leave residues or pollute the environment.

The Principle

The scientific principle involved in heat therapy is that the pathogen present in seed material is ***selectively inactivated*** or ***eliminated at temperatures*** that are non-lethal to the tissues.

The exact mechanism by which heat inactivates the pathogen is not fully understood. It is universally accepted that heat cause ***inactivation and immobilization*** of the pathogen. There are two thoughts regarding inactivation of pathogen (viruses) by heat.

1. It may be the chances that heat leads loosening of bonds both in nucleic acid and the protein components of the virus. In the nucleic acid, when the bonds are disrupted the linear arrangement of nucleotides is disrupted and thus the virus looses infectivity.
2. In proteins, the bonds holding the chains of amino acids may be destroyed. Disruption of bonds causes denaturing of protein molecules which become less soluble in water and finally leads to coagulation.

The rate at which the pathogen is inactivated is determinate by temperature and the duration of treatment.

Methods for managing Seed-borne Pathogens

- ❑ The physical agents *like as;* hot water or hot air or steam is used to eliminate the seed-borne infection.
- ❑ These methods are successfully used in controlling certain **internally seed-borne** diseases like loose smut of wheat and systemically infected diseases caused by bacterial, virus and phytoplasma.
- ❑ Following physical methods are employed for reduction and/or elimination of primary inoculums that may be present in seed or planting material:

1. **Hot Water Treatment:** Hot water treatment is widely used for the control of seed-borne pathogens, especially bacteria and viruses. A list some important seed-borne diseases, which can be controlled by hot water treatment is given below:

Crop	Disease	Treatment
Cluster bean	Blight	50° C for 10 min
Pearl millet	Downy mildew	55° C for 10 min
Sugarcane	Grassy shoot	50° C for 2 hr
Sugarcane	Mosaic	20 min treatment for 3 successive days at 52° C
Rice	Udbatta	54° C for 10 min
Rice	White tip	51-53° C for 15 min

2) **Hot Air Treatment:** Hot air treatment is less injurious to seed and easy to operate but also less effective than hot water treatment. It has been used against several diseases of sugarcane. For example, complete control of red rot in varieties Co 527, CoS 510, Bo 3 and Bo 32 is obtained by hot air treatment of 54°C for 8 hours. Hot air treatment at 54°C for 8 hours effectively eliminates ratoon stunting pathogen without impairing the germination of buds.

3) **Solar Heat Treatment:** The treatment has been developed in India to eliminate the pathogen of loose smut of wheat. Previously the hot water treatment was followed to eliminate loose smut. As the thermal death point of the fungus and the embryo are very close, extensive care should be taken to avoid killing of the embryo.

- ✓ ***Luthra in 1953***, devised a method to eliminate the deep seated infection of *Ustilago nuda*. The method is popularly known as ***solar heat or solar energy treatment.***
- ✓ **Luthra's solar energy treatment:** The seeds are soaked in cold water for 4 hours in the forenoon on a bright summer day followed by spreading and drying the seeds in hot sun for four hours in the afternoon in summer season.

4. Aerated Steam Therapy (AST)

- ✓ Sugarcane setts are also exposed to aerated steam at 50^0C for 3 hrs to eliminate mosaic virus.

5. Moist Hot Air Treatment (MHAT)

- ✓ This method is effectively used in sugarcane to eliminate grassy shoot disease. Initially the setts are exposed to hot air at 54^0C for 8 hrs, then exposed to aerated steam at 50^0C for 1 hr and finally to moist hot air at 54^0C for 2 hours.

Methods for managing soil borne pathogens

Soil harbours a large number of plant pathogens. Soil treatment is largely curative in nature as it mainly aims at killing the pathogens in soil and making the soil 'safe' for the growth of the plant. Some of the commonly followed physical methods for soil-borne pathogens are:

1) Soil Solarization

- ✓ ***Soil solarization*** *is an eco friendly method of using solar power for the control of soil borne pathogens by covering soil with a transparent polyethylene cover.*
- ✓ It is a **hydrothermal method** of soil disinfestations. Soil solarization was first time developed **in Israel** (Egley and Katan) for the management of plant pathogenic pests, diseases and weeds.
- ✓ The principal involve in this method is that if moistened soil covered under very thin (25 µm) transparent polyethylene film during summer to prevent dissipation of the solar heat, the temperature of soil rises to a level, sometimes 13^0 C higher than the temperature outside, reaching 50-55^0 C at 8 cm depth. This temperature is lethal to many pathogens.

Procedure for Soil Solarization

- ✓ The nursery bed/field is first to be levelled and pebbles present on the surface removed before solarization.
- ✓ Incorporate the required quantity of organic manure in the soil and irrigate the soil.
- ✓ Cover the beds with 100 gauge (25 µm) transparent polythene sheets.
- ✓ Seal the edges of the sheet with soil to keep it in position in order to maintain the temperature and moisture inside the polythene mulch.
- ✓ Adequate care is also to be taken to see that the sheet is in close contact with the surface of soil to prevent the formation of air pockets between the soil and polythene sheet.
- ✓ Keep the sheet in this way for 1-2 month. Protect it from stray animals and birds. After the period of solarization, remove the sheet and the bed/field is ready for sowing and transplanting

Advantages of soil solarization

- ✓ It is a non chemical method which is not hazardous to the environment, human and animals.
- ✓ It is less expensive than other methods. This technology can easily be transmitted to the ordinary farmers and can be applied in large areas manually and mechanically.
- ✓ It may have a long term effect, since effective disease control lasts for more than one season.
- ✓ It control soil borne pathogen *like as;* fungi, nematode etc. It also control certain weed. It also increase the plant growth response.

Limitations of soil solarization (Disadvantage)

- ✓ It can only be used in regions where the summer temperature are relative high and the soil is free of crops for about one month or more at a time of trapping with PE sheets.
- ✓ It is time consuming method requires longer time more than 1-2 month for entire process.

- ✓ It is expensive for some crops and also ineffective in the control of certain diseases. Heat tolerant pathogens might develop after repeated application.
- ✓ Sometime this method causes harmful effect on certain beneficial microorganism present in the soil.
- ✓ It is difficult to protect PE sheets from wind and animals.

Example

- ➢ Soil solarization with transparent polyethylene sheet mulch (25µm) for 40 days is effective for the control of collar and root rot of strawberry.
- ➢ Fungal diseases such as damping-off, root rots, stem rots, fruit rots, wilts and blights have been successfully managed by soil solarization.
- ➢ Nematode *Heterodera* spp. and *Meloidogyne* spp. have been successfully managed by soil solarization.
- ➢ Bacterial canker of tomato is successfully managed by Soil solarization for 1-2 months.

2) Steam Sterilization: Steam is passed through perforated pipes at a depth of 15 cm to sterilize the upper layers of soil. It is mostly practiced under glass house and green house conditions.

3) Hot Air Sterilization: Hot air is also passed through pipelines to sterilize the soil in the nursery areas.

4) Hot Water Treatment: It is mainly done in pot culture studies to kill the fungi and nematodes. The pots containing soil are immersed in boiling water at 98^0C for 5 minutes or drenching boiling water @ 20 litres/m^2.

5) Burning: Burning of plant wastes, crop residues, stubbles in the areas selected for raising nurseries for vegetable crops heats the soil and kills the inoculum of the pathogens present in the top layer of the soil. When nurseries are raised in these areas incidence of damping off disease is highly reduced. This practice is also followed in pits made for planting coconut, banana and other fruit trees. Burning of wheat plant every second or third year is suggested for eradication of pathogen from the field.

6) Flooding: Flooding fields and orchards to reduce or eliminate soil-borne inoculum of plant pathogens by creating anaerobic condition in soil is an ancient practice. Flooding has been recognized to be one of the key factors for the low incidence of soil-borne disease and nematodes.

Physical Methods for managing post-harvest losses

1) **Refrigeration:** Low temperature at or slightly above the freezing point checks the growth and activities of all such pathogens that cause a variety of post harvest diseases of vegetables and fruits. Therefore, most perishable fruits be transported and stored in refrigerated vehicles and stores.

2) **Radiation:** Electromagnetic radiations such as ultraviolet (UV) light, X-rays and Y-rays, as particulate radiations can be used to management of post-harvest diseases of horticultural crops. Y rays controlled post harvest fungal infections in peaches, straw berries and tomatoes but higher radiation doses required to kill pathogens which found injurious to host tissues.

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III. REGULATORY (LEGAL) METHODS

➢ **Legal control:**

A legal restriction on the movement of agricultural commodities for the purpose of exclusion, prevention or delaying the spread of the plant pests and diseases in uninfected areas" or holding of important plant in isolation for a specific period to ensure their freedom from disease/pest.

The **principle involve in this method** is **exclusion** which means to prevent entry of pathogen into new area where it is not prevailing.

Some examples of diseases which are introduced into India from foreign countries

Sr. No.	Disease	Country	Year
1	Coffee rust	Sri Lanka	1879
2	Late blight of potato	England	1883
3	Flag smut of wheat	Australia	1906
4	Downy mildew of grapes	Europe	1910
5	Downy mildew of maize	Java	1912
6	Black rot of crucifers	Java	1929
7	Foot rot of rice	South East Asia	1930
8	Black shank of tobacco	Holland	1938
9	Powdery mildew of rubber	Malaya	1938
10	Bunchy top of banana	Srilanka	1940
11	Wart of potato	Netherlands	1953
12	Golden nematode of potato	Europe	1961

❑ **Plant Quarantine**

✓ The term '***Quarantine***' has been derived from ***Latin word*** '***Quarantum***' means simply forty i.e., 40 days period.

✓ '***Plant Quarantine***' refers to the holding of plants in isolation until they are believed to be healthy. Now, broader meaning of the plant quarantine covers all aspects of the regulation of the movement of living plants, living plant parts/plant products between politically defined territories or ecologically distinct parts of them.

✓ ***It is a legal restriction on the movement of agricultural commodities for the purpose of exclusion, prevention or delaying the spread of the plant pests and diseases in uninfected areas.***

❑ History of Plant Quarantine

- ✓ The **first plant quarantine law** was promulgated in ***Rollen, France*** in 1660 to suppress and prevent barberry bush, the alternate host for wheat stem rust.
- ✓ In 1891-92, the first plant quarantine measure discovered was initiated in USA by setting up a sea port inspection station at San Pardo, California.
- ✓ In 1903, quarantine law was passed in Denmark.
- ✓ In 1909, Federal Plant Quarantine service was established in Australia.
- ✓ In 1912, first plant quarantine law was passed in USA.
- ✓ Among other countries, the first few to establish plant quarantine services were Germany, France, Australia and the U.S.A.
- ✓ In India, legislative measures against crop pests and diseases was initiated under the **Destructive Insects and pests Act of 1914 (DIP act)** and it was passed by Governor General of India on 3rd February, 1914.

❑ Set Up of Plant Quarantine in India

Government of India

↓

Ministry of Agriculture and Farmers Welfare

↓

Directorate of plant Protection, Quarantine & Storage

↓

Technical officer of
Directorate of plant Protection, Quarantine & Storage
(Enforcement of Plant Quarantine regulation)

↓

Plant Protection Advisor

Plant Quarantine station in India

Presently, there are **35 Plant quarantine Stations** at different **International Airports, Seaports and Land frontiers** implementing the Plant Quarantine regulations with its **headquarters at Faridabad**.

List of the some of the Plant Quarantine station in India

Seaports	Airports	Land Frontiers
1. **Bhavnagar - Gujarat** 2. Calcutta - West Bengal 3. Chennai - Tamil Nadu 4. Cochin - Kerala 5. Mumbai - Maharashtra 6. Nagapattinam - Tamil Nadu 7. Rameswaram - Tamil Nadu 8. Tuticorin - Tamil Nadu 9. Visakhapatnam – Andhra Pradesh 10. **Kandla-Gujarat** 11. Kakinada- Andhra Pradesh 12. Mangalore- Karnataka	1. Amritsar - Punjab 2. Calcutta - West Bengal 3. Chennai - Tamil Nadu 4. Hyderabad - Andhra Pradesh 5. Mumbai - Maharashtra 6. New Delhi - New Delhi 7. Patna - Bihar 8. Tiruchirappalli - Tamil Nadu 9. Trivandrum - Kerala 10. Varanasi - Uttar Pradesh 11. Bangalore- Karnataka	1. Amritsar Railway Station - Punjab 2. Attari Railway Station - Punjab 3. Attari-Wagah Border- Punjab 4. Bangaon Benapol Border - West Bengal 5. Gede Road Railway Station West Bengal 6. Kalimpong - West Bengal 7. Sukhia Pokhri – West Bengal

Research material is examined by three agencies

1. **National Bureau of Plant Genetic Resources (NBPGR)- New Delhi** for agricultural and horticultural crops.
2. **Forest Research Institute (FRI) - Dehra Dun** for forestry plants and
3. **Botanical Survey of India (BSI) - Calcutta** for other plants other plant of economic importance.

❑ **Quarantine Procedure**

1. **Inspection and Treatment:** It involves the initial examination after that if required; treatment is given to the commodities. Sometime fumigation with methyl bromide or phosphine is given.
2. **Inspection and certification:** It involves pre-shipment inspection by the importing country in cooperation with exporting country and certification in accordance with quarantine requirements of importing country.
3. **Inspection at the point of entry**: It involves inspection of plant material immediately upon arrival at the prescribed port of entry and if necessary treatment is given to the importing material.
4. **Utilization of post entry plant quarantine facilities**: It involves growing of introduced plant propagating material under isolated or confined conditions.

❑ Phytosanitary Certificate

Phytosanitary or health certificate is a certificate which should accompany a plant or plant material or seed which is to be moved from one place to another place. This certificate indicates or certifies that the material under transit is free from pests or diseases. It is an official certificate from the country of origin, which should accompany the consignment without which the material may be refused from entry.

❑ Plant quarantine methods

✓ There are three types of p**lant quarantine methods.**

1. **Domestic quarantine:** When there is restriction on the movement agriculture commodities from one state/region to another state/region with in India is called domestic quarantine. Domestic quarantine in India exists for two pests (*Rooted scale and San jose scale*) and three diseases.

✓ **Bunchy top of banana:** The export and the transport of banana plant parts *like as;* sucker, stem, leaf, flower and any other part from Assam, Kerala, Orissa, West Bengal, Tamil Nadu is completely prohibited.

✓ **Banana mosaic:** The export and transport of banana plant parts *like as;* sucker, stem, leaf, flower, and any other part from Maharashtra and Gujarat is prohibited.

✓ **Potato wart**: The export to potato tubers from West Bengal to any other State or territory of India is prohibited.

2. Foreign quarantine

When there is restriction on the import of any agriculture commodities from other foreign countries to India is called foreign quarantine. Foreign quarantine rules may be general or specific.

3. Total embargoes:

Total restriction on import and export of agricultural commodities.

Usefulness/ Advantage of Plant quarantine

✓ Restrict the movement of infected material and thereby spreading of plant diseases.
✓ Intercept plant shipment containing nematodes and other pest.
✓ The disease/contaminated plant material treated at the entry point to kill the pest.
✓ It also prevents the movement of contaminated soil.
✓ Useful in controlling vector.

Limitations/ Disadvantage of Plant quarantine

✓ Only useful when disease is discovered.
✓ Very expensive method
✓ These regulations can create many obstacles in trades.

- ✓ These regulations can create many obstacles in import of food grains when it urgently required.
- ✓ When food materials imported/exported cannot be treated by toxic materials.

❑ **The quarantine may fail or does not work when:**

- ✓ The pathogen is air borne.
- ✓ Smuggling of seeds from country to country.
- ✓ Some pathogens may get transported by clothes and aircraft.
- ✓ Sea water, birds also carry pathogens from one region to another.
- ✓ Free trade policy

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IV. BIOLOGICAL CONTROL

Biological control is defined *as the reduction of inoculum density or disease producing activities of a pathogen or parasite in its active or dormant stage by one or more organisms accomplished naturally or through manipulation of the environment, host or by introduction of one or more antagonists or by mass introduction of one or more antagonists*. (Baker and Cook, 1974)

Or

Biological control of plant disease is a condition or practice whereby survival or activity of a pathogen is reduced through the agency of any other living organism (except human beings), with the result that there is reduction in incidence of the disease caused by the pathogen (Garett, 1965).

The four main mechanisms involved in the biocontrol are...

- ✓ The biological agent (antagonist) may parasite the other harmful organism.
- ✓ Antagonist may secrete metabolites (antibiotics) harmful to the pathogens (Antibiosis).
- ✓ Antagonist may compete with the pathogens for nutrients or space (Competition).
- ✓ It may cause death of the parasite by producing enzymes (Lysis).

Mechanisms of Biological Control

1. Competition

Most of the biocontrol agents are fast growing and they compete with plant pathogens for space, organic nutrients and minerals. Most aerobic and facultative anaerobic micro-organisms respond to low iron stress by producing extracellular, low molecular weight (500-1000 daltons) iron transport agents, known as **Siderophores**, which selectively make complex with iron (Fe^{3+}) with very high affinity.

Siderophore producing strains are able to utilize Fe^{3+} - Siderophore complex and restrict the growth of deleterious micro-organisms mostly at the plant roots. Iron starvation prevents the germination of spores of fungal pathogens in rhizosphere as well as rhizoplane. Siderophores produced by *Pseudomonas fluorescens* (known as **pseudobactins** or **pyoveridins**) helps in the control of soft rot causing bacteria.

2. Antibiosis

Antagonism mediated by specific or non-specific metabolites of microbial origin, by lytic enzymes, volatile compounds or other toxic substances is known as antibiosis.

- ❑ **Antibiotics**: Antibiotics are generally *considered to be low molecular weight organic compounds produced by microbes which at low concentrations deleterious to the harmful micro-organisms.*

- ✓ *Gliocladium virens* produces **gliotoxin** that was responsible for the death of *Rhizoctonia solani* on potato tubers.
- ✓ Colonization of pea seeds by *Trichoderma viride* resulted in the accumulation of significant amount of the antibiotic **viridin** in the seeds, thus controlling *Pythium ultimum*.
- ✓ Some strains of *Pseudomonas fluorescens* produce a range of compounds, viz., **2,4-diacetyl phloroglucinol (DAPG), phenazines, pyocyanin,** which have broad spectrum activity against many plant pathogenic bacteria and fungi.

- ❑ **Bacteriocins**: These are antibiotic like compounds with bactericidal specificity closely related to the bacteriocin producer. Ex: The control of crown gall (caused by *Agrobacterium tumefaciens*) by the related *Agrobacterium radiobacter* strain K 84 is by the production of bacteriocin, **Agrocin K84**.

Antibiotic	Source	Disease
2, 4-diacetyl-phloroglucinol	*Pseudomonas fluorescens*	Damping off
Agrocin 84	*Agrobacterium radiobacter*	Crown gall
Bacillomycin D	*Bacillus subtilis*	Aflatoxin contamination
Bacillomycin, fengycin	*Bacillus amyloliquefaciens*	Wilt
Gliotoxin	*Gliocladium virens*	Root rots
Zwittermicin A	*Bacillus cereus*	Damping off

- ❑ **Volatile compounds**: Antibiosis mediated by volatile compounds has been observed in the management many soil borne pathogens. The volatile fraction responsible for inhibition was identified as ammonia.

3. Mycoparasitism or Hyperparasitism

Mycoparasitism or hyperparasitism occurs when the antagonist invades the pathogens by secreting enzymes *such as;* chitinases, celluloses, glucanases and other lytic enzymes.

Mycoparasitism is the phenomenon of one fungus being parasitic on another fungus. The parasiting fungus is called **hyperparasite** and the parasitized fungus as **hypoparasite**. The biocontrol agent parasitizes the pathogen by coiling around the hyphae. *Trichoderma harzianum* parasitize and lyse the mycelia of *Rhizoctonia* and *Sclerotium*.

4. Lytic enzymes production

Lysis is the complete or partial destruction of a cell by enzymes. In lysis, destruction of pathogen cell take place by the enzymes *like as;* chitinases, cellulases

which are produce by bioagent. *Trichoderma* sp. produces chitinases and β-1,3 glucanases which lyses the cell wall of *Rhizoctonia solani.*

5. Production of hydrogen cyanide

Many rhizobacteria produce hydrogen cyanide and this has been shown to play direct as well as indirect role in biological control of plant diseases and increasing the yields. *Pseudomonas fluorescens* produce HCN for suppression of pathogens.

6. Induction of host resistance

Plants respond to a variety of chemical produced by soil and plant associated microbes. Such chemical activate host defences mechanism of host plant and enhance resistance of plant against variety of pathogens.

7. Plant growth promotion

Biocontrol agents also produce growth hormones like, auxins, cytokinin, gibberellins etc. These hormones suppress the deleterious pathogens and promote the growth of plants and simultaneously increase the yield. *Pseudomonas* spp. increase plant growth by producing gibberellins like substances and also by mineralizing phosphates.

Advantage of Biological Control

1. Biological control is less costly and cheaper than any other methods.
2. Biocontrol agents give protection to the crop throughout the crop period.
3. They are highly effective against specific plant diseases.
4. They do not cause toxicity to the plants.
5. Application of biocontrol agents is safer to the environment and to the person who applies them.
6. They multiply easily in the soil and leave no residual problem in soil.
7. Biocontrol agents can eliminate pathogens from the site of infection and also enhance the root and plant growth.
9. Biocontrol agents are very easy to handle and apply to the target.
10. Biocontrol agents can be combined with biofertilizer.
11. They are easy to mass produce.

Disadvantage of Biological Control

1. Biocontrol agents can only be used against specific diseases.
2. They are less effective than the fungicides.
3. Biocontrol agents have slow effect in the control of plant diseases.
4. At present, only few biocontrol agents are available for use and are available only in few places.

5. This method is only a preventive measure and not a curative measure.
7. Biocontrol agents should be multiplied and supplied without contamination and this requires skilled persons.
8. The shelf life of biocontrol agents is short.
9. The efficiency of biocontrol agents is mainly decided by environmental conditions.

Some Examples of Biocontrol agents used in Plant Diseases Management

Fungal Biocontrol agent	Pathogen/Disease
1. *Ampelomyces quisqualis*	Powdery mildew fungi
2. *Darluca filum, Verticillium lecanii*	Rust fungi
3. *Pichia gulliermondii*	*Botrytis, Penicillium*
4. *Trichoderma* sp.	Many soil borne diseases
5. *Paecilomyces lilacinus*	Egg parasite of root knot nematodes

Bacterial Biocontrol agent	Pathogen/Disease
1. *Pasteuria penetrans*	*Juvenile parasite of root knot nematode*
2. *Pseudomonas fluorescens*	Many soil borne diseases
3. *Bacillus subtilis*	Many soil borne diseases
4. *Agrobacterium radiobacter* K-84	Crown gall of stone fruits caused by *Agrobacterium tumefaciens*

❑ **Some commercially available formulations of biocontrol agents**

- ✓ *Trichoderma viride* (**Ecofit**, **Bioderma** in India),
- ✓ *Gliocladium virens* (**GlioGard** in USA),
- ✓ *Trichoderma harzianum* (**F-Stop** in USA) **(Sawaj in JAU, Junagadh)**
- ✓ *Trichoderma polysporum* (**BINABT**)
- ✓ *Pseudomonas fluorescens* (**Dagger-G** in USA)
- ✓ *Bacillus subtilis* (**Kodiak** in USA)
- ✓ Agrobacterium radiobacter K-84 (**Gallex** or **Galltrol**)

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V. HOST PLANT RESISTANCE

The inherent ability of a crop plant to resist or withstand against the pathogen is called **resistance.** This disease resistance character of the plant transmissible from parent to off-spring by Mendelian law fashion was first time given by **Biffen (1905)** in his work on ***yellow rust of wheat.*** Since then, intensive work has been done on this aspect which has proved the value of applying genetical principles in developing disease resistant varieties of plants for effective control of diseases.

Resistant varieties can be the simplest, practical, effective and economical method of plant disease control. The use of resistant varieties cannot only ensure protection against diseases but also save the time, energy and money spent on other measures of control.

The resistance of a crop against pathogen depends not only on the genotype of the host but also depend upon genotype of the pathogen. **Flor (1942)** proposed the ***gene-for-gene hypothesis,*** according to which, ***for every gene for resistance in the host, there is a corresponding gene for pathogenicity in the pathogen.***

- **Disease resistance**

It is the ability of a plant to overcome completely or in some degree the effect of a pathogen or damaging factor.

- **Susceptibility**

The inability of a plant to resist the effect of a pathogen or other damaging factor.

- **Disease escape**

It is a preventive mechanism that causes the host to escape from pathogenic infection. It is ***not true genetic resistance***. It is due to the other characters of the plant which helps the plant to escape from the disease and these characters may be governed by genetically. e.g. Mechanical and anatomical barriers such as thick cuticle, waxy bloom on leaves and stem, stomatal regulation prevent penetration of spores.

- **Disease endurance**

A host or population of host plants is defined as tolerance if the signs and symptoms produce on it due to the disease are visually similar to those observed on a susceptible variety but damage on it is relatively lesser than observed in susceptible variety. It is a well-known phenomenon that plants fertilized with phosphatic and potash manures are more tolerant to disease.

- **True resistance**

It is the ability of the host plant to resist or withstand the attack of a pathogen. True resistance is inheritable and less influence by environmental. It is specific in character. This type of resistance may be morphological, functional, structural or protoplasmic. Functional nature of resistance is determined by opening of the stomata, time of opening of flowers and time of maturity, rate of cork formation and cambial activity.

Types of genetic resistance

- **Van der Plank** (1960) has discussed on the types of genetic resistance. He gave concept of vertical resistance and horizontal resistance.

1. **Vertical resistance:** When a variety is more resistant to some races of the pathogen than others, the resistance is called vertical resistance (race-specific resistance, qualitative resistance, discriminatory resistance). Vertical resistance is usually governed by single gene and is unstable.
2. **Horizontal resistance:** When the resistance is uniformly spread against all the races of a pathogen, then it is called horizontal/generalized/non-specific/field/qualitative resistance. Horizontal resistance is usually governed by several genes and is more stable.

Feature	Vertical resistance	Horizontal resistance
Pathogen specificity	Race specific	Race nonspecific
Nature of gene action	Monogenic, Oligogenic	Polygenic; rarely oligogenic
Resistance is governed by	Major gene	Minor gene
Response to pathogen	Usually, hypersensitive	Resistant response
Phenotypic expression	Qualititative	Quantitative
Stage of expression	Seedling to maturity	Expression increases as plant matures (Adult plant)
Selection and evaluation	Relatively easy	Relatively difficult
Risk of boom and Burst cycle	Present (rarely durable)	Absent (durable)
Need for other control measures	Likely (Required)	Much less likely (Less Required)
Host-pathogen interaction	Present	Absent
It is also called	Perpendicular resistance, physiological resistance, seedling resistance, race specific resistance, qualitative resistance, monogenic or oligogenic resistance	Non-specific, general, polygenic, minor gene, mature plant, adult, quantitative resistance, partial or field resistance

3. **Monogenic resistance:** When the defense mechanism is controlled by a single gene pair, it is called monogenic resistance.

4. **Oligogenic resistance:** When the defense mechanism is governed by a few gene pairs, it is called oligogenic resistance.

5. **Polygenic resistance:** When the defense mechanism is controlled by many genes or more groups of supplementary genes, it is called polygenic resistance.

Methods for development of disease resistance varieties

1. Introduction

It is a very simple and inexpensive method. Varieties resistant to a particular disease elsewhere may be thoroughly tested in the regions in which they are proposed to be introduced. It is possible that a variety resistant in one region need not be resistant in another region due to variation in the physiological race of the pathogen or due to a much different agroclimatic condition in the new location. Introductions have served as a useful method of disease control. For example; rice variety introduced in Karnataka from the Philippines has tolerance to blast, bacterial leaf blight and sheath blight.

2. Selection

This is better method than introduction and has more chances of success in obtaining disease-resistant plants. The work of selection is carried out either in the naturally infected fields under field conditions or under artificially inoculated conditions. The resistance in such individuals will occur in nature by mutation. Rice varieties *like as;* Sudha, Janaki, Ambika are developed by selections which is found resistant to one or more diseases.

3. Hybridization

When selection of resistant varieties is not feasible, resistant varieties may be evolved by crossing the susceptible popular variety with resistant wild variety where in the resistant gene or genes transferred into the genetic makeup of susceptible variety. Rust resistance gene transferred from *Triticum durum* to *Triticum aestivum* with hybridization method.

5. Induced mutagenesis

While following mutation breeding for disease resistance, a large number of mutation progeny should be produced and screened under artificial epiphytotic condition to select resistant plants. MCU10 cotton, a resistant variety to bacterial blight was evolved in Tamil Nadu by subjecting seeds of a susceptible variety CO4 to gamma rays followed by screening and selection.

5. Tissue culture technique

In this method; meristem culture techniques is utilized for produce virus free planting material. While protoplasts culture techniques are important tools for inter specific, intra specific and intra generic hybridization of various biocontrol agent for strain improvement which enhance the biocontrol potential for the management of pathogenic fungi.

6. Genetic engineering

Genetic Engineering is the technology by which a particular gene is isolated from one organism and inserted into the genome of another organism and made to express at the right time. E.g. Bt cotton.

Advantages of host plant resistance method

1. Resistant varieties can be the most simple, practical, effective and economical method of plant disease management.
2. They not only ensure protection against plant diseases but also save the time, energy and money spent on other measures of control.
3. Resistant varieties, if evolved can be the only practical method of control of diseases such as wilts, viral diseases, rusts etc.
4. They are non-toxic to human beings, animals and wild life and do not pollute the environment.
5. They are effective only against the target organisms whereas, chemical methods are not only effective against target organisms but also effective against non-target organisms.
6. The resistance gene, once introduced is inherited and therefore permanent at no extra cost.

Disadvantages of host plant resistance method

1. Development of resistant varieties is a slow and expensive process.
2. Resistance of the cultivar may be broken down with the evolution of the pathogen.

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Chapter: 8

Ecological Management of Crop Environment / Eco-friendly IDM

IDM/IPM which lays more importance on environmental safety. All methods except the use of chemical pesticides are encouraged. '***Organic farming***' is a new concept where no chemical pesticide or fertilizer is used in agriculture. Ecofriendly IDM/IPM may be followed in organic farming. Ecofriendly IDM/IPM uses methods like biological control, physical and cultural methods. Here more emphasis is given to environmentally sustainable diseases management.

Indigenous/Traditional technologies in IDM/IPM

1. Cultural methods/Practices for IDM

Farm level and community level various methods for plant disease management are utilized and these methods are originally practiced by farmers. The purpose of the culture methods is to make the environment less favourable for pathogen and/or favourable for its antagonist and thereby reducing diseases intensity or severity.

Since cultural control are based on habitat management and one of the component of agro-ecosystem in which the pathogen decrease and antagonist increased, this approach has also been called as "***Ecological management" or "Environmental control***". Cultural practices include.....

(a) Planting time

- ✓ **Delayed Sowing-** Late sowing of winter wheat and barley is considered to be the most effective measures in reducing take all disease of wheat.
- ✓ **Early Sowing –** The incidence of leaf spot of ground nut is reduced, if the crop sown early in June.

(b) Spacing

- ✓ **Closer spacing** – Incidence of downy mildew and bunch rot of grapes is higher in dense planting.
- ✓ **Wider spacing –** Virus diseases like groundnut rosette the incidence is observed to be less when wider spacing is adopted.
- ✓ **Closer spacing** – Lower incidence of cotton wilt (*Verticillium albo-atrum*) in closer planting.

(c) Tillage/Ploughing

- ✓ Deep ploughing in summer season affects the soil environment and destroy pathogens. Groundnut stem rot is controlled by deep ploughing in summer.

(d) Intercropping

- ✓ Inter cropping with moth bean in a cotton field reduced the root rot incidence. Intercropping of sorghum in pigeon pea field reduced the wilt incidence.

(e) Crop rotation

- ✓ Most of the diseases caused by soil borne pathogens can be significantly reduce by crop rotation. Crop rotation of banana crop with sugarcane or paddy is effective for control of panama wilt of banana incited by *Fusarium oxysporum* f. sp. *cubense*).

(f) Trap Cropping

- ✓ Decoy/Trap crops (hostile crops) are sown with the purpose of making soil borne pathogens waste their infection potential. Different trap crop *like as;* sunflower, safflower, lucerne, chickpea are grown for the control of flowering plant parasite *Orobanche* spp. incidence in tomato and tobacco.

(g) Seed rate

- ✓ Use of higher seed rate in the nursery creates favorable microclimate for the pathogens causing damping-off disease in vegetables crops.

(h) Nutrient management and soil amendment

- ✓ Application of high rate of nitrogen increase the incidence of rust powdery mildew, blast, tobacco mosaic and some bacterial diseases.
- ✓ Repeated application of phosphatic fertilizers delays the onset and lessens the severity of take-all disease of barley.
- ✓ Potassium application reduces the disease incidence in many crop diseases probably by increasing phenolics compound synthesis in plants.
- ✓ Application of potash induces resistance in groundnut against root rot disease.
- ✓ Application of 5 kg of neem cake/tree three times in a year reduces the basal stem rot (Ganoderma lucidum) of coconut.

(i) Irrigation

- ✓ Root rot of cotton increase, when the soil temperature rises and there is water stress. By irrigating the field, soil temperature is brought down, stress is removed and the disease is suppressed.
- ✓ Due to excess irrigation humidity in soil increase which favors damping off disease in many crop.

(j) Sanitation

- ✓ Destroying or removing of crop residues to eliminate pathogen from overwintering sites and thereby reduce the spreading of disease. Removal of malformed flowers from mango tree helps in reducing the spreading of mango malformation disease in orchard.

2. Physical Method

(a) Hot water treatment

- ✓ Hot water treatment is widely used for the control of seed-borne pathogens, especially fungi, bacteria and viruses.
- ✓ Hot water treatment to pearl millet (Bajara) seed at 55ºC for 10 minute successfully controls downy mildew disease.

(b) Hot air treatment

- ✓ Red rot disease in sugarcane controlled by hot air treatment to sugarcane setts at 54ºC for 8 hours.

(c) Solar heat treatment

- ✓ Many internally seed borne diseases easily controlled by sun heat treatment to the seed *like as;* loose smut of wheat.

(d) Soil solarization

- ✓ When the soil is covered with white polythene sheets during hot seasons, soil temperature increases. Increased soil temperature eliminates wilt pathogens from tomato field. High soil temperature also favours antagonistic fungi.

(e) Burning

- ✓ Burning of plant wastes, crop residues, stubbles in the areas selected for raising nurseries for vegetable crops, tobacco, chillies which heats the soil and kills the inoculum of the pathogens present in the top layer of the soil.
- ✓ Due to burning of rice crop residues, stem rot disease of rice is managed successfully.

(f) Flooding

- ✓ Root knot nematodes and many soil born diseases controlled by flooding.

3. Biological control

- ✓ It is a process in which disease producing activities of the pathogen or parasite reduce by another species of microorganism by mechanisms such as antibiosis, hyper-parasitism, competition.
- ✓ The fungal biocontrol agent *Trichoderma* sp. and bacterial biocontrol agent *Pseudomonas fluorescens* is utilized for the control the control of many soil borne pathogen.

4. Botanical pesticides

- ✓ Plant products play an important role in evolving an ecologically sound and environmentally acceptable disease management system. Plant products have been found to have fungicidal, bactericidal and antiviral properties.
- ✓ Application of 3% neem oil @ 500 lit/ha reduces rust disease in ground nut.
- ✓ The leaf extract of tulsi is found effective against paddy brown spot.

3. Farmers' own wisdom on disease control

- ✓ e.g. Spraying of chilli mash or garlic juice or buttermilk for the control of various disease and insect. Removing the virus infected plant from the field helps in controlling viral diseases.

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Chapter: 8
Introduction to Conventional Pesticides for Plant Diseases Management

- Plant products play an important role in evolving an ecologically sound and environmentally acceptable disease management system. Plant products have been found to have fungicidal, bactericidal and antiviral properties.
- It is well established that about 346 plant products have fungicidal properties, 92 have bactericidal and 90 have antiviral properties. This clearly indicates that the plant kingdom is a vast storehouse of chemicals that can check several plant pathogens.
- ***Vriksha Ayurveda*** compiled by **Surpala** describes the use the various plant based preparations to protect crop against various diseases.
- ***Vriksha Ayurveda*** reflects the ethos of organic way of managing crop pests, making us believe that sustainable agriculture was very much the way of life in ancient time.
- Use of several plants such as adhatoda, garlic, lantana, mahuva, neem, nirgundi, peepli, pongamia, tulsi etc. has been mentioned and their role in crop protection has been described in the ancient manuscripts.

Some Conventional Pesticides Used for Plant Diseases Management

No	Plant Use	Pathogen	Chemical Compound
1	*Azadirachta indica* (Neem) Leaf extract, Kernel extract, neem oil etc.	Many soil borne disease Foliar disease & Nematodes	Azadirachtin, Meliantriol, Nimbinin, Nimbidin, Salannin, Nimbin
2	*Pongamia pinnata* (Karanja)	Reniform Nematodes *Fusarium* sp.	Pongamol, Pongapin, Pongaglabrone, Pongallone
3	Lantana sp.	Soil & Foliar disease	Lantic acid, Lantanolic acid
4	*Garlic bulb extract*	*Alternaria* sp.	Allicin, Diallyl sulphide and Diallyl disulphide
5	Marigold	Root knot nematodes	Tagetone
6	Turmeric extract	Many Foliar disease	Curcumol, curcumin
7	Ginger extract	Many Foliar disease	Gingerols, zingiberene
8	Onion bulb extract	Many Foliar disease	Oleic acid, alpha and beta-tocopherols

Anti- Viral property of plant product

- ❑ Plants are also known to contain some compounds which are inhibitory to virus.
- ❑ The leaf extracts of sorghum, coconut, bougainvillea, *Prosopis juliflora and Cyanodon dactylon* are known to contain virus inhibiting principles.

❑ Advantages of Conventional Pesticides

1. Easily degradable through the action of air, water and sunlight and thereby possessing less residual toxicity problem.
2. Act quickly on pathogen and inhibit the spore germination in fungi.
3. Possess low to moderate toxicity to mammals.
4. In most cases non-toxic to plants (not phytotoxic).
5. Broad-spectrum.
6. May be used shortly before harvesting.
7. Farmers can make the local formulations on their own based on their requirement.
8. Resistance development in the target organisms is not as quick as in synthetic pesticides.
9. Mostly compatible with biocontrol agents.
10. Multiple modes of action like antifungal, antibacterial, and antiviral properties as well.
11. Well-fit for the sustainable farming.
12. Relatively safer to the user during handling, other non-target organisms in the environment.

❑ Disadvantage of Conventional Pesticides

1. Easily degradable and therefore required to be applied more often.
2. May not cause death of insects.
3. Sometime it may be poisonous to humans or the environment.
4. May not be readily available all throughout the season and everywhere.
5. Broad-spectrum and therefore sometimes toxic to beneficial organism as well.
6. Generally have poor water solubility.
7. Non-systemic in nature.

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Chapter No 10: Survey Surveillance and Forecasting of disease

Survey : It means to view the situation comprehensively and extensively at different periodicity
Surveillance: It is vigilant supervision of situation (to keep the close and constant watch).
Plant disease surveys are basic guide to disease progress that help in fixing the priorities. It may serve as forewarning for certain actions to be taken. It also tells about the impact of the changing agricultural technology, especially plant disease tools.
Survey procedure: The procedure adopted for disease survey will depend on the objectives and the types of information(primary and secondary) required. Following are some common ways used for disease surveys:
Random samplings: Area sampling stratified or purposive (only specific area or growers to be covered).
Trap nurseries: Generally used for detecting new virulence in area and screening of germplasm.
Mobile units: The mobile plant clinic vans go in different directions , on pre-plant routes, and collect samples at predefined distance to cover large area.
Spore trapping: Spore trapping devices or spore trap nurseries give information about arrival (time and quantity) of air borne inoculum.
Vector population: It is an indicator of pathogen activity and threat potential to the crop in the season. Aphid populations are monitored by the use of suction traps in Europe. This helps in developing distribution and movement maps of vector species.

Plant Disease Forecasting

Forecasting of plant diseases means predicting for the occurrence of plant disease in a specified area ahead of time, so that suitable control measures can be undertaken in advance to avoid losses. Disease forecasts are predictions of probable outbreaks or increase in intensity of disease. It involves well organized team work and expenditure of time, energy and money. It is used as an aid to the timely application of chemicals. Among the first spray warning services to be established for growers, were the grapevine downy mildew forecasting schemes in France, Germany and Italy in the 1920s.
Miller and O' Brien (1952) proposed a descriptive definition of disease forecasting that stands valid even today. They stated, forecasting involves all the activities in ascertaining and notifying the growers of community that conditions are sufficiently favourable for certain diseases, that application and control measures will result in economic gain or on the other hand, and just as important that the amount expected is unlikely to be enough to justify the expenditure of time, energy and money for control. The above statement made explicit distiunction between positive forecast and negative forecast and both have value for growers as well as society in general.
Positive forecast: Employs need based chemicals sprays, provides adequate protection to crop and reduce damage to environment.
Negative forecast: Avoids unnecessary chemical sprays, no risk to the crop health and no disruption of environment.

Informations needed for disease forecasting

Forecasting of diseases is a part of applied epidemiology. Hence, knowledge of epidemiology (development of disease under the influence of factors associated with the host,

pathogen and environment) is necessary for accurate forecasting. The factors of epidemic and its components should be known in advance before forecasting is done.

The informations required for forecasting are:

1. Host Factors

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

2. Pathogen factors

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

3. Environmental factors

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

warning or predictions.

Pre- requisites for developing a forecasting system

Disease forecasting is complicated, expensive and risky venture as several biotic and abiotic factors influence appearance and development of plant disease. The advancements in science and technology have made Disease warning system a working proposition, it requires lot of expenditure in terms of time, money and technical personnel. Following points should be considered before starting a project on disease forecasting:

1. The crop must be cash crop (economic value)
2. The disease must have potential to cause damage (yield losses)
3. The disease should not be a regular feature (uncertainity)
4. Effective and economic control known (options to growers)
5. Reliable means of communication with farmers.
6. Farmers should be adaptive and have purchase power.

Principles of plant disease forecasting

- ✓ The nature of pathogen (monocyclic or polycyclic)
- ✓ Effect of the environment on the stages of pathogen development
- ✓ The response of host to infection (age-related resistance)
- ✓ Activities of growers that effects the pathogen or the host

Models for disease prediction

1. Empirical models: Based on experience of growers , scientist or both.
2. Simulation models: Based on theoretical relationships. These models try to simulate or mimic life situations under study.
   Examples:
   EPIMAY: Southern leaf blight of maize
   EPIVEN: Apple Scab
   EPIDEM: Alternaria blight of potato and tomato
   EPIPRE: Cereal rusts and aphids

Methods of disease forecasting

Disease forecasting requires field observations on the pathogen characters, collection of weather data, variety of the crop and certain investigations and their correlations. Usually the following methods are employed in disease forecasting.

1. Forecasting based on primary inoculums

Presence of primary inoculum, its density and viability are determined in the air, soil orplanting material. Occurrence of viable spores or propagules in the air can be assessed by using different air trapping devices (spore traps). In the case of soil-borne diseases the primary inoculum in the soil can be determined by monoculture method. Presence of loose smut of wheat, ergot of pearlmillet and viral diseases of potato can be detected in the seed lots at random by different seed testing methods. Seed testing methods can be used to determine potential disease incidence and enable decision to be made on the need for chemical seed treatment. The extent of many virus diseases is dependent on the severity of the preceding winter which affects the size of vector population in the growing season. e.g.Sugarbeet yellows virus.

2. Forecasting based on weather conditions

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

3. Forecasting based on correlative information

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

4. Use of computer for disease forecasting

Forecasting of disease can also be made by the use of computers. This system gives the results quickly. One such computer based programmes in the USA is known as '**Blitecast**' for potato late blight.

Importance and uses of disease forecasts

Forewarning or assessment of disease important for crop disease management

1. **For timely plant protection measures:** Information whether the disease status is expected to be below or above the threshold level is enough, models based on qualitative data can be used.- qualitative models
2. **Loss assessment:** Forewarning actual intensity is required- quantitative model
3. **For making strategic decision:** Prediction of the risks involved in planting a certain crop. Deciding about the need to apply strategic control measures (soil treatment, planting a resistant cultivar, etc)
4. **For making tactical decision:** Deciding about the need to implement disease management measures

Devices used in forcasting:

GPS (Global Positioning System) is satellite system that gives information to GPS receivers on ground, enabling users to determine latitude and longitude coordinates.
GIS (Geographic Information System) is a computer system designed to capture, store, manipulate, analyze, manage and present all types of spatial or geographical data. It is an important tool that can be applied in predicting, monitoring and controlling diseases.

Chapter: 11
Implementation and Impact of IDM/IPM

Although IPM/IDM has been accepted in principles as the most attractive option for the protection of agriculture crops from different diseases, yet implementation at the farmer level has been rather limited. Some important constrains in IPM/IDM adoption or implementations have been listed by as follows:

Constrains in IPM/IDM Implementation

1. Institutional constraint

- ✓ IPM/IDM requires interdisciplinary approach to solve pest problem. Lack of coordination among different institution is a constraint.
- ✓ Research programme based on farmer's need is lacking.

2. Informational constraint

- ✓ Lack of information on IPM/IDM among farmers and extension worker.
- ✓ Lack of training on IPM/IDM.

3. Sociological constraint

- ✓ Some farmers feel it is risky to adopt IPM/IDM compared to use of pesticides alone.
- ✓ Our farmers are habituated to using more pesticides.

4. Economic constraint

- ✓ Lack of funds for training farmers and extension workers on the use of IPM/IDM.

5. Political constraint

- ✓ More interest associated with pesticide trade.
- ✓ Pesticide subsidy by Government.

Options/Strategies in IPM implementation

Acceleration of IPM implementation requires the following

1. Farmer's participation

- ✓ Farmers must be encouraged to participate in IPM/IDM and give their views.

2. Government support

- ✓ Government can remove subsidies on pesticides and allot more fund for IPM/IDM implementation.

3. Legislative measures

- ✓ Suitable legislation (law) may be passed for adopting IPM/IDM by all farmers (IPM/IDM will be successful only if adopted on community basis).

4. Improved institutional infrastructure

- ✓ National level institution for implementation of IPM/IDM is required. Data base on role of biotic and abiotic factors on pest population, crop yield are required.

5. Improved awareness

✓ Awareness should be created at all levels on IPM/IDM i.e. Policy makers, farmers, consumers and general public. NGOs (Non Governmental Organization) should be made aware of the advantages of IPM/IDM.

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Chapter: 12
IDM Modules for Different Crop Diseases

1. Integrated Management of Groundnut Diseases

- Deep ploughing in hot summer.
- Remove plant debris from the previous crop to avoid soil borne primary inoculum.
- Use well decomposed FYM.
- Apply castor cake or neem cake 500 kg/ha at the time of sowing for management of stem rot, collar rot and nematode.
- Crop rotation with sorghum, cotton, maize, chick pea or wheat reduce leaf spot and rust infection.
- Intercropping with castor, sorghum, pearl millet also effective for control of nematode and peanut bud necrosis virus vector (thrips).
- Timely/Early sowing may reduce bud necrosis disease incidence.
- Use healthy and damage free seed for sowing purpose.
- Give the seed treatment of thiram or mancozeb @ 3-4 gram/ per kg seed before sowing.
- Destroy volunteer (self sown) plants in early stages and alternative weed hosts.
- For reducing collar rot incidence, avoid deep planting.
- Plant population per unit area should be increased to reduce PBND disease.
- After harvesting of groundnut, seed should be dry in sunlight than store it in dry place.
- Apply bio-control agent *Trichoderma harzianum* along with well decomposed FYM or castor cake @ 2.5-4 kg in 300 kg can be applied in furrow just before sowing for the control of stem rot disease.
- For the control of chlorosis, solution of 100 g ferrous sulphate and 10 g. citric acid in 10 liter of water sprayed at regular interval.
- Spraying of carbendazim (5 gram in 10 liter water) or hexaconazole (5 ml in 10 liter water) at regular interval for the control of foliar diseases.
- Application of systemic insecticides to control the thrips vector of PBND.
- For the chemical control of root knot nematode; nematicides such as carbofuran (furadon 3 G) 33 kg/ha is used.

2. Integrated Management of Wheat Diseases

- Burning of crop debris.
- Deep ploughing in during hot summer.
- Use disease free seed for sowing.
- Hot water treatment at 20 ^{0}C for 5 hrs. then transferred into 49 ^{0}C for about 1 min. and again 52 ^{0}C for 11 min and immediately place in cold water for control of seed borne disease.
- For control of seed borne disease apply solar heat treatment to seed.

- Seed soaking in brine solution to separate out the galls from nematode infected seed lot.
- Seed treatment with vitavax @ 2.5 g./kg seeds or carboxin or carbendazim @ 2 g./kg of seeds.
- Follow strict quarantine measures. Destroy the alternate host for the control of rust.
- Growing of repellent crop marigold for the control of nematode.
- Judicious application of nitrogenous fertilizers.
- Adjusting sowing date, to escape the crop from disease.
- Intercropping with gram or lentil for control of soil pathogen.
- Crop rotation with legume, maize, mustard for control of soil borne diseases.
- Grow resistant variety *like as;* N.P.700 and N.P. 800, Lerma Rojo, Safed Lerma and Sonalika are highly resistant varieties to all the three rusts.
- Spraying of Mancozeb (0.2 %) 25-30 gram in 10 liter water or Hexaconazole 10 ml in 10 liter water for the control of foliar disease.
- Spray streptocycline @ 1g in 10 liters of water for control of yellow ear rot disease.

3. Integrated Management of Potato Diseases

- Field must have proper drainage and high level of fertility.
- Grow resistant varieties *such as;* Kufri Sindhuri, Kufri Badshah, Kufri Jyothi, Kufri Sherpa, Kufri Naveen etc.
- Use of clean and disease free tuber for seed purpose. Such seeds should be obtained from certified sources.
- Dip sprouted tubers in 0.2% Metalaxyl for 30 min for the control of late blight disease.
- High soil temperature and moisture should be avoided at the time of planting.
- Destruction of infected plant debris before harvesting.
- Green manuring with green gram or sun hemp in kharif.
- Crop rotation with legume crops.
- Avoid use of acidic fertilizers like CAN (calcium ammonium nitrate) and hold the soil pH at about 5.3 by addition of Sulphur for control of potato scab disease.
- Spray Mancozeb (27 gram/10 liter water) or chlorothanonil (27 gram/10 liter water) for the control of foliar diseases.
- Spray systemic insecticides regularly for the control of insect vector of various viral disease.
- Tubers must be harvested carefully to avoid wounding that would allow storage rot fungi and bacteria to gain entrance into the tuber. Wounded tubers, if any, must be sorted out and discarded.
- Healthy tubers should be stored at about 15^0 C for healing of wounds and then at about 10^0 C to prevent development of fungus rot in storage.

4. Integrated Management of Mango Diseases

- ✓ Only certified scion/stock from healthy tree should be used for propagation.
- ✓ Nutrient management of orchard trees, pruning, sanitation, proper aeration etc. practices are very important for disease reduction.
- ✓ For powdery mildew management spray wettable sulphur or dinocap or carbendazim or triazole group fungicide immediately after initiation. Avoid spray at full bloom.
- ✓ Once malformation initiated in orchard: Remove malformed panicles along with 15-20 cm healthy portion of branch and burn. This is to be followed by spraying of carbendazim 50 WP (5g/10 l).
- ✓ Spray Naphthalene acetic acid (NAA) (2g/10 l) before bud differentiation (October). Spray acaricides to reduce further transmission of malformation disease.
- ✓ Spray carbendazim (5g/10 l) or copper oxychloride 50 % WP (40 g/ 10 l) after pruning and also for the control of different fungal diseases.
- ✓ Fruit harvesting should be done in clean and sunny weather condition.
- ✓ It is desired that the harvesting of fruits is done carefully as any injury to fruit predisposes it to the attack of both the rots.
- ✓ Fruits should not be allowed to touch soil.
- ✓ Stem end rot disease starts from the stem end. It is desired that the fruits to be harvested along with 5 cm stalk, so the disease can be checked or delayed. If fruits are harvested without stalk, the opening should be closed with wax.
- ➢ Post harvest rots will not developed under temperature below 10° C.
- ➢ Practice of sanitation and cleanliness at all times in transit, storage, containers and market stalls will reduce incidences.

5. Integrated Management of Rice Diseases

- ➢ Summer ploughing of fields also expose soil borne pathogen in sun heat.
- ➢ Green manuring with sesbania or sunhemp.
- ➢ Select suitable resistant or moderately resistant variety like as; Rasi, Vikas, Krishna Hamsa, Tulasi, IR 64, Aditya, Swarnadhan, Himalaya 1 *etc.*
- ➢ Use disease free pure seed.
- ➢ Removal and destruction (burn) of diseased infested plant.
- ➢ Timely planting/sowing.
- ➢ Raising of healthy nursery.
- ➢ Balanced use of fertilizers and micro-nutrients as recommendations. Reduce nitrogen application and apply if needed only small dose of N in more split doses.
- ➢ Seed treatment with carbendazim @ 2 g/kg seed or *Trichoderma/Pseudomonas* @ 5-10 g/kg of seed for seed or soil borne diseases and carbosulfan 2 g/kg of seed for root nematodes.

- Spray carbendazim or tricyclozole or ediphenphos or isoprothiolan for the control of blast and sheath blight diseases.
- Spray streptocycline 100 to 150 ppm solution for the control of bacterial blight disease.

6. Integrated Management of Cotton Diseases

- Field sanitation.
- Soil solarization and use only well decomposed manure.
- Deep ploughing during summer.
- Crop rotation with millet, sorghum, ground nut.
- Mixed cropping with mothbean, sorghum, urdbean are useful for reducing the incidence of wilt in cotton.
- Use delinted healthy seeds.
- ✓ Use resistant variety *like as;* Jayadhar, Jarila, Vijay, Pratap, Verun, BDS, Sujatha, CRH 71, HC-9, Reba-B-50, Badnawar-1 etc. In Gujarat Digvijay, Gujarat cotton-17, Gujarat cotton- 23 and G.Cot.-13 are resistant to wilt of cotton.
- Amendment with Zinc with balanced use of NPK reduce soil borne disease.
- Soil application of *Trichoderma* spp. before sowing along with organic manures reduce soil pathogen.
- ✓ Spraying streptocycline (100 ppm) 1 gram + copper oxychloride (blue copper) 20 g. in 10 liter water for the control of angular leaf spot of cotton.
- ✓ Seed treated with *Pseudomonas fluorescens* or spraying of *Pseudomonas fluorescens* powder (20 gram in 10 liter water) for the control of angular leaf spot disease.
- ✓ Grey mildew and fungal leaf spot diseases effectively controlled by spraying with carbendazim (10 gram in 10 liter of water) or copper oxychloride (40 gram in 10 liter of water).

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Chapter: 13

SAFETY ISSUES IN USE OF PESTICIDES

Pesticides are toxic to both pests and humans. Most pesticides will cause adverse effects if intentionally or accidentally ingested or if they are in contact with the skin for a long time. Pesticide particles may be inhaled with the air while they are being sprayed. An additional risk is the contamination of drinking-water, food or soil.

Special precautions must be taken during transport, storage and handling. Spray equipment should be regularly cleaned and maintained to prevent leaks. People who work with pesticides should receive proper training in their safe use.

Safe issues and Handling of Pesticides

- Always read the label and get advice before using pesticides.
- Do not buy damage packs.
- Do not buy packs with broken seals.
- Keep pesticides away from passengers and foodstuffs.
- Use suitable equipments for proper measuring and mixing.
- Never scoop or stir pesticides with bare hands.
- Use the recommended dose only and wear recommended protective clothing.
- Keep children and animals always away.
- Wear clothing to cover as much of the body as possible.
- Do not spray or dust against the wind.
- Do not apply pesticides when it is likely to rain.
- Do not use leaking or defective equipment.
- Do not allow pregnant woman and children for the spray.
- Clean sprayers with water or a soft probe.
- Do not blowout clogged nozzles with the mouth.
- Clean and check equipment at the end of each day's work.
- Wash hands and face before eating, drinking or smoking.
- Wash contamination skin and clothing.
- Wash all clothing and equipment after each day's use.
- Remove contamination clothing immediately and wash skin.
- Wash pesticides splashes from the eyes with clean water for 10 minutes.
- Start artificial respiration immediately, if breathing stops.
- Obtain medical attention as quickly as possible and show the label.

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Chapter: 14

CASE HISTORIES OF IMPORTANT IPM PROGRAMMES

Huffaker Project

It was the first major IPM project in the USA, spanned 1972-1978 and covered six crops viz., alfalfa Citrus, cotton, pine pome and stone fruits and soyabean. The crop was selected on the basis of 3 criteria *i.e.* 1) Level of insecticide use (very high in cotton and citrus and very low in soyabean) 2) potential for successful biological control (alfalfa, citrus, pome and stone fruits) 3) Representation of non agricultural system (pines). The outcome of the program was 40 – 45 percent reduction in the use of environmentally polluting insecticide with in 5 year period and 70 - 80% percent in 10 years. Advances efforts were made in many aspects for implementing IPM strategies for all crops.

Consortium for IPM

A group of scientist under the leadership of P.L. Adkisson secured funding for the **second load** scale large scale IPM Project in the US, called Consortium for Integrated Pest Management 1979-1985. The project focused on for major crops viz., alfalfa, Apple, cotton and soyabean. It was claimed that the average adoption of IPM for 4 crops was 66 per cent over 5.76 million ha. The main indicators of adoption were the use of Economic threshold and economic injuries levels for spray decision, use of selective pesticide or application of low dosage of broad spectrum insecticides. A significant achievement of the program was the genuine attempt to integrate weed sciences and plant pathology and the emphasis on economic assessment of IPM adoption.

National IPM initiative

The US Department of Agriculture USDA launched national IPM initiative in 1993 the goal of which was implement IPM practices on 75 percent of the nation's crop area by 2000. It has been estimated that some level of IPM has been implemented on about 70% of the US crop acerage. The highest percentage of cropland under IPM was in case of cotton and vegetables followed by soyabean other crops and pastures, fruits and nuts.

IPM in cotton

In India, the Indian Council of Agricultural Research, sponsored a village level IPM project to test and demonstrate the efficacy, practicability and economics of IPM in cotton in the Punjab in 1957. Initially, two or three villages were adopted for a period of 3 years after which the areas was shifted to new villages in other development blocks. In this way, a total of 15 villages in the main cotton growing districts of the state were covered during a period of 15 years. Farmers where educated regarding different insect pest problems, side effects of indiscriminate use of

insecticides, cultural and mechanical practices for minimizing the incidence of insect pest and cultivation of short duration varieties. The project resulted in substantial gains. Within a period of 4 years, long duration varieties like LSS where replaced by short duration jassid -tolerant varieties. IPM Technology resulted in 73. 7 and 12. 4% reduction in number of insecticides sprays for the control of sucking pest and bollworms, respectively. In addition to farmers obtain 23. 2 percent higher yield and 31. 7% higher net income.

A landmark in cotton IPM has been the validation of the cropping system based Holistic community approach of IPM at village Ashta (1998-2001) in Nanded dist. Of Maharashtra. the baseline information indicated less than 1 quintal average seed cotton yield per hectare in the *Helicoverpa armigera* epidemic year of 1997 when the farmers had spray more than 12-13 chemical pesticides spray rounds. All the farmers of the village were involved and the IPM approach covered 180 hectare cotton area. The offseason practices included management of *H.armigera* on pigeon pea and chickpea through use of neem seed kernel extract (NSKE) and HaNPV, field sanitation and deep plouging. In the pre sowing practices, resistant cultivars and treating the seed with Imidacloprid. By adopting IPM programme farmer of this area got higher seed cotton yield and income.

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PRINCIPLES OF PLANT DISEASE MANAGEMENT

Information on etiology, symptoms, pathogenesis and epidemiology of plant diseases are intellectually interesting and scientifically justified but most important of all they are useful as they help in formulation of methods developed for successful management of disease and thereby increasing the quantity and improving the quality of plant and plant products. Practices of disease management vary considerably from one disease to another depending upon the type of pathogen, the host and the biotic and abiotic factors involved. Contrary to management of human and animal diseases where every individual is attended, the plants are generally treated as populations and measures used as preventive rather than curative. Methods for plant diseases control were first classified by Whetzel (1929) into exclusion, eradication, protection and immunization. Further advances in plant pathology leading to development of newer methods. Two more principles - avoidance and therapy were created (NAS, 1968).

Disease Control vs. Disease Management

In past, plant pathologists always aimed at the impossible task of eradicating pathogen to control diseases. However, diseases really controlled are very few. No plant pathogen has ever wiped out from the face of earth. So long as the pathogen survives and its host is cultivated, the chances of disease incidence will persist. It is different matter that due to some direct or indirect efforts on the part of man the disease causing agent has been reduced to an innocuous level.The efforts better called management practices whereby the populations of the pathogen or its disease causing abilities have been kept under check so that it is unable to cause noticeable damage to the crop.

Although disease control is an established and widely understood term, there is convincing rationale supporting the substitution of management for control. The word control evokes the notion of finality, the disposal of problem (Apple, 1977). Management conveys the concept of a continuous process rather than an event accomplished through application of an intrinsic factor. Recommendation for disease control are the followed almost every year, suggests that these control methods are also a part of continuous process, to manage the disease under certain limits. The plant pathogens strategically are inherent components of ecosystem, which, must be dealt with on a continuous and strategic basis. Management should be on the principle of maintaining the damage or loss below and economic injury level or at least minimizing occurrence above that level.

Essential considerations in plant disease Management:

1. Benefit-cost ratio
2. Procedures for disease control should fit into general schedule of operations of crop production
3. Control measures should be adopted on a **co-operative basis** over large adjoining areas which reduces frequency of applications, cost of control and increases chances of success of control measures
4. Knowledge aspects of disease development is essential for effective economical control. General information is needed on the following aspects

a. Cause of a disease b. Mode of survival and dissemination of the pathogen c. Host parasite relationship d. Effect of environment on pathogenesis in the plant or spread in plant population

5. Prevention of disease depends on management of primary inoculum

6. Integration of different approaches of disease management is always recommended

General principles of plant disease management

1. **Avoidance**: 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 (Immunization)**: Preventing infection or reducing effect of infection by managing the host through improvement of resistance in it by genetic manipulation or by chemical therapy.
6. **Therapy** : It is the treatment of infected host plant, which is attempted in case of economically important horticulture plants. As a principle of plant disease control, it provides an opportunity to cure or rejuvenate the diseased host plant by use of physical or chemical agents.

The first five of these principles are mainly preventive (prophylactic) and constitute the major components of plant disease management. They are applied to the population of plants before infection takes place. Therapy is a curative procedure and is applied to individuals after infection has taken place.

Under the concept of disease management these principles have been classified into following five categories (Horsfall and Cowling, 1977):

1. Management of physical environment (cultural control)
2. Management of associated micro biota (biological antagonism)
3. Management of host genes (host resistance)
4. Management with chemicals (Chemical control)
5. Management with therapy (Physical, chemical etc)

The six principles that characterize the modern concept of plant disease management should be viewed from three stand points:

(a) Reduction in the initial inoculums or the rate of disease development.
(b) Management of the pathogen population, the cure or induce defense of the suscept or modify the environment as it influences disease and
(c) Interruption of dispersal, survival or the course of disease development.

These interactions are originally proposed by Baker (1968) and Roberts and Boothroyd (1972) and subsequently modified for the readers are illustrated as below :

Principles of plant disease management

I. Avoidance of the pathogen: These methods aim at avoiding the contact between the pathogen and susceptible stage of the crop. This is achieved by a. Proper selection of geographical area

a. Proper selection of geographical area
b. Proper selection of the field
c. Adjusting time of sowing
d. Disease escaping varieties
e. Proper selection of seed and planting material

a) Proper selection of geographical area: Many fungal and bacterial diseases are more severe in wet areas than in dry areas. Cultivation of bajra in wet areas is not profitable due to the diseases, smut (*Tolyposporium penicillariae*) and ergot (*Claviceps microcephala*).

b) Proper selection of the field: Proper selection of field will help in the management of many diseases, especially the soil borne diseases. Raising of a particular crop year after year in the same field makes the soil sick, where disease incidence and severity may be more.

Ex: Wilt of redgram, late blight of potato (*Phytophthora infestans*), green ear of bajra (*Sclerospora graminicola*), etc.

c) Time of sowing: Generally pathogens are able to infect the susceptible plants under certain environmental conditions. Alteration of date of sowing can help in avoidance of favourable conditions for pathogen.

Ex: *Rhizoctonia* root rot of redgram is more severe in the crop sown immediately after the rains. Delayed sowing will help in reducing the incidence of disease.

Ex: Infection of black stem rust of wheat (*Puccinia graminis tritici*) is more in late sowing, hence, early sowing helps in reduction of stem rust incidence.

d) Disease escaping varieties: Certain varieties of crops escape the disease damage because of their growth characteristics. Ex: Early maturing varieties of wheat or pea escape the damage due to *Puccinia graminis tritici* and *Erysiphe polygoni*, respectively.

e) Proper selection of seed and planting material: Selection of seed and seedling material from healthy sources will effectively manage the diseases such as loose smut of wheat (*Ustilago nuda tritici*), bunchy top of banana (*Banana virus-1*), Panama wilt of banana (*Fusarium oxysporum f.sp. cubense*) and whip smut of sugarcane (*Ustilago scitaminae*). Potato seed certification or tuber indexing is followed for obtaining virus free seed tubers. Citrus bud wood certification programme will help in obtaining virus free planting material.

f) Manipulation of environment: The biotic environment of the phyllosphere also influences the pathogens of aerial parts. The environments may, therefore, require modifications to combat a disease. The measures under modification of environments are basically cultural practices aimed at changing the crop and soil environments in such a way that growth of the plant is favoured while there is an adverse effect on the pathogen and disease cycle.

II. Exclusion of the pathogen: These measures aim at preventing the inoculum from entering or establishing in the field or area where it does not exist. Different methods of exclusion are seed treatment, seed inspection & certification, and plant quarantine regulation.

a) Seed inspection and certification: Crops grown for seed purpose are inspected periodically for the presence of diseases that are disseminated by seed. Necessary precautions are to be taken to remove the diseased plants in early stages, and then the crop is certified as disease free. This practice will help in the prevention of inter and intra regional spread of seed borne diseases.

b) Plant quarantine regulation: Plant quarantine is defined as " a legal restriction on the movement of agricultural commodities for the purpose of exclusion, prevention or delaying the spread of the plant pests and diseases in uninfected areas".

Plant quarantine laws were first enacted in **France** (1660), followed by Denmark (1903) and USA (1912). These rules were aimed at the rapid destruction or eradication of barbery bush which is an alternate host of *Puccinia graminis tritici*.

III. Eradication: These methods aim at breaking the infection chain by removing the foci of infection and starvation of the pathogen (i.e., elimination of the pathogen from the area by destruction of sources of primary and secondary inoculum). It is achieved by

a) Rouging: Removal of diseased plants or their affected organs from field, which prevent the dissemination of plant pathogens.

Ex: Loose smut of wheat and barley, whip smut of sugarcane, red rot of sugarcane, ergot of bajra, yellow vein mosaic of bhendi, katte disease of cardamom, etc. During 1927-1935, to eradicate citus canker bacterium in USA, 3 million trees were cut down and burnt.

b) Eradication of alternate and collateral hosts: Eradication of alternate hosts will help in management of many plant diseases.

Eg: Barbery eradication programme in France and USA reduced the severity of black stem rust of wheat

Eg: Eradication of *Thalictrum* species in USA to manage leaf rust of wheat caused by Puccinia recondita.

Eradication of collateral hosts, such as *Panicum repens*, *Digitaria marginata* will help in the management of rice blast disease (*Pyricularia oryzae*)

c) Crop rotation: Continuous cultivation of the same crop in the same field helps in the perpetuation of the pathogen in the soil. Soils which are saturated by the pathogen are often referred as **sick soils**. To reduce the incidence and severity of many soil borne diseases, crop rotation is adopted. Crop rotation is applicable to only root inhabitants and facultative saprophytes, and may not work with soil inhabitants. Ex: Panama wilt of banana (long crop rotation), wheat soil borne mosaic (6 yrs) and club root of cabbage (6-10 yrs), etc.

d) Crop sanitation: Collection and destruction of plant debris from soil will help in the management of soil borne facultative saprophytes as most of these survive in plant debris. Collection and destruction of plant debris is an important method to reduce the primary inoculum.

e) Manures and fertilizers: The deficiency or excess of a nutrient may predispose a plant to some diseases. Excessive nitrogen application aggravates diseases like stem rot, bacterial leaf blight and blast of rice. Nitrate form of nitrogen increases many diseases, whereas, phosphorous and potash application increases the resistance of the host. Addition of farm yard manure or organic manures such as green manure, 60-100 t/ha, helps to manage the diseases like cotton wilt, Ganoderma root rot of citrus, coconut, etc.

f) Mixed cropping: Root rot of cotton (*Phymatotrichum omnivorum*) is reduced when cotton is grown along with sorghum. Intercropping sorghum in cluster bean reduces the incidence of root rot and wilt (*Rhizoctonia solani*)

g) Summer ploughing: Ploughing the soil during summer months expose soil to hot weather which will eradicate heat sensitive soil borne pathogens.

h) Soil amendments: Application of organic amendments like saw dust, straw, oil cake, etc., will effectively manage the diseases caused by *Pythium, Phytophthora, Verticillium, Macrophomina, Phymatotrichum* and *Aphanomyces*. Beneficial micro-organisms increases in soil and helps in suppression of pathogenic microbes.

Eg: Application of lime (2500 Kg/ha) reduces the club root of cabbage by increasing soil pH to 8.5

Ex: Application of Sulphur (900 Kg/ha) to soil brings the soil pH to 5.2 and reduces the incidence of common scab of potato (*Streptomyces scabies*).

i) Changing time of sowing: Pathogens are able to infect susceptible plants under certain environmental conditions. Alternation in date of sowing can help avoidance of favourable conditions for the pathogens.

Eg: Rice blast can be managed by changing planting season from June to September/October.

j) Seed rate and plant density: Close spacing raises atmospheric humidity and favours sporulation by many pathogenic fungi. A spacing of 8'X8' instead of 7'X7' reduces sigatoka disease of banana due to better ventilation and reduced humidity. High density planting in chillies leads to high incidence of damping off in nurseries.

k) Irrigation and drainage: The amount, frequency and method of irrigation may affect the dissemination of certain plant pathogens. Many pathogens, including, *Pseudomonas solanacearum, X. campestris pv. oryzae* and *Colletotrichum falcatum* are readily disseminated through irrigation water. High soil moisture favours root knot and other nematodes and the root rots caused by species of *Sclerotium, Rhizoctonia, Pythium, Phytophthora, Phymatotrichum*, etc.

IV. Protection : Reduce the level of initial infection by means of a toxicant or other barrier to infection by protecting the infection court against the inoculums of many fast spreading pathogens by several ways:

a) Chemical treatments: The aim of most chemical sprays,dusts and seed treatments is to form a protective toxiv layer on the host surface so that when the pathogen comes in contact with the surface it is killed or prevented from growth. The chemicals used for this type of protection are called protective chemicals.

b) Control of insect Vectors: Many species of insects are important vectors of viral and other diseases.Timely and effective destruction of these vectors is the most important approach in the management of such diseases.

c) Modification of Environment: Improvement of aeration under crop canopy reduces humidity on leaves and other aerial parts and checks the growth of fungi which flourish in humid atmosphere. Eg: Root diseases favoured by high temperature are often managed by irrigation. Post harvest decay of fruits and vegetables is reduced or prevented by cold storage which provides a modified environment unfavourable for decay causing organisms.

d) Modifications of Host nutrition: Host nutrition often influences development of a disease in the plant. It generally acts through strengthening of the tissues. Many leaf diseases are favoured by high level of nitrogen in soil. Eg: In case of rice application of 100kg N/ha instead of 120 kg is recommended to prevent losses from leaf spots, blast and sheath blight. Deficiency of potash in plants renders the tissue susceptible to water soaking and susceptible to many diseases. High calcium increases resistance to wilt and soft rot diseases through strengthening of pectic substance in the cell walls and pectic enzymes having destructive activity against the pathogen.

V. Resistance

a) Selection of hybridization: Selection of resistant individual with poor commercial qualities and hybridizing them with susceptible plants of high commercial qualities is the aim of developing resistance through hybridization.

b) Genetic manipulation through biotechnology: Manipulation , genetic modification and multiplication of plants through such techniques as tissue culture and genetic engineering is now used in many crops.

c) Induction of Acquired resistance: Plants acquire localized and systemic resistance during its life time through effect of chemicals or microorganisms. Use of phosphates and carbonates as foliar therapy have been found turn on the resistance genes. The rhizobacteria also known to induce systemic acquired resistance in the foliar parts against many diseases simultaneously.

d) Resistance through chemotherapy: Temporary physiological resistance in plants can be developed through chemotherapy by using systemic fungicides and antibiotics .

VI. Therapy: Applied to diseased plant

a) **Chemotherapy:** The principal underlying is that the chemical used against the target pathogen is absorbed by leaves and roots and on reaching the cell protoplasm, act as a chemical defence mechanism by destroying the pathogen or suppressing its growth and sporulation and persisting for some time in the plant to provide temporary resistance against invasion. These chemicals can also act by detoxifying the toxins produced by the pathogen. In these manner, the tissue not invaded by the pathogen is saved and the plant is cured. Chemicals used for chemotherapy are systemic fungicides and antibiotics. The systemic fungicides when absorbed by roots are translocated through the xylem to upper parts of the plant.

a) **Heat therapy:** Plants which can tolerate the thermal inactivation or death point of pathogens can be treated by heat to destroy the pathogen. Eg. Ratoon stunting and many virus diseases of sugarcane are also eradicated by hot water or hot air treatment of seed setts

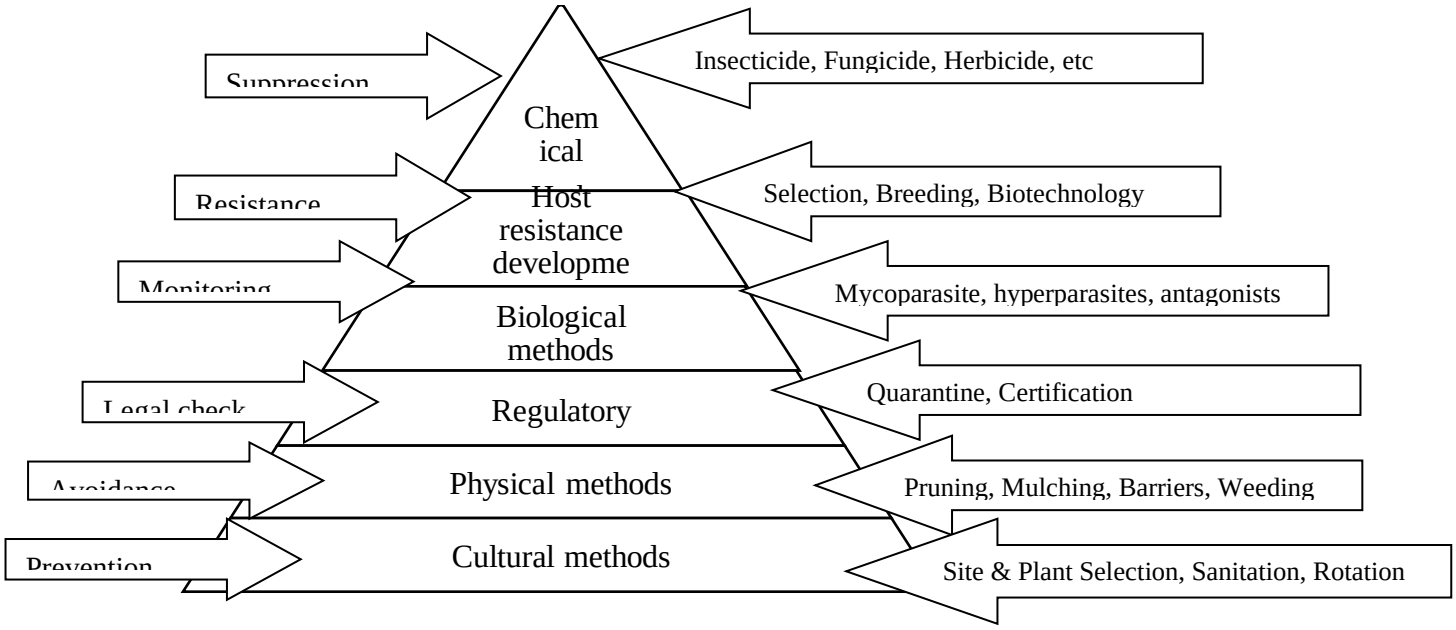
b) **Tree surgery:** Large sized fruit trees are cleaned of infection by pruning of incfeted branches or scrapping of the diseased portion, disinfection of the wound with some chemical and then applying a fungicidal paste to allow self healing of the wound by the tree. Eg. Stem diseases of apple.

Methods of plant diseases control

The main objective of plant pathology is the economic control of plant disease. However, the term control evokes the notion of finality or completeness, which is not true in nature. Whereas the word "Management" conveys the concept of a continuous process and management is based on the principle of maintaining the damage or less below an "economic injury level".

Methods of plant diseases control involves following practices:

1. Cultural methods
2. Physical methods
3. Regulatory methods
4. Biological methods
5. Host resistance development
6. Chemical methods



I. CULTURAL CONTROL

The term cultural control describes the activities of humans aimed at controlling disease through the cultural manipulation of plants.The success of cultural control practices ultimately depends on understanding the biology of the pathogen and the response of the host to infection. Such knowledge facilitates management decisions which 'attack' parasites at vulnerable stages of their life cycles. It may be difficult to assess their effectiveness because the farming operations are often performed well in advance of expected pathogen attacks.

Cultural practices involves all the activities carried out on the farm before, during and after planting of crops. They are grouped into: pre-planting, planting and postplanting operation. So pre planting operations can include choice of site, then clearing of land, Stumping, Ploughing, Harrowing and Ridging. So, these are some of the practices that are normally done before planting of the crop. Similarly, planting operations include Planting, Transplanting and Nursery practices. So these are operations that are done during the plantation of the crop and the post planting operations include thinning, supplying, mulching, manuring, watering, weeding, Pest and disease

management, harvesting, processing and storage. So, the post planting operation is also significant in reducing the disease inoculum.

Pre planting operations:

1.Crop rotation

Crop rotation is essentially a preventive measure and has its effect mainly on the succeeding crop. Crop rotation is the oldest and cheapest method adopted in agriculture for eradication of certain types of pathogens from infested soil. Continuous cropping or monoculturing provides opportunity for perpetuation of pathogenic organisms in the soil when the same crop is raised year after year in the same field. Crop rotation with sugarcane or paddy is effective in the control of 'Panama wilt' of banana (*Fusarium oxysporum* f.sp. *cubense*) and crop rotation with paddy or green manures is effective in the control of red rot of sugarcane (*Colletotrichum falcatum*). Rotation of cereal crops like pearlmillet, finger millet or fox-tail millet is recommended for the control of *Macrophomina* root rot of pulse crops.

2. Fallowing

Fallowing starves the pathogen and helps in reduction of the inoculum by elimination of the host. Diseases like *Macrophomina* root rot on different crop plants is controlled by following this method. Flood fallowing is to a depth of 0.6 to 1.5 m for 4 to 6 months markedly reduced the Panama wilt pathogen *Fusarium oxysporum* f.sp.*cubense* inoculum in banana. Flooding the soil strewn with debris infected by *Xanthomonas axonopodis* pv. m*alvacearum* for 4 days reduced the inoculum level and thus the incidence of disease was only 2.1% as against 69.5% in unflooded fields. Wet fallowing reduces saprophytic survival of *Alternaria solani* on crop debris.

3. Application of organic manures

Addition of organic manures like farm yard manure or green manures or oil cakes to the soil increases the antagonistic microorganisms in the soil. Build up of antagonistic microorganisms reduces the population of soil borne plant pathogens and the diseases caused by them. Application of farm yard manure at the rate of 12.5 tonnes/ha reduced the incidence of *Macrophomina* root rot of cotton. In the control of sesame root rot *(Macrophomina phaseolina)* application of neem cake at the rate of 150 kg/ha is recommended.

4. Soil amendment

It has been proved that the organic amendments rich in carbon and deficient in nitrogen control the take-all disease (O*phiobolus graminis*) of wheat. There is considerable liberation of CO_2 by soil saprophytes which suppresses the pathogenic activity of this fungus .In the process of survival also, low nitrogen content in the soil reduces the longevity of the fungus. *Phytophthora* root rot of avocado is controlled by amending the soils with alfalfa meal- a material of low C/N ratio. The other diseases are pea root rot *Aphanomyces euteichus* when cruciferous plant residues were incorporated into the soil.

5 .Summer ploughing

Deep ploughing during summer periods buries the inocula of fungi of soil borne nature. Fungal propagules, sclerotia and different types of spores conidia on plant refuses die when exposed to sunlight due to the higher temperature prevailing during the summer. Further infected self sown plants, volunteer hosts plants, weed hosts, regrowths from the plant roots, alternate hosts

and alternative hosts are also destroyed. Here, the spread of the disease is avoided. Groundnut blight (*Corticium rolfsii*) is controlled by ploughing the soil to a depth of 20 cm. The inverted plough sole soil buries the sclerotia of the fungi, *Claviceps, Sclerotium* and *Sclerotinia* in association with plant or alone.

7. Leveling of the field and provision of drainage facilities

Water stagnation in different patches of field favours the fungi like *Pythium, Phytophthora, Rhizoctonia solani*, etc., for which proper leveling of the field before sowing or planting is very essential.

7. Burning of stubbles and crop residues

Burning of plant wastes, crop residues, stubbles, *etc.*, in the areas selected for raising nurseries for vegetable crops, tobacco, chillies and forest trees etc. heats the soil and kills the inoculum of the pathogens present in the top layer of the soil. When nurseries are raised in these areas incidence of damping off disease is highly reduced. Otherwise, debris in the field helps the perpetuation of the pathogen and the disease. Burning of rice crop residues avoid carryover of sheath blight (*Rhizoctonia solani*); stem rot (*Sclerotium oryzae*) of rice and bacterial blight of cotton.

<u>During Crop growing seasons</u>

1. Adjustment of sowing time

In many diseases the incidence is more severe when the susceptible stage of the plant growth and favourable conditions for the pathogens coincides. While choosing the time of sowing it should be taken into consideration that susceptible stage of the crop growth and soil conditions and other environments favourable for maximum activity of the pathogen does not fall at the same time. Avoiding cool and cloudy days for planting will help to reduce red rot of sugarcane. Late sowing of winter wheat and barley is considered to be the most effective measures in reducing take all disease of wheat. Rapeseed sown in mid to late August is more liable to attack by leaf spot (*Alternaria brassicae*) than late-sown crops. Stem rust of wheat damages the late sown crop more than the early sown crop. Because, time of onset of disease and ear formation coincides.

2. Selection of seeds and seed materials

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

The diseases like foot rot, brown spot, short smut of sorghum, loose smut of wheat, bacterial blight of rice, bacterial blight of cotton, leaf crinkle of blackgram etc., are transmitted through seeds.

3. Seed rate

Use of higher seed rate in the nursery creates favourable microclimate for the pathogens causing damping-off in vegetables, tobacco, chillies and forest nurseries. Hence, use of optimum seed rate should be adhered in such crops.

4. Depth of sowing

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

5. Spacing

Closer spacing invariably alters the microclimate underneath the canopy of the crop which may provide favourable environment for development of diseases. Closer spacing favours many air borne diseases because of high humidity in the crop canopy. Early and late blight of groundnut and blister blight of tea are more in dense canopy. Early spread of black rot of cabbage takes place in closer spacing. Crowded stands may reduce some systemic diseases. Cotton wilt caused by *Verticillium albo-atrum* will be less in closely planted crop if the fungal inoculum is less in the soil.

6. Method of sowing/planting

In places where water accumulation is a problem to the crop growth sowing of seeds on the sides or ridges is found effective in reducing the incidence of *Sclerotium rolfsii* on groundnut and vegetable crops and *Sclerotinia sclerotiorum* and *Rhizoctonia solani* on vegetable crops and *Phytophthora* blight of pigeonpea.

7. Mixed cropping

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

8. Intercropping

Intercropping is also a device in the control of some soil borne diseases. Intercrops should be properly chosen so that they should not have any common pathogen for e.g., *Macrophomina phaseolina* has got wide host range and hence common host should not be grown as intercrops. Intercropping with moth bean (*Phaseolus aconitifolius*) in a cotton field reduced the root rot (*M.phaseolina*) incidence.

9. Decoy crop and trap crop

Decoy crops (hostile crops) are non-host crops sown with the purpose of making soil-borne pathogens waste their infection potential. This is effected by activating dormant propagules of fungi, seeds of parasitic plants, etc. in absence of the host. A list of pathogens that can be decoyed is given in table.

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

Post planting operations:

1. Mulching

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

2. Irrigation water management

Irrigation to the crop in the field is to wet the soil to the extent that roots easily get water and nutrients. If excess water is added to soil, it may directly affect activity of pathogens and/or it may affect disease incidence through the effect on the host. Scab attack on potato tubers is prevented by maintaining soil moisture near field capacity during tuber formation. Bacterial flora antagonistic to *Streptomyces scabies* increases under high moisture conditions. When excess irrigation is made the juvenile stage of plants is lengthened making it susceptible to attack of fungi like *Pythium*. Supply of frequent but low quantity of irrigation water is, therefore, recommended for reducing chances of damping off in nurseries.

3. Roguing

Roguing consists of completely removing or uprooting the diseased plants to prevent further spread of the disease. This method is widely adopted in the control of virus diseases spread by insects (cassava mosaic, yellow mosaic of blackgram and greengram, citrus tristeza, katte disease of cardamom, bunchy top of banana) and basal stem rot of coconut, green ear of pearlmillet and broomrape (*Orobanche*) in tobacco. The whip smut of sugarcane (*Ustilago scitaminea*) in the canal areas of Bombay in Co.475 variety has been greatly checked by roguing carried out over wide areas and long period.

4. Management of plant nutrients

The plant nutrients in general when applied in excess may increase or reduce the resistance in plants to diseases. Increased application of nitrogenous fertilizers increases the incidence of many diseases. Crops fed with heavy doses of nitrogenous was fertilizers grow robust with foliage and succulent tissue but become highly susceptible to the attack of diseases like rust powdery mildew, blast, tobacco mosaic and some bacterial diseases .In the case of blast of rice optimum dose of nitrogenous fertilizers are recommended and it is applied in 3 split doses viz. 50% as based at transplanting, 25% at tillering and 25% at panicle initiation stage. Late application of nitrogenous fertilizers increases wheat leaf blotch (*Septoria nodorum*) and powdery mildew (*Erysiphe graminis tritici*).

6. Avoiding injury

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

7. Time of harvesting

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

8. Avoiding ratooning

Ratooning is a general practice in sugarcane when the incidence of grassy shoot disease and red rot are very high. Hence ratooning should be avoided.

II. PHYSICAL METHODS

Introduction

Physical methods for disease control as alternative to fungicides for the management for seed and soil-borne pathogens, and for integration in disease management programs. They usually do not leave residues or pollute the environment.

The Principle

The scientific principle involved in heat therapy is that the pathogen present in seed material is selectively inactivated or eliminated at temperatures that are non-lethal to the tissues. The exact mechanism by which heat inactivates the pathogen is not fully understood. It is universally accepted that heat cause inactivation and immobilization of the pathogen. There are two of thoughts regarding inactivation of pathogen (viruses) by heat.May be the chances that heat leads loosening of bonds both in nucleic acid and the protein components of the virus. In the nucleic acid, when the bonds are disrupted the linear arrangement of nucleotides is disrupted and thus the virus looses infectivity. In proteins, the bonds holding the chains of amino acids may be destroyed. Disruption of bonds causes denaturing of protein molecules which become less soluble in water and finally leads to coagulation. The rate at which the pathogen is inactivated is determinate by temperature and the duration of treatment.

Methods for managing Seed-borne Pathogens

Some of the seed treating procedure do not involve the use of fungicides, the physical agents like hot water or hot air or steam is used to eliminate the seed-borne infection. These methods are successfully used in controlling certain internally seed-borne diseases like loose smut of wheat and systemically infected diseases caused by bacterial, virus,viral and phytoplasma. Following physical methods are employed for reduction and/or elimination of primary inoculums that may be present in seed or planting material:

1) Hot Water Treatment: Hot water treatment is widely used for the control of seed-borne pathogens, especially bacteria and viruses. A list some important seed-borne diseases, which can be controlled by hot water treatment is given below:

Crop	Disease	Causal Organism	Treatment
Brassica spp.	Black rot	*X. campestris* pv. *campestris*	50ºC for 20 or 30 min
Cluster bean	Blight	*X. campestris* pv. *cyamopsidis*	50ºC for 10 min
Pearl millet	Downy mildew	*Sclerosporagraminicola*	55ºC for 10 min
Sugarcane	Red rot	*Colletotrichum falcatum*	54ºC for 8 hr
Sugarcane	Mosaic	Virus (Potato Virus Y group)	20 min treatment for 3successive days at 52ºC
Rice	Udbatta	*Ephelisoryzae*	54ºC for 10 min
Rice	White tip	*Aphelenchoidesbesseyi*	51-53ºC for 15 min

2) Hot Air Treatment: Hot air treatment is less injurious to seed and easy to operate but also less effective than hot water treatment. It has been used against several diseases of sugarcane. For example, complete control ofred rot in varieties Co 527, CoS 510, Bo 3 and Bo 32 by hot air treatment of 54ºC for 8 hours. Hot air treatment at 54ºC for 8 hours effectively eliminates RSDpathogen without impairing the germination of buds.

3) Solar Heat Treatment: The treatment has been devised in India to eliminate the pathogen of loose smut of wheat.Previously the hot water treatment was followed to eliminate loose smut. As the thermal death point of the fungus and the embryo are very close, extensive care should be taken to avoid killing of the embryo. Luthra in 1953, devised a method to eliminate the deep seated infection of *Ustilago nuda*. The method is popularly known as solar heat or solar energy treatment.

Luthra's solar energy treatment: The seeds are soaked in cold water for 4 hours in the forenoon ona bright summer day followed by spreading and drying the seeds in hot sun for four hours in the afternoon.

Methods for managing Seed-borne Pathogens

Soil harbours a large number of plant pathogens and the primary sources ofmany plant pathogens happens to be in soil where dead organic matter supports active or dormant stages of pathogens. Soil treatment is largely curative in nature as it mainly aims at killing the pathogens in soil and making the soil 'safe' for the growth of the plant. Some of the commonly followed physical methods for soil-borne pathogens are:

1) Soil Solarization: Soil solarization is conduction of heat by entrapment of solar irradiation through the greenhouse effect. Very thin (25-50 µm) transparent polyethylene (PE) mulch is commonly used to trap solar heat because it is permeable to the short-wavelength solar radiation, but does not transmit longer-wavelength radiation (heat) from the ground back into the atmosphere. Solar radiation with short wavelengths (about 120 - 400 nanometers) carries higher amounts of energy than does radiation with longer wavelengths (infrared radiation). The transparent plastic tarp used in solarization transmits ultraviolet (UV) and visible light but not infrared radiation (IR) known as photoselective PE plastic. As the short-wavelength solar radiation passes through the plastic layer, it loses energy; the wavelengths increase in length and the radiation essentially becomes infrared radiation that generate heat. This heat is trapped beneath the plastic where it warms the soil.

Soil solarization with transparent polyethylene sheet mulch (25μm) for 40 days is effective for the control of collar and root rot of strawberry caused by *Sclerotium rolfsii*, Fungal diseases such as damping-off, root rots, stem rots, fruit rots, wilts and blights caused by *Pythium* spp., *Phytophthora* spp., *Fusarium* spp., *S. rolfsii, R. solani, Sclerotiniasclerotoirum, T. basicola* and *Verticillium* spp. have been successfully managed by soil Solarization.

Nematode diseases such as *Ditylenchus dipsci, Globoderarostochiensis, Heterodera*spp., and *Meloidogyne* spp. have been successfully managed by soil solarization.

Bacterial canker of tomato (*Clavibactermichiganensis*subsp. *michiganensis*) is successfullymanaged by Soil solarization for 1-2 months.

2) Steam Sterilization: Steam is passed through perforated pipes at a depth of 15 cm to sterilize the upper layers of soil. It is mostly practiced under glass house and green house conditions.

3) Hot Air Sterilization: Hot air is also passed through pipelines to sterilize the soil in the nursery areas.

4) Hot Water Treatment: It is mainly done in pot culture studies to kill the fungi and nematodes. The pots containing soil areimmersed in boiling water at 980C for 5 minutes or drenching boiling water @ 20 liters/m^2.

5) Burning: Burning helps in destroying inoculum crop debris or pasture grasses which can periodically be freed from inoculum before they make new growth. It has also been realized that the increasing success of non-tillage in some crops and the resultant problems of debris management can make burning an attractive and effective proposition.

6) Flooding: Flooding fields and orchards to reduce or eliminate soil-borne inoculum of plant pathogens is an ancient practice. Flooding has been recognized to be one of the key factors for the low incidence of soil-borne disease.

Physical Methods for managing post-harvest losses

1) Refrigeration: Low temperature at or slightly above the freezing point checks the growth and activities of all such pathogens that cause a variety of post harvest diseases of vegetables andfruits. Therefore, most perishable fruits be transported and stored in refrigerated vehicles and stores.

2) Radiation: Electromagnetic radiations such as ultraviolet (UV) light, X-rays and Y-rays, as particulate radiations can be used to management of post-harvest diseases of horticultural crops.

III) REGULATORY METHODS – PLANT QUARANTINE AND INSPECTION

Plant Quarantine: The term 'Quarantine' has been derived from Latin word '*Quarantum*' means simply forty i.e., 40 days period. This was more commonly referred to the period of detention for ships arriving from countries subject to epidemic diseases such as the Bubonic plague, cholera and yellow fever. The crew and the passengers used to be compelled to remain isolated on board for

sufficient period to permit the diseases to develop and be detected. The purpose of the health authorities was to establish adequate detention period. Later on, the term 'Quarantine' came to be only used for the detention and the practices connected with it. The term got associated from the human disease field to the animal disease field and later on adopted to cover protective methods for the exclusion of pests and diseases of agricultural and horticultural crops.

'Plant Quarantine' refers to the holding of plants in isolation until they are believed to be healthy. Now, broader meaning of the plant quarantine covers all aspects of the regulation of the movement of living plants, living plant parts/plant products between politically defined territories or ecologically distinct parts of them. Intermediate quarantine and post entry quarantine are used respectively to denote the detention of plants in isolation for inspection during or after arrival at their final destination.

History: The first plant quarantine law was promulgated in Rollen, France in 1860 to suppress and prevent the spread of common barberry, the alternate host for wheat stem rust. Among other countries, the first few to establish plant quarantine services were Germany, France, Australia and the U.S.A. In India, legislative measures against crop pests and diseases was initiated under the Destructive Insects and pests Act of 1914 (DIP act) and it was passed by Governor General of India on 3 rd February, 1914. Under this Act, rules governing the import and movement of plants and plant materials, insects and fungi are framed.

The Act provides:

- It authorizes the Central Government to prohibit or regulate the import into India or any part there of any specific place therein, of any article of class of articles.

- It authorizes the State Governments to make rules for the detention, inspection, disinfection or destruction of any insect or class of insects or of any article or class of articles, in respect of which the Central Government have issued notifications. It also authorizes the State governments for regulating the powers and duties of the officers whom it may appoint on this behalf.

Importance of Plant Quarantine: The adoption Quarantine Regulations and acts by different countries of the world has arisen out of fact that extensive damages often sudden in nature, have been caused not by indigenous organisms but by exotic ones which have been cause not by indigenous organisms but by exotic ones which have been introduced along with plants, plant parts or seeds in the normal channel or trade or individual transit.

Introduction of new pathogen: Often a new pest, disease or weed has accidentally entered a country where it did not exist before and has multiplied, spread and caused enormous damage to the crops of that country.

Table: Diseases believed to have been introduced into India from foreign countries, some examples are as follows:

S. No.	Disease	Country	Year
1	Coffee rust *(Hemileia vastatrix)*	Sri Lanka	1879
2	Late blight of potato (*Phytophthora infestans*)	England	1883
3	Rust of chrysanthemum (*Puccinia carthami*)	Japan / Europe	1904
4	Flag smut of wheat	Australia	1906
5	Downy mildew of grapes (*Plasmopara viticola*)	Europe	1910

6	Downy mildew of maize (*Scleropora phillipinensis*)	Java	1912
7	Downy mildew of cucurbits (*Pseudoperonospora cubensis*)	Srilanka	1918
8	Black rot of crucifers (*Xanthomonas campestris*)	Java	1929
9	Foot rot of rice (*Fusarium moniliforme*)	South East Asia	1930
10	Leaf spot of sorghum (	South Africa	1934
11	Powdery mildew of rubber (*Oidium heveae*)	Malaya	1938
12	Black shank of tobacco	Holand	1938
13	Fire blight of pear	England	1940
14	Crown gall of apple/pear (*Agrobacterium tumefaciens*)	England	1940
15	Bunchy top of banana	Srilanka	1940
16	Canker of apple	Australia	1943
17	Wart of potato (*Synchytrium endobioticum*)	Netherlands	1953
18	Golden nematode of potato	Europe	1961
19	Sunflower downy mildew (*Plasmopara holstedi*)	Australia	1985

Losses caused by introduced plant diseases: The entry of a single exotic insect or disease and its establishment in the new environment continues to cause great, national loss. In certain cases a country has to spend a few million rupees before success in controlling the introduced insect pest or disease is achieved.

Table: Losses caused by introduced plant diseases

S. No.	Disease	Crop	Host Country	Introduced from	Losses caused
1	Bunchy top	Banana	India	Sri Lanka	Rs.4 crores
2	Wart	Potato	India	Netherlands	2500acres infected
3	Canker	Citrus	U.S.A	Japan	$ 13 million; 19.5 million trees destroyed
4	Downy mildew	Grapevine	France	U.S.A	$ 50,000 million
5	Powdery mildew	Grapevine	France	U.S.A	80% in wine production

Plant quarantine methods

There are number of plant quarantine methods which are used separately or collectively to prevent or retard the introduction and establishment of exotic pests and pathogens. The components of plant quarantine activities are:

1. Complete embargoes

It involves absolute prohibition or exclusion of specified plants and plant products from a country infected or infested with highly destructive pests or diseases that could be transmitted by the plant or plant products under consideration and against which no effective plant quarantine treatment can be applied or is not available for application.

2. Partial embargoes

Partial embargoes, applying when a pest or disease of quarantine importance to an importing country is known to occur only in well defined area of the exporting country and an effectively operating internal plant quarantine service exists that is able to contain the pest or disease within this area.

3. Inspection and treatment at point of origin

It involves the inspection and treatment of a given commodity when it originates from a country where pest/disease of quarantine importance to importing country is known to occur.

4. Inspection and certification at point of origin

It involves pre-shipment inspection by the importing country in cooperation with exporting country and certification in accordance with quarantine requirements of importing country.

5. Inspection at the point of entry

It involves inspection of plant material immediately upon arrival at the prescribed port of entry and if necessary subject to treatment before the same related.

6. Utilization of post entry plant quarantine facilities

It involves growing of introduced plant propagating material under isolated or confined conditions.

Agencies involved in plant quarantine

The authority to implement the quarantine rules and regulations framed under DIP Act rests basically with the Directorate of plant Protection, Quarantine & Storage, under the Ministry of Agriculture. This organization handles bulk import and export of seed and planting material for commerical purpose. Under this organization 9 seaports, 10 airports and 7 land frontiers are functioning. These are the recognized ports for entries for import of plant and plant material.

The names and places of the ports and stations are as follows:

Seaports (9)	Airports (10)	Land Frontiers (7)
1. Bhavnagar - Gujarat 2. Calcutta - West Bengal 3. Chennai - Tamil Nadu 4. Cochin - Kerala 5. Mumbai - Maharashtra 6. Nagapattinam - Tamil Nadu 7. Rameswaram - Tamil Nadu 8. Tuticorin - Tamil Nadu 9. Visakhapatnam - Andhra Pradesh	1. Amritsar - Punjab 2. Calcutta - West Bengal 3. Chennai - Tamil Nadu 4. Hyderabad - Andhra Pradesh 5. Mumbai - Maharashtra 6. New Delhi - New Delhi 7. Patna - Bihar 8. Tiruchirappalli - Tamil Nadu 9. Trivandrum - Kerala 10. Varanasi - Uttar Pradesh	1. Amritsar Railway Station - Punjab 2. Attari Railway Station - Punjab 3. Attari-Wagah Border- Punjab 4. Bangaon Benapol Border - West Bengal 5. Gede Road Railway Station - West Bengal 6. Kalimpong - West Bengal 7. Sukhia Pokhri - West Bengal

Certification:

Phytosanitary or health certificate is a certificate which should accompany a plant or plant material or seed which is to be moved from one place to another place. This certificate indicates or certifies that the material under transit is free from pests or diseases.

Domestic Quarantine

Under the DIP Act, the Directorate of Plant Protection, Quarantine and storage has the responsibility to take the necessary steps and regulate the inter-state movement of plants and plant material in order to prevent the further spread of destructive insects and diseases that have already entered the country. The sole object of enforcing domestic quarantine is to prevent the spread of these diseases from infected to non-infected areas. Currently, domestic plant quarantine exists in four diseases:

1. Bunchy top of banana: The export and the transport from the States of Assam, Kerala, Orissa, West Bengal, Tamil Nadu to any other State of Banana plant or any other plant of the genus *Musa*, including sucker, stem, leaf, flower, and any other part thereof which may be used for propagation, or the materials of banana plant or any other plant of the genus *Musa*, which are used for packing and wrapping, excluding the banana fruit is prohibited.

2. Banana mosaic: The export and transport from the States of Maharashtra and Gujarat of any plant of Banana or any other plant of genus *Musa* including the sucker, stem flower and any other part thereof, but excluding leaf and fruit thereof is prohibited.

3. Potato wart: The export to potato tubers from the State of West Bengal to any other State or territory of India is prohibited.

4. Apple scab: The Directorate of Horticulture, Himachal Pradesh worked out a detailed scheme for the eradication of scab, and prohibiting the export of planting material of apple outside the State.

IV. BIOLOGICAL CONTROL

Definition: "Biological control of plant disease is a condition or practice whereby survival or activity of a pathogen is reduced through the agency of any other living organism (except human beings), with the result that there is reduction in incidence of the disease caused by the pathogen (Garett, 1965).

According to Baker and Cook, 1974 "Biological control is the reduction of inoculum density or disease producing activity of a pathogen or a parasite in its active or dormant state by one or more organisms accomplished naturally or through manipulation of the environment of host or antagonist by mass introduction of one or more antagonists".

The four main mechanisms involved in the biocontrol are (i) the biological agent (antagonist), may parasite the other organism, (ii) antagonist may secrete metabolites (antibiotics) harmful to the pathogens (Antibiosis) (iii) antagonist may compete with the pathogens for nutrients or space (Competition) and (iv) may cause death of the parasite by producing enzymes (Lysis).

Parasitism and Lysis

The biocontrol against parasitizes the pathogen by coiling around the hyphae, e.g., *Trichoderma* viride; various bacteria and fungi secrete hydrolytic about the degradation of cell wall of pathogens.

e.g. (i) *Bacillus* sp. causes hyphal lysis of *Gaeumanornyces* graminis

(ii) The chitnolytic enzymes of *Serratia marcescens* caused cell wall lysis of *Scierotium rolfsii.*

(iii) *Trichoderma* sp. produces chitinases and β-1,3 glucanases which lyses the cell wall of *Rhizoctonia solani.*

Antibiosis

The antibiotic compounds secreted by the biocontrol agent suppress the growth of the pathogen. e.g. Phenazine-l-carboxylic acid produced by *P fluorescens* plays an important role in suppressing the take all disease of wheat.

Competition

The biocontrol bacteria and fungi compete for food and essential elements with the pathogen thereby displacing and suppressing the growth of pathogen.

e.g. (i) The competition for nutrients between *Pythium aphanidermatum, P ultimum* and bacteria suppress the damping off disease in cucumbers.

(ii) Fluorescent siderophores (iron chelaters) such as pseudobactinis & *pyoverdins* produced by P *fluorescons* chelates iron available in the soil, thereby depriving the pathogen of Its Fe requirements.

Biocontrol agents for the management of plant pathogens

Biocontrol agent	**Pathogen/disease**
1. *Ampelomyces quisqualis*	Powdery mildew fungi
2. *Darluca filum, Verticillium lecanii*	Rust fungi
3. *Pichia gulliermondii*	*Botrytis, Penicillium*

Biocontrol agent	**Nematode**
1. *Pasteuria penetrans* (Bacteria)	Juvenile parasite of root knot nematode
2. *Paecilomyces lilacinus* (Fungus)	Egg parasite of *Meloidogyne incognita*

<u>Important fungal biocontrol agents</u>:

Most of the species of *Trichoderma*, viz., *T. harzianum, T. viride, T. virens* (*Gliocladium virens*) are used as biocontrol agents against soil borne diseases, such as, root rots, seedling rots, collar rots, damping off and wilts caused by the species of *Pythium, Fusarium, Rhizoctonia, Macrophomina, Sclerotium, Verticillium,* etc.

Formulations of biocontrol agents available: *T. viride* (**Ecofit**, **Bioderma** in India), *G. virens* (**GlioGard** in USA), *T. harzianum* (**F-Stop** in USA) and *T. polysporum* (**BINAB-T**)

<u>Important bacterial biocontrol agents</u>:

1. *Pseudomonas fluorescens* (**Dagger-G** against damping off of cotton seedlings in USA)

2. *Bacillus subtilis* (**Kodiak** against damping off and soft rot in USA)
3. Agrobacterium radiobacter K-84 (**Gallex** or **Galltrol** against crown gall of stone fruits caused by *Agrobacterium tumefaciens*)

V. HOST PLANT RESISTANCE

A physiological deviation from the normal functioning of the organism (i.e., the crop plant) caused by pathogenic organisms is a disease and may be caused by fungi, bacteria or viruses. The inherent ability of an organism (i.e., the crop plant) to resist or withstand the pathogen is called resistance. Disease resistance commonly met with in the plant kingdom relative in nature, total immunity being too rare. Its hereditary transmission from parent to off-spring is essentially "Mendalian", but often polygenic.

The earliest demonstration of the behaviour of "disease-resistance" as a character transmissible from parent to off-spring in the "Mendelian" fashion was given by Biffen (1905) in his work on yellow rust of wheat. Since then, intensive work has been done on this aspect which has proved the value of applying genetical principles in developing disease-resistant varieties of plants for effective control of diseases.

Resistant varieties can be the simplest, practical, effective and economical method of plant disease control. The use of resistant varieties cannot only ensure protection against diseases but also save the time, energy and money spent on other measures of control.

The resistance of a crop to a physiological race of the pathogen depends not only on the genotype of the host for resistance, but also upon the genotype of the pathogen for virulence or aggressiveness. Flor (1942) proposed the gene-for-gene hypothesis, according to which, for every gene for resistance in the host, there is a corresponding gene for pathogenicity in the pathogen.

It means that there are atleast two alleles at a locus controlling resistance/susceptibility in the host (R-r) and two alleles at a corresponding locus in the pathogen (V-v) controlling virulence / aggressiveness. Out of the four possible interactions between these alleles, only one combination leads to the expression of resistance. The demonstration of gene-for-gene relationship requires genetic studies of both the host and the pathogen.

Vertical resistance (VR) and horizontal resistance(HR)

Van der Plank (1960) has discussed the whole issue of disease resistance in a different perspective. He calls the unstable and often complete type of resistance as vertical resistance and the more stable but somewhat incomplete resistance as horizontal resistance. If resistance to some races of a pathogen is more than to other races, it is called Vertical resistance. It is also called Perpendicular resistance, Physiological resistance, seedling resistance, hypersensitivity, race specific resistance or qualitative resistance. As it is conditioned by one or a few genes, it is called major gene or monogenic of oligogenic resistance.

Resistance to more than one race of the pathogen or to many or all races of the pathogen is called Horizontal Resistance. It is non-specific resistance governed by polygenes. It is severally termed as non-specific, general, polygenic, minor gene, mature plant, adult, quantitative resistance, partial or field resistance or tolerance. HR causes reduction in the number and rate of sporulation of the pathogen on the host and slows down the infection rate. HR includes tolerance slow development of disease, escape and exclusion mechanisms besides hypersensitive reaction.

a. Disease escape

It is a prevention mechanism that causes the host to escape pathogenic infection. Early or late maturity of the crop may prevent physical contact of the pathogen with the host. Mechanical and anatomical barriers such as thick cuticle, waxy bloom on leaves and stem, stomatal regulation prevent penetration of spores. Ergot, a fungal disease of inflorescence in cereals caused by *Claviceps purpurea* does not affect varieties of wheat and barley in which the flowers remain closed until pollination occurs. Erect leaves of barley avoid deposition of spores of *Erysiphe graminis tritici* in contrast to prostrate leaves. Early maturing varieties of groundnut escape early leaf spot infection (*Cercospora arachidicola*) and early varieties of wheat escape rust and loose smut infection.

b. Disease endurance

The host after being infected by the pathogen tolerates the infection and suffers less damage. It does not result in any substantial decrease in yield. This is brought about by influence of external factors. It is a well-known phenomenon that plants fertilized with phosphatic and potash manures are more tolerant to disease; this is the case in wheat against rust infection. Rice crops fertilized by silicates are "resistant" to blast (P*yricularia oryzae*) in Japan. Wheat crops fertilized by potash and phosphatic manures are highly tolerant to mildew and rust infection. The fertilizers act indirectly to arrest vegetative growth and promote early maturity, better straw and strengthening tissues to protect the plant which form a bulwark against pathogenic invasion.

c. True resistance

It is the ability of the host plant to resist or withstand the attack of a pathogen. True resistance is inheritable and much less subject to environmental influence. It is specific in character. The basis of resistance may be morphological, functional, structural or protoplasmic. Functional nature of resistance is determined by opening of the stomata, time of opening of flowers and time of maturity, rate of cork formation and cambial activity.

True resistance, however, is of a specific character and is determined by the defence equipment and activities of the plant itself against the parasitic invasion and is therefore not subject to any appreciable modifications by external factors.

Methods of breeding for disease resistance

The methods of breeding varieties resistant to diseases do not differ greatly from those adopted for other characters. The following methods are used:

1.Introduction

1. Selection

2. Hybridization followed by selection

3. Back cross method

4. Induced mutagenesis

5. Development of multilines

6. Tissue culture techniques

7. Genetic engineering

1. Introduction

It is a very simple and inexpensive method. Varieties resistant to a particular disease elsewhere may be thoroughly tested in the regions in which they are proposed to be introduced. Their yield performance and disease resistance should be confirmed by large scale cultivation. It is possible that a variety resistant in one region need not be resistant in another region due to variation in the physiological race of the pathogen or due to a much different agroclimatic condition in the new location.

Introductions have served as a useful method of disease control. For example, from Manila, a rice variety introduced in Karnataka from the Philippines, has tolerance to blast, bacterial leaf blight and sheath blight. Early varieties of groundnut introduced from U.S.A. have been resistant to leaf spot (*Cercospora arachidicola).*

2. Selection

This is better method than introduction and has more chances of success in obtaining disease-resistant plants. The work of selection is carried out either in the naturally infected fields under field conditions or under artificially inoculated conditions. The resistance in such individuals will occur in nature by mutation. To ensure the resistant character of a plant, large population of crop plant may be exposed to the attack of pathogen under artificial conditions and the non-infected plants may be chosen. Sugandh of Bihar is a selection from Basmati rice of Orissa tolerant to bacterial leaf blight.

3. Hybridization

When selection of resistant varieties is not feasible, resistant varieties may be evolved by crossing the susceptible popular variety with resistant wild variety where in the resistant gene or genes transferred into the genetic make up of susceptible variety. Very often the F1 from crosses may be resistant but carry the other undesirable qualities of the resistant parent. The bad qualities are removed by several back crossing of F1 with the susceptible parent may ultimately yield a resistant progeny with good agronomic characteristics.

Under certain circumstances pedigree or bulk method of selection is followed to obtain a resistant variety. In this method, the crosses are made till F2 population is got. Selections are made in F2 generation for superior genetic traits including disease resistance. By continued selfing, selections are made through F3 to F5 or F6 generations and the best variety is selected. This method is suited for small grains and beans but unsuited to fruits and vegetables.

4. Back cross method

Back cross method is widely used to transfer disease resistance from wild species. Wild species are rice sources of disease resistance. Interspecific hybridization is made to transfer the gene or genes for resistance to the cultivated species. Resistance to grassy stunt virus from *Oryza nivara* to *O.sativa*, late blight resistance from *Solanum demissum* to cultivated potato, rust resistance from *durum* to *aestivum* wheat are some of the examples involving interspecific hybridization. Depending upon the number of genes governing resistance and the nature of the gene, whether dominant or recessive, the procedure varies. The number of back crosses to the cultivated species may be five to six. Once the back cross progeny resemble the cultivated parent, then they are selfed and segregating progeny screened for disease resistance.

5. Induced mutagenesis

While following mutation breeding for disease resistance, a large number of mutation progeny should be produced and screened under artificial epiphytotic condition to select resistant plants. MCU10 cotton, a resistant variety to bacterial blight was evolved in Tamil Nadu by subjecting seeds of a susceptible variety CO4 to gamma rays followed by rigorous screening and selection 6. Development of multilines The concept of multilines was first suggested by Jensen(1952) and developed by Borlaug (1959) for evolving multiline varieties to resist stem rust in wheat. A multiline variety is a composite of genetically similar lines, except that each line possesses a different gene for resistance to the pathogen.

Lines that are genetically similar, except for one gene, are called isoline. It is assumed that gene for resistance in each isoline contributes resistance to a separate physiological race or group of races. Genes for disease resistance are transferred by backcrossing from donor varieties to a common disease susceptible, but agronomically superior, recurrent parent. Isolines are generated differing only in the gene for disease resistance. The isolines are composited to synthesize a multiline variety. The isolines are maintained for resynthesizing the multiline whenever needed. A multiline variety is composed of a mixture of resistant and susceptible genotypes and provides a buffering effect against rapid development of disease. It will provide resistance or tolerance to a broad spectrum of races of a pathogen. If new races of the pathogen are identified at a later stage, additional isolines resistant to the newly arisen races may be constituted and incorporated. Kalyan sona and Sonalika-based multilines of wheat resistant to different races of rust have been developed in India.

7. Tissue culture technique

Tissue culture techniques to produce somaclonal variation for disease are developed in different crops. Somaclonal variations for disease resistance are reported in *Zea mays* for *Drechslera maydis* race T-toxin resistance, in *Brassica napus* for resistance/tolerance to *Phoma lingam*, early and late blight resistance in potato, *Pseudomonas* and *Alternaria* resistance in tobacco, besides smut and rust disease resistance in sugarcane.

8. Genetic Engineering is the technology by which a particular gene is isolated from one organism and inserted into the genome of another organism and made to express at the right time.

VI. CHEMICAL CONTROL

CHEMICAL CONTROL OF PLANT DISEASES: STUDY OF DIFFERENT FUNGICIDES & ANTIBIOTICS

Use of chemicals in plant diseases management is included in the principles of **protection or therapy**.

❖ **Protection**

The prevention of the pathogen from entering into the host or checking the further development in already infected plants by the application of chemicals is called ***protection*** and the chemicals used are called ***protectants***.

❖ **Therapy**

Therapy means cure the disease, in which fungicide is applied after the pathogen is in contact with the host. Chemicals used are called ***therapeutants***.

❖ **Fungicides**

The word fungicide originated from two **Latin** words, viz., "***fungus***" and "***caedo***". The word "***caedo***" means **to kill**. Thus, these are biocidal chemical compounds used to kill fungi or fungal spores.

❖ **Fungistat**

Some chemicals which do not kill fungi, but simply inhibit the fungus growth temporarily which is known as ***fungistat*** and the phenomenon of temporarily inhibiting the fungal growth is termed as ***Fungistatics***.

❖ **Antisporulant**

The chemical which inhibits spore production without affecting vegetative growth of the fungus.

Characteristics of a good (ideal) fungicide

- ✓ Low phytotoxicity: Ideal fungicide should not be toxic to the plants.
- ✓ It should have long shelf life.
- ✓ It should be less toxic to human being and cattle etc. It should not be toxic to earthworms and beneficial soil microorganisms.
- ✓ It should have high toxicity to a wide range of fungi
- ✓ It should not cause accumulation of toxic chemicals in soil or in plants or in plant products.
- ✓ It should combine with important or commonly used insecticides or acaricides without any deleterious effects.
- ✓ It should be cheap (economical) and easily available in the market and easily transportable.
- ✓ It should be available in different formulations.
- ✓ It should easily spread on the host surface when applied.
- ✓ It should not cause environmental pollution.

CLASSIFICATION OF FUNGICIDE

1. BASED ON MODE OF ACTION

(I) Protectant: Fungicide which protects a plant from a pathogen if it is applied prior to infection. They are prophylactic in their behaviour. Contact fungicides which kill the pathogen present on the host surface when it comes in contact with the host are called protectants. These are applied to seeds, plant surfaces or soil. These are non-systemic in action (i.e. they cannot penetrate plant tissues). e.g. Sulphur, Zineb.

(II) Therapeutant: Fungicide which eradicates a fungus after it has caused infection thereby curing the plant is called therapeutant. Usually chemotherapeutants are systemic and eradicates deep seated infection. e.g. Carboxin, Oxycarboxin, Aureofungin

(III) Eradicant: A chemical substance that destroys (eradicate) pathogen after its establishment in the host plant is known as eradicant. It eradicates the dormant or active pathogen from the host. It also acts as protectant. They can remain on/in the host for some time. e.g. Organomercurials, Lime sulphur, Dodine.

2. BASED ON GENERAL USE

1	**Seed protectants**	Organomercurials, Captan, Thiram, Carbendazim, Carboxin
2	**Soil fungicides (Pre-plant)**	Bordeaux Mixture, Copper Oxychloride, Vapam, Chloropicrin, Formaldehyde
3	**Soil fungicides (when plants are in field)**	Bordeaux Mixture, Copper Oxychloride, Thiram, Captan
4	**Foliage and blossom protectants**	Bordeaux Mixture, Copper Oxychloride, Ferbam, Zineb, Mancozeb, Captan, Carbendazim, Chlorothalonil
5	**Fruit protectants**	Maneb Mancozeb, Captan, Carbendazim, Thiabendazole
6	**Tree wound dresser**	Bordeaux Paste, Chaubattia Paste
7	**Antibiotics**	Streptomycin, Streptocycline, Griseofulvin

3. BASED ON CHEMICAL COMPOSITION

(A) Sulphur Fungicides

Sulphur is probably the oldest chemical used in plant disease management for the control of powdery mildews. **Robertson** first recommended to use sulfur as fungicide for control of powdery mildew of peach.

(I) Inorganic Sulphur: Lime Sulphur and Elemental Sulphur fungicides.

(II) Organic Sulphur: It also called as carbamate fungicides. e.g. Zineb, Thiram.

(I) Inorganic Sulphur

Common name	Trade name	Disease managed
1. Lime sulphur	It is manually prepared by mixing rock lime and sulphur along with water	Powdery mildews of various crops
2. Sulphur dust	Kolo dust, Mico- 999	
3. Wettable sulphur	Sulfex, Thiovit, Cosan	

(II) Organic Sulphur Compounds

Organic sulphur compounds are derived from **dithiocarbamic acid** and are widely used as spray fungicides. In 1931, **Tisdale and Williams** were the first to describe the fungicidal nature of **Dithiocarbamates**.

Dithiocarbamates can be categorized into two groups, *viz.,* dialkyl dithiocarbamates and monoalkyl dithiocarbamates.

Common name	Trade name	Disease managed
1. Ziram	Ziride	Anthracnose of pulses, tomato, beans.
2. Thiram	Thiram, Thiride	Soil borne diseases
3. Zineb	Dithane Z-78,	Chilli die-back and fruit rot, early blight of potato
5. Mancozeb	Dithane M-45, Indofil M-45	Early and late blight of potato and tomato, rust diseases of field and fruit crops

(B) Copper Fungicides: Copper fungicides can be classified as preparatory and proprietary copper compounds.

PREPARATORY COPPER FUNGICIDES

Common name	Chemical composition	Diseases managed
Copper Sulphate Preparation		
1. Bordeaux Mixture (PMA Millardet)	Copper Sulphate + Hydrated Lime + Water	Downy mildew of grapes, Citrus canker etc.

2. Bordeaux Paste	Copper Sulphate + Hydrated Lime + Water	It is a wound dressing fungicide and applied to the pruned parts of the plant. Citrus gummosis, Stem bleeding of coconut

3. Burgundy Mixture (Mason)	Copper Sulphate + Sodium Carbonate + Water	Fungal diseases of trees and fruit plant
4. Cheshunt Compound (Bewley)	Copper Sulphate + Ammonium Carbonate	It is used for soil drenching only. Damping-off diseases of solanaceous crops
Copper Carbonate Preparation		
5.Chaubattia paste (Singh)	Copper Carbonate + Red Lead + Lanolin Or Linseed Oil	Pink disease of citrus, stem canker and collar rot of apple and pears

PROPRIETARY COPPER FUNGICIDES or FIXED or INSOLUBLE COPPER FUNGICIDES

In the fixed or insoluble copper compounds, the copper ion is less soluble than in Bordeaux mixture. So, these are ***less phytotoxic than*** Bordeaux mixture but are effective as fungicides.

Common name	Trade name	Disease managed
1. Copper Oxychloride	Blitox-50, Blue copper- 50	Anthracnose of Grapevine, Tikka leaf spot of groundnut, Citrus Canker
2. Cuprous Oxide	Fungimar, Perenox	-"-
3. Copper Hydroxide	Kocide	Blister blight of tea, False smut of rice, Tikka leaf spot of groundnut

(C) Mercurial Fungicides

Mercury compounds have been in use for a long time for treating seeds, rhizomes, corms, bulbs etc. of vegetables and flowering plants for the control of seed-borne diseases. It have fungicidal and bactericidal activity. They are highly toxic towards animals and human beings and at times even phytotoxic. Because of their extreme toxicity the usage on foliage is limited. Mercury fungicides are **banned** in almost all countries **including India** because of their toxic nature and persistence to get into the food chain.

Common name	Trade name	Disease managed
I. Inorganic Mercury 1. Mercuric chloride 2. Mercurous chloride	Merfusan, Mersil Cyclosan, M-C Turf	Seed Borne Disease
II. Organomercurials Methoxy ethyl mercury Chloride (MEMC)	Agallol, Aretan, Emisan	

(D) Heterocyclic Nitrogenous Compounds

Captan, folpet and captafol belong to dicarboximides and are known as phthalamide fungicides. ***First heterocyclic nitrogenous compounds Captan*** was discovered by **Kittleson.**

Common name	Trade name	Disease managed
1.Captan **(Kittleson's killer)**	Captan 50W, Captan 75 W	Seed borne disease
2.Captafol	Difosan, Difolaton	
3. Iprodione	Rovral, Glycophene	Diseases caused by *Alternaria, Sclerotinia, Helminthosporium*

(E) Benzene Fungicides

Common name	Trade name	Disease managed
1.Chlorothalonil	Bravo, Kavach	Foliar Diseases
2. Dinocap	Karathane	Powdery mildews diseases in many crops

(F) Organo Phosphorus Fungicides: Ediphenphos

(G) Quinone Fungicides: Chloranil, Dichlone *etc*.

(H) Organo Tin Compounds: Du-ter, Brestanol *etc*.

(I) Systemic Fungicides

These are fungi toxic compounds which when applied on different parts of the plant are absorbed by the plant tissues and then translocated upwards, downwards and both ways and act on the pathogen either directly or through its metabolic products and control plant diseases away from the point of application.

The systemic fungicides (**Oxathiin**) was first discovered by **Von Schelming and Marshall Kulka** in 1966. On the basis of chemical nature these fungicides are classified as follows.

Common name	Trade name	Disease managed
Acylalanines		
1. Metalaxyl	Ridomil, Apron	It is highly effective against *Pythium*, *Phytophthora* and many downy mildew fungi.
Aromatic Hydrocarbons		
1. Chloroneb	Demosan	Seedling diseases of cotton, peanut and peas.
Benzimidazoles Compound		
1. Carbendazim	Bavistin	Many Foliar and Soil borne diseases
2. Benomyl	Benlate	Highly effective against diseases caused by the species of *Rhizoctonia, Theilaviopsis* and *Cephalosporium*
3.Thiabendazole	Mertect	Blue and green molds of citrus, loose smut of wheat, Tikka leaf spot of groundnut
Oxathins or Carboximides		
1. Carboxin	Vitavax	Seed bone diseases
2.Oxycarboxin	Plantavax	Highly effective against rust diseases.
Morpholines		
1. Tridemorph	Calixin (Now Banned in India)	Powdery mildew, Rusts
Organophosphates		
1. Iprobenphos	Kitazin	Rice blast, stem rot and sheath blight of rice
2. Ediphenphos	Hinosan	Rice blast, stem rot and sheath blight of rice
Alkyl Phosphonates		
1. Fosetyl-Al or Aluminium Tris	Aliette	Specific against Oomycetes fungi
Thiophanates		
1.Thiophanate methyl	Topsin M	Blast and sheath blight of rice, sigatoka leaf spot of banana
Triazoles		
1. Hexaconazole	Contaf	Rust, Powdery Mildew and other foliar diseases
2. Propiconazole	Tilt	

3. Difenconazole	Score	
4. Tebuconazole	Folicure	
5. Flusilazole	Nustar, Cursor	
6. Myclobutanil	Systhane	
Strobilurins (Isolated from wood rotting mushroom fungi, (*Strobilurus tenacellus)*		
1. Azoxystrobin	Amistar, Quadris	Broad spectrum fungicide
2. Kresoxim methyl	Ergon, Discus	Commonly used for control of ornamental diseases
3.Pyraclostrobin	Headline	Early blight

(II) Based on Chemical Composition

(A) Sulphur fungicide

- **Organic Sulphur:** Zineb, Mancozeb, Ziram, Thiram *etc*.
- **Inorganic Sulphur:** Elemental Sulphur, Sulphur Dust, Lime Sulphur *etc*.

(B) Copper fungicide

- **Copper Sulphate Preparations:** Bordeaux mixture, Bordeaux paste, Burgundy mixture, Cheshunt compound, Fytolan, Copper oxychloride *etc*.
- **Copper Carbonate Preparation:** Chaubbatia paste *etc*.

(C) Mercury fungicide

- **Inorganic Mercury:** Mercuric chloride, Mercurous chloride *etc*.
- **Organomercurials:** Methoxy ethyl mercury chloride, Ethyl Mercury Chloride *etc*.

(D) Quinone Fungicides: Chloranil, Dichlone *etc*.

(E) Heterocyclic Nitrogenous Compound: Captan, Captafol *etc*.

(F) Organo Phosphorus Fungicides: Ediphenphos

(G) Benzene Fungicides: Chlorothalonil, Dinocap, Penta chloro nitrobenzene *etc*.

(H) Organo Tin Compounds: Du-ter, Brestanol *etc*.

(I) Systemic Fungicides

- ✓ **Oxathilin and Related Compounds:** Carboxin, Oxycarboxin *etc*.
- ✓ **Benzimidazoles compound:** Benomil, Thiabendazole, Carbendazim *etc*.
- ✓ **Thiophanates:** Thiophanate methyl
- ✓ **Morpholines:** *Tridemorph*
- ✓ **Sterol Biosynthesis Inhibitor Fungicides:** Propiconazole, Triadimefon *etc*.
- ✓ **Acylalanines or Acylanilides:** Metalaxyl, Cyprofuram *etc*.
- ✓ **Strobilurins Fungicides:** Azoxystobin, Picoxystrobin *etc*.

- ✓ **Organic Phosphate:** Fosetyl- Al, Kitazin *etc*.
- ✓ **Miscellaneous Systemic Fungicides:** Ethazole, Chloroneb *etc*.

(J) Antibiotics

- ➢ **Antibacterial Antibiotics:** Streptomycin, Tetracyclines *etc*.
- ➢ **Antifungal Antibiotics:** Aureofungin, Griseofulvin, Blasticidin, Kasugamycin *etc*.

(K) Fumigant: Formalin 40%, Chloropicrin, Ethylene dibromide *etc*.

FUNGICIDES AND THEIR MODE OF ACTION

Group	Member	Mode of Action	Pathogen
Systemic Fungicides			
Carboximides	Carboxin, Oxycarboxin	**Mitochondrial Electron Transport Inhibitors**	*Puccinia, Ustilago, Tilletia,*
Dinitrophenols	Dinocap		Powdery mildews
Strobilurins	Azoxystrobin Picoxystrobin Kresoxim-Methyl		All the pathogens group
Phenylamides	Metalaxyl	**Nucleic acid biosynthesis inhibitors**	Oomycetes *Phytophthora, Pythium,*
Benzimidazoles	Carbendazim, Benomyl, Thiabendazole Thiophanate-Methayl	**Mitosis and cell division inhibitors**	All fungal pathogens except Oomycetes
Dicarboximides	Iprodione	**Lipid and membrane synthesis inhibitors**	*Botrytis, Septoria, Alternaria*
Organophosphates	Edifenphos, Iprobenfos		*Pyricularia, Rhizoctonia*
Triazoles	Hexaconazole, Propiconazole	**Sterol biosynthesis inhibitors**	All pathogens except Oomycetes
Morphilines	Tridemorph		
Non-Systemic (Contact) Fungicides			

Copper fungicides	Copper	**Multisite contact inhibitors** (Disrupt function of enzymes, energy transport systems, integrity of membranes)	Oomycetes, *Septoria, Fusarium*
Inorganic Sulphur	Elemental sulphur	**Multisite contact inhibitors** (Inhibit electron movement and interferes with cellular respiration)	Powdery mildews
Dithiocarbamate	Mancozeb, Thiram	**Multisite contact inhibitors** (Reacts with protein)	Leaf spots, blights
Heterocyclic nitrogenous compounds	Captan	**Multisite contact inhibitors** (Inhibits enzymes and interferes with decarboxylation process and citrate synthesis)	Oomycetes, *Alternaria, Cercospora, Rhizoctonia*
Phosphonate	Fosetyl- Al	**Multisite contact inhibitors** (Inhibits oxidative phosphorylation)	*Phythium Phytophthora Plasmophora*

Difference between Systemic fungicides and Non-systemic fungicides

	Systemic fungicides	Non-systemic fungicides
1	Any compound capable of being translocated after penetrating the plant & reaches to the point of infection.	Do not get absorbed and translocated into the plant.
2	Movement of fungicide may be **apoplastic** (Upward movement) or **symplastic** (Downward movement)	No movement of fungicides (contact in nature)
3	Single site inhibitor	Multisite inhibitor
4	Long persistence	Short persistence
5	More chances of development of fungicidal resistance	Minimum chances of development of fungicidal resistance

6	Used in low concentration i.e. 0.025 to 0.05%	Used in higher concentration i.e. 0.2 to 0.3%
7	e.g. Carbendazim, Hexaconazole etc.	e.g. Thiram, Captan, Mancozeb etc.

Some Examples of Ready-Mix (Combination Fungicides)

Sr. No.	Chemical name of fungicides	Disease Managed	Trade name
1.	Carbendazim 12% + Mancozeb 63% WP	Rust & Leaf Spot of groundnut	SAAF, Sixer
2.	Azoxystrobin 11% + Tebuconazole 18.3% SC	Fruit rot, Powdery mildew, Die back	Custodia
3.	Carboxin 37.5% + Thiram 37.5 WS%	Seed borne diseases	Vitavax Power
4.	Tebuconazole 50% + Trifloxystrobin 25% WG	Foliar Diseases	Nativo
5.	Metalaxyl 8% + Mancozeb 64% WP	Downy mildew or disease caused by Oomycota fungi	Ridomil MZ

Antibiotics in Plant Disease Management

- **Antibiotics**

Antibiotic is defined as a chemical substance produced by one micro-organism which is low concentration can inhibit or even kill other micro-organism. They have systemic action in plants moving in both directions. They act as eradicant and protectants as well. Antibiotics are antibacterial and antifungal in nature. Some examples of antibiotics have been mentioned below:

I. Antibacterial Antibiotics

1. Streptomycin

Streptomycin is an antibacterial, antibiotic produced by *Streptomyces griseus*. This was the **first broad spectrum antibiotics** discovered by **SA Waksman and A Schatz**. Streptomycin are streptomycin sulphate is sold as Agrimycin,-100, Streptomycin sulphate, Plantomycin *etc*. This group of antibiotics act against a broad range of bacterial pathogens causing blights, wilt, rots diseases.

The primary mechanism of action of streptomycin is binding irreversibly to bacterial ribosomes and thereby inhibiting protein synthesis. At high concentrations, streptomycin can be phytotoxic to plants; thus it is applied to the surface of plants and not injected.

2. Tetracyclines

Antibiotics belonging to this group are produced by many species of *Streptomyces*. All these antibiotics are bacteriostatic, bactericidal and mycoplasmastatic. These are very

effective against seed-borne bacteria. This group of antibiotic is very effective in **managing MLO** (Phytoplasma) **diseases** of a wide range of crops.

Tetracyclines inhibit protein synthesis by binding reversibly to bacterial ribosomes. Tetracycline derivatives are the only antibiotics that are registered for internal use in plants. They are injected into the trunks of palm and elm trees to treat lethal yellows diseases caused by phytoplasmas.

II. Antifungal Antibiotics

1. Aureofungin

It is fungal antibiotic produced in sub-merged culture of *Streptoverticillium cinnamomeum* var. *terricola*. It is used for control of citrus gummosis, powdery mildew of apple, apple scab *etc.*

2. Griseofulvin

This antifungal antibiotic was first discovered to be produced by *Penicillium griseofulvum* and now by several species of *Penicillium*, viz., *P.patulum, P.nigricans.* It is commercially available as Griseofulvin, Fulvicin and Grisovin. It is highly toxic to powdery mildew of beans and roses, downy mildew of cucumber.

3. Cycloheximide

It is obtained as a by-product in streptomycin manufacture. It is produced by *Streptomyces griseus* and S. *nouresi.* It is commercially available as Actidione and Actispray. It is active against a wide range of fungi and yeast. Its use is limited because it is extremely phototoxic. It is also used against leaf spot, leaf blast of paddy etc.

4. Blasticdin

It is a product of *Streptomyces griseochromogenes* and specifically used against blast disease of rice. It is commercially sold as Bla-s.

5. Kasugamycin

It is obtained from *Streptomyces kasugaensis.* It is also very specific antibiotic against rice blast disease. It is commercially available as Kasumin.

6. Thiolution

It is produced by *Streptomyces albus* and effectively used to control late blight of potato and downy mildew of cruciferous vegetables.

7. Endomycin

It is a product of *Streptomyces endus* and effectively used against leaf rust of wheat and fruit rot of strawberry *(Botrytis cinerea).*

8. Bulbiformin

It is produced by a bacterium, *Bacillus subtills* and is very effectively used against wilt diseases, particularly red gram wilt.

9. Nystatin

It is also produced by *Streptomyces noursei.* It is successfully used against anthracnose disease of banana and beans. It is commercially marketed as Mycostain and Fungicidin.

10. Eurocidin

It is an antibiotic produced by *Streptomyces anandii*. It is effectively used against diseases caused by several species of *Colletotrichum* and *Helminthosporium.*

FUNGICIDE FORMULATION

Fungicides formulation is a process by which the fungicides is put into a form which can be easily produced, stored, transported and applied by practical methods in order to achieve a safe, convenient, economic and effective methods of disease control. It is a mixture of the active and inert ingredients in the fungicide. Pure chemicals are rarely used to control pathogens. They are usually combined with other inert materials that improve the physical and chemical properties of the product. Commercial fungicides are formulated in various ways *like as;*

1. Liquid Formulation

- **Emulsifiable concentrates (EC):** They contain a liquid active ingredient, one or more petroleum-based solvents and an agent that allows the product to be mixed with water to form an emulsion. e.g. Tridemorph 80% EC, Dinocap 48% EC, Hexaconazole 5% EC.
- **Solutions:** Solutions are formulations in which active ingredients readily dissolve when mixed with a solvent such as water or a petroleum-based solvent. Solutions can be used in any type of sprayer.
- **Suspension or Slurries:** These are formulations in which a dry form of the active ingredients is mixed with liquid along with an inert ingredient to form a suspension. These are generally used as a seed dresser in different seed processing companies.

2. Dry Formulations

- **Wettable powder:** They are dry, finely ground formulations that look like dusts. They are usually mixed with water for application as a spray. e.g. Mancozeb 75% WP, Copper oxychloride 50% WP, Carbendazim 50% WP, Chlorothalonil 75% WP.
- **Dust:** Ready to use formulations which usually contain 1-10% of active ingredient combined with a fine, dry inert carrier made from talc, chalk, clay, or volcanic ash. e.g. Sulphur dust.

- **Granules (Pellets):** Granules formulations are similar to dust formulations, except granular particles are larger and heavier. The granules contain normally 3-10% of the active ingredients. e.g. Carbofuran 3G, Phorate 10G.

Different Types of Pesticides Formulations

C = Concentrate	L = Liquid
CS: Capsule suspension	LC = Liquid concentrate
DP: Dustable Powder	LV = Low volatile
DS: Powder for Dry seed Treatment	P = Pellets
D = Dust	PA: Paste
DC: Dispersible concentrate	PS = Seed coated with Pesticides
EG: Emulsifiable Granules	RTU = Ready to use
EP: Emulsifiable Powder	S = Solution
ES: Emulsion for seed Treatment	SC: Suspension Concentrate
WP = Wettable powder	SE: Suspension Emulsion
WDG = Water-dispersible granules	SL: Soluble concentrate
WSP = Water-soluble powder	SP = Soluble powder
WSC = Water-soluble concentrate	WSL = Water-soluble liquid

METHODS OF FUNGICIDE APPLICATION

I. Foliar Application

- **Spraying**

Many inorganic or organic fungicides including systemic/ non-systemic and antibiotic are generally applied by spraying either with high volumes or with low volumes sprayers. The different equipments used for spray application are foot-operated sprayer, rocking sprayer, knapsack sprayer, motorized knapsack sprayer (power sprayer), tractor mounted sprayer, mist blower and aircraft or helicopter (aerial spray). The main objective of spraying is to provide protective covering over surface before arrival of the pathogen.

- **Dusting**

In soluble or non suspended materials are used for dusting on foliage. Dust formulations are dusted with the help of dusters like hand duster, bellows duster, rotary hand duster etc; e.g. Sulphur duster for Powdery mildew of coriander.

II. Seed Treatment

Seed tubers, bulb and other planting materials are given chemical treatment for eradication of pathogen present on them and for preventing in soil after planting.

Sometimes for seed borne disease, seed treatment is the only method of control. e.g. cover Smut of barley and grain smut of bajra.

(A) Dry Seed Treatment: Dressing of seed with dry fungicide. In this method, the fungicide adheres in a fine form on the surface of the seeds. A calculated quantity of fungicide is applied and mixed with seed either manually with hands or using machinery specially designed for the purpose. e.g. Captan, Thiram, Sulphur dust @ 2-3g /kg seed. It is very effective against externally seed borne pathogen causing disease.

(B) Wet Seed Treatment: This method involves preparing fungicide suspension in water and dipping the seeds or seedlings or propagative materials in it for a specified time. It is very effective against internally seed borne disease. Seed treatment is given by various compounds e.g. Organomercurial PCNB, Streptomycin, Thiram, Captan, carbendazin etc.

(C) Slurry Treatment (Seed Pelleting): In this method, chemical is applied in the form of a thin paste (active material is dissolved in small quantity of water) to the seed. The required quantity of the fungicide slurry is mixed with the specified quantity of the seed so that during the process of treatment slurry get deposited on the surface of seeds in the form of a thin paste which later dries up. Seed processing units have usually slurry treaters, which mix the fungicide slurry with specified quantity of seeds before the seed lot is bagged.

III. Soil Treatment

Soil treatment is given to reduce the amount of inoculum present in soil. This treatment is effective for soil borne pathogens. The fungicides are applied in soil in different forms by different methods:

(A) Soil Drenching

A suspension of chemical before sowing or after emergence of seedling is applied with the help of sprinkler or water can in a unit area. This method is mostly employed for the control of damping off disease, root rot or any soil borne pathogen at ground level e.g. Bordeaux mixture (BM) for foot rot of Papaya. Carbendazim for control of wilt in different cultivated crops.

(B) Broadcasting

In this method, fungicide is to be applied in a same way as in case of fertilizer application. In this methods, generally granular fungicides is utilised which is broadcasted nearer to the plant. By this method, more quantity of fungicide is required and hence it is not commonly employed. e.g. sulphur is applied at the rate of 25kg in Bajra and Sorghum.

(C) Furrow Method

Whenever the crop is planted in rows this method is employed. A required quantity of chemical to be applied in opened furrow in soil and mixed. Sow the seeds and irrigate the field. e. g. Carbofuran, Phorate

(D) Soil Fumigation

Application of certain chemicals to the soil can control fungi and nematodes. Such material usually produce a gas that distributes itself through the soil. This method is usually restricted to small areas and high value crops. Volatile toxicants (fumigants) such as methyl bromide, chloropicrin, formaldehyde and vapam are the best chemical sterilants for soil to kill fungi and nematodes as they penetrate the soil efficiently. Fumigations are normally done in nursery areas and in glass houses.

(E) Chemigation

The fungicides are directly mixed in the irrigation water. It is normally practiced when sprinkler or drip irrigation system is followed in a field or garden.

IV. Paste and Paint

In fruit orchard, the wound created during pruning and trimming of trees and often served as opening for entry of the pathogen. The fungicide paste or paints are used as protective layers. The paste is prepared with chemical like $CuSO_4$ + Lime, $CuSO_4$ + $CaCO_3$ etc. The residual effect is long lasting enough to permit natural healing of cut surface e.g. Control of Margo Dieback, Citrus Gummosis.

Difference between Spraying of fungicides and Dusting of fungicides

	Spraying of fungicides	**Dusting of fungicides**
1	Formulation used in liquid form either wettable powder (WP) or EC formulation	Formulation is used in powder form
2	Can be used in high wind velocity	Not used in high wind velocity
3	More area covered	Less area covered
4	Material needs preparation	Materials is ready to use
5	More effective	Less effective
6	Particle size biger	Particle size smaller
7	Sprayer is used	Duster is used
8	Water is required	Water not required
9	e.g. Mancozeb, Carbendazim	e.g. Sulphur dust

Auxiliary Spray Materials (ADJUVANTS)

(1) Wetting agents: They facilitate contact between spray and sprayed surface. They reduce the surface tension of the spray liquids and improve the surface of contact and help to spread. e.g. Alcohol, Ester of Fatty acid.

(2) Spreaders: They improve the contact between spray materials and plant surface. They help in the good coverage of the fungicide. e.g., Soap, Saponins, Mineral Oils, Glyceride Oil, Terpene Oil etc.

(3) Stickers: They are materials added to increase tenacity e.g. Fish Oil, Milk, Gelatin.

(4) Deflocculating agents: Materials added to keep particles away from each other to prevent flocculation to ensure dispersion and retard sedimentation eg. Gum, Gelatin and Milk Products.

(5) Safeners: A chemical which reduces the phytotoxicity of another chemical is called safener. Lime is added to copper sulphate in the preparation of Bordeaux mixture as a safener. Glyceride oils are also used as safener.

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Theory Note

B. Sc (Agril.)

Course No. Pl.Path.5.4

DISEASES OF FIELD AND HORTICULTURAL CROPS AND THEIR MANAGEMENT-I(CREDIT 2+1)

Semester-V

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Course Title: Diseases of Field and Horticultural Crops and Their Management- I

Course No. Pl. Path. 5.4 Course Credit: 2 + 1 = 3

SYLLABUS

Theory:

Economic importance, symptoms, etiology, epidemiology, disease cycle and integrated management of diseases of Groundnut, Sesamum, Castor, Cotton, Bajra, Finger millet, Sorghum, Maize, Rice, Pigeon pea, Soybean, Black gram, green gram, Tobacco, Coconut, Pomegranate, Tea, Coffee, Banana, Papaya, Tomato, Okra, Brinjal, Cluster bean, Beans and Colocasia.

Practical:

Identification and histopathological studies of selected diseases of field and horticultural crops covered in theory. Field visit for the diagnosis of field and horticultural crop diseases.Collection and preservation of plant diseased specimens for Herbarium.

Suggested readings:

1. Sanjeev Kumar (2016). Diseases of Field Crops and Their Integrated Management. New India Publishing Agency, New Delhi – 110 034
2. Shahid Ahamad and Udit Narain (2007). Ecofriendly management of plant diseases. Daya Publishing house, New Delhi – 110 035.
3. Rangaswami, G. and Mahadevan, A. (2008). Diseases of crop plants in India. PHI Learning Pvt. Ltd., New Delhi – 110 001.
4. Sanjeev Kumar (2015). Diseases of horticultural crops: Identification & management. New India Publishing Agency, New Delhi – 110 034.
5. Singh,R. S. (2018). Diseases of fruit crops. MEDTECH *A division of* Scientific International Pvt. Ltd. New Delhi – 110 002.
6. Sharma,I. M. and Gautam,H. R. (2017). About diseases of horticultural crops. Neoti Book Agency Pvt. Ltd., New Delhi – 110 002.
7. Gupta, S. K. Sharma, R. C. and Monica Sharma (2017). Disease of Vegetable Ornamental and Spice Crops. Scientific Publishers (India) P.O. Box. 91, Jodhpur – 342 001.
8. Singh R. S. (2014). Plant Diseases. OXFORD & IBH Publishing Co. Pvt. Ltd. New Delhi - 110 049
9. Singh, R. S. (2016). Diseases of vegetable crops. CBS Publishers & Distributors Pvt. Ltd. Delhi – 110 092.

10. Singh, R. S. (2017). Diseases of Fruit Crops. OXFORD & IBH Publishing Co. Pvt. Ltd. New Delhi - 110 049
11. Steven, T. Koike, Peter Gladders and Albert O. Paulus. (2017). Vegetable Diseases. CRC Press, Taylor & Francis Group, Boca Raton, FL.

Introduction:

Among various causal agent of plant disease, fungi were the first to be recognized. The contagious nature of wheat smut was shown by Prevost in 1807. From this basic report, many others reported fungal diseases of crops. But systematic investigation o the pathogenic nature of plant diseases goes to the great German scientist Anton de Berry (1831- 1888) who is considered as father of plant pathology. Of the 30,000 plant diseases recorded from different countries about 5,000 diseases are present in India.

There are no dependable survey of number of humans living on earth. The world population was 6.2 billion in 2000 and expected 7.1 billion by the year 2010. Currently it increased by 1 billion every 11 years. In which 50-80% of the population is engaged in agriculture. Even though 2 billion people suffer from hunger or malnutrition. To feed this people and the additional millions to come in next few years all possible methods of increasing world food supply are being pursued including crop protection i.e. disease management.

It is estimated that the total annual production for all agricultural crops world wide is about $ 12000-13000 billion. Of this, about $ 500 billion worth of produce is lost annually to diseases, insects and weeds. Approximately 26 $ billion

is spent annually for pesticides alone fungicides, insecticides and herbicides. In United States alone each year crop worth 9.1$ billion are lost to diseases.

Significance of plant diseases:

Plant diseases are important because they cause economic losses to growers. It may affects:

1. Plant diseases may limit the kinds of plants and industries in an area.
2. Plant diseases reduce the quantity and quality of plant produce.
3. Plant diseases may make plant poisonous to humans and animals.
4. Plant diseases may cause financial losses.

What is Plant Pathology?

Plant Pathology is the study of the microorganisms and of the environmental factors that cause disease in plants, of the mechanisms by which these factors induce disease in plants, and of the methods of preventing or controlling disease and reducing the damage it causes.

When we consider plant is healthy?

When plant carry out its physiological functions to the best of its genetical potential.

Functions:

- ✓ Photosynthesis

- ✓ Translocation - Apoplast and symplast
- ✓ Metabolism
- ✓ Storage of photosynthetic products
- ✓ Reproduction- seed, flowering and fruiting

Pathogenic microorganism that is the biotic agents generally referred as pathogens usually cause disease by

- ✓ Disturbing the metabolism of plant cell
- ✓ By production of enzymes, toxins, growth regulators and other secretions and there by absorbing food stuffs from host cell for their own life.

Microorganisms:

- ✓ Fungi (Eukaryotes)
- ✓ Bacteria (Prokaryotes)
- ✓ Mollicutes: RLO- Rickettsia like organism- Fastidious Vascular Bacteria causing stunting yellowing and proliferation. MLO – Phytoplasma , SLO
- ✓ Viruses: Mosaic, yellowing and leaf curl
- ✓ Viroid: Smallest entity have only infectious RNA and no protein.
- ✓ Nematodes
- ✓ Flowering plant parasite
- ✓ Protozoa
- ✓ Algae

Terminology

Plant Disease:

Any malfunctioning of host cells and tissues that results from continuous irritation by a pathogenic organisms or environmental factors that leads to development of symptoms.

Infectious disease:

Those diseases that results from infection of plant by a pathogen. In such disease pathogen can grow and multiply rapidly on diseased plants.

Symptoms:

The external and internal reactions or alteration of a plant as a result of disease.

Sign:

It is the characteristic of causal organism which will indicate the presence of causal organism.

Pathogen:

An entity that can cause the disease.

Parasite:

An organism living upon or in another living organism (the host) and obtaining its food from the invading host is called a parasite.

Parasitism:

Parasitism is a non-mutual relationship between organisms of different species where one organism, the parasite, benefits at the expense of the other, the host.

Host:

A living organism containing or invaded by a parasite, which obtains its nutrients from the organism. or a plant that supports the growth and development of the parasite that has infected it.

Infection:

The initiation and establishment of a parasite within a host plant.

Obligate parasite:

A parasite that can grow only on living organisms.

Propagule:

One unit of inoculum of any pathogen is called a propagule.

Inoculum:

The pathogen(s) that lands on or is otherwise brought into contact with the plant is called the inoculum.

Primary inoculum:

An inoculum that survives dormant in the winter or summer and causes the original infections in the spring or in the autumn is called a primary inoculum.

Primary infections:

The infections causes by primary inoculum is called primary infections.

Secondary inoculum:

An inoculum produced from primary infections is called a secondary inoculum.

Secondary infections:

The infections causes by secondary inoculum is called secondary infections.

A**lternate host:**

Plants not related to the main host of parasitic fungus, where it produces its different stages to complete one cycle (heteroecious).

A**lternative host:**

A plant other than the main host that a parasite can colonize; alternative hosts are not required for completion of the developmental cycle of the parasite.

Collateral host:

The wild host of same families of a pathogen is called as collateral host.

Epidemiology:

The study of the inter relationships between a given pathogen, the environment and groups or populations of the relevant hosts.

1. Pearlmillet/Bajra (*Pennisetum typhoides* L.)

Pearlmillet/Bajra is one of the common millets that serve as the poor man's food grain in India, Pakistan and some part of Africa. In India it covers an area of about 10 million hectares, distributed almost over the entire country. It comes up well in any kind of soil and for this reasons poor soils not fit for growing rice, wheat& Sorghum usually are used to grow. It is grow extensively in Tamil Nadu, Gujarat, Rajasthan, Madhya Pradesh and Uttar Pradesh. About twenty diseases of this crop are known, only a few are important.

1.Ergot

C.O. : *Claviceps fusiformis*

Subdivision : Ascomycotina

Order : Sphaeriales

Family : *Clavicipitaceae*

Occurrence & Economic importance:

- ✓ This disease is reported from many parts of Africa & India.
- ✓ In our country, it was first time reported from Satara area Maharashtra in 1956 in epiphytotic form. More severity is due to introduction of new hybrid HB-l, 2.
- ✓ It is also found in Delhi, Rajasthan, Karnataka, Tamil Nadu and Gujarat.
- ✓ During 1967-68 it has broken out in epiphytotic form affecting most of the newly introduced hybrid bajara varieties in all areas.
- ✓ Loss in hybrid varieties 58-76 %.
- ✓ This fungus produces ergotoxin/ergotin (alkaloid), which is poisonous to animals & human beings.

Symptoms:

- The disease occurs only at the time of flowering.
- Small droplets of a pinkish or light honey colored dew like substance exudes from infected spikelet. Later these droplets become darker, coalesce and cover larger area of the cob. A few to many such spikelets may be found in a group which darken with age, and small greyish or dark brown sclerotia are formed.
- These sclerotia replace the ovary or grain and are about 0.5-1 cm in length 1-2 mm in diameter, are hard and woody, during harvesting and threshing sclerotia get mixed with grains.
- The fungus attacks the ovary and grows profusely producing masses of hyphae to form the Sclerotium.
- Small conidiophores on which conidia are produced formed from the hyphae.
- The conidia are hyaline, one celled and lunate.
- The honey dew like droplets in the affected ears are full of conidia.
- The sclerotia germinate in about a month's time producing one or two stripes on which the asci are produced.
- This sclerotia contain ergotoxin which when consumed in excess quantities is toxic to animal life.

Favourable conditions:

- ✓ High humidity- spreads rapidly in field through secondary inoculum (60-90 % RH at morning followed by 45-50 % at evening).
- ✓ Rain at flowering-favours the disease.
- ✓ Cloudy weather, low sunshine during flowering, daily showers, moderate temp. (28-30^0 C), air movement during flowering favors the disease development.

Disease cycle:

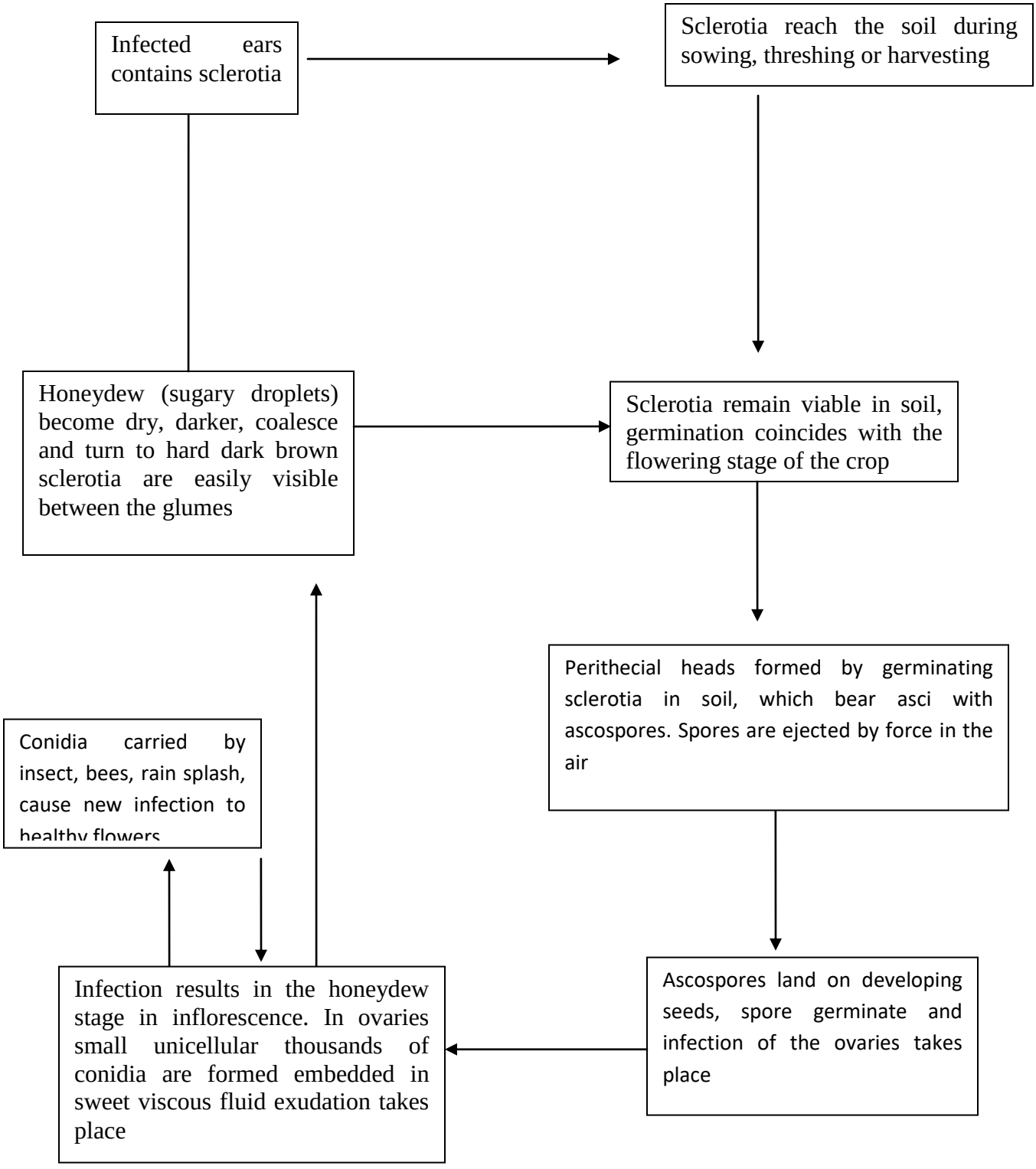
- ✓ The disease is soil and seed borne in nature. Primary source of infection - sclerotia left in the field during harvesting or mixed with seed serve as primary source of infection.
- ✓ Secondary source of infection -Dissemination begins with the appearance of honey dew, which contains numerous conidia of pathogen.

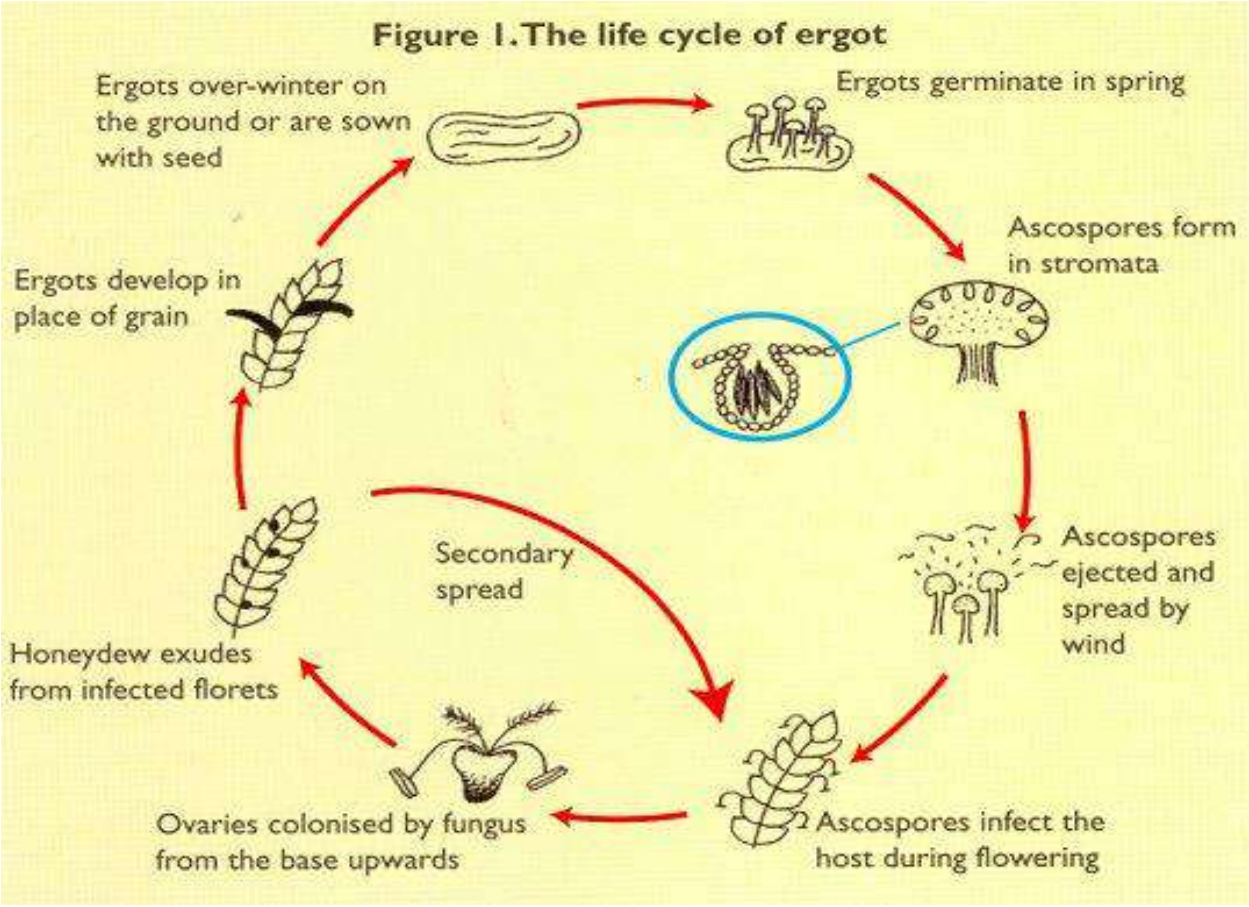
- ✓ These conidia disseminated by splashing rain, wind, insects and physical contact.

Management:

- ➢ Use certified, healthy and clean seeds.
- ➢ Seed soaking in 20% brine solution (NaCl) to separate out the sclerotia from seed.
- ➢ Spraying of cuman-L or ziram 0.2% @ 2 gram/ 1 liter or ziram + copper oxychloride 2-3 times at 5-7 days interval starting just prior to ear head emergence.
- ➢ Eradication of collateral hosts.
- ➢ Long crop rotation.
- ➢ Deep ploughing reduces sclerotial load.
- ➢ Timely sowing after onset of monsoon.

Disease cycle of Ergot of Bajra


Infected ears contains sclerotia
Sclerotia reach the soil during sowing, threshing or harvesting
Honeydew (sugary droplets) become dry, darker, coalesce and turn to hard dark brown sclerotia are easily visible between the glumes
Sclerotia remain viable in soil, germination coincides with the flowering stage of the crop
Perithecial heads formed by germinating sclerotia in soil, which bear asci with ascospores. Spores are ejected by force in the air
Conidia carried by insect, bees, rain splash, cause new infection to healthy flowers
Ascospores land on developing seeds, spore germinate and infection of the ovaries takes place
Infection results in the honeydew stage in inflorescence. In ovaries small unicellular thousands of conidia are formed embedded in sweet viscous fluid exudation takes place



Terminology:-

- ✓ **Epiphytotic or Epidemic: -**It is a sudden outbreak of a disease periodically over a widespread area in a devastatingly severe form causing severe losses or complete destruction. This is constantly present in a locality but it assumes severe form only on occasions. This is because of the occurrence of favorable environment responsible for the rapid development of disease.

- ✓ ***Perithecium*:-** These are flask shaped structures opening by a pore or *ostiole* (short papilla opening by a circular pore) through which the ascospores escape. The *ostiolar canal* may be lined by hair-like structures called *periphyses*.

2. Downy Mildew or Green Ear

C.O. :*Sclerospora graminicola*

Subdivision : Mastigomycotina

Class : Oomycetes

Order : Peronosporales

Family :*Peronosporaceae*

Occurrence & Economic importance:

- ✓ It occurs in many parts of Africa, and in India, **It was first reported by Butler in 1907 from India.**
- ✓ Losses due to the disease may be as high as 30 per cent in the high yielding varieties. During 1975, there was severe outbreak of downy mildew in Karnataka and Maharashtra causing100 % loss in thousands of hectares.
- ✓ It is locally known as "*Kutul*" disease

Symptoms:

- ➢ The infected plants become dwarf because of shortening of the internodes and excessive tillering.
- ➢ Foliage become pale and chlorotic and broad streaks are formed extending from the base to the tip of the leaf.
- ➢ On close examination, downy, dirty white fungal growth is seen on lower surface of the leaf during rainy & humid seasons.
- ➢ The leaves become distorted and later dry up leaf shreds due to formation of oospores.
- ➢ The principal symptoms of this disease are produced in the inflorescence.
- ➢ Badly infected plants fail to form ears or if formed they are malformed in to green leafy structures, hence it is known as "**Green ear disease**".

- Mostly the entire ear is transformed into leafy' structure, but sometimes a part of ear alone is affected, while other parts produce normal grains.
- As the disease advance, the green leafy structures become brown and dry.

Pathogen:

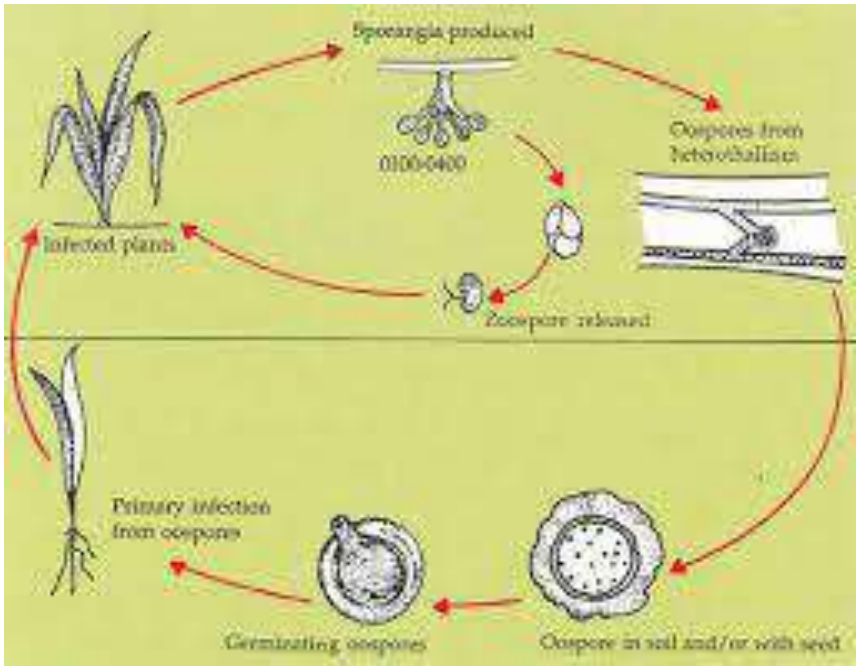
The fungus is an obligate parasite. It causes systemic infection in plant and send haustoria in plant cell.

Favorable condition:

- ✓ The disease is more in low lying and poorly drained soil.
- ✓ More humidity (90 %) and temp. between 22-25 ^{0}C, favours the disease.
- ✓ Presence of water on the leaves.

Disease cycle:

- ✓ Primary source of infection seed borne/soil-borne/plant debris. Oospores remain viable for 1 to 10 years. Primary infection takes place by direct penetration of root hairs and the coleoptiles by the germ tubes.
- ✓ Secondary spread starts form sporangia (zoospores), which are most active during the rainy seasons. The germ tubes of zoospores enter the leaf stomata to initiate the infection.



Management:

- Avoidance of low laying and water logging fields for sowing.
- Seed treatment with metalaxyl @ 2.0 g a.i./kg (apron SD 25 % @ 8.0 g/kg) or Metalaxyl-MZ @ 2 g a.i./kg (Ridomil MZ- 72 @ 8 g/kg) followed by two sprays of Metalaxyl- MZ 0.2 % at 25 and 35 days after sowing.
- Rough out infected plants carefully during thinning operation.
- Grow resistant variety GHB-351, GHB-316, NH-179, MH-169,GHB-30, GHB-32, GHB-526 (For Summer), GHB- 558, GHB- 744, GHB-732 and GHB 719.
- Sow the crop immediately after the onset of monsoon.

3. Smut

C.O. : *Tolyposporium penicillariae*

Subdivision : Basidiomycotina

Order : Ustilaginales

Family : *Ustilaginaceae*

Occurrence and Economic importance:

- ✓ This disease occurs in Pakistan, Africa and India.
- ✓ It is prevalent in Tamil Nadu, Andhra Pradesh and Maharashtra.
- ✓ It generally cause limited damage to the crop, the heavy incidence in certain years may cause considerable loss in grain yield.

Symptoms:

- ✓ The disease becomes apparent at the time of grain setting. The pathogen infects few florets and transforms them into large oval shaped sacs (sori) containing black powder (smut spores).
- Only individual grains irregularly distributed in an ear are infected.
- A few grains sporadically distributed or the ear, may be replaced by oval to top-shaped sori, which are generally two or three times the size of the normal grain.

- ✓ Initially the sori are larger and greener than normal healthy grains and when the sori mature they become dark brown and are easily broken and release millions of black smut spore balls.

Disease cycle:

- ✓ Primary inoculum consists of smut spore balls which are soil borne or is brought to soil as contaminant with the seed.
- ✓ These smut balls germinate in soil and produce sporidia which are disseminated by wind and reach individual florets where they cause infection.
- ✓ This infection is local and restricted to the individual flowers.
- ✓ The kernel is replaced by smut sorus.
- ✓ The spores that fall on the ground may germinate and produce sporidia which can caused secondary infection of flowers appearing late.
- ✓ It survives as spore balls in the **seed** and **soil** and serves as primary source of inoculum. The air borne spores germinate to produce the sporidia that enter the spikelets and infect the ovary.

Favorable conditions:

- ✓ High humidity.
- ✓ Monoculturing of the susceptible variety is favouring the disease.

Management:

- ➢ No effective measures available so resistant variety could be used for sowing.
- ➢ Immediate sowing after onset of monsoon. Early sowing have been found effective to reduce disease incidence.
- ➢ Deep ploughing during summer.
- ➢ Crop rotation
- ➢ Removal of infected plant from the field.
- ➢ Spraying of 0.1% vitavax at boot leaf stage to avoid the infection.

4. Rust:

C. O. : *Puccinia penniseti*

Subdivision : Basidiomycotina

Order : Uredinales

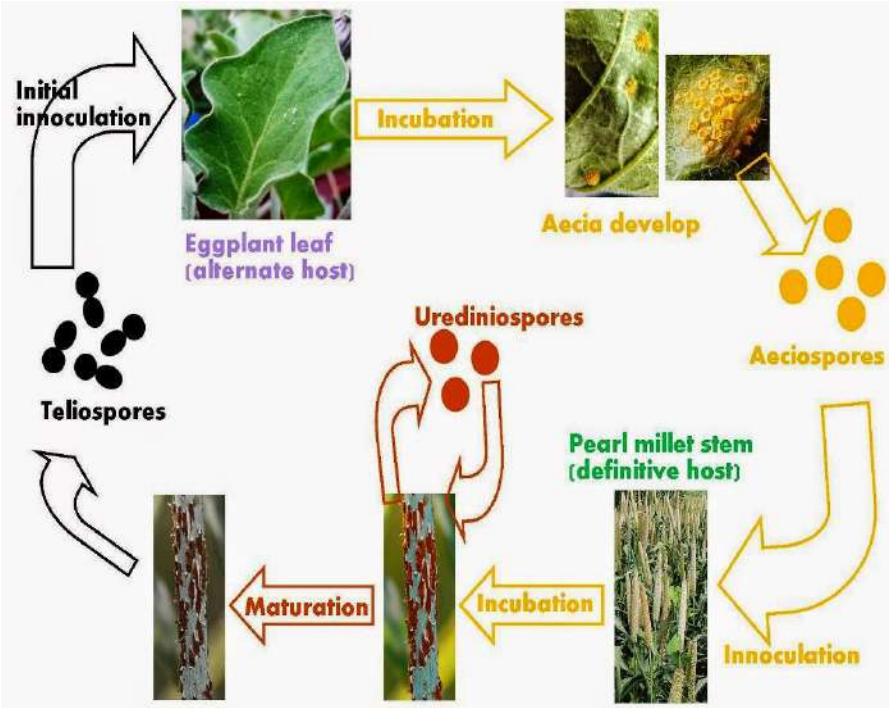
Family : *Pucciniaceae*

Occurrence & Economic Important:

- ✓ This disease observed in warmer parts of India.
- ✓ If infection is at early stage then there will be severe loss.
- ✓ It is found under dry and irrigated crop condition.

Symptoms:

- ➢ Symptoms first appear mostly on lower leaves as minute, round raised reddish brown pustules. The minute round uredosori occur in group on both surfaces of leaves, particularly towards the distal end of the blade, on the leaf sheath. The pustules may also be formed on stem and peduncles.
- ➢ The telia which appear later in the season are black elliptical and sub-epidermal, and are also distributed over the leaf blade, leaf sheath and stem.
- ➢ In severe infections, whole leaf may wither completely presenting a scorched appearance to the field. The infected plants appear slightly stunted compared with healthy ones.



Disease cycle of Rust of Bajra

Disease cycle:

- ✓ The rust is **heteroecious**. The fungus has a long life cycle producing uredial and telial stages on bajra and aecial and pycnial stages on several species of ***Solanum***, including **brinjal**. *(Solanum melongena)*. Uredospores are known as repeating spores and cause infection in the field.
- ✓ Primary infection is from the alternate host, brinjal, in nature. Secondary spread is through wind borne uredospores. The uredial stages also occur on several species of *Pennisetum.*

Management:

- ➢ Two sprays mancozeb or zineb @ 0.2% at 15 days interval starting from the initiation of disease.
- ➢ Destroy alternate host/grasses.

- Immediately sowing after onset of monsoon.
- Grow resistant varieties like RT 814-3, PT 826/4, PT 829/5.

…..x…..x…..x…..

2. Groundnut/Peanut (*Arachis hypogaea* L.)

Among all the state, Gujarat is a pre-dominating groundnut growing state. The crop is affected by several diseases causing large losses in both yield and quality of seeds. About more than 55 pathogens including viruses are affected the crop. However, only a few are economically important in India.

Major diseases	**Minor diseases**
(1) Early leaf spot	(7) Bud necrosis
(2) Late leaf spot	(8) Chlorosis (Nutritional Deficiency)
(3) Rust	(9) Afla rot
(4) Collar rot	
(5) Stem rot/ sclerotial rot/ pod rot	
(6) Root rot/ charcoal rot	

Leaf spot (Tikka / Early & Late) diseases

Occurrence:

- Tikka disease is the major diseases of groundnut in India.

- It occurs in groundnut growing areas of the world, including United States of America, most countries of Africa, the Philippines, Indonesia, Sri Lanka, China, Malaysia and Australia.
- All the groundnut verities grown in India are susceptible to this disease.

Economic Importance:

- Losses from 20 to 50 per cent have been reported.
- When tikka disease is associated with rust (*Puccina arachidis*) the loss may be up to 70 per cent.

(1) Early leaf spot:

C.O. : ***Cercospora arachidicola***

Subdivision : Deuteromycotina

Order : Moniliales

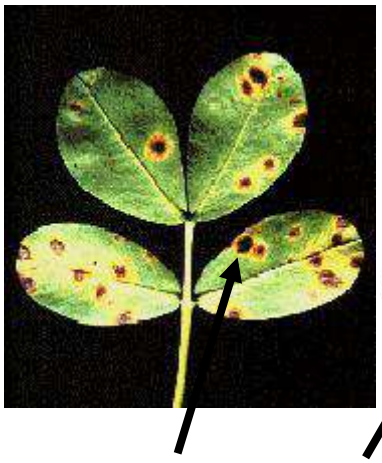
Class : Hyphomycetales

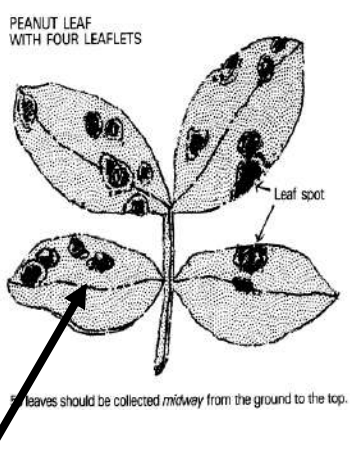
Family : *Dematiaceae*

Per. stage : ***Mycosphaerella arachidicola***

Symptoms:

- This spots are generally occurs early in the season i.e. 10 to 18 days after emergence.
- The early leaf spots caused by *C. arachidicola* are small circular to irregular, 1-10 mm in diameter but larger than late leaf spot.
- They are surrounded by yellow halo with dark brown center from the very beginning.
- On the upper surface, the necrotic area is reddish brown to black while on the lower surface it is light brown.
- The yellow halo is indistinct or not present on lower leaf surface.
- The fungus do not produce haustoria.
- The spores are produced mostly on the upper leaf surface.





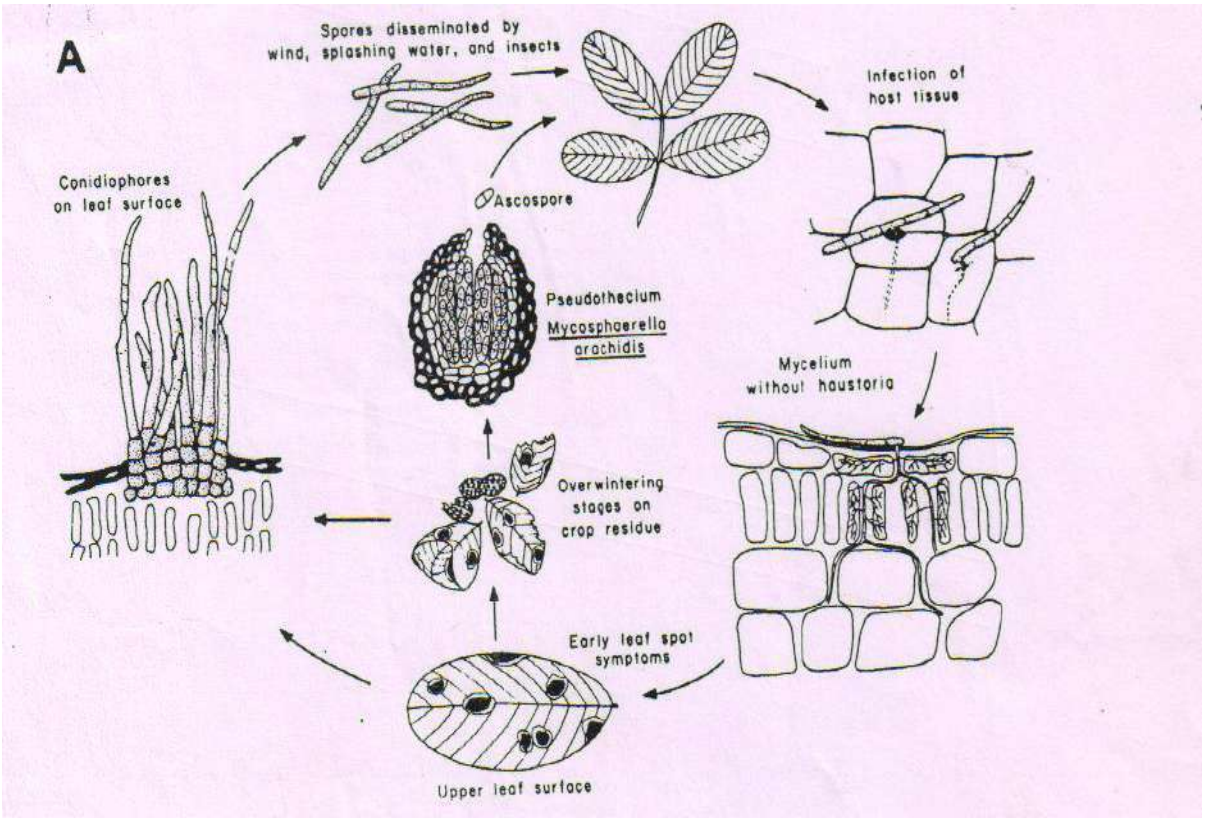
Reddish brown to black spot on leaves along with yellow halo

early leaf spot sketch

yellow halo with dark brown center

Disease cycle:

- ✓ The groundnut leaf spot fungi perpetuate through conidia lying in the soil or diseased plant debris and through conidia carried on the shell of the nuts.
- ✓ **The primary source** of infection seed borne as well as conidia present in plant debris in soil.
- ✓ **Secondary spreads** through airborne conidia.



Disease cycle of early leaf spot disease of Groundnut

(2) Late leaf spot:

C.O. : *Cercosporidium personatum* (**Revised : *Phaeoisariopsis personata***)

Sub-div. : Deuteromycotina

Order : Moniliales

Class : Hyphomycetales

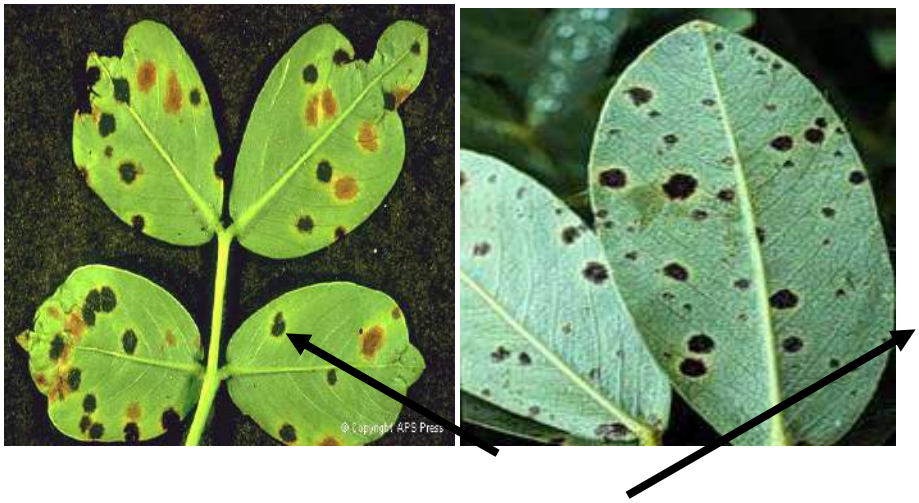
Family : *Dematiaceae*

Perfect stage: ***Mycosphaerella berkeleyii***

Symptoms:

- This disease comes late in season.

- *C. personatum* produces smaller, more circular spots which are 1-6 mm in diameter and dark brown to black in color.
- These brown spots **are not surrounded by yellow halo** in early stages.
- The lower surface of the spots is carbon black in colour.
- Pathogen produced conidia are restricted to the lower surface.
- Severely infected leaves may drop off prematurely.
- Minimum after 1 month of sowing and becomes severe in between flowering & harvesting stage under favorable climatic condition.
- Late spot develop 2-4 weeks later than the early spots.
- This disease is more damaging, causing spots and spread faster and cause early defoliation.



Circular spots with dark brown to black in color on leaves of groundnut

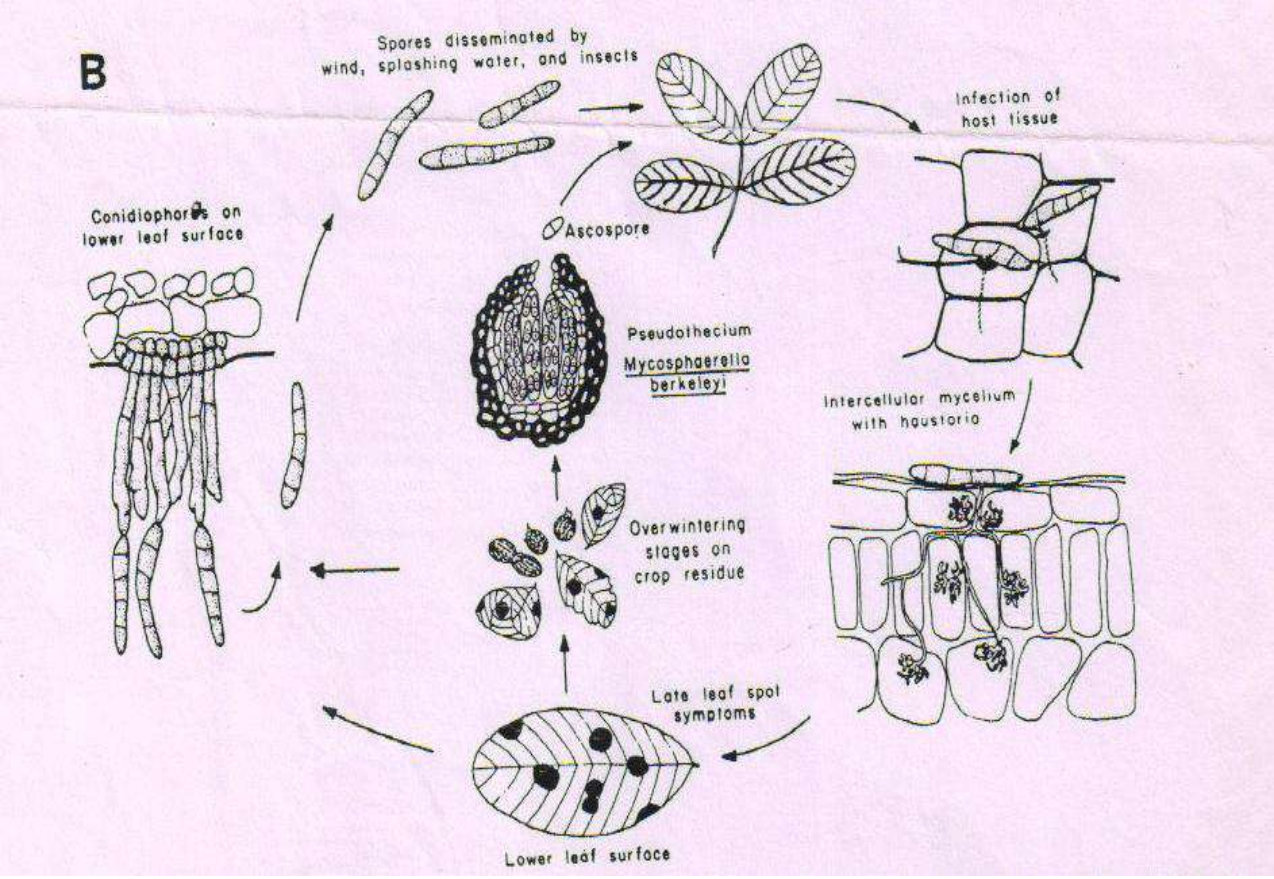
Favourable conditions:

- ✓ Relative humidity is the most important factor for infection. Constant 3 days of high humidity is essential for maximum infection. The disease intensity is maximum at the end of August or in September due to high humidity period.
- ✓ Prolonged low temperature and dew also favors the disease.
- ✓ More application of N_2, P_2O_5 increase disease intensity, while K_2O decreases slightly.

- ✓ Deficiency of Magnesium in plant either directly or indirectly increases the susceptibility of plants to disease.

Disease cycle:

- ✓ The groundnut leaf spot fungi perpetuate through conidia lying in the soil or diseased plant debris and through conidia carried on the shell of the nuts.
- ✓ **The primary source** of infection seed borne as well as conidia present in plant debris in soil.
- ✓ **Secondary spreads** through airborne conidia.



Disease cycle of late leaf spot disease of Groundnut

Management:

- Cultural practices promote vigorous growth, which helps to minimize infection.
- Destroy volunteer (self sown) plants in early stages.
- Plant debris from the previous crop should be burnt to avoid soil borne primary inoculum.
- Rotation and deep burying of the debris may also help to destroy, the soil borne inoculum.
- Seed treatment is essential to eliminate seed borne inoculum. Seed treatment with thiram or captan @ 3 to 4 g / kg seed.
- Alteration in the date of sowing can help in avoiding the damage by the disease.
- Early maturing varieties usually escape the losses caused by the disease.
- Select resistant variety.
- In chemical control- spraying of carbendazim 0.05 % @ (5 gram in 10 liter water) twice or 3 spray of chlorothalonil 0.2 % + mancozeb 0.2 % @ (25 gram in 10 liter water) or hexaconazole 0.005 % @ (5 ml in 10 liter water) at 10-15 days interval for the control of both leaf spots.

(3) Rust:

Groundnut rust occurs in central and south America, West Indies, Venezuela, China, USSR, USA, and India. **In India first time reported in 1971** in Punjab, Tamil Nadu, West Bengal, Andhra Pradesh and in Gujarat. It is locally known as **Geru**.

C.O. **: *Puccinia arachidis***

Sub-div: Basidiomycotina

Order : Uredinales

Family : *Pucciniaceae*

Economic importance:

- ✓ In southern parts of India, the loss in yield due to rust alone has been reported in the range of 10 to 29 %.
- ✓ Capital loss in pod yield due to rust alone in India has been reported in the range of 14 to 52 %.
- ✓ The loss in oil content 7 to 10 %.

Symptoms:

- ➢ The disease is usually found when the plants are over 6 weeks old.
- ➢ Small brown to chestnut brown dusty pustules occur on the lower surface of leaves.
- ➢ The epidermis ruptures and exposes a powdery mass of uredospores.
- ➢ Corresponding to the sori, small, brown, necrotic spots appear on the upper surface of leaves.
- ➢ The pustules are 0.3 to 2.0 mm in diameter and usually surrounded by a yellow halo.
- ➢ In severe infection lower leaves wither and drop prematurely.
- ➢ The kernels formed in the affected plants are shriveled and small.

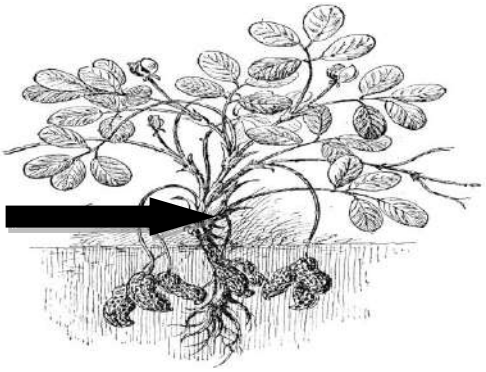
Small, brown, necrotic spots on the upper surface of infected groundnut leaf

Favourable condition:

- ✓ Intermittent rain.
- ✓ High relative humidity.
- ✓ 20 to 26^0 C temp.

Disease cycle:

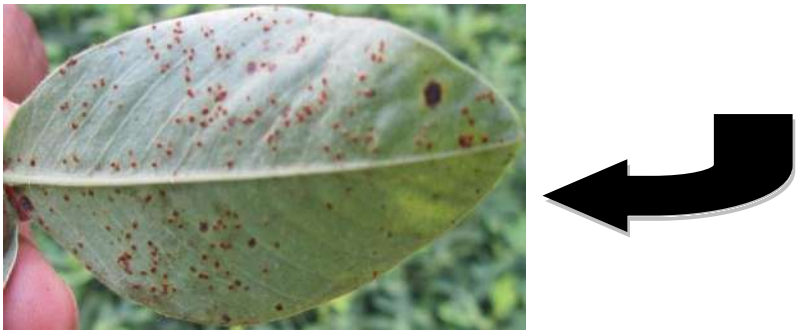
- ✓ There is no report of existence of collateral/ alternate host however due to continuous cultivation of groundnut there is overlapping of harvesting and sowing of the crop.
- ✓ Uredospores of ground nut rust remain viable for about 20-30 days and hence they do not find any difficulty in carrying themselves from one crop season to other crop season.
- ✓ The fungus survive in uredial stage in south and central India on ground nut plant. Diseased plant debris play important role.
- ✓ Wind borne uredospores play role as secondary spread of the disease.



Uredospore on the leaves

Uredospore survive on Groundnut plant or on infected plant debris

Wind borne Uredospore cause secondary infection



Infected Leaf contain uredospore

Disease cycle of Groundnut Rust

Management:

- Destroy the diseased plant debris by burning.
- Sowing of the crop should be done in first week of June.
- Three sprays (40, 55 and 70 days of germination) of tridemorph @ 0.04 % or mancozeb @ 0.2 %
- For the control of rust & tikka (both mix infection) 3 spraying of mixture of carbendazim 0.025 % and mancozeb 0.2 % at 30, 42 & 54 days after sowing OR Alternative spraying of 0.2 % mancozeb OR 0.025 % carbendazim & 0.2 % mancozeb OR three spray of 0.2 % chlorothalonil at 30, 50 and 70 days after germination.
- Removal of volunteer groundnut plants/ ground keepers.

(4) Collar rot / Crown rot and Seeding blight:

Collar rot was first reported from Saurashtra. It was then reported to occur in Australia, East Africa, Fiji, Island, India, Iran, Israel, Sudan and the US. This disease is most common in Saurash1ra region of Gujarat.

C.O. : ***Aspergillus niger***

Sub-div. : Ascomycotina

Class : Plectomycetes

Order : Eurotiales

Family : *Eurotiaceace*

Economic Importance:

- ✓ As the disease causes considerable seedling mortality in the initial stage of crop growth.
- ✓ Crop yield is directly affected by reductions in the plant stand of the crop.
- ✓ In India under Punjab condition, the losses may about to 40-50 % in term of mortality of plants.

Symptoms:

- ➢ The disease appears in two phases *viz* pre-emergence and post-emergence.
- ➢ In the pre-emergence phase, the seeds may rot and become covered with sooty black mass of spores (seed rot).
- ➢ On germination the emerging hypocotyls is rapidly killed by lesion below ground, resulting in rotting of the seedling before their emergence from the soil (pre- emergence). In the post-emergence phase initially a circular light brown lesion appears on the cotyledons and as the time advances the hypocotyls tissue or stem region becomes water-soaked and shows light brown discoloration.
- ➢ The seedlings then collapse and die due to rotting of succulent hypocotyls.
- ➢ As disease advances collar region becomes shredded and dark browns.
- ➢ Mature plants may also attack at ground level due to collar rot there is girdling of the collar region and the leaves become chlorotic.
- ➢ This is followed by drooping, wilting and death of' the affected branches.
- ➢ The affected collar region becomes shredded & soon covered with profuse growth of conidiophores and conidia.

Black mass of spore on cotyledon

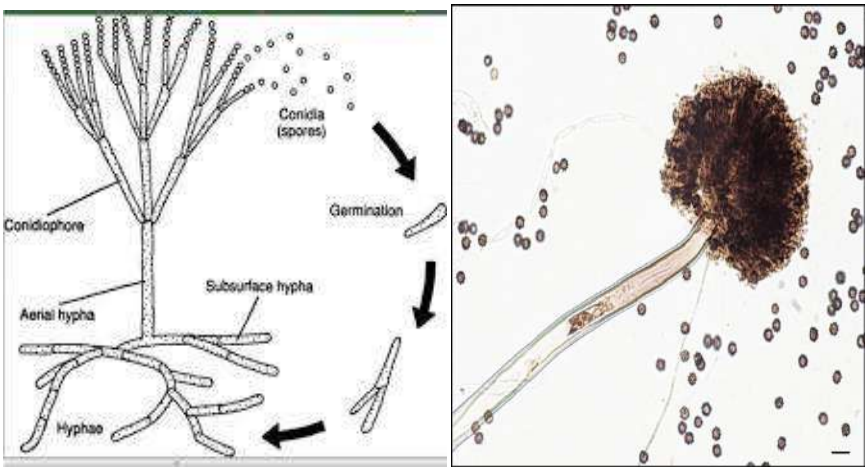
Collar region becomes shredded & covered conidiophores and conidia of *Aspergillus niger*

Favorable conditions:

- ✓ High soil and air temperature predispose the plants to infection.
- ✓ Maximum seed rot occurs at 15^0 C and 40^0 C.
- ✓ Temp. 30-35^0 C favors the disease.
- ✓ Monoculturing increase the inoculum in soil.
- ✓ Infection occurs from seed borne as well as soil-borne inoculums of the fungus.
- ✓ Seed is infected if stored in damp condition.
- ✓ Deep sowing, late germination, heavy soil and high air temperature favors the disease.

Disease cycle:

- ✓ Primary source of infection: Seed-contaminated with spores, soil -debris in the soil
- ✓ Secondary source of infection: Conidia dissemination through irrigation/rain water and farm implements.



Spore (Conidia) and Conidiophore of *Aspergillus niger* under Microscopic field

Management:

- Seed dressing with organomercurials. In Gujarat- treat seeds with thiram 3 g/kg or captan 4 g/ kg seed.
- Use healthy and high quality seed for sowing. Avoid to damage seed for sowing.
- Application of castor cake or neem cake 500 kg/ha at the time of sowing.
- Resistance variety runner type more susceptible than Spanish type.
- Avoid deep planting.
- Deep ploughing during summer.
- Crop rotation with chick pea and wheat.
- After harvesting of groundnut, seed should be dry in sunlight than store it in dry place.

(5) Stem rot / Sclerotial rot / pod rot

The disease occurs thought groundnut growing areas of the world in the tropic and in warmer parts of the temperate zone. This is the most serious disease in Saurashtra due to monoculturing of the crop.

- **Economic importance**: About 27% or more yield loss due to the disease has been reported from India.

C.O. : *Sclerotium rolfsii*

Sub-div : Deuteromycotina

Order : Moniliales

Family : Mycelia sterilia

Per. stage : *Athelia rolfsii*

Symptoms:

- The Pathogen attacks on all parts of groundnut but stem infection is most common and serious hence the commonly name 'Stem rot' is given.
- First symptom observed as sudden wilting of branches which come in contact with the soil.
- Usually the disease appears more frequently as the plant approaches maturity.
- Infection may take place on the stem just above the soil surface or at the foot of the plant.
- In the beginning a deep brown lesion around the main stem at soil level is observed
- Soon after, the lesion become covered with white mycelium which encircles the affected portion of the stem.
- Black sclerotia appear gradually on infected areas.
- Entire plant or branches dries, Pod formation is poor causing infection to pegs. On pods orange colored lesion are observed. Shriveling and rotting of kernels takes place.
- Roots are also affected showing white fungal growth (Root rot).

White mycelium on infected stem and sclerotia on infected areas

Favorable conditions:

- ✓ Monoculturing have been found to induce severe occurrence of the disease. Frequent rains/irrigation favors the disease.
- ✓ Moderate to high temperature (28 to 30^0 C) and moist condition. In sandy or sandy-loam well aerated soil, disease development take place more, rapidly & severely than heavy or poorly aerated soil.
- ✓ High P_2O_5, gypsum, undecomposed FYM increases disease.

Disease cycle:

- ✓ Primary source of inoculum: soil bone/ seed borne/sclerotia present in soil. Sclerotia remain viable for **8 years in** soil.
- ✓ Secondary source of infection: Conidia dissemination through irrigation water /rain & farm equipment.

Management:

- ➢ Deep ploughing during the summer.
- ➢ Crop rotation- maize- wheat- sorghum.
- ➢ Use healthy seed (without damage).
- ➢ Use well decomposed FYM.
- ➢ Remove the infected plant from the field.

- Give the seed treatment of thiram or Mancozeb @ 3-4 g/1 kg of seed before sowing.
- Crop sanitation-burning of crop residues, deep ploughing.
- Bio-control agents-In Gujarat application of *T. harzianum* mixed with well decomposed FYM or castor cake @ 1.5 kg in 300 kg can be applied in furrow just before sowing.
- Control moisture: Practices that reduce moisture retention in the canopy.
- In groundnut due to tikka disease, leaves earlier defoliated and this fungus utilized it as a food. So proper precaution should be taken for the control of leaf spot of groundnut.

(6) Root rot/ charcoal rot

Causal organism: *Macrophomina phaseolina*

Symptoms

- In the early stages of infection, reddish brown lesion appears on the stem just above the soil level.
- The leaves and branches show drooping, leading to death of the whole plant. The decaying stems are covered by whitish mycelial growth.
- The death of the plant results in shredding of bark. The rotten tissues contain large number of black or dark brown, thick walled sclerotia.
- When infection spreads to underground roots, the sclerotia are formed externally as well as internally in the rotten tissue. Pod infection leads to blackening of the shells and sclerotia can be seen inside the shells.

Favourable Conditions

High soil temperature, prolonged drought.

Disease cycle

- The fungus remains dormant as sclerotia for a long period in the soil and in infected plant debris.
- The primary infection is through **soil-borne and seed-borne sclerotia.**
- The secondary spread of **sclerotia is aided by irrigation water, human agency, implements, cattle etc.**

Management

- Deep ploughing during summer.
- **Give sufficient irrigation as and when required.**
- **Pod should not be injured at the time of harvesting.**
- Treat the seeds with **thiram 4g or Carbendazim 2g/kg.**
- Spot drench with carbendazim at 0.5 g/lit.

Viral Diseases of Groundnut:

1. Chlorotic rosette 2. Green rosette 3. Bud necrosis,

4. Mosaic 5. Bunchy plant 6. Rugose leaf curl 7. Peanut mottle

Loss in yield from the mosaic disease may range from 25-100 per cent.

(7) Bud necrosis:

C.O.: **Tomato Spotted Wilt Virus (TSWV)**

Tomato spotted **wilt virus was first seen in India in 1964**.

- ✓ The bud necrosis disease (BND) of ground nut is now considered to be one of the most damaging disease of the crop in India.
- ✓ It is widely distributed in the major growing region of India.
- ✓ Disease incidence has been observed in parts of Maharashtra, Gujarat, Rajasthan & Western UP.

Economic Importance:

- ✓ Under Indian condition the incidence of the disease appears to more in the wet crop season (*Kharif*) than in summer & winter crop seasons.
- ✓ The incidence of the disease ranges from 5 to 80% in different parts of India. Yield losses from Australia reported to the tune of 50 to 90%.

Symptoms:

- ➢ Chlorotic speckling of ring spot on the terminal leaf (about 30-40 days after planting) followed by necrosis of the terminal but.
- ➢ The buds get killed, which later dries and becomes brown.
- ➢ The necrosis may proceed down ward and the whole branch become blighted.
- ➢ Necrosis may also be seen on the petioles
- ➢ Proliferation of axillary shoots takes place but the leaves of such shoots remain smaller than normal and show a wide range of symptoms including distoration, mosaic, mottling and general chlorosis.
- ➢ Affected plants remain stunted & show bushy appearance.

On infected plant proliferated of axillary shoots and Chlorosis and mosaic on leaves	**Transmitted by thrips**

Disease Transmission:- Two types of Thrips, *Frankliniella schultzei* and *Scirtothrips dorsalis* have been found to transmit the virus.

Management:

- Bud necrosis of groundnut is primarily associated with infestation by thrips during the month of August, September, January & February, adjustment of sowing dates may reduce disease incidence means early sowing of the crop.
- Intercropping groundnut with pearl millet, sorghum, maize shows lesser incidence of disease.
- Control the alternative weed hosts.
- Application of systemic insecticides to control the thrips.
- Plant population per unit, area should be increased.

(8) Chlorosis (Iron deficiency)

Symptoms:

- Plants grown on soils with high pH (7.6 to 8.3 pH) frequently shows iron deficiency symptoms.
- In Gujarat, highly calcarious soil results in chlorosis of groundnut.
- Young leaves turn yellow & then white, and the plant become dwarf.
- Under such induced iron deficiency, chlorotic symptom appears more rapidly under low light intensity.
- The chlorosis of leaves without any mottling is the most prominent symptom.

Management:

- A solution of 100 g ferrous sulphate and 10 g. citric acid in 10 litre of water sprayed at 10 days interval for 2-3 times.
- Chelated iron 2 % sprayed at 10 days interval for 2-3 times.
- Spray 40 g.$FeSO_4$ + 20 g. lime + 10 lit. of water.

(9) Afla Rot:

C. O.: ***Aspergillus flavus***

Economic importance:

In England, thousands of turkey poults died with Turkey X – disease caused by eating moldy peanuts infected with Aspergillus flavus. A toxic fungal metabolite aflatoxin is the causative agent in this disease. Aflatoxin B1 (AFB1) is toxic to many animals, was discovered to be a strong hepatocarcinogen for rats, trouts (fish) and human beings. Krishnamachari et al (1975) reported an outbreak of hepatitis in human beings and dogs caused by consumption of toxin contaminated maize in Rajasthan and Gujarat in 1974.

Peanuts and their products, cotton seeds and copra are the most favourable substrates and are called “high aflatoxin risk materials”. Aflatoxins are

produced by *Aspergillus flavus*, *A. parasiticus* and *Penicillium puberulum*. In human, chronic AFB_1 has been linked to liver cancer, but in some cases, it has been found associated with lung, kidney and colon tumers. The bacterium *Flavobacterium* removes aflatoxin completely from contaminated milk, peanut, peanut oil, butter and corn.

Occurance:

It is prevalent in almost all groundnut growing states. The yellow mold fungus is commonly found in the seed of both rotten and apparently healthy pods of groundnut.

Symptoms:

- It first appears on cotyledons after the emergence of seedlings. Infected plants generally become stunted and leaf lamina is drastically reduced with a pointed tip. Vein clearing and chlorosis of the leaflets is also visible. Infected seedlings lack a secondary root system a condition known as aflaroot.
- Such plants do not produce flowers and hence become unproductive.
- Soil borne inoculum is the primary source of infection.
- The pathogen can tolerate low soil moisture and the fungus develops best at temperature between 25 and 35 º C.

Epidemiology:

- The extent of yellow mould damage and aflatoxin production is dependent on the environmental conditions and production, harvesting and storage practices.
- The pathogen is seedborne and soilborne, and active in high humidity (90-98%) and low soil moisture.
- Temperatures conducive to growth are 17-42°C with aflatoxin production between 25-35°C.

Management

- Since the fungus is a weak parasite, agronomic practices which favour **rapid germination and vigorous growth** of seedling will reduce the chance of *A. flavus* infection.
- Seed treatment with carbendazim @ 2g/kg seed or captafol @ 3 g/kg seed or thiram @ 3-5 g/kg seed has given good results.

.....x.....x.....x.....

3. SORGHUM / GREAT MILLET / JOWAR (*Sorghum vulgare*)

Jowar is an important food and fodder crop of India. There are seven smut diseases of jowar, four of which occur in India.

(1) Grain smut/ Covered Smut/ Kernel Smut/ Short Smut

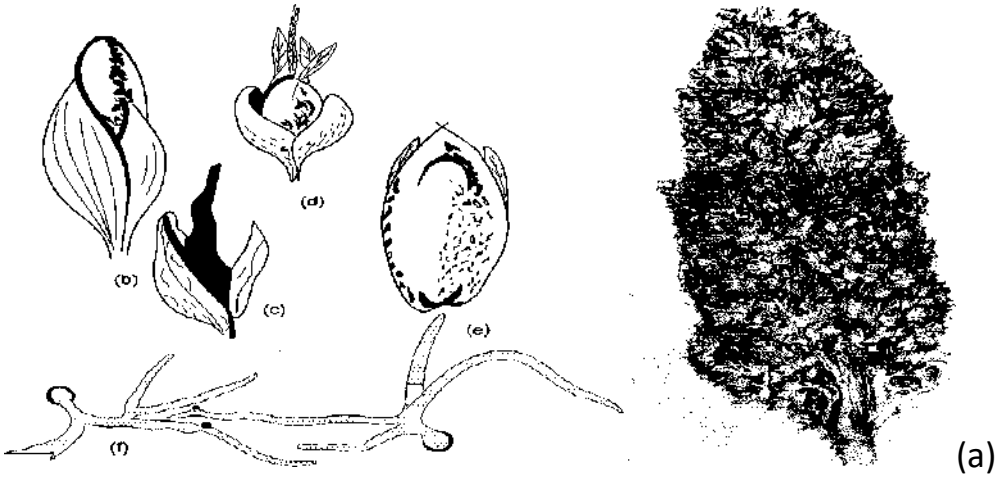
It is the most common destructive of all the smuts, causing extensive damage to grain yield all over India. It is found in rainfed as well as irrigated sorghum. In certain areas it is reported to cause damage up to 25% incidence is recorded.

Local Known as **:** Dana no angariyo (Aanjiyo) or Anavrut angariyo (Aanjiyo)

C.O. **:***Sphacelotheca sorghi* (Link)

Symptoms:

- The disease becomes apparent only at the time of grain formation in the ear at which time smut sori are formed in the place of healthy grains.
- The size of the sori varies with crop variety, but generally they are larger than the normal grain.
- The **individual grains** are replaced by **smut sori** which can be localized at a particular part of the head or occur over the entire inflorescence. The sori are dirty white to gray in color, oval or cylindrical and are covered with a tough white cream to light brown skin (**Peridium**) which often persists unbroken up to threshing.
- Most of the grains of an infected ear are replaced by the smut sori, but in some cases only a few grains may be smutted. Ratoon crops exhibit higher incidence of disease.



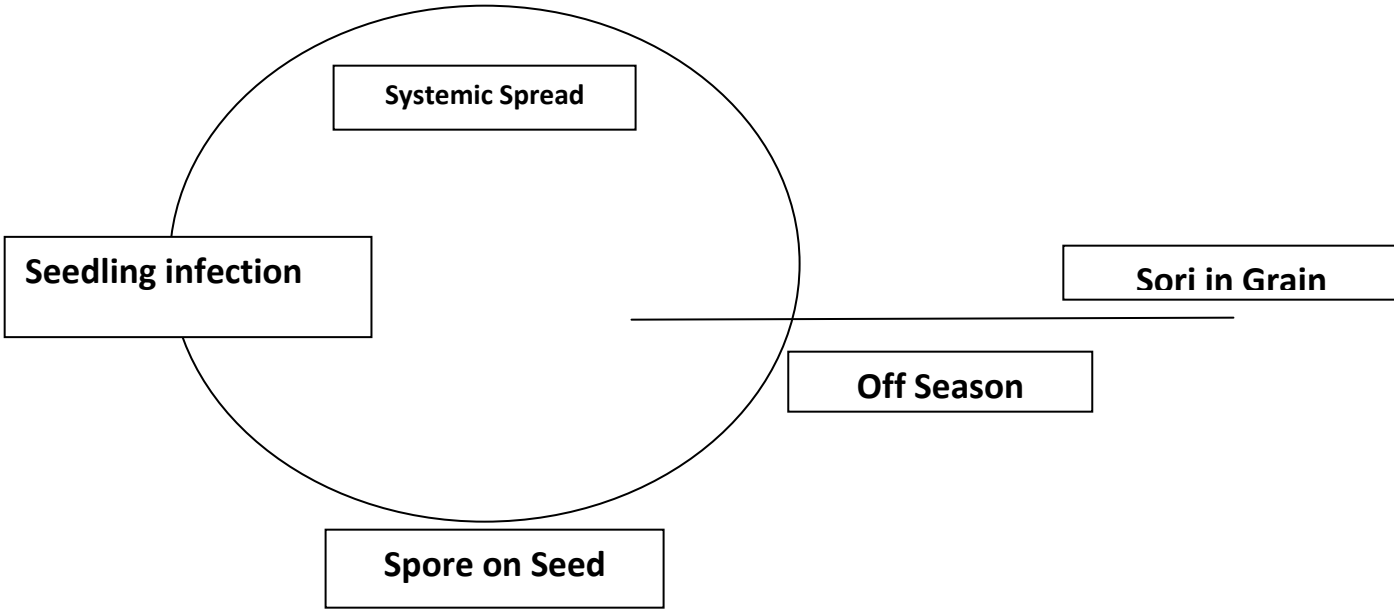
(a)

Grain smut of jowar (Sphacelotheca sorghl):(a) symptoms (b) a sorus magnified (c) columella (d) sorus not involving stamens (e) sorus with stamens involved (f) germinating spores.

Diseases cycle:

- ✓ The disease is **externally seed borne** and **systemic**. The spores are attached on the surface of seeds.
- ✓ They germinate with the seed and infect the seedlings by penetrating through radicle or mesocotyl region to establish systemic infections.

- ✓ At the time of flowering the fungal hyphae get converted into spores replacing the ovary with the smut sori.
- ✓ If diseased ears are harvested with healthy ones and threshed together the healthy grains become contaminated with smut spore released from the bursting of sori.
- ✓ The spores remain dormant on the seed until sown next season.
- ✓ The smut spores remain viable indefinitely, depending on how they are stored. In some cases they may be viable even after 10 years.



Disease cycle of Grain smut/ Covered Smut/ Kernel Smut/ Short Smut

Favourable conditions:

- ✓ Infection occurs best on slow germinating seeds.
- ✓ Optimum temperature for disease development is 20 to 30 ^{0}C.
- ✓ High temperature following sowing less infection than did moderate temp.

Management:

- ➢ Since the spores are seed borne externally, seed treatment with sulphur @ 4 g / kg seed or organomercurials (MEMC) @ 2 g/ kg of seeds.
- ➢ Use resistance variety, GJ-38, GJ-39, GJ-40, GJ-4, CSH- 5, CSH- 6, CSH-1, CSH-9, CSH-7.
- ➢ Use clean seeds free from smut sori.

- Solar energy treatment (Asthana, 1947) soaking of seeds in water at ordinary temperature during summer for 4 hours in the morning and then spreading in the sun to dry for better disease control.
- Formalin wash to seed- immersion of seeds in 0.5 % for 2 hours and then dry it.
- Seed soaking in copper sulphate @ 0.5 to 3.0 % solution for 10-15 min. and then dry it and use for sowing.
- Field sanitation.

(2) Loose Smut

In India it occurs in Tamil Nadu, Karnataka, Andhra Pradesh, Maharashtra and Gujarat.

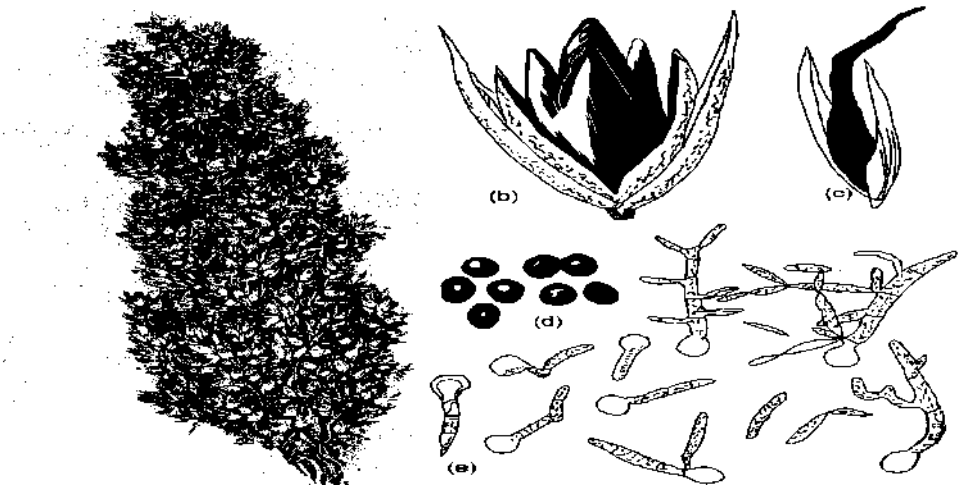
Local name : Anavrut anjario

C. O. :*Sphacelotheca cruenta* (Kuhn) Potter

Longer, bigger, more curved than grain smut columellum is present externally seed borne.

Symptoms:

- The affected plants can be detected before the ears come out. The affected plants are stunted, produces thinner stalks, more tillers and earlier flowering than the healthy plants.
- All the spikelets (glumes) of the infected ear are malformed and hypertrophied. The sorus replaces the pistil and stamens.
- The size of the sori ranges from 3-18 mm in length and 2-4 mm in thickness. The affected ear appears like a leafy or leathery structure.



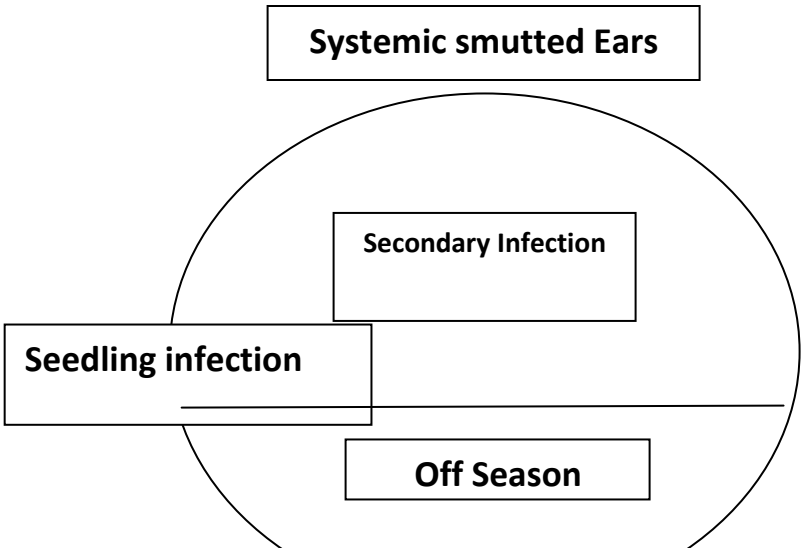
Loose smut of jowar (Sphacelotheca cruenta): (a) smutted ear (b) Sorus involving stamens (c) Exposed columella after the spores have fallen (d & e) spores and their germination

Disease cycle:

- ✓ Primary source of infection: Externally seed borne.
- ✓ It is soil borne to some extent in dry soil.
- ✓ The fungus enters the seedlings through the radicle mesocotyle or epicotyle and then become systemic causing smuting of the ovaries at the time of earhead formation.
- ✓ Secondary spread: By wind borne conidia.

Favourable condition:

- ✓ Temp. 30^0 C is favorable for the disease development.
- ✓ Soil pH- 3 to 9



Spore on Seed and soil

Disease cycle of Loose smut of sorghum

Management:

- Seed treatment as per grain smut.
- Crop rotation.
- Field sanitation.
- Resistance variety GJ-38, GJ-39, GJ-40, GJ-41.

(3) Long Smut

This smut is sporadic and has been reported from some parts of Africa, Iraq, Egypt and Pakistan and in India it occurs in Tamil Nadu, Karnataka, Andhra Pradesh, Maharashtra, Madhya Pradesh and Uttar Pradesh. Since disease affects only a few grains in an ear the damage caused is not significant.

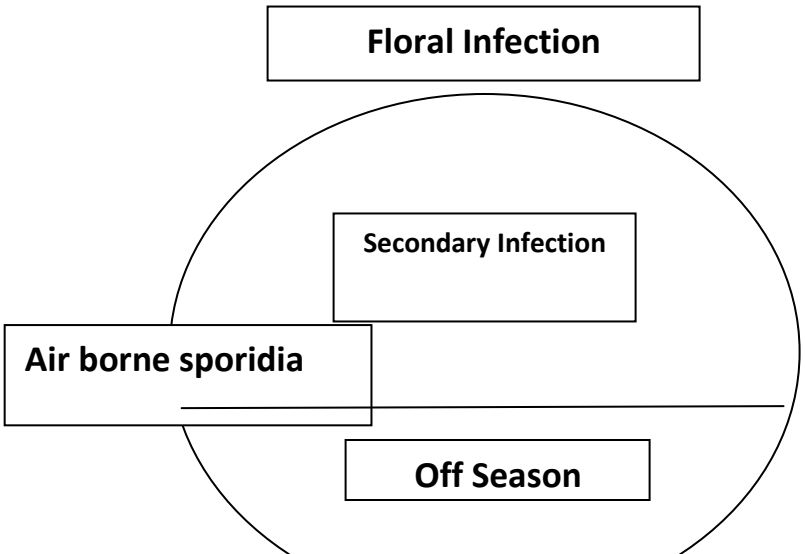
C.O. :*Tolyposporium ehrenbergii* (Khun) Potter

Symptoms:

	➢ Only close examination of the ear in the field reveals the presence of long smut. ➢ Usually a few smut sori are scattered sporadically through the ear; rarely are most of the grains in an ear smutted. ➢ The sorus is covered by a whitish to dull yellow fairly thick membrane and is much longer than those of the other two smuts. ➢ It is cylindrical measuring 4 cm. in length and 6-8 mm. in width. ➢ Usually the membrane remains intact until broken mechanically.

Disease cycle:

- ✓ It is believed that floral infection occurs by the means of sporidia which enter the floral parts, producing sori about 12 to 15 days later.
- ✓ Primary source of infection: The spore in the soil may germinate to produce cluster of sporidia which become air borne and cause primary infection.
- ✓ Secondary infection take place through the spores released from the smutted ears in the field.



Sori in Ears

Sporeball in soil

Disease cycle of Long smut of sorghum

Management:

- Early sowing reduces the incidence of the disease.
- Crop rotation.
- Field sanitation to keep down the buildup of inoculum.
- Resistance variety GJ-38, GJ-39, GJ-40, GJ-41.

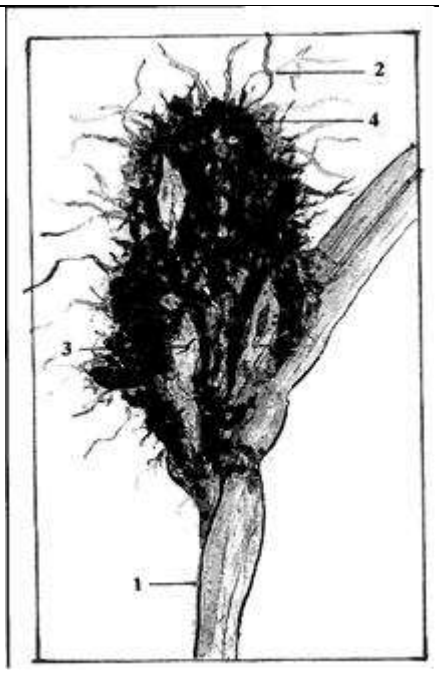
(4) Head Smut

The disease is reported from many countries and in India it is reported from Tamil Nadu, Karnataka, Andhra Pradesh, Maharashtra, Madhya Pradesh, Uttar Pradesh and Punjab. The disease cause significant damage to sorghum as well as maize, as it affects entire ear, transforming it into a smutted head.

Locally known as: Dunda no angariyo

C.O.: ***Sporisorium relianum*** **(Old name:-** ***Sphacelotheca reiliana)***

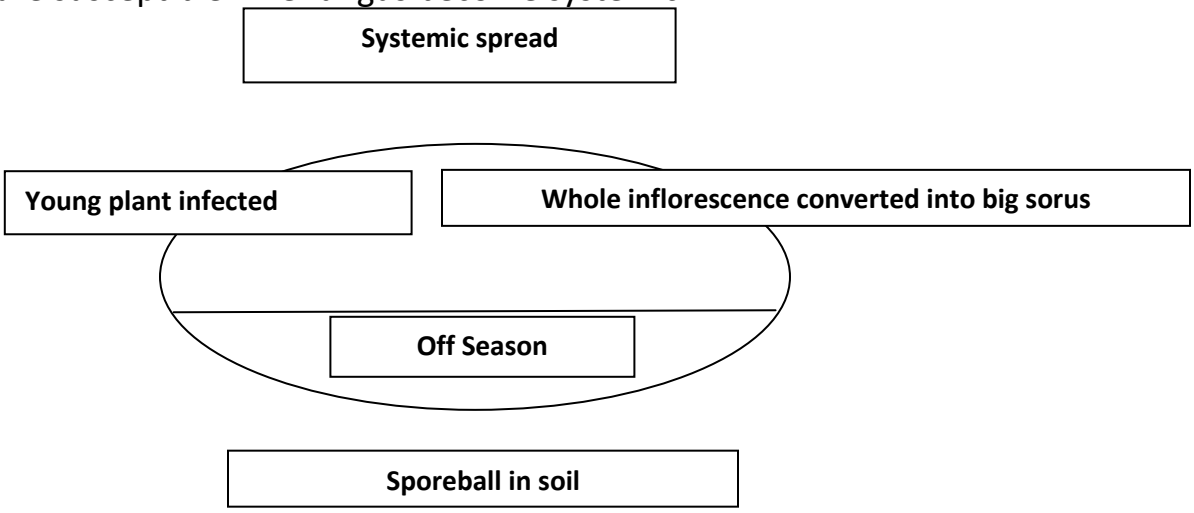
Symptoms:



- The disease becomes apparent only at the time of flowering.
- The entire inflorescences is converted in to a big sorus, a sorus fully covered with a grayish white membrane emerges from the boot leaf.
- The cylindrical sours usually 8-10 cm. long and 2.5-5.0 cm. wide.
- When it is fully emerged the fungal membrane rupture, exposing large mass of black powdery spores, if the wind blowing at this time, air borne spores resemble a smoky cloud around the head.
- When blown, only filamentous vascular tissues of the host spore is exposed.

Disease cycle:

Soil as well as seed borne. Spore present in the soil cause infection. Only young plants are susceptible. The fungus become systemic.



Disease cycle of Head smut of sorghum

Management:

- Seed treatment as per grain smut.

- Collection of smutted head in cloth bags and dipping in boiling water to kill the pathogen will reduce the inoculum potential for the following crop. If this is done over several years, the disease may be checked considerably.

Comparison of characters of four smuts of sorghum

Character	Grain smut	Loose smut	Long smut	Head smut
Organism	*S. sorghi*	*S. cruenta*	*T. ehrenbergii*	*S. reiliana*
Host	Stunted, heading premature	Not- Stunted, heading normal	Not- Stunted, heading normal	Not- Stunted, heading premature
Ear infection	All or most grains smutted	All or most grains smutted	Only about 2% of grains are infected	The entire inflorescence is converted into a big sorus
Site	Ovary	Ovary	Ovary	Inflorescence
Mode of infection	Systemic	Systemic	Non systemic	Systemic
Sori	Small 5-15 X 3-5 mm	Small 3-18X 2-4 mm	Long 4 X 6-8 mm	Very large 7.5-10 X 2.5-5 cm
Membrane	Rather tough and persist	Rapture easily	Relatively thick membrane	Rapture easily
Columella	Short columella present	Long columella present	Coluella absent, but 8-10 vascular strands present	Columella absent but a network of vascular tissues present
Spores	In single round to oval, olive-brown, smooth walled 5-9 M in diameter	In singles spherical or elliptical, dark-brown, spore walls pitted, 5-10 M in diameter	Always in spore balls, globose, brownish green warty spore wall, 12-16 M in diameter	Loosely bound into balls, spherical or angular, dull brown, minutely 10-16 M in diameter
Viability	Over 10 years	About 4 years	About 2 years	Up to 2 years

In culture	Yeast-like growth with sporidia	In colonies with sporidia and resting spores 40 X 50 M in diameter	In colonies with masses of sporidia	In colonies germ tubes and sporidia
Spread	Externally seed-borne	Externally seed-borne	Air-borne	Soil-borne and seed-borne

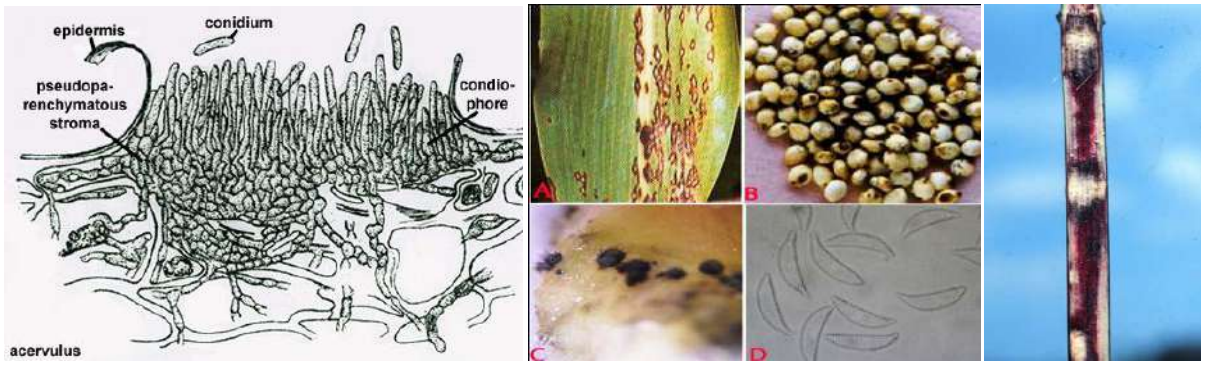
(5) Anthracnose

C.O: *Colletotrichum sorghi*

Symptoms:

- The fungus causes both leaf spot (anthracnose) and stalk rot (red rot) in sorghum.
- The disease appears as small red coloured spots on both surfaces of the leaf.
- The center of the spot is white in colour encircled by red, purple or brown margin.
- Numerous small black dots are seen on the white surface of the lesions which are the fruiting bodies (acervuli) of the fungus. Depending upon severity of infection a few to twenty or more spots may be seen on a single leaf.
- Many lesions coalesce and kill large leaf portions.
- In midrib region, elongate elliptical, red or purple regions with black acervuli are formed.
- The infection is found mostly on the basal and older leaves, the young leaves usually being free from infection.
- The infection may spread to the stem, causing reddish streaks. The affected young seedlings shows blighting.
- Stalk and inflorescence infection can be characterized externally by the development of circular cankers.

- Infected stem when split open shows discoloration, which may be continuous over a large area or more generally discontinuous giving the stem a marbled appearance. The stem lesion also shows acervuli.



Favourable conditions:

Continuous rain, Temp. 28-30^0 C, High humidity

Disease Cycle:

Fungus has wide host range and survives on **Johnson grass, Sudan grass, maize, barley** and **wheat**. Also survives in seed and infected plant debris. Primary infection is from the conidia produced on the infected plant debris and infected seed. Disease spread within the season is through air borne conidia, which are produced on first infected plants.

Management:

- Seed treatment with captan or thiram @ 4 g/kg seeds.
- Three sprayings of carbendazim @ 0.025 % beginning from initiation of disease at an interval of 15 days.
- Destruction of infected plant debris and collateral hosts.
- Crop rotation with non-host crops.

(6) Striga / Witch weed (Partial / Semi root parasite)

Scientific names :*Striga parasitica; Striga densiflora; Striga asiatica*

Family : Scrophulariaceae

Genus : Striga

Hosts: Corn, Sugarcane, Cereals, Tobacco, Millets, Paddy etc.

- Obligate parasites. Striga plants usually develop in clusters around each stool of sorghum/ sugarcane. The seeds are very minute and are produced in great abundance.
- The seeds remain viable for number of years.
- The stimulus provided by the root exudates of specific hosts helps the seeds to germinate.
- After germination the parasite grows below the soil surface for about 1-2 months and produce underground stems and roots.
- The root has haustoria, which penetrate the host root, drawing nourishment from the host and destroying the host.
- For complete life cycle 90-120 days required and resting period is 15 days.

Favourable conditions:

- ✓ Heavy loam soil is favorable.
- ✓ Temperature is also favouring the weed growth and development.

Management:

- Avoid soil contamination. The seeds are disseminated through equipment. It should be washed before use.
- Avoid water contaminations from infested to healthy field.
- Grow trap crop- non-host crop e.g. legume crops due to this- starvation and death may occur.Four to six hand weedings.
- Grow trap crops before sowing the main crop.
- Chemical control: copper sulphate solution 2-3% in upper 4-inch soil. Spraying 2-4-D @ 2% found effective (fernoxone) or 1% tetrachloro dimethyl phenoxyacetic acid. Flooding the field before sowing the main crop.
- Use resistant/ tolerant variety. Follow crop rotation. Use clean, striga free seeds for sowing.

…..x……x…..x…..

4. Castor (*Ricinus communis* L.)

(1) Fusarium Wilt

- **C.O** : *Fusarium oxysporium* f.sp. *ricini* (Nanda and Prasad)
- **Sub division** **:** Deuterormycotina
- **Order** **:** Moniliales
- **Family** **:** Tubercullariaceae
  - ✓ In India, the incidence of disease has been observed to the extent of 20 % on variety Gujarat Hybrid-3.
  - ✓ Losses cause up to 80 %.

Symptoms:

- ***Fusarium*** **wilt can appear in two forms:** A quick/short from or a protracted (to delay) form of the disease.
- Quick form seen in young plants during period from sprouting to formation of two to three real leaves. In diseased plants the color of the hypocotyls gets a dull tinge and apex leaves lose their turgor, within two to three days leaves wilt and dry out without any change in their green color. In some plants wilting is proceeded by the appearance of spots or blight.
- In later phase of growth, the disease develops up to harvesting. The first symptom of disease appears on individual plant or in a small circle of plants, which increase and spread all over the field.
- Infected plant appears as yellow sickly and marginal necrosis of leaves, which later advances to inter veinal areas and covers the leaves completely.

Castor wilt

- Leaves shrivel, lower ones drop away leaving few top leaves. Finally resulting in development of irreversible wilting of the affected plants.
- In later stage, individual branches or the entire plants acquire dark violets tinges and during moist weather luxuriant mycelium of the fungus develop near the stem neck.
- Infected stem show intracellular mycelium in vessels and hypertrophy *of* xylem parenchymatous cells.
- Formation of tyloses is seen in root tissues of affected plants which cause necrosis of pith. Sick plants either do not bear capsules or give poor seed. Partial wilting is observed commonly.

Favourable conditions:

- ✓ Disease appears in the month of October-November when the plants are 3-4 months old and more prominent in February-March.
- ✓ For infection temp. 13-15^{0}C
- ✓ For symptoms expression temp. 22-25^{0}C

Disease cycle:

- ✓ Causal agent is seed borne as well as soil borne.
- ✓ **Primary source** **:** Chlamydospore, Infected plant debris
- ✓ **Secondary spread :** Macro and micro conidia
- ✓ The fungus enters the young host roots through incidental wounds and injury caused by insects and eelworms.
- ✓ After getting entry inside the host the fungus multiplied and moves to the xylem vessels, where the mycelium is formed in abundance to partially or completely plug the vessels, interrupting the transport of nutrients to the above ground plant parts.
- ✓ The fungus induces production of tyloses, which when present in abundance in xylem vessels, prevent movement of nutrient.
- ✓ Fungus also produces toxic substance *i.e* fusaric acid which is responsible for wilting and retard translocation of water nutrients.
- ✓ Primary infection is soil borne. Pathogen remains viable in stubbles of disease plants and seeds.

- ✓ Secondary spread through water, irrigation rain and soil contamination by implements.
- ✓ The association of fungus with nematodes the disease is more severe. The funguses can survive in soil as saprophyte for many years. It's chlamydospore act as a resting spores.

Management:

- ➢ Crop rotation with non-host crop for long duration. Green manuring.
- ➢ Deep ploughing during summer.
- ➢ Seed treatment with carbendazim 3g/kg seeds or thiram or captan 3-4 g/kg seeds at the time of sowing.
- ➢ Application of organic manures i.e. FYM, neemcake, castor cake@ 5 t /ha.
- ➢ Antagonistic fungi- *Trichoderma* spp. @ 2.5 kg mix in 500 kg/ha.well decomposed FYM or neem cake or castor cake than apply in furrow at the time of sowing or seed treatment @ 8 g/kg seeds.
- ➢ Growing of resistant varieties like as**GCH-7**.

(2) Root Rot

Wilting / rotting up to 40% have been reported affecting plant stand and yield. Root rot is a devastating disease of castor. Dry climate prevailing during cropping season plays a very significant role on the occurrences and development of the disease.

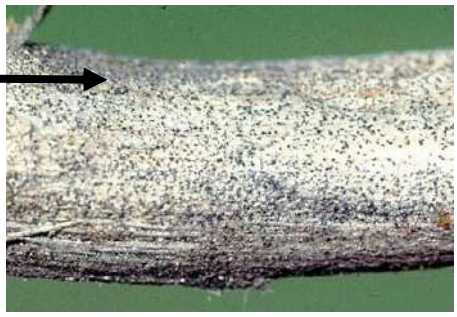
- ❖ **Causal organism :** *Macrophomina phaseolina* **(Pycnidial stage)**
- ❖ **Sub division :** Deuterormycotina
- ❖ **Order :** Agonomycetes (*Mycelia sterilia*)
- ❖ *Thanetophorus cucumeris* **(Perfect stage)**
- ❖ *Rhizoctonia bataticola***(Sclerotial stage)**

Symptoms:

- ➢ This disease appears at different growth stage of the crop. Hence name given variously as spike blight, stem blight, twig blight, collar rot and root rot.
- ➢ The symptoms of root rot appear after 45 days of planting. The primary infection starts as

small brown depressed lesion at collar region which later on become sunken and spread upward and downward on the stem and plant girdle at this point causing wilt and leaf drops.

- Pith region shows brown discoloration due to formation of sclerotia. The discoloration extends to the root system (root rot), the bark shreds and several minutes dots like black structure (sclerotia) are seen on decayed root system.
- The entire plant dries up and gets easily pulled up, leaving behind roots in soil. ***Pycnida are produced on the outer surface of the stem and capsules.***
- The tip of the terminal buds get discolored, blackish and gradually dries up from tip to downward (twig blight). In case of spike blight, disease start in a few capsules, get discolored turn black and drying up of capsules in course of time.



Favorable conditions:

- ✓ Soil temperature 30-35^{0}C
- ✓ Soil moisture 15-20%

Disease cycle:

- ✓ Disease is soil borne and sclerotia provide primary source of inoculum.
- ✓ Scleroria of the fungus can survive free or embedded in host tissues in soil. Survival is longer in dead tissue than in free state in the soil. Dry soils prolong survival of these sclerotia.
- ✓ In wet soil sclerotia cannot survive for more than 7-8 weeks. While the mycelium cannot survive for more than 7 days.
- ✓ The fungus is a poor competitor in soil but readily colonizes plant debris. Populations of the fungus in soil increase when hosts are grown continuously in the same field and the disease becomes more severe in the succeeding crops.
- ✓ Sclerotia germinate and produced numerous germ tubes, penetration in the root occurs by appressoria formed over the epidermal cells or natural openings.

- ✓ The fungus hyphae grow inter and intra cellularly through xylem where they formed sclerotia and plug the vessels, Pathogenesis of the pathogen probably involves mechanical plugging of vessels by sclerotia, toxins production and enzymatic action.

Management:

- ✓ **Removal and destruction** of infected plants from the field.
- ✓ Adjustment in sowing date when soil moisture is at proper level.
- ✓ Crop rotation or mixed cropping with sorghum, urid, mung.
- ✓ Summer deep ploughing, green manuring.
- ✓ In stress condition give light irrigation in short duration to decrease the soil temperature.
- ✓ Application of organic manure, neem cake, mustard cake helps in reduction of inoculum.
- ✓ Soil application of *Trichoderma harzianum* with oil cakes (neem, mustard, castor) before sowing.
- ✓ Seed treatment with **thiram or captan @ 3 g/kg seeds.**
- ✓ Antagonistic fungi- ***Trichoderma* spp. @ 2.5 kg / 500 kg of neem cake**
- ✓ **Apply need base irrigation** to the crop under stress condition.
- ✓ Drenching the plant near soil base with **copper oxychloride @ 40 g/10 liter** of water to minimize inoculum.

…..x…..x…..x…..

5. Cotton (*Gossypium herbaceum* L.)

About 20 diseases have been reported on cotton in India. It is roughly estimated the total annual loss in cotton yield due to various diseases is about ₹ 1200 million.

1. Wilt

Cotton plant suffer from two destructive wilt disease one caused by *Fusarium* and the other by *Verticillium dahlia* (*V. albo-atrum*) which is not common in India. In India *Fusarium* wilt was first reported from Nagpur by Evans in 1908.

C. O. : *Fusarium oxysporum* f. sp. *vasinfectum*

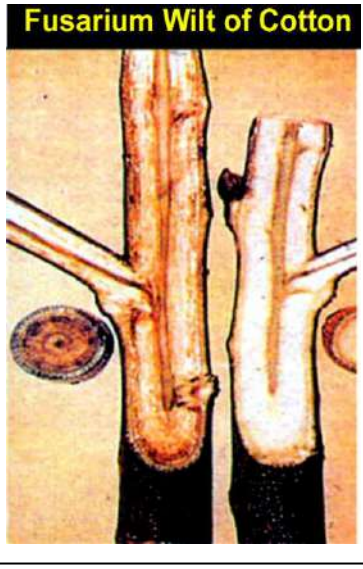
Sub-div : Deuteromycotina

Order : Moniliales

Family : *Tubercullariaceae*

Symptoms:

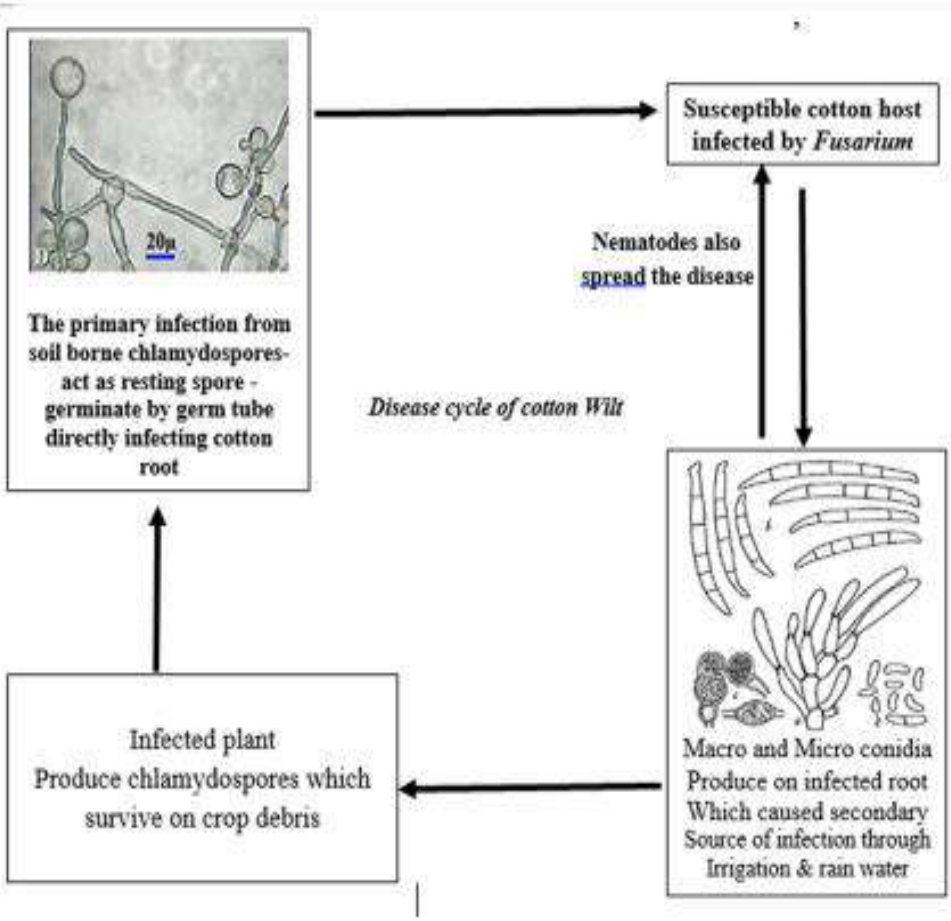
- The disease affect plants at all stages of growth.
- The first symptom on young seeding.
- Vein clearing of the leaves, followed by necrosis of interveinal tissue.
- There is yellowing & browning of the cotyledons, and formation of a **brown ring on the petiole**.
- The seedling soon wilts & dies.
- If plants are affected little later, the leaves become turgid, resulting in drooping and wilting, starting from the lower leaves at the base and proceeding upward, finally involving the branches and whole plants.
- Often there may be complete defoliation, leaving the stem alone standing in the field. Discoloration of the stem is also seen.
- In young plants the basal portion of the stem turns black.



- Some times partlia1 wilting confined to one side of the stem observed.
- Growth is retarded & the plant eventually wilts. Affected plants remain stunted with smaller leaves and bolls.
- **The vascular system of basal stem becomes brown and filled up with a gummy substance consisting of fungal hyphae and conidia**.

Disease cycle:

- ✓ The fungus is soil borne and seed borne and weak saprophyte. Fungus produce toxic substance i.e. fusaric acid which is responsible for wilting. Pectinolytic enzyme produced by the fungus cause breakdown of the cell wall components. The degradation product add to vascular system which retard translocation of water & nutrients.
- ✓ The primary infection is soil borne chlamydospores.
- ✓ Secondary spread through macro and micro conidia with irrigation & rain water, soil contamination by implements. The association of fungus with, nematodes the disease is more severe.



Disease cycle of cotton Wilt

Favorable conditions:

- ✓ The disease is favored by soil temperature 20°-30^{0}C, the optimum being 24-28^{0}C.
- ✓ Hot and dry periods followed by rain seems to favor the disease.
- ✓ **Zn deficient soil**, low potash & high acidic and heavy black soil favors the disease.

Management:

- ➢ Crop rotation with millet, sorghum, ground nut.
- ➢ Field sanitation and mixed cropping with Mothbean, Urdbean are useful for reducing the incidence of wilt in cotton.
- ➢ Use delinted seeds.

- Use resistant variety Jayadhar, Jarila, Vijay, Pratap, Verun, BDS. In Gujarat Digvijay, Gujarat cotton- 17, Gujarat cotton- 23 and G.Cot.-13 are resistant to wilt of cotton.
- Amendment with Zinc with balanced use of NPK.
- Soil solarization in combination with fumigation with vapam reduces the inoculum in soil.
- Organic amendment. Use only well decomposed manure.
- Deep ploughing during summer.
- Irrigation at short intervals
- Green manuring
- Soil application of *Trichoderma* spp. before sowing along with organic manures.

Comparison of Wilt & Root rot of cotton

Sr. No.	**Wilt**	**Root rot**
1	*Fusariumoxysporum*f. sp.*vasinfectum*	*Rhizoctonia bataticola*
2	Heavy black cotton soil	Sandy soil favorable
3	Infected plant can not be easily pulled out.	Infected plants can be easily pulled out.
4	Spreads in fields scattered patches.	Spreads in concentric circle.
5	Infection confined only in vascular system.	Necrosis in the parenchymetous region.
6	Also called as vascular wilt.	Also called as dry root rot.
7	No rotting of roots, formed chlamydospores, no sclerotial bodies formed.	Rotting of root, no vascular infection, minute blackish sclerotial bodies on woody tissues

8	Temp. 28- 30^0 C favourable	35^0 C & above favorable
9	No bark shredding symptoms on infected roots	bark shredding symptoms on infected roots always observed

2. Angular leaf spot / Blackarm

Alabama State of USA in 1891.The disease has been reported from South America, Egypt, Sudan, African countries, USSR, China, Australia, Sri Lanka, Pakistan and India. It was first observed in India in Tamil Nadu in 1918. Annual losses vary from 5 to 25 % due to this disease. The common name of the disease is bacterial blight.

C. O : *Xanthomonas campestris* pv. *malvacearum*

Now : ***Xanthomonas axonopodis* pv. *malvacearum***

Family : *Pseudomonadaceae*

The bacterium is a short motile rod, single or in pairs & equipped with a single polar flagellum. It is gram negative, non-acid fast, non-spore forming.

Symptoms:

The bacterium attacks all the above ground plant parts, causing various types of symptoms. The disease has four distinct phases depending on the plant part affected.

- **Seedling stage (seedling blight)**: The earliest symptoms is found on the seedling cotyledons in form of water soaked circular lesions, which later enlarge to become irregular & brown causing distortion & withering of the cotyledons. Severely affected seeding withers & wilt.

- **<u>Leaf spot (angular leaf spot)</u>**: On the leaves water soaked lesions first appear & they become angular, bound by veinlets and turn dark brown to black. The infection spreads along veins & veinlets of the leaf to cause finger like lesions and known as finger blight or vein blight and then down ward reach to the petiole & later stem. On the surface of affected tissues, profuse exudation of bacterial ooze/slime is seen which dry & form thin film.

- **<u>Black arm (stem infection)</u>:** Fruiting branches become dark brown to black, linear, sunken lesions are formed, cause premature dropping off of the leaves resulting in the characteristic black arm or dry black twigs standing in the place of healthy plants. The affected stems show cracks and gummosis and are easily broken by wind.

- **<u>Boll lesions (boll rot)</u>:** The bacterium also infects the boll. Lesions are initially water soaked later turn into dark brown to black, sunken irregular spots. Such spots are deep seated and spread inside the boll to involve the lint fiber and seed, and cause sever reduction in quality of lint. As the disease advances the seeds invaded by the bacterium causing severe reduction in size and loss of viability. Young infected bolls fall down prematurely.

Favorable condition:

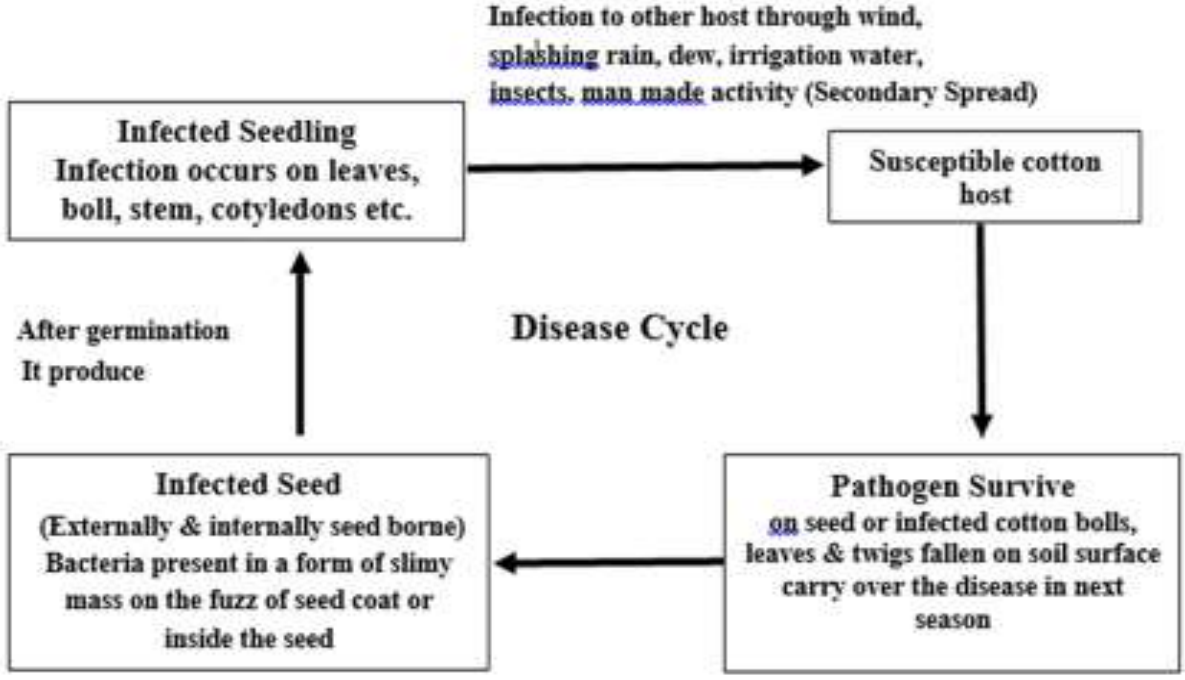
- High relative humidity following rains

- Atmospheric temp. around 280 C.

Disease cycle:

The bacterium is a short motile rod, single or in pairs with a single polar flagellum. It is gram negative, non-acid fast, non-spore forming. The bacteria may remain viable in dry **leaves for 17 years** and in dry or moist soil for 8 days at 21° - 33° C.

- The primary source of infection is mainly through, both externally & internally seed borne. Bacteria present in a form of slimy mass on the fuzz of seed coat or inside the seed. Infected cotton bolls, leaves & twigs fallen on soil surface also carry over the disease.
- Secondary source of infection- Through wind, splashing rain, dew, irrigation water, insects, man made activity in seed production plots.



Management:

- Use healthy seed.
- Seed treatment with thiram, agrosan GN, Ceresan @ 3-4g/kg. after delinting with sulpharic acid
- Spraying streptocycline 0.01 % (100 ppm) 1 gram + copper oxychloride (blue copper) 20 g. in 10 liter water 2-3 spray at 15 days intervals.

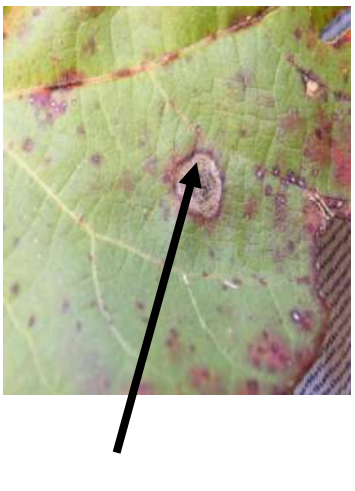
- Grow resistant variety *Gossypium herbaceum* and *G. arboreum* are mostly immune. For example variety like as; Sujatha, CRH 71, HC-9, Reba-B-50, Badnawar-1 etc.
- Sanitation to prevent survival of pathogen on old infected debris. Decomposition of debris should be done.
- Crop rotation.
- Addition of potash to the soli reduce intensity of the disease.
- Biological control: seed treated with *Pseudomonas fluorescens (Strain -1)* or spraying of *Pseudomonas fluorescens* powder @ 0.2 % (20 gram in 10 liter water) after 30,50,70 and 90 days after sowing.

(3)Alternaria blight:

C.O. : *Alternaria macrospore*

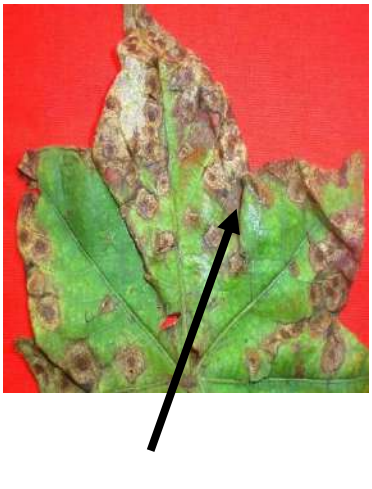
Symptoms:

- Small, pale to brown, irregular to circular spots appear on the leaves.
- Each spot has a central necrotic lesion surrounded by concentric rings.
- Several spots coalesce to form blighting.
- Infected leaves become brittle and fall prematurely.
- Some times lesions appears on bolls, bracts and stem.
- Disease appears in 45-60 days old crop.



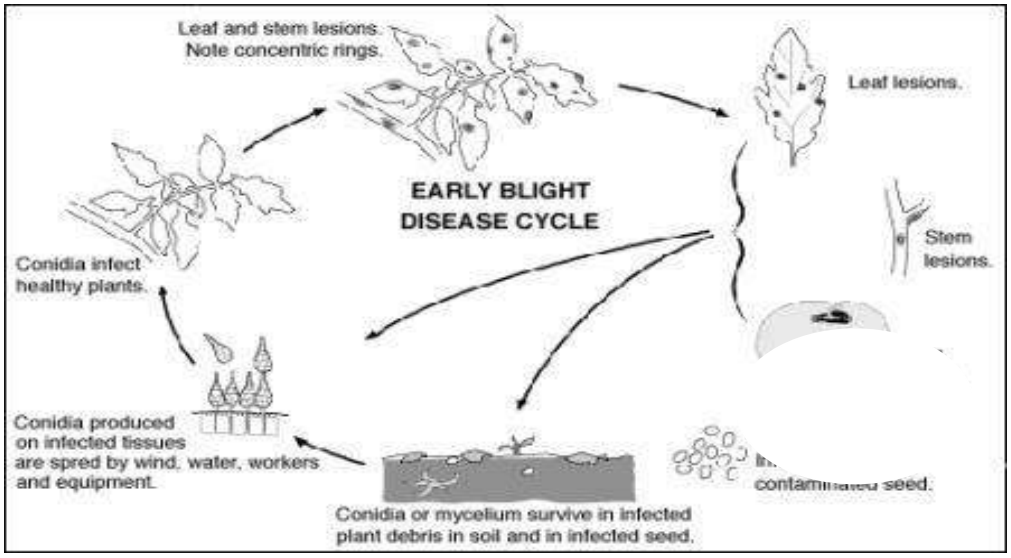
Necrotic lesion surrounded by concentric rings

Brown, irregular to circular spots appear on the leaves



Disease cycle:

- Primary source: Pathogen survives in the dead leaves as dormant mycelium.
- Secondary spread: Air borne conidia.



Favorable condition:

- ✓ High humidity 90-95 %.
- ✓ Intermittent rains.
- ✓ Temperature 25-28^0 C.

Management:

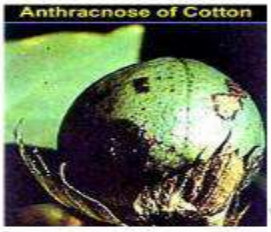
- Remove and destroy the infected plant residues.
- Spray Mancozeb 0.2% @ 27 gram in 10 liter water or copper oxycholoride @ 2 kg/ha at the initiation of the disease. Four to five sprays may be given at 15 days interval.

5) Anthracnose

C. O.: ***Colletotrichum capsici***

Symptoms:

- The pathogen infects the seedlings and produces small reddish circular spots on the cotyledons and primary leaves.
- The lesions develop on the collar region, stem may be girdled, causing seedling to wilt and die.
- In mature plants, the fungus attacks the stem, leading to stem splitting and shredding of bark.
- The most common symptom is boll spotting. Small water soaked, circular, reddish brown depressed spots appear on the bolls.
- The lint is stained to yellow or brown, becomes a solid brittle mass of fibre.
- The infected bolls cease to grow and burst and dry up prematurely.



Pathogen

- The pathogen forms large number of acervuli on the infected parts.
- The conidiophores are slightly curved, short, and club shaped. The conidia are hyaline and falcate, borne single on the conidiophores.
- Numerous black coloured and thick walled, setae are also produced in acervulus.
- **The fungus is both inter and intra cellular,** producing numerous acervuli on affected leaves, stems and bolls.

Favourable conditions

- Prolonged rainfall at the time of boll formation
- Close planting.

Disease cycle

- The pathogen survives as dormant mycelium in the seed or as conidia on the surface of seeds for about a year.
- The pathogen also perpetuates on the rotten bolls and other plant debris in the soil.
- The secondary spread is by air-borne conidia. The pathogen also survives in the weed hosts viz., *Aristolachiabractiata*and *Hibiscus diversifolius.*

Management

- Treat the delinted seeds with carbendazim or carboxin or thiram or captan @ 2g/kg seeds.
- Remove and burn the infected plant debris and bolls in the soil.
- Rogue out the weed hosts.
- Spray the crop at boll formation stage with m ancozeb 2 kg or copper oxychloride 2.5 kg or carbendazim 500g/ha.

(5) Leaf reddening

Leaf reddening in cotton is also known as red leaf disease (lal patti). This disorder is an outcome of interaction of location, variety, environmental condition and nitrogen supply. In general, some of the *Hirsutum* varieties and a few inter and intra specific tetraploid hybrids are sensitive and vulnerable to this malady.

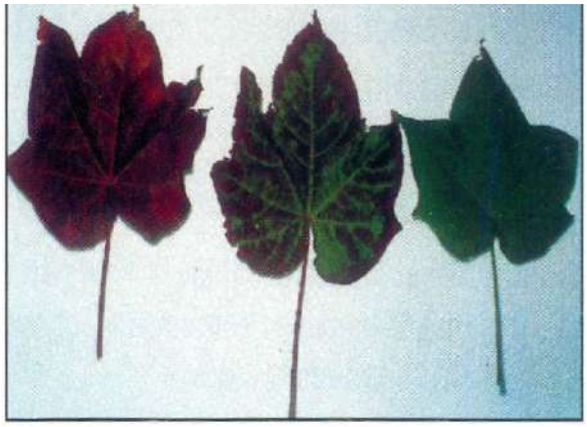
Appearance of red leaf symptom is primarily, due to the accumulation of anthocyanin pigment. Leaf reddening may occur at any growth stage of the crop. However, it is quite often confused with the reddening of leaves caused by sucking pest damage at early growth stages. At grand growth phase (flowering and boll development) any hindrance in the assimilate production, translocation and distribution intensifies the leaf reddening effect. The factors

affecting ideal source-sink relationship promote leaf reddening and symptoms are prolific in nature under extreme stress situations.

Symptoms

Leaf reddening is initially seen in the mature leaves and gradually spreads throughout the canopy. To begin with, leaf margin turns yellow and later red pigmentation is formedon the whole leaf area. In due course of time the leaf becomes dry and subsequentlyprone to shedding.

Leaf reddening in the plant canopy



Intensity of leaf reddening as compared to normal leaf

Causes

The reddening appears in the plants due to various reasons-

Lowered nitrogen level in the leaves (below the critical limit) due to -

- Low availability in the soil
- Impaired uptake under water deficit and waterlogging conditions
- Diversion of N to the developing bolls
- Synchronized boll development and high boll demand
- Desiccation caused by high wind velocity

Anthocyanin (red) pigmentation due to -

- Abrupt changes or drop in night temperature (below 15° C)
- Nitrogen deficiency
- Magnesium deficiency
- Chlorophyll degradation

Management

- Adjustment of sowing time for enabling the crop to skip over the adverse environmentalcondition during boll development stage.
- One or two sprays of urea (1%) at appropriate times.
- Application of magnesium sulphate (0.5%)
- Adequate drainage to avoid waterlogging of the fields
- Leaf reddening incidence due to sucking pests may be overcome bysprayingrecommended insecticides
- Boll load management
- Supplyof adequate nutrients during flowering and boll development particularly inhybrids
- Timely inter-culture and weeding operations and other agronomic practices
- Avoidance of susceptible cultivars
- Adequate irrigation if available

- Adoption of crop rotation and growing of intercrop to maintain the soil health andnutritional status

.....x.....x.....x.....

6. Maize

1. Downy mildew

C.O.: ***Peronosclerospora sorghi***

Economic importance:

- The most common and dangerous in India are caused by *P. sorghi, P. philippinensis* and *P. sacchari.*
- The complete loss of the plant during its early stage of growth or varying degrees of grain loss if cobs are formed.
- If normal looking kobs do develop they rarely reach maturity or bear mature grains.

Symptoms:

- The development of chlorotic stripes along almost the entire length of the leaves.
- Stripes are yellowing white in the beginning, continue as such and turn darker quite late.
- Plants exhibit a stunted growth and bushy appearance.
- White downy growth is seen on the lower surface of leaf.

- Downy growth also occurs on bracts of green unopened male flowers in the tassel.
- Small to large leaves are noticed in the tassel.
- Proliferation of auxilliary buds on the stalk of tassel and the cobs is common (Crazy top).

Disease cycle:

- PSI is through oospores in soil or dormant mycelium present in the infected maize seeds.
- The oospores in the soil germinate in response to root exudates from susceptible maize seedlings.
- The germ tube infects the underground sections of maize plants leading to systemic infection including extensive chlorosis and stunted growth.
- Oospores survive in nature for up to 10 years.
- Once the fungus has colonized host tissue, sporangiophores emerge from stomata and produce sporangia which are wind and rain splash disseminated and initiate secondary infections.
- Sporangia are always produced in the night. They are fragile and do not remain viable for more than a few hours.
- As the crop approaches senescence, oospores are produced in large numbers.

Favourable conditions:

- Optimum temperature is 21-25º C
- High relative humidity: 90 per cent
- Free water on leaf surface
- Young plants are more susceptible

Management:

- Deep ploughing.
- Treat the seeds with metalaxyl at 6 g/kg seeds

- Spray the crop with metalaxyl + mancozeb @ 0.2% on appearance of disease.
- Crop rotation with pulse crops.
- Rough out infected plants.
- Grow resistant varieties and hybrids *viz*., CO1, COH1 and COH2.

2. Stalk rot

C.O.: ***Fusarium moniliformae***

Economic importance:

- The disease was first reported from United States of America by Pammel (1914) as a serious root and stalk diseases.
- In India *Fusarium* stalk rot was first reported from Mount Abu, Rajasthan (Arya and Jain, 1964).
- It has been infecting maize in north and central Karnataka causing economic losses up to 85 per cent.

Symptoms:

- The infection of plants begins from seed germination to tassel emergence, but symptoms will be visible later.
- The plants expressed symptoms of drying from margin of leaves extending towards mid rib covering entire leaf lamina.
- Subsequent death of all leaves leads to drying of whole plant before seed set.
- Stalk rot in maize is usually noticed at the time of tassel emergence.
- **Pith shredding is indicative of *Fusarium* stalk rot.**

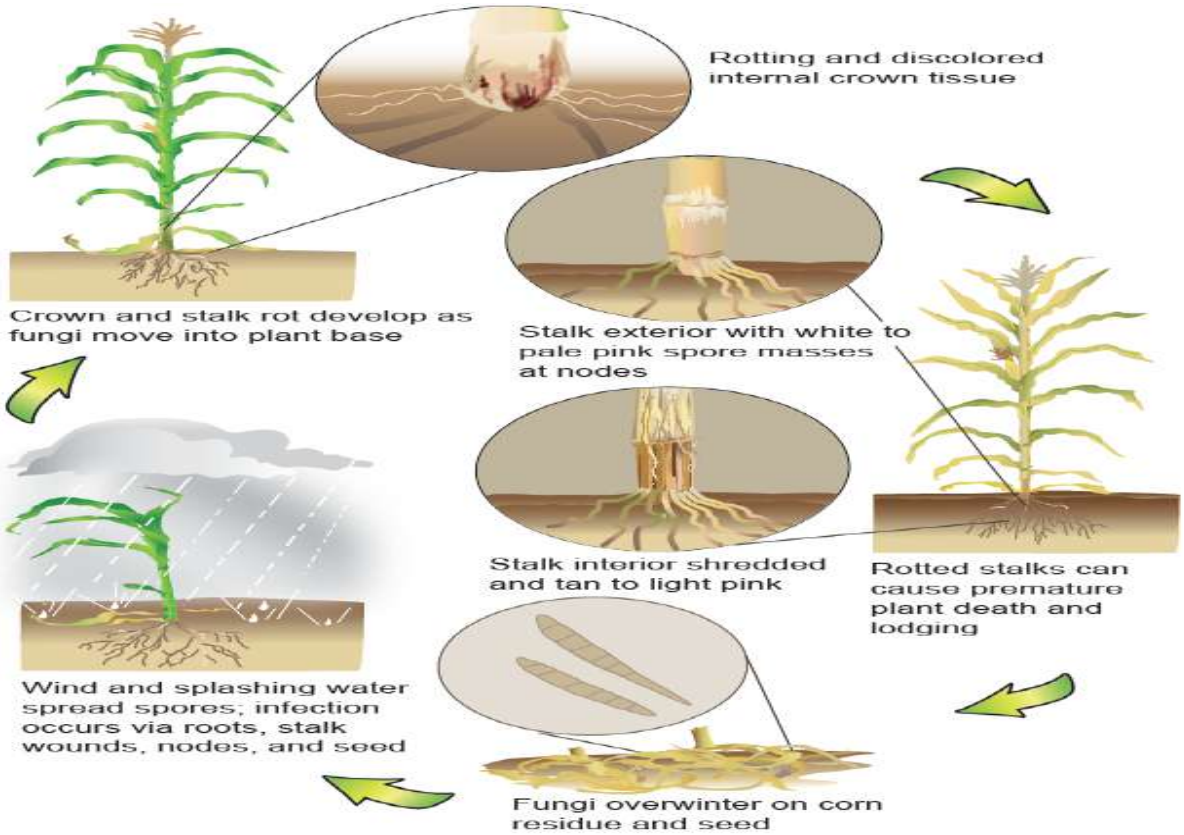
- The affected plant exhibited from a healthy green color to a dull green and the lower stalk becomes yellowed.
- Premature plant death may occur and lodging due to disintegration of the internal stalk pith tissue, the stalk often collapses.
- **Mycelium is often seen at the nodes.**
- When split open the stalks may reveal a **pink discoloration** often confused with the red color of *Gibberella* stalk rot.

Favorable conditions:

- Disease development is favored by warm temperatures (80-100°F)
- Common stresses that lead to stalk rot include; high nitrogen, low potassium, high moisture in the mid to late season, moisture stress early in the season and during grain fill.
- Physical damage that creates wounds allowing the pathogen to enter such as insect damage or hail storms may also predispose corn plants to stalk rot.

Disease cycle:

- The fungi overwinter as mycelium in corn residue and in corn seed.
- The fungus often found growing in healthy stalks and may cause rot.
- Spores are spread by wind and splashing water.
- Infection takes place through the roots, wounds in the stalk or leaf scars.
- Two types of conidia are produced, **macroconidia and microconidia**, that can be splash dispersed onto leaves and washed down the leaf into the sheath and infect at the nodes.
- **European corn borer [*Ostrinia nubilalis* (Hübner)]** has been shown to be an important vector moving *F. verticilliodes* from plant to plant (adult stage) and creating wounds necessary to allow the fungus to enter the plant (laval stage).
- Fusarium root and stalk rots typically occur in complexes with other root and stalk rots such as *Gibberella*, *Diplodia*, and *Colletotrichum*.



Disease cycle

Management:

- Seed treatment with mancozeb 50% + carbendazim 25% WS or thiram 75WP or carbendazim or captan @ 2g/kg of seed.
- Seed treatment with *T. viride* and *P. fluorescens* at 2.5g each/kg of seed and soil application of 1.25kg/h each fortified with 250 kg of FYM.

3. Leaf Blight / Turcicum Leaf Blight

C. O. : *Helminthosporium turcicum*

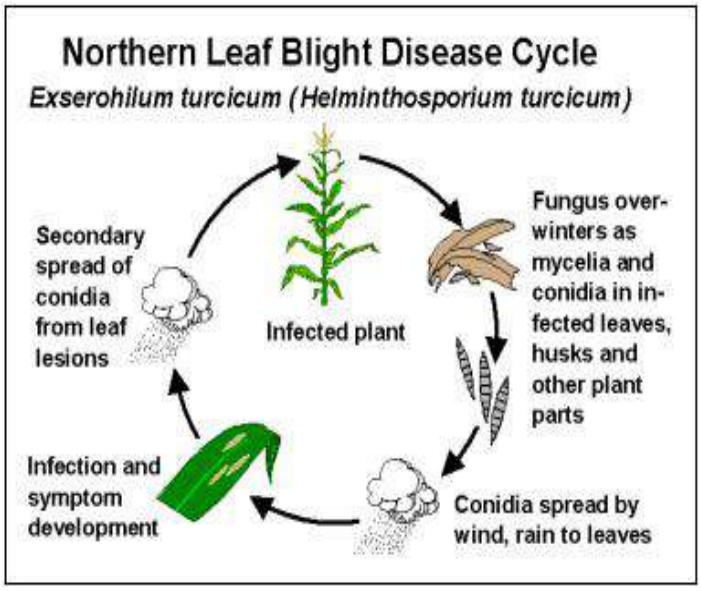
Perf. Stage : *Trichometasphaerica turcicum*

Symptoms:

- ✓ Disease is characterized by long ellipticalgrayish green or tan lesions on the leaves. Boat shaped leaf spots (4-15 cm) are developed on leaves.
- ✓ The fungus affects the maize plant at young stage.
- ✓ Small yellowish round to oval spots are seen on the leaves. The spots gradually increase in area into bigger elliptical spots and are straw to grayish brown color in the center with dark brown margins.
- ✓ The spots coalesce to form bigger spots and gives blighted appearance.
- ✓ The surface is covered with olive green velvety masses of conidia and conidiophores.
- ✓ Under high humidity the whole leaf area becomes necrotic and plant appears as dead. Lesions may be extended to husk.
- ✓ If disease appear in early stage, no cob formation takes place.

Disease cycle:

- ✓ Fungus survives in plant debris, seed and collateral hosts. The fungus is externally seed borne.
- ✓ It also infects Sudan grass, Johnson grass, sorghum, wheat, barley, oats, sugarcane and spores of the fungus are also found to associate with seeds of green gram, black gram and cowpea.
- ✓ Secondary spread is through wind borne conidia.



Favorable conditions:

- ➢ High humidity.
- ➢ Cloudy atmosphere.

- Heavy doses of N_2 fertilizer.

Management:

- Destroy plant debris.
- Seed treatment with thiram / captan @ 3 g / kg seeds.
- Spraying of mancozeb or chlorothalonil @ 0.2 %.
- Use resistant varieties; GM-2, GM-3, GM-4, GM-6, Narmada Moti, Ganga safed -2 and Ganga-11.

4. Maydis Leaf Blight

C.O : *Helminthosporium maydis*

Class : Mastigomycota

Order : Chytridiales

Family : *Physodermateaceae*

Symptoms:

- The disease occur on leaf blade, leaf sheath and outer husk of ear.
- The spots are water soaked, light green at first reddish brown and finally become brown. The lesion enlarge gradually and are restricted by the adjacent veins.
- Spots are more concentrated on basal leaves of plant.
- Affected leaves may die prematurely.

Disease cycle:

- Primary source: Infected seed, crop residue containing conidia/mycelium in soil.
- Secondary spread: Air borne spores.

Favourable conditions:

- High temp. 28^0C- 29^0C
- Abundant moisture
- Water logging

Management:

- Field sanitation.
- Timely sowing of the crop.
- Use of healthy and disease free seed for sowing.
- Crop rotation.
- Avoid water logging condition in field.
- Spray mancozeb or chlorothalonil @ 1.25 kg/ha.
- Use resistant varieties: GM-2, GM-3, GM-4, GM-6, Narmada Moti, Deccan-10 etc.

5. Banded leaf and sheath blight

C.O.: *Rhizoctonia solani f. sp. sasakii*

Perfect stage: *Thanatephorus cucumeris*

Yield losses due to this disease is ranged from 12-41 per cent, but in severe cases grain yield losses may be up to 97 per cent.

Distribution:

- Bertus reported BLSB as minor disease from Sri Lanka for the first time and described it as a 'Sclerotial' disease of maize.
- In India, the disease was first reported in 1960 from *Tarai* region of Uttar Pradesh. Since then its occurrence has been reported in the states of Himachal Pradesh, Uttar Pradesh, Haryana, Punjab, Madhya Pradesh, Rajasthan, Jharkhand, West Bengal, Meghalaya, Assam and Orissa.

Symptoms:

- The symptoms of the banded leaf and sheath blight were observed on all aerial parts of the maize plant except tassel.
- The disease manifests itself on leaf, leaf sheaths, stalks and ears as leaf and sheath blight.
- Under natural conditions, disease appears at pre-flowering stage on 30 to 40 day old plants but infection can also occur on young plants which may subsequently result in severe blighting and death of apical region of growing plants.
- The affected areas appear bleached, soon they become straw colored and necrotic.
- The spread of the disease on leaves is more rapid and extensive than on sheaths.

- The lesions and blotches cover greater areas and the alternating narrow purple or brown zones become more prominent, resulting in the characteristic symptoms of banded leaf and sheathblight.
- In general, the symptoms on leaf sheaths resemble those described for leaves.

Disease cycle:

Primary infection: Sclerotia fallen in the soil

Secondary spread: On contact with diseased plant through fungi.

Favourable conditions:

- High relative humidity (>80%)
- 28° C temperature
- Rainfall during crop growth
- High dose of nitrogen

Management:

- Removal, destruction and burning of infected crop debris.
- Seed treatment with *Trichoderma harzianum*@ 4g/kg seed or *Pseudomonas fluorescens* @ 10 g/kg seed.

- Foliar spray with carbendazim or propiconazole @ 0.025% two to three times.
- Use of neem oil as seed treatment and spray for effective and eco-friendly strategy in managing the BLSB.

6. Late Wilt of Corn or Black Bundle Disease

C. O.: *Harpophora maydis*

Synonyms: *Cephalosporium maydis*

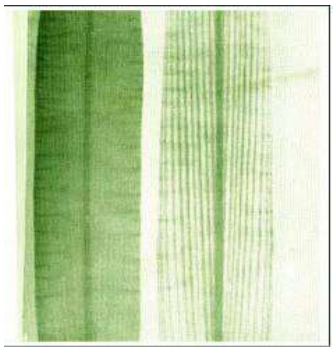
Acremonium maydis

Economic importance:

- In Egypt 100% infection occurs yield losses approached 40%
- Destructive in India, with incidence as high as 70% and economic losses up to 51%.
- Late wilt occurs in Andra Pradesh, Uttar Pradesh, Bihar and Rajasthan provinces of India.

Symptoms

- Root tips of infected corn plants are stained red during early stages of infection, but above ground parts generally remain symptomless until tasseling when a rapid wilting of lower leaves progresses upward.
- Leaves appear streaked as tissue between the veins becomes dull green and then chlorotic eventually rolling inward and appearing scorched while retaining somewhat of a green color.



Spread

- *H. maydis* is a soilborne vascular wilt pathogen that also is seed-borne.
- The pathogen survives as sclerotia on corn debris and infects seedlings through the roots or mesocotyl.
- *H. maydis* initially grows epiphytically on roots and produces short, thick-walled hyphae with swollen cells.
- Penetration can occur anywhere on the root system or mesocotyl (except root tips), but is most common where lateral roots originate or in the zone of root elongation.
- As epidermal cells start to collapse, the fungus penetrates directly and grows intra and intercellularly to the xylem.
- Root injury predisposes plants to the disease and insect or nematode damage provides additional avenues for entry.

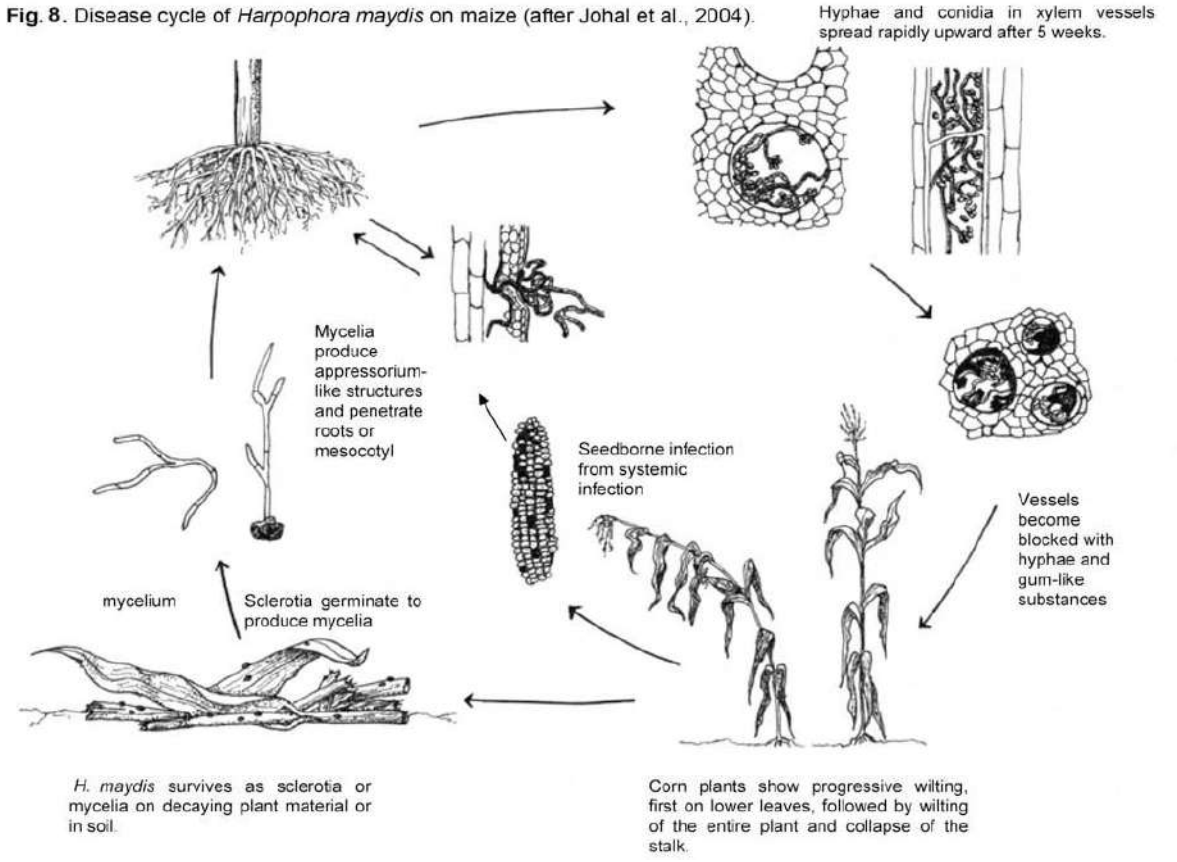


Fig. 8. Disease cycle of *Harpophora maydis* on maize (after Johal et al., 2004).

Favourable condition

- Late wilt develops rapidly at 20-32° C, with optimum disease development at 21-27° C.
- Growth of *H. maydis* in soil is sharply inhibited above 35° C.
- pH from 4.5-10, with an optimum at pH 6.5.
- Low soil moisture (25% saturation) favors sclerotial survival.

Disease management:

- Disease-free seed (inspected, certified seed) could limit further distribution of the pathogen
- Soil solarization
- Early sowing
- Moisture stress is a major predisposing factor for late wilt and frequent watering or saturated soils reduced late wilt.
- Seed treatments with captan.
- Development of genetically resistant corn lines.

…….x…..x…..x…..

7. Sesame (*Sesamum indicum* L.)

1. Root rot/stem rot/charcoal rot

C. O.: *Macrophomina phaseolina*

Symptoms:

- ✓ Disease symptoms found on the root, stems, capsules and seeds of the infected plants.
- ✓ Roots and collar regions of the stems show brown discoloration that extends upwards, resulting in discoloration of the most of the part of the stem.

- ✓ Severely infected plants fail to produce seeds. If seeds are formed they remain light in weight, small in size and shrivelled in appearance.
- ✓ Minute pycnidial bodies may be seen on the infected parts and sclerotia are observed with infected roots. Black colour bodies also found on seed.

Favorable Conditions

Day temperature of 30°C and above and prolonged drought followed by copious irrigation.

Disease cycle:

- ➢ The fungus remains dormant as sclerotia in soil as well as in infected plant debris in soil. The infected plant debris also carries pycnidia.
- ➢ The fungus primarily spreads through infected seeds which carry sclerotia and pycnidia. The fungus also spreads through soil borne sclerotia.
- ➢ The secondary spread is through the conidia transmitted by wind and rain water.

Management:

- ➢ Use healthy and disease free seed for sowing.

- Seed treatment with thiram @ 3 g/kg seed.
- Spraying of mancozeb 75% WP (20 gram) or carbendazim 50% WP (10 gram) or hexaconazole 5% EC (10 ml) in 10 liter of water. Second spray should be done as and when required.

2. Phytophthora blight:

C.O.:*Phytophthora parasitica* var. *sesame.*

First reported from India by Butler in 1918.

Loss: Plant mortality 72-79 % reported.

Symptoms:

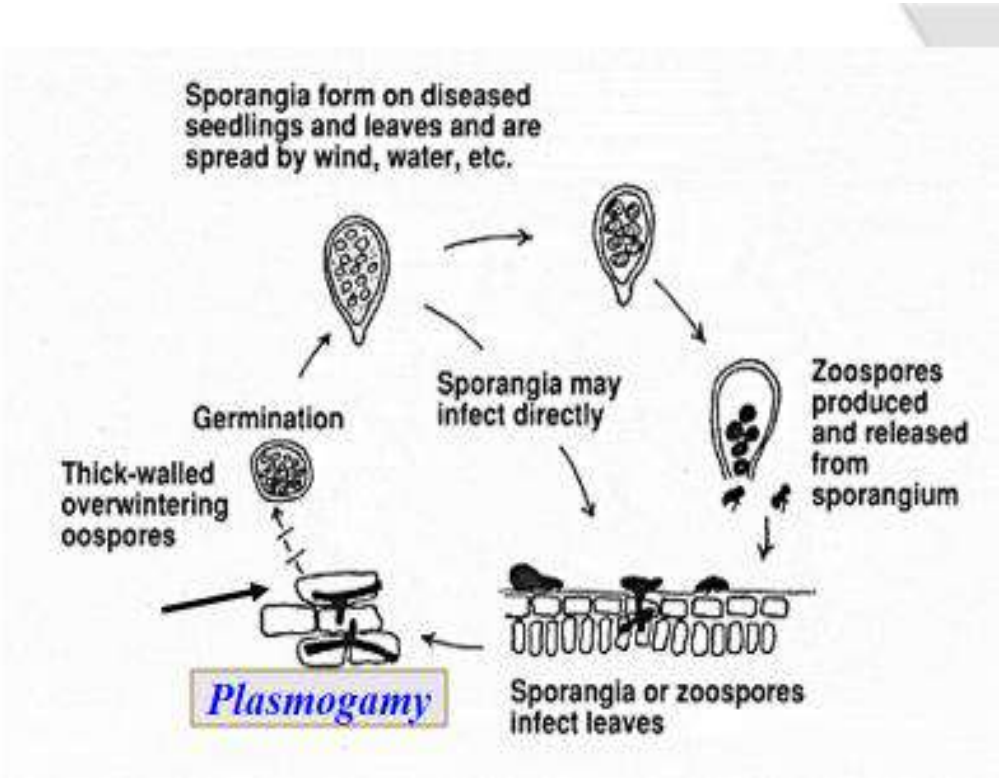
- Appearance of small and water soaked irregular spots on the leaves is the characteristic symptoms of the disease. Water soaked brown spots also found on stem.
- Spots increases in size and cover entire leaf surface and destroy whole leaf.
- Discolouration of stem at basal portion. **Black coloured lesions on stem near soil level at collar region observed**.
- Capsules are also affected. Appearance of cottony growth on the capsules may also be observed.
- As a result of the disease, plant may fail to produce flower, causing considerable damage to the crop.
- Poor seed development and seeds shrivelled

Black coloured lesions on stem near soil level at collar region and on stem

Water soaked brown spots also found on leaves and stem

Disease cycle:

- **Primary source**: Fungus survives in soil as dormant mycelium or chlamydospores. Infected seeds also play important role. The mycelium remains in embryo of seed.
- **Secondary spread**: It is through sporangiophores and zoospores through irrigation water.



Disease cycle of Phytophthora blight of sesame

Favourable condition:

- ✓ Heavy rains for 2 weeks.
- ✓ Temperature 25^0 C.
- ✓ R.H. 90 % and above for 3 weeks.

Management:

- Seed treatment with thirum @ 3 g/kg.
- Use well drained soil for sowing of the sesame crop.
- Three sprays of B.M. 0.6% or mancozeb 75% WP (26 g/10 litre water) or copper oxycholoride 0.3%. **or**
- Spraying of copper oxycholoride @ 40 gram in 10 litre of water after the initiation of the disease and second spray should be done after 15 days of first spray. **or**
- Two sprays of Metalaxyl 8% + Mancozeb 64% mixture @ 20 g/10 litre water required at 15 days interval for effective control of the disease.
- Crop sanitation, proper drainage, rouging etc.
- Use resistant varieties-T-11, NP-3, NP-4 & NP-8.

3. Phyllody:

Mc Gibbon was first to report its occurance in Burma in 1924.

Phyllody of sesame is caused by *Candidatus* phytoplasma. It is a serious and widespread disease of sesame in India. It occurs in Sudan, Uganda, Venezuela, Tanzania, Egypt, Pakistan and other countries of the world.

C.O.: *Candidatus* Phytoplasma

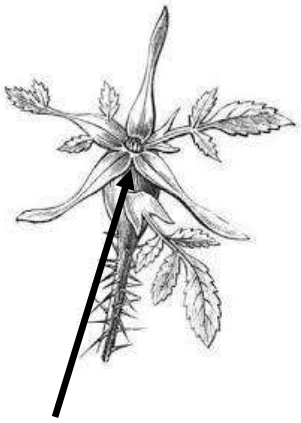
- Bouded with unit membrane
- Unculturable
- Spherical, oval or pleomorphic
- Phloem parenchyma cell

Yield loss: 39 to 74%.

Symptoms:

- Disease manifests in the flowering stage.
- Floral parts transformed in to green leafy structure which grow profusely.
- The sepals are transformed into leaf-like structure and the corolla, stamens and carpels turn green and leafy structure.

- Leaves become clustered, Chlorotic, remain small in size, and flowers malformed. Entire floral structure appears leafy. Internodes remains short and abnormal branching take place so that entire plant malformed.
- Flowers become sterile, veins become thick and prominent.
- Stamens/ ovary becomes leaf like and elongated.



Floral parts transformed in to green leafy structure ***Leaf hopper***

Disease Cycle:

- ✓ The pathogen survive on many other host plants like as; Groundnut, *Crotalaria spp., Cicer arietinum L., Trifolium* spp*., Brassica* spp.which serve as source of **primary inoculum**.
- ✓ Disease is transmitted through leaf hopper **(*Orosius albicinctus).*** **Minimum acquisition feeding period** (time period necessary for successful acquisition of the virus by its vector which then become viruliferous) of the vector is **8 hrs** and **incubation period** (the period between exposure to an infection and the appearance of the first symptoms) is **about 3 weeks**.
- ✓ It is not mechanically or seed transmissible.

Favourable condition:

- ✓ Higher temperature 30-35^{0}C.
- ✓ Higher moisture in plant.

- ✓ High N_2 application

Management:

- Late sowing of the crop helps in reduce disease incidence.
- The disease is transmitted by sucking pest so spraying of effective insecticides like as; methyl-o-dematon 0.05% @ 10 ml in 10 litre water or triazophos 0.05% to reduce further transmission of the phytoplasma.
- Rouging of infected plants helps in checking the spread of disease.

4. Powdery mildew:

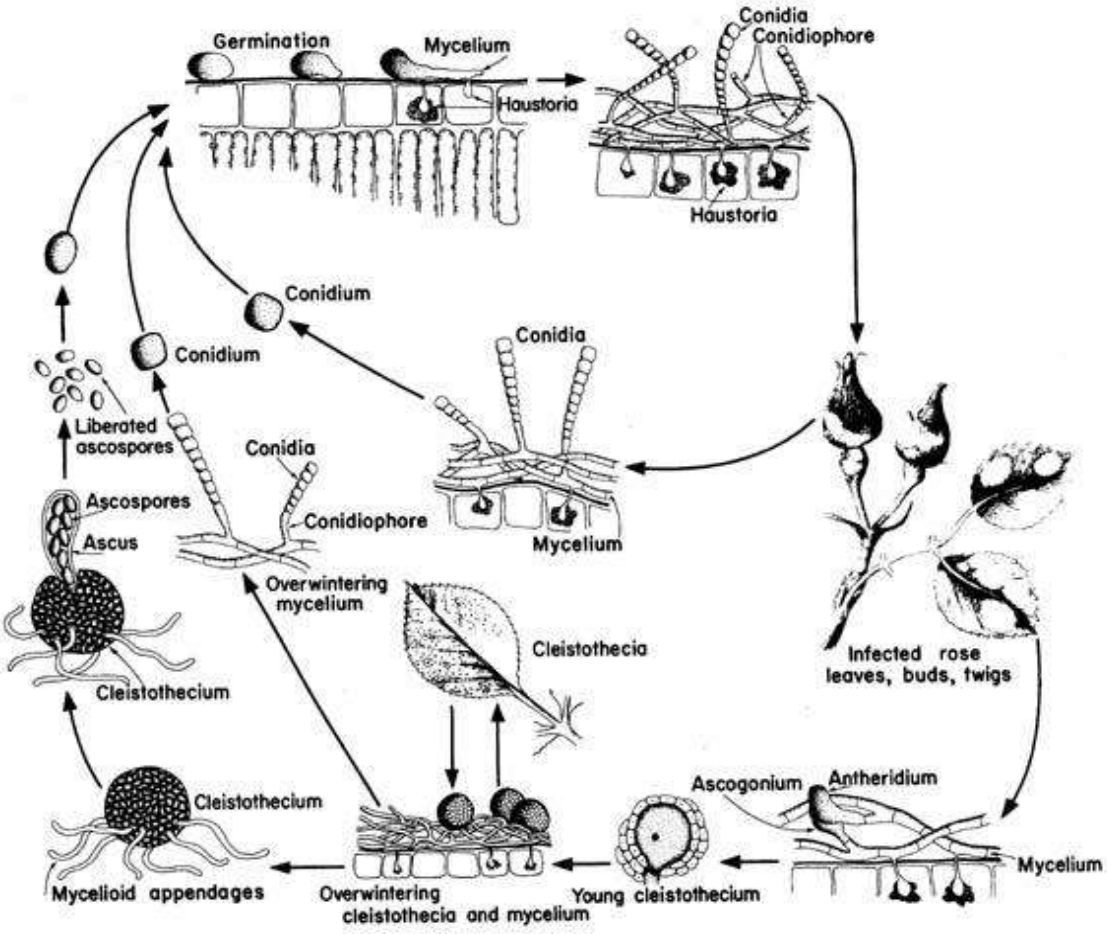
C.O.: *Erysiphae cichoracearum*

Symptoms:

- Greyish white powdery growth found on upper surface the affected leaf.
- Spots coalesce causing entire leaf covered with white power. Affected leaves fall down prematurely.
- Infection also spreads to flowers and capsules.
- Premature shedding of flowers and capsules.
- Seeds are shrivelled and oil content is reduced.

Disease cycle:

- **Primary source**: Pathogen perpetuate in cleistothecia in infected plant debris in soil. The ascospores from cleistothecia cause primary infection. Fungus is obligate parasite.
- **Secondary spread**: Wind borne conidia.



Disease cycle of Powdery mildew of Sesame

Favourable condition:

- ✓ Cool and dry climate.
- ✓ Low R.H.

Management:

- Remove the infected plant debris and destroy it.
- Spray wettable sulphur 80 % WP @ 30 gram in 10 litre of water or sulphur dust @ 25 kg /ha and repeat after 15 days.
- Grow late maturing variety *like as;* SI-1516, SI-1683, SI-2177 and SI-2183.

5. Bacterial leaf blight

Causal Organism: *Xanthomonas campestris pv. sesami*

Kingdom: Bacteria

Phylum : Proteobacteria

Class : Gamma proteobacteria

Order : Xanthomonadales

Family : Xanthomonadaceae

Symptoms:

- Water soaked spots developed on the cotyledons of the seedlings coming out of the infected seeds.
- In older seedling, these spots appear dark brown to black in colour.
- Irregular to round spot found on the leaves, these spot coalesce, forming bigger spots at the later stage of the disease development.
- Black colour lesions developed on petioles.
- Infected leaves may fall down prematurely.

Favourable condition:

High humidity, Hot and cloudy weather favours disease development.

Disease cycle:

- Primary source: The disease is seed and soil borne. The bacteria also survive on weed host.
- Secondary spread: by wind, water, rain, man, implements.

Management:

- Seed treatment should be done with Agrimycin-100 (250 ppm) or streptocycline suspension (0.05%) for 30 minutes for preventing seed borne infection.
- Spray mixture of streptocycline (0.5 gram) + copper oxychloride (40 gram) in 10 litre water after initiation of the disease. Second spray should be done after 15 days.

6. Cercospora leaf spot

C. O.: *Cercospora sesami* Zimm.

Symptoms

- The disease manifests itself on the leaves as minute water socked lesions, which enlarge to form round to irregular spot 5-15 mm in diameter on both leaf surfaces.
- The characteristic symptoms of the disease usually appear at the time of flowering.
- The spotted area is light brown to white in the beginning which later becomes dark grey. Several spots produce on leaf blade.
- Infection also found on petiole, stem and capsules, producing linear, dark coloured, deep seated lesions.
- Leaf blight and defoliation occurs in severe infection.

Favourable condition:

High humidity and low temperature

Disease cycle:

- Primary source: The fungus is seed born, both internally and externally. It can also survive in plant debris.
- Secondary spread: wind borne conidia

Management:

- Seed treatment with thiram @ 3 g/kg seed before sowing for eradication of externally seed borne fungus and hot water treatment to the seed for 30 minutes at 53° C for eradication of internally seed borne fungus.
- Spraying of propiconazole 0.025% (10 ml/10 litre water) or mancozeb 0.2% (25 g/10 litre water) after initiation of the disease. Two sprays should be done after 12 days interval.

.....x.....x.....x.....

8. Soybean *(Glycine max)* (L). Merr.

1. Rhizoctonia blight of soybean

C.O.: ***Rhizoctonia solani***

Economic importance:

- *Rhizoctonia* root rot considered one of the most common soilborne disease of soybean worldwide.
- The fungus is long-lived and ubiquitous in most field soils and can cause seed rot, damping-off, root rot and stem rot.

Symptoms:

- Pre and post-emergence damping-off.

- Infected seedlings produced reddish-brown lesions on the hypocotyls at the soil line.
- These lesions are sunken and remain firm and dry.
- The root rot phase may persist into late vegetative to early reproductive growth stages.
- Affected plants remain stunted in growth.

[A] **[B]** **[C]**

Damping off caused by *Rhizoctonia*:(A) seedling blight and (B) root rot [C] Hypocotyl lesions

Disease cycle:

- The fungus survives on plant residue or in soils as sclerotia.
- Infection may occur soon after seed is planted.

Favourable condition:

- Disease is more severe on light and sandy soils, and can be more common on the slopes of fields.
- Many strains of *Rhizoctonia* can infect corn, alfalfa, dry bean, and some cereal crops.

Management:

- Eliminating stress factors, such as use of herbicides that cause injury to soybean roots, can help reduce root rot problems.
- Fungicide seed treatments are effective against *Rhizoctonia*.
- Rotation with small grains and corn may reduce pathogen populations in a highly infested field.

2. Soybean mosaic:

C. O.: Soybean mosaic virus (SMV)

- SMV occurs to a limited extent in all soybean producing areas.

Symptoms:

- Infected plants can be recognized by their stunted growth, distorted and puckered leaves. Leaves are narrower and deeper green than normal.
- The leaves are dwarfed, crinkled and narrow with their margins turned downwards. In severe cases, dark green blister like puckering along the veins takes place.
- Leaf margins are turned down and blades are severely ruffled along the edge of the main veins.
- Pod setting is drastically reduced. Infected plants produce distorted pods and fewer seeds.
- Seed discoloration can be seen under severe infection. The infected plants remain green even at the end of the growing season.
- In susceptible variety 100% seed transmission is noticed.

Favourable Conditions

- SMV vary depending on the soybean cultivar, the age of the soybeans, the virus strain, and the temperature. Symptoms are most noticeable under cool temperatures of 18 to 24^0 C.
- When temperatures rise above 30^0 C., leaf symptoms may be masked. The youngest and most rapidly growing leaves show the most severe symptoms.

Virus Transmission: Several species of aphids spread the virus from plant to plant in the field.

Disease cycle:

Primary infection through seeds.The virus remains in a viable state in the seeds for at least two years. The virus is also transmitted by several species of aphid vectors such as *Aphis craccivora*, *A. fabae, A. gossypii, Liphaphis erisimi, Myzus percsicae* etc in a non persistent manner. The vectors become viruliferous by feeding on the leaves, stems or branches of infected plants.

Management:

- Diseased plants should be rouged out from the fields where soybeans are frown for seed production.
- Use disease free seeds for planting.
- Spray systemic insecticides to control aphids i.e. methyl-o-demeton and other sucking pest insecticides.
- Keep down weeds and aphid population.
- Grow resistant cultivars.

…..x…..x…..x…..

9. Red Gram / Pigeonpea (*Cajanus cajan*)

1. Wilt:

C.O. : *Fusarium oxysporum f. sp udum* (Butler) Snyder & Hansen.

Class : Deuteromycets

Order : Moniliales

Family : Tuberculariaceae

Distribution

Maharashtra, Uttar Pradesh, Madhya Pradesh, Bihar, Tamil Nadu and Gujarat.
This is the worst disease of red gram in India causing severe damage wherever the crop is grown.

Symptoms:

- The diseases may appear from early stages of plant growth (4-6 week old plant) up to flowering and pod formation stage.
- The disease appears as gradual withering and drying of plants, as if they were suffering from drought.
- In the beginning, yellowing of leaves and blackening of the stem region appear, starting from collar to branches.
- Leaves turn yellow prematurely. Foliage droops down and within 3 to 4 days plant wilt. Disease appears in patches and spreads in concentric circles. Black lesions of varying sizes also appear on tap root and stem.
- Vascular tissues exhibit brown discoloration. Discoloration of roots and vascular bundles causes plugging of xylem and phloem vessels.
- Many times only one side of the stem and root system is affected resulting in partial wilting.

Favorable climatic condition:

- pH. range 4 to 9

- Soil temperature 35^{o} C
- The fungus can with stands adverse climatic condition

Disease cycle:

- Fungus remains longer time as saprophytes in soil.
- Primary source of infection is chlamydospores perpetuating in infected stubbles.
- The secondary spread takes place through macro and micro conidia with irrigation water/interculturing implements.

Management:

- Seed treatment with carbendazim @ 4 g/kg of seed.
- Enrich soil with green manuring and oil seed cake.
- Soil application of *Trichoderma viride* (Monitar, Bioderma or Ecoderma) @ 2.5 kg/300 kg FYM in open furrows before sowing.
- Seed treatment with *Trichoderma viride* @ 6-8 g/kg of seed.
- Follow crop rotation with sorghum, pearl millet, tobacco etc.
- Deep ploughing during summer.
- Removal of infected plants with their roots.
- Use resistant variety-ICP-8862, ICPL-88046 and ICPL-227, BDN-2, G-1, N-15 and ND-38.
- Addition of solution of boron, manganese or zinc protect the seedlings during germination.
- Application of press mud 10 tone/ha after solarization the field with 25 LDPE clear polyethylene film for reduced the wilt.

2. Phytophthora blight/Stem blight

- **Causal organism:** ***Phytophthora drechsleri f. sp .cajani***
- **Class** **:** Oomycetes
- **Order** **:** Pythiales
- **Family** **:** Pythiaceae

Symptoms:

- Purple to dark brown necrotic lesions girdle the basal portion of the stem later may occur on aerial parts of the seedlings.
- Initially the lesions are small and smooth, later enlarging and slightly depressed.
- Infected tissues become soft and whole plant wilts. In the adult plants infection is mostly confined to basal portions of the stem.
- The infected bark become brown and the tissue softening causing the plants to collapse. The infected branches may break off in wind.

	➢ Swelling of the stem at ground level, stem lesion later develop in to cankerous structure at the edges.

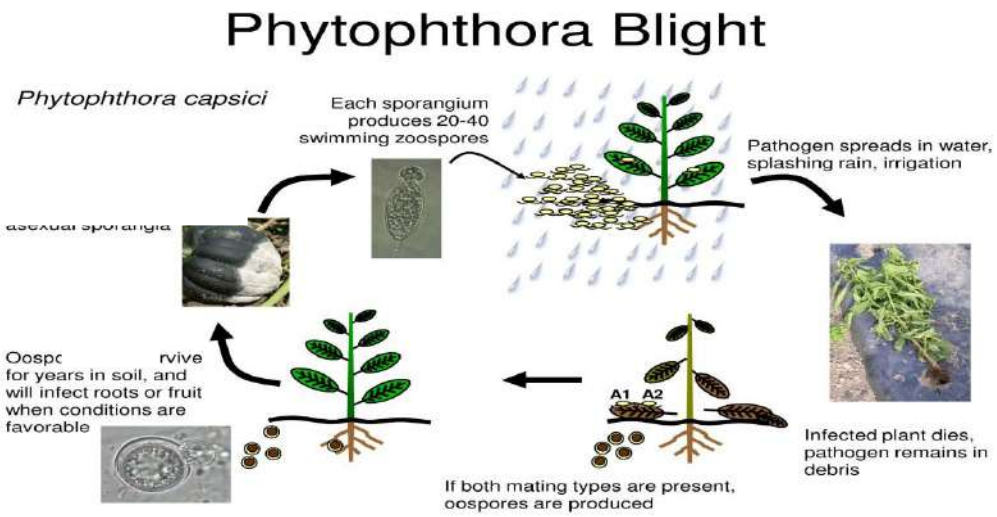
Infected Plants

Favorable condition

- Soils with poor drainage.Low lying areas.
- Heavy rain during the months of July-Sept.High temperature 28^0-30^0 C.

Disease cycle:

- The fungus survives in the soil and plant debris in the form of oospores, and dormant mycelium.
- Primary infection is from oospores and secondary spread by zoospores from sporangia.
- Rain splash and irrigation water help for the movement of zoospores.
- *Cajanus scarabaeoides* var. *scarabaeoides*, a wild relative of pigeon pea is also a host of the blight pathogen.



Management:

- Treat the seeds with metalaxyl @7 g/Kg seed.
- Spray metalaxyl @ 500g/ha.
- Adjust the sowing time so that crop growth should not coincide with heavy rainfall.
- Sowing of red gram should be done in disease free field.
- Avoid sowing of the crop in the field where water logging conditions occur.

3. Sterility Mosaic Virus (SMV)/Sterility Mosaic Disease (SMD).

C.O : *Sterility Mosaic Virus*

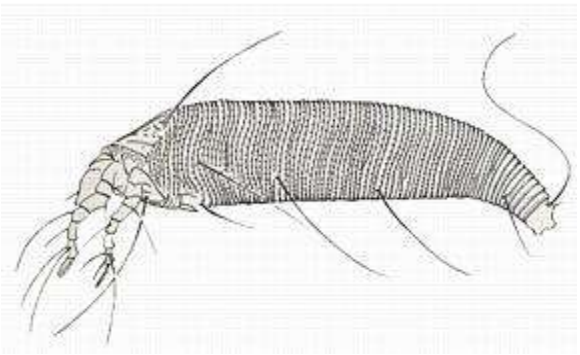
Sterility mosaic disease (SMD) was first described in 1937 from Pusa (Bihar, India).

Symptoms:

Appears at any stage of crop growth. Mottling, yellowing and puckering of leaves.

- Leaves become smaller with clustering near tip and shows stimulation of auxiliary buds. Small size of the leaves gives a bushy appearance to the top of the plant.

- Internodes become short and plant shows stunting appearance. Plant does not produce flowers and pods. Plants with complete sterility gives 95 % yield loss. Partial sterility symptoms are observed in older plants.
- The seeds from infected plant are small, shriveled, immature and non-viable.



Eriophyid mite Aceria cajani ***Infected Plant***

Mode of spread and survival

- The disease is transmitted by an eriophyid mite *Aceria cajani*.
- The mite and virus are highly host specific with a narrow host range confined to pigeon pea and it's wild relatives *Cajanus scarabaeoides* and *C. cajanifolius.*
- The vector can retain virus for up to 13 hours. The transmission is semi-persistent.
- The single viruliferous mite is able to transmit SMD. Self-sown red gram plants and perennial types of plant serves as primary source of inoculums.

Management:

- ✓ Rough out infected plant and crop rotation.
- ✓ Spraying of acaricides like as;dicofol 15ml/ 10 liter water.
- ✓ Grow resistant genotypes-ICP-7035, VR-3, Purple-1, DA-11, DA-32, ICP-6997, Bahar, Bhavanisagar-1, BSMR-235, ICP-7198, PR-5149, ICP-8861.

…..x…..x…..x…..

10. Black gram (*Phaseolus mungo*)

(1) Powdery mildew

C.O.: *Erysiphe polygoni*

Symptoms

- Small, irregular powdery spots appear on the upper surface of the leaves, sometimes on both the surfaces.
- The disease becomes severe during flowering and pod development stage.
- The white powdery spots completely cover the leaves, petioles, stem and even the pods.
- The plant assumes grayish white appearance leaves turn yellow and finally shed.
- Often pods are malformed and small with shriveled seeds.

Favorable conditions

- Warm humid weather.
- The disease is severe generally during late *kharif* and *rabi* seasons.

Disease cycle:

- The fungus is an obligate parasite and survives as cleistothecia in the infected plant debris.
- **Primary infection is usually from ascospores** from perennating **cleistothecia**.
- The secondary spread is carried out by the **air borne conidia**.
- Rain splash also helps in the spread of the disease.

Management

- Remove and destroy infected plant debris.
- Spray carbendazim 500 g. or wettable sulphur 1.5 kg. or tridemorph 500 ml./ha at the initiation of disease and repeat 15 days later.
- Grow resistant varieties like LBG-17, PDU- 10, ICI -12/2 and PLU- 322.

(2)Anthracnose

C.O. : *Colletotrichum lindemuthianum*

Sex. Stage: *Glomerella lindemuthianum*

Symptoms

- The symptoms can be observed in all aerial parts of the plants and at any stage of crop growth.
- The fungus produces dark brown to black sunken lesions on the hypocotyls area and cause death of the seedlings.
- Small angular brown lesions appear on leaves, mostly adjacent to veins, which later become grayish white centre with dark brown or reddish margin.
- The lesions may be seen on the petioles and stem.
- The prominent symptom is seen on the pods.
- Minute water soaked lesion appears on the pods initially and becomes brown and enlarges to form circular, depressed spots join to cause necrotic

areas with black dots (Acervuli). The infected pods have discolored and shriveled seeds.

Favourable conditions:

- High relative humidity (< 90 %), low temperature (15-20^{o}C) and cool rainy days.

Disease cycle:

- The fungus is seed-borne and cause primary infection.
- It also lives in the infected plant tissues in soil.
- The secondary spread by air borne conidia produced on infected plant parts.
- Rain splash also helps in dissemination.

Management

- Remove and destroy infected plant debris.
- Treat the seeds with carbendazim at 2 g/kg.
- Spray carbendazim 500 g. or mancozeb 1 kg/ha soon after the appearance of disease and repeat after 15 days.

…..x…..x…..x…..

11. Green gram (*Phaseolus aureus*)

(1) Cercospora leaf spot:

C.O.: *Cercospora canescens*

Symptoms

➢ Small, circular spots develop on the leaves with grey centre and brown margin. ➢ The several spots coalesce to form brown irregular lesions. ➢ In severe cause defoliation occurs. ➢ The brown lesions may be seen on petioles and stem in severe cases. ➢ Powdery growth of the fungus may be seen on the centre of the spots.	

Favourable climatic conditions

- Humid weather and dense plant population.

Disease cycle:

- Primary source: The fungus survives on diseases plant debris and on seeds.
- Secondary spread by air-borne conidia.

Management

- Remove and burn infected plant debris.
- Spray mancozeb at 1 kg/ha or carbendazim 250g/ha.

(2)Yellow mosaic virus

Symptoms

➢ Initially small yellow patches or spots appear on green lamina. ➢ The young leaves are the first to show the symptoms. ➢ The infected plants normally mature later and bear a very few flowers and pods. ➢ The pods are small and distorted. ➢ The early infection causes death of the plant before seed set.	

Favourable climatic conditions

- Summer sown crops are highly susceptible.
- The presence of weed hosts viz. *Croton sparsiflorus, Acalypha indica, Eclipta alba* and *Cosmos pinnatus* and other legume hosts.

Disease cycle:

- The virus survives in the weed hosts and other legume crops.
- The disease spreads through wind-borne viruliferous white fly, *Bemisia tabaci*.

Management:

- Diseased plants should be rouged out from the fields.
- Use disease free seeds for planting.
- Spray systemic insecticides to control sucking pests i.e. methyl-o-demeton @ 500 ml/ha.

…..x…..x…..x…..

12. Ragi/Finger millet (*Eleusine coracana*)

(1) Blast

C.O.: ***Pyricularia grisea***

Symptoms

Infection may occur at all stages of plant growth. Young seedlings may be blasted or blighted in the nursery bed as well as developing young plants in the main field. There are three stages in disease development.

- ***Leaf blast***: It is more severe in tillering phase. The disease is characterized by spindle shaped spots on the leaves with gray centers surrounded by reddish brown margins.
- ***Node blast***: Infection on stem causes blackening of the nodal region and the nodes break at the point of infection. All the parts above the infected node die.
- ***Neck blast***: At flowering stage, the neck just below the ear head is affected and turns sooty black in color and usually breaks at this point. In early neck infections, the entire ear head becomes chaffy and there is no grain set at all. If grain setting occurs, they are shriveled and reduced in size.

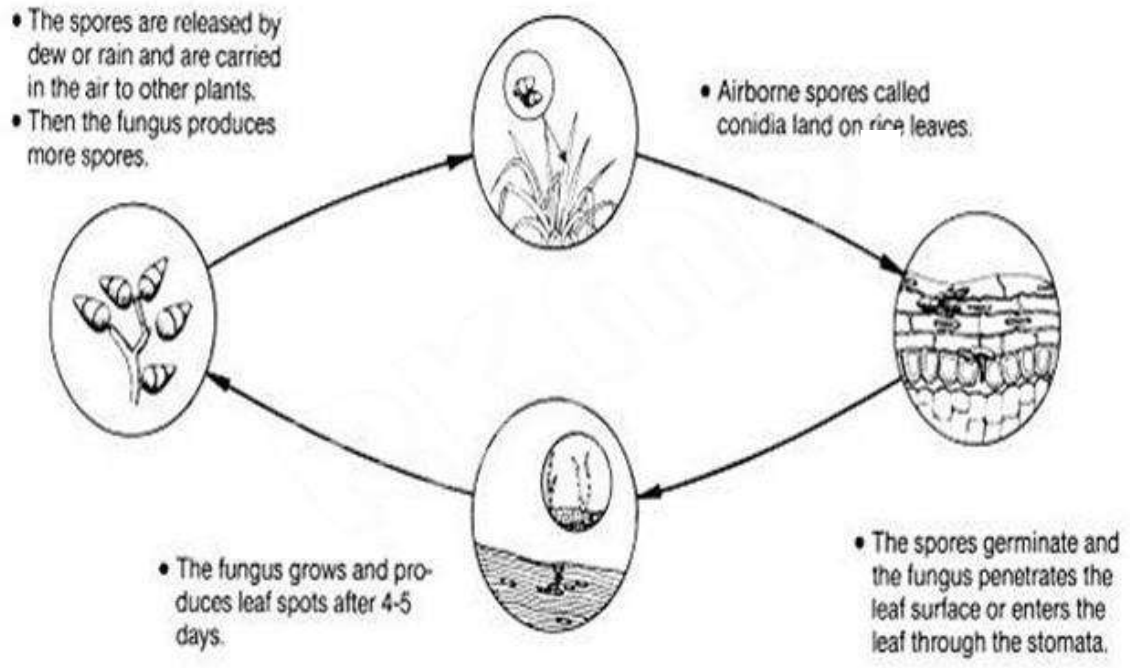
Favourable conditions:

- ✓ Cloudy weather with high relative humidity 93-99 %

- ✓ Intermittent drizzles and more no. of rainy days
- ✓ Slow wind movement
- ✓ Presence of collateral hosts like bajra, wheat, barley and oats.

Disease Cycle:

- ✓ Primary source: Mycelium and conidia in infected debris serves as inoculum.
- ✓ The secondary spreads takes place through wind born conidia.
- ✓ The mycelium survives in straw for 1-2 years under dry condition.
- ✓ The fungus is seed-borne and the primary infection takes place through the seed-borne conidia and also through diseased plants, stubbles and weeds.



Management:

- ➢ Use disease free seeds.
- ➢ Seed treatment with organomercurials @ 2-3 g/kg of seed e.g. MEMC
- ➢ Seed treatment with captan, thiram, carbendazim, carboxin or tricyclazole @ 2 g/kg

- Spray the main field with edifenphos 250 ml or iprobenphos 500 ml carbendazim 250 g or tricyclozole 400 g or thiophanate methyl 500 g or pyroquilon 500 g/ha.
- Spray blasticidin (20 ppm) or kasugamycin (20 ppm) or hinosan 0.1%.
- Two sprays of 0.05% carbendazim or 0.035% thiophanate methyl at boot leaf stage and flowering stage.
- Immediately sowing after onset of monsoon.

…..x…..x…..x…..

13. Paddy (*Oryza sativa* L.)

(1) Blast

C.O. : *Pyricularia oryzae*

Class : Deuteromycetes

Order :Moniliales

Family :*Moniliaceae*

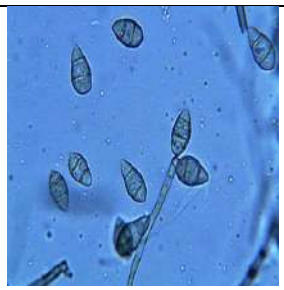
Per (Sexual). Stage : *Magnaporthe grisea*

The causal organism was first detected by Cavara in 1891 from Italy. The pathogen produces few toxins namely, Pyricularin and pyriculol. First reported from China in 1637. In India first time reported from Tanjore district of Tamil Nadu in 1918. It is common in over 85 rice growing countries. It cause yield loss up to 70 – 80 per cent. In Italy the disease called "**brusone**" was reported in 1828.

Symptoms:

The fungus attacks the crop at all stages from seedlings in nursery to heading in main field. The typical symptoms appear on leaves, leaf sheath, rachis, nodes and even the glumes are also attacked.

- **Leaf blast:** On the leaves, the lesions start as small bluish green flecks, soon enlarge under moist weather to form the characteristic spindle shaped spots with grey centre and dark brown margin. The spots join together as the disease progresses and large areas of the leaves dry up and wither. Similar spots are also formed on the sheath. Severely infected nursery and field show a burnt appearance.
- **Nodal blast:**Black lesions appear on nodes girdling them. The affected nodes may break up and all the plant parts above the infected nodes may die. The affected nodes break up and all the plant parts above the infected nodes may die.
- **Neck blast:** After flower / panicle emergence the fungus attacks the peduncle. On neck a brownish to black coloured lesion appears, seeds become black and shriveled.Partial grain filling is observed in late infected panicles. Infected panicle hangs down.

			
Leaf blast	**Nodal blast**	**Neck blast**	**Spores**

Favourable conditions: -

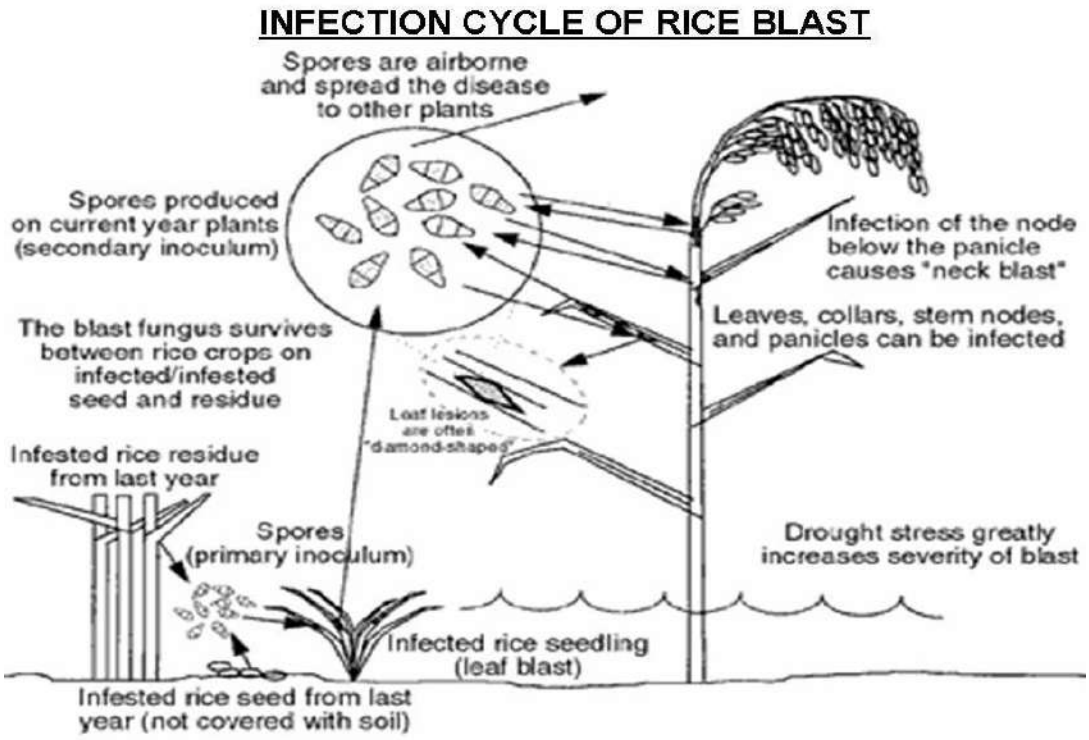
- ✓ Excessive application of nitrogenous fertilizers.
- ✓ Cloudy weather
- ✓ High relative humidity 90-99 %
- ✓ Intermittent drizzles
- ✓ Low night temperatures 15^{o}-20^{o} C
- ✓ More number of rainy days
- ✓ Longer duration of dew, slow wind movement, Presence of collateral hosts.

Pathogen

- Mycelium is hyaline to olivaceous and septate.
- Conidia are produced in clusters on long septate, olivaceous conidiophores.
- Conidia are pyriform to ellipsoid, attached at the broader base by a hilum.
- Conidia are hyaline to pale olive green, usually 3 celled.
- The perfect state of the fungus is *M. grisea* producing perithecia.
- The ascospores are hyaline, fusiform, 4 celled and slightly curved.

Disease Cycle:

✓ **Primary source:** Mycelium and conidia in infected straw and seeds are serves as inoculum. The most probable source of perennation and initiation of the disease appear to be the grass hosts and early sown paddy crop.



✓ The fungus also survives on collateral hosts *viz., Brachiaria mutica, Echinochloa crusgalli, Panicium repens, Digitaria marginata, Dinebra mutica, Leersia hexandra, Dinebra retroflea* etc.

✓ The disease cycle is short and most damage is caused by secondary infections.

- ✓ **The secondary spreads** takes place through wind born conidia.
- ✓ The mycelium survives in straw for 1-2 years under dry condition.

Forecasting

- Forecasting blast of rice can be made on the basis of minimum night temperature range of 20-26^0 C in association with a high relative humidity range of 90 per cent 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.
- In Japan, the first leaf blast model was developed and named as **BLAST**.
- Later based on different field experiments various models were developed namely, **PYRICULARIA, PYRIVIEW, BLASTAM**, and **P- BLAST**.
- A model to forecast the disease called **"Epi-Bla"** has been evolved in India.

Management:

- Use disease free seeds.
- Seed treatment with carbendazim or tricyclazole @ 2 g/kg seeds or *Pseudomonas fluorescens* @ 10 g/kg seed.
- Spray nursery with carbendazim 0.2 g/lor tricyclazole 0.3g/l.
- Spray the main field with iprobenfos 500 ml or carbendazim 250 g or tricyclazole 400 g or thiophanate methyl 500 g or pyroquilon 500 g /ha.
- Spray blasticidin or kasugamycin @ 20 ppm.
- Middle Gujarat-Transplant GR-11 or Pankhali-203 between 1st July to1st August.
- Remove and destroy the weed hosts in the field bunds and channels.
- Splitapplication of nitrogenand judicious application of nitrogenous fertilizers.
- Two sprays of tricyclazole @ 0.045% or isoprothiolane @ 0.04% or or prochloraz @ 0.05% first at boot leaf stage and second at flowering stage.

(2) Bacterial Leaf Blight

C.O. **: *Xanthomonas oryzae* pv*. oryzae***

Class : Schizomycetes

Order :Pseudomonodales

Family :*Pseudomondaceae*

Economic Importance

- First reported from Japan and Philippines sixty years ago. In Indonesia, **Kresek** disease was reported to kill young seedlings completely in 1950.
- In India Srinivasan described disease in 1959.
- Distribution: Sri Lanka, China, Indonesia, Philippines, India
- Yield loss:The extent of loss has been reported to vary from 6-60%.
- Disease appeared in epidemic form in 1962 in Bihar due to introduction of exotic rice variety- ***Taichung Native* - 1**.

Symptoms:

- The bacterium induces wilting and yellowing of leaves or wilting of seedlings known as **Kresek** is seen in seedlings within 3-4 weeks after transplanting of the crop. Kresek results either in the death of whole plant or wilting of only a few leaves.
- The bacterium enters through the hydathodes and cut wounds in the leaf tips, becomes systemic and cause death of entire seedling.
- In grown up plants dull greenish, water soaked translucent lesions appear (5-10 mm) on leaf tip and margins giving marginal drying.
- Several such lesions may coalesce to form straw coloured brown large lesions giving blighted appearance.
- Milky or pale amber colour bacterial ooze may observe on infected portions.

Bacterial leaf blight

- In severe infections all the leaves are attacked and premature drying noticed.
- If cut end of infected leaf is dipped in water, bacterial ooze makes the water turbid.
- Unlike plants infested with stem borer, rice plants with Kresek are not easily pulled.

Quick diagnose bacterial blight on leaf:

- Cut a young lesion across and place in a transparent glass container with

clear water.

- After a few minutes, hold the container against light and observe for thick or turbid liquid coming from the cut end of the leaf.

Pathogen:

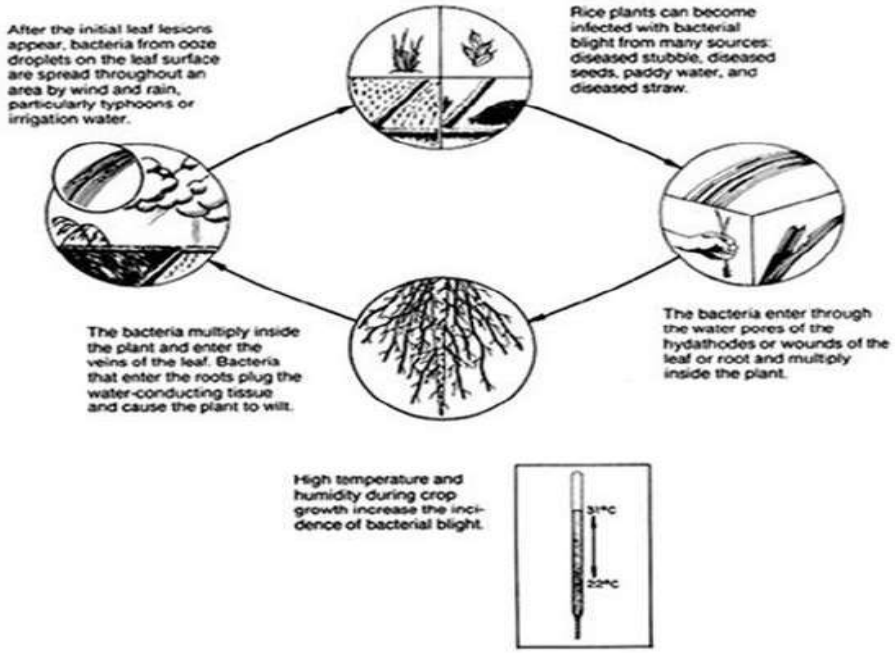
The bacterium is aerobic,Gram–ve, Non-spore forming, capsulated, rod shaped with 0.05-0.08 x 1- 2 μ with monotrichous polar flagellum.

Favourable conditions:

- ✓ Clipping of tip of the seedlings at the time of transplanting.
- ✓ Heavy rain, heavy dew, flooding, deep irrigation water.
- ✓ Severe wind, temp. $25^{o} – 30^{o}$ C.
- ✓ Application of excessive nitrogen, especially late top dressing.

Disease cycle:

- ✓ The pathogen survives in soil in infected stubbles and on collateral hosts like *Leersiahexandra, Cyperus rotundus, Cenchrus ciliaris, Cynadon dactylon*, *Plantago major*.
- ✓ The bacterium is also present in seed husk as well as in the endosperm.
- ✓ Secondary spread is through irrigation water and wind borne as well as through rain storms. The bacterium enters into the host through hydathodes, wounds and stomatal openings or through injuries in roots or leaves.
- ✓ The bacterial ooze also serves as secondary inoculum and cause secondary infection.



Management:

- Use disease free healthy seeds.
- Avoid clipping of tip of seedling at time of transplanting.
- Avoid flood irrigation.
- Remove weed hosts.
- Use optimum doses of nitrogenous fertilizers.
- Seed soaking in streptocyline (3 g/10 lit of water) for 8 hrs.
- Seed soaking in agrimycin (0.025%) followed by hot water treatment at 52^{o} - 54^{o} C for 10 minutes.
- Treating seeds with hot water 52^{o} C for 10 min.
- Spray streptocycline (agrimycin or phytomycin) 1 g + copper oxychloride 30 g /10 lit.
- Use resistant varieties-TKM-6, IR-42, IR-24, IR-28, Ratna, Sathi, Ambika.
- Affected stubbles are to be destroyed by burning or through ploughing.

(3) Brown leaf spot

C.O. : *Helminthosporium oryzae*

Class : Deuteromycetes
Order : Moniliales
Family :*Dematiaceae*

Per. Stage : *Cochliobolus miyabeanus*

Economic Importance

- ✓ This is major disease of paddy occurring in almost all the rice growing areas of the world. In, India it occurs more or less every year in mild to severe form, occasionally taking epiphytotic form.
- ✓ The disease was also one of the principal cause of the famous Bengal famine of 1943.
- ✓ The first report of disease made in India from Madras in 1919 by Sundararaman and now is reported from all of the rice growing states.
- ✓ This fungus produces terpenoid phytotoxins called **ophiobolin** or **cochliobolin.**
- ✓ The disease causes 5% yield loss across all lowland rice production in South and Southeast Asia. Severely infected field can have as high as 45% yield loss.

Symptoms:

- ✓ Infected seedlings have small circular yellow brown or brown lesions that may girdle the coleoptile and distort primary and secondary leaves.
- ➢ The fungus attacks the crop from seedling in nursery to milk stage in main field. Symptoms appear as lesions on the coleoptile, leaf blade, leaf sheath and glumes, being most prominent on the leaf blade and glumes.
- ➢ The disease appears first as minute brown dots, later becoming cylindrical or oval to circular. The several spots coalesces and the leaf dries up.
- ➢ The seedlings die and affected nurseries can be often recognized from a distance by their brownish scorched appearance. Neck region become brown or grayish brown. Infected grain become shriveled and discoloured.

- Low percentage of germination is obtained with the infected grains.
- The disease is associated with a physiological disorder known as **akiochi** in Japan. Abnormal soil conditions (Deficiency of potassium) predispose the plants to heavy infection.

Favourable conditions:

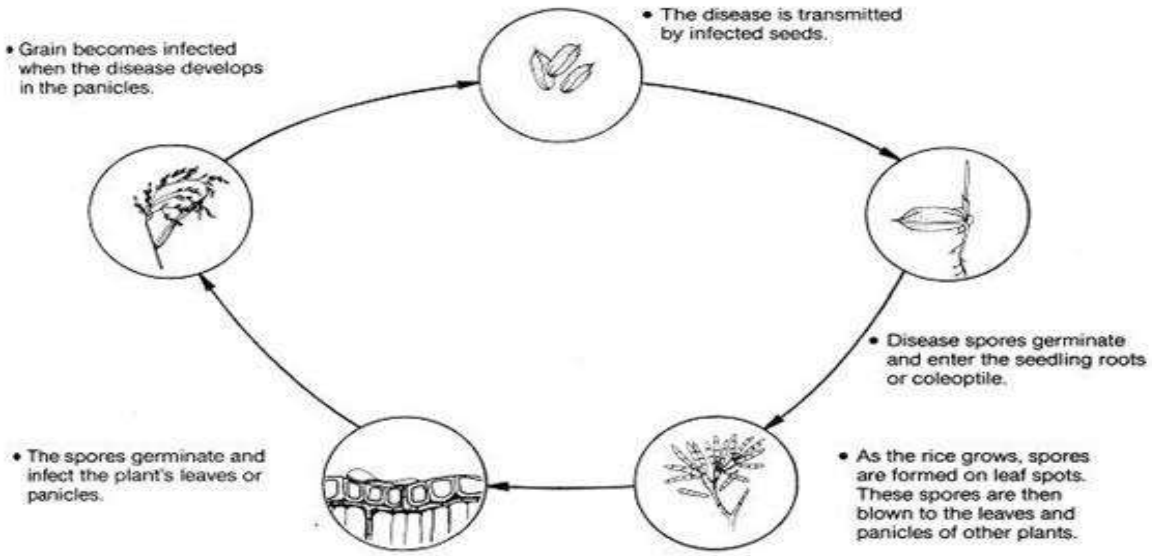
- Cloudy days.
- Heavy monsoon.
- Temp. 25-30^0 C.
- Excessive application of N_2 fertilizers.
- Humidity 90-95% and above.

Pathogen

- The fungus produces septate mycelium.
- Conidiophores arise singly or in small groups, geniculate and brown in colour.
- Conidia are usually curved with a bulged center and tapered ends.
- They are pale to golden brown in colour and are 6-14 septate.
- Peritheciawith asci containing 6-15 septate, filamentous or long cylindrical hyaline to pale olive green ascospores.

Disease cycle:

- **Primary source of inoculum: Soil, stubbles and infected seeds**. The fungus overwinters mainly in the infected plant parts. It is not soil borne.
- The infected seeds are the most common source of primary infection. The fungus also survives on collateral hosts like *Digitaria sanguinalis, Leersia hexandra, Echinochloa colonum, Pennisetum typhoides, Setaria italic*a and *Cynodon dactylon*.
- **Secondary source of inoculum: Wind borne conidia.**



Management:

- Destruction of stubbles and grass hosts from the field.
- Use disease free seeds.
- Cleaning of bunds.
- Proper use of N_2 fertilizers.
- Seed treatment with thiram or captan @ 2-3 g/kg seeds.
- Spraying of chlorothalonil @ 0.2% or mancozeb @ 0.25% or propiconazole @ 0.005%.
- Treat seeds with hot water (53-54° C) for 10-12 minutes before planting, to control primary infection at the seedling stage. To increase effectivity of treatment, pre-soak seeds in cold water for 8 hours.
- Growing resistant varieties- Orissa T-141, Navagam-19.

(4) False Smut or Green Smut

C.O. :*Ustilaginoidea virens*

Class : Deuteromycetcs

Order : Moniliales

Family :*Dematiaceae*

Syn. :*Claviceps oryzae*

Economic Importance

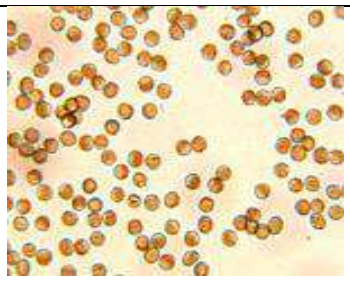
- This disease is known to occur in all the rice growing areas of the world.
- In India, false smut was first reported by Cooke in 1878 from Tirunelveli in Tamil Nadu.
- The disease affects the early flowering stage of the rice crop when the ovary is destroyed.
- The second stage of infection occurs when the spikelet nearly reaches maturity.
- It also causes a reduction in seed germination of up to 35%.
- In India, yield loss of 7-8% was observed.

Distribution:

Japan, U.S.A., Philippines, India, Burma, Sri Lanka, and China. There was severe epidemic in Burma in 1935.It is more prevalent in Eastern Part of India.

Symptoms:

- The disease appears on the ears only.
- Individual ovaries are transformed into large velvety yellow or green masses.
- Only few spikeletes in the ear are affected.
- The glumes are not affected but are covered superficially by the yellow or green mass of spores.

	
False smut on ear	**Smut spores**

- The infected ovaries become yellowish brown which releases the spores of false smut.

Favourable conditions:

- High relative humidity
- Temperature ranging 25-35° C
- Rainfall and cloudy weather during the flowering and maturity periods.

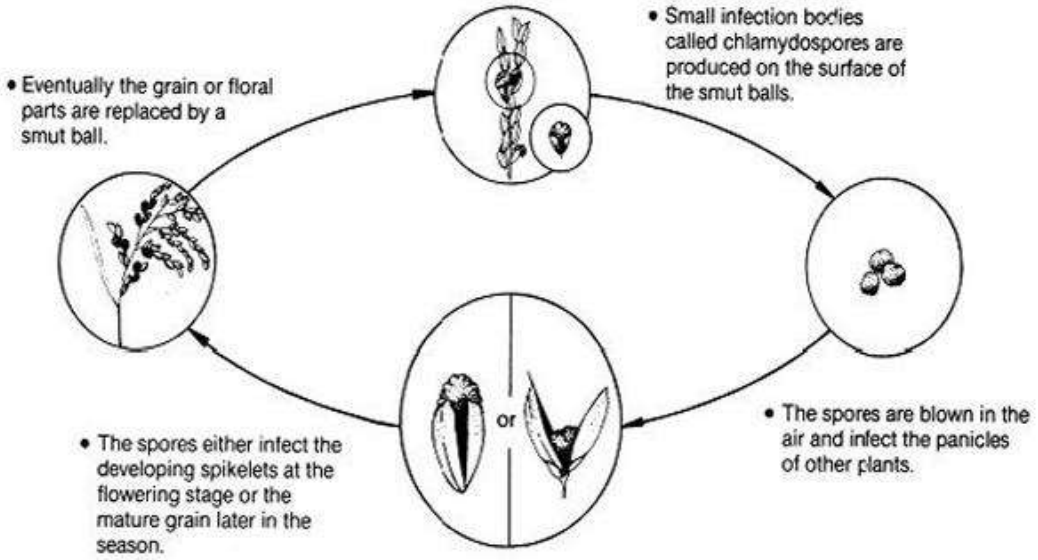
Pathogen

- The mycelia filling in the ovary are united, fine and colorless and they develop conidia pseudosclerotial and sclerotial stages.
- The conidia are spherical, echinulate, olivaceous in color and measure 4-6 x 25 µm.
- The conidia germinate in water giving rise to short germ tube, 1-3 secondary conidia are produced on each germ tube usually after 12-24 hours.
- The secondary conidia are subglobose or globose to oblong, hyaline, granulate and measure 4-8 x 2-5 µm.
- Late in the season, the conidial masses in smut balls harden and form pseudosclerotia.
- The sclerotia are formed in each green ball.
- The sclerotia are hard, variously shaped, and 5-13 x 2-5 mm in size.
- The sclerotia germinate to produce stipes bearing perithecial stroma.
- Perithecia are ovate to pyriform measuring 150-430 x 86-193 µm.
- They bear hyaline, elongated, cylindrical and each containing eight ascospores.
- Ascospores are hyaline, unicellular: germinate in water giving rise to 1-2 germ tubes, which bear secondary conidia.

Disease cycle:

- The fungus survives the winter through sclerotia as well as through chlamydospores. Ascospores produced on the over wintered sclerotia apparently start primary infection.
- Primary infection is believed to be caused by ascospores produced from the sclerotia produced on grasses and wild rice.
- The disease is not seed borne.
- The primary inoculums also come from the collateral hosts.
- It is considered however, that conidia play an important role in causing secondary infection during the growing season.

- Chlamydospores are air-borne which can cause infection in flowers at flowering time.



Management:

- Field sanitation, collection and destruction of smutted ear heads from field.
- Destruction of wild collateral hosts.
- Treat seeds with hot water (52° C) for 10 minutes.
- Seed disinfections with copper oxychloride is reported to reduce the incidence.
- Spraying of copper hydroxide at 2.5 g/l or propiconazole at 1.0 ml/l of water.

(5) Sheath blight:

***C.O.: Rhizoctonia solani* Kuhn.**

(Deuteromycotina: *Mycelia sterilia*)

Perfect stage: *Thanetophorus cucumeris*

- Vernacular Name: *Snake skin disease*

Economic Importance

- Sheath blight was first reported from Japan in 1910 by Miyake.
- The disease known to occur in most rice growing countries like India, Sri Lanka and China etc.
- **In** the United States, a yield loss of 50% was reported when susceptible

cultivars were planted.

- Sheath blight is a fungal disease, more common in rainy season than in dry season.
- This disease is problematic in areas where irrigation facilities are abundant.
- Due to blighting of the leaf sheaths, it is commonly called as sheath blight.
- Sheath blight occurs on leaves and lower portions of the plant and will not interfere with panicle exertion.

Symptoms:

- Initial symptoms are noticed on leaf sheaths near water level.
- On the leaf sheath oval or elliptical or irregular greenish grey spots are formed.
- As the spots enlarge, the centre becomes greyish white with an irregular blackish brown or purple brown border.
- Lesions on the upper parts of plants extend rapidly coalescing with each other to cover entire tillers from the water line to the flag leaf.
- The presence of several large lesions on a leaf sheath usually causes death of the whole leaf, and in severe cases all the leaves of a plant may be blighted.
- The infection extends to the inner sheaths resulting in death of the entire plant.
- Plants heavily infected in the early heading and grain filling growth stages produce poorly filled grain, especially in the lower part of the panicle.
- The fungus affects the crop from tillering to heading stage.

Lesions on leaves	**Lesions on leaf sheaths**	**Panicle exertion**

Favourable condition:

- High temperature (28-32°C)

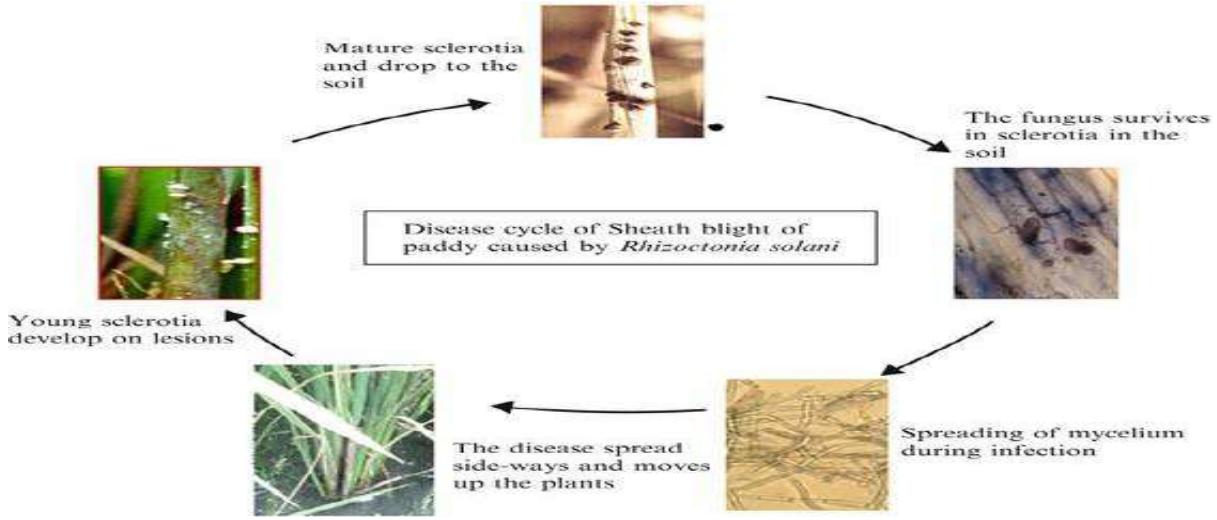
- High relative humidity (>96%)
- Frequent rainfall
- High doses of N
- Close planting

Pathogen

- The fungus produces septate mycelium which are hyaline when young, yellowish brown when old.
- **It** produces large number of spherical brown sclerotia.

Disease Cycle

- The pathogen has a wide host range *viz*,sugarcane, bean, soybean, tomato, eggplant, tobacco, water hyacinth and green gram
- The pathogen can survive as sclerotia or mycelium in infected plant debris in soil.
- Sclerotia spread through irrigation water.



Disease cycle of sheath blight of paddy

Management

- Deep ploughing in summer and burning of stubbles/ weeds, *etc*.
- Moderately Resistant Varieties: Swarnadhan, Vikramarya, Radha, Pankaj, Manasarovar, Mandya Vijaya.
- Avoid using infected seed.
- Apply moderate 'N' levels (80-100 kg/ha) in 3-4 splits.
- Avoid excess 'N', skip final 'N' in sheath blight infected fields.
- Apply organic amendments *viz.*, neem cake @ l50 kg/ha or FYM 12.5 tons/ha.

- Soil application of *P. fluorescens* @ 2.5kg/ha mixed with 50 kg of FYM after 30 days of transplanting.
- Avoid flow of irrigation water from infected fields to healthy fields.
- Seed treatment with carbendazim 2.0g/kg of seeds.
- Spraying fungicides of 1g carbendazim 50% WP or 2.0 g mancozeb 75WP or 1 ml hexaconozole in 1 liter of water.

(6) Tungro

C.O.: Rice tungro baciliform virus (RTBV) and
Rice tungro spherical virus (RTSV).

Economic Importance

- Tungro is one of the most damaging and destructive diseases of rice in South and Southeast Asia.
- In severe cases, Tungro susceptible varieties infected at an early growth stage could have as high as 100% yield loss.

Symptoms

- Plants affected by tungro exhibit stunting and reduced tillering.
- Leaves become yellow or orange-yellow, may also have rust-colored spots.
- Discoloration begins from leaf tip and extends down to the blade or the lower leaf portion
- Delayed flowering - panicles small and not completely exerted.

	➢ Most panicles sterile or partially filled grains. ➢ Tungro virus disease affects all growth stages of the rice plant specifically the vegetative stage.
Nephotettix virescens **Affected plant**	

Pathogen

- Two morphologically unrelated viruses present in phloem cells.
- The disease complex is associated with rice tungro baciliform virus (RTBV) bacilliform capsid, circular ds DKA genome and rice tungro spherical virus (RTSV)isometric capsid, ssRKA genome.RTBV cannot be transmitted by leafhoppers unless RTSV is present. Rice tungro bacilliform virus (RTBV),

- RTBV particles are rod-shaped and 100-300 nm in length and 30-35 nm in width.
- RTSV particles are isometric and 30 nm in diameter.

Favourable condition:

- Presence of the virus sources.
- Presence of the vector.
- Age and susceptibility of host plants.
- Synchronization of the three above factors.
- All growth stages of the rice plant specifically the vegetative stage

Disease cycle:

- The most efficient vector is the green leafhopper *Nephotettix virescens*.
- Leafhoppers can acquire the viruses from any part of the infected plant by feeding on it even for a short time.
- It can, then, immediately transmit the viruses to other plants within 5 to 7 days.
- The viruses do not remain in the leafhopper's body unless it feeds again on an infected plant and re-acquires the viruses.
- Seedlings raised in nursery can also infected with Tungro prior to transplanting and can be a primary source of virus.

Management

- Light traps are to be set up to attract and control the leaf hopper as well as to monitor the population.
- Spray systemic insecticide to manage the vector.
- Use Resistant varieties like IR 36, IR 50, ADT 37, Ponmani, Co 45, Co 48, Surekha, Vikramarya, Bharani, IR 36 and white ponni
- Adjusting the date of planting.
- Plouging and harrowing the field to destroy stubbles right after harvest in order to eradicate other tungro hosts.
- Destruction of weed hosts on bunds.

(7) Khaira disease of rice (Zinc deficiency):

Cause: The disease is caused due to **zinc deficiency** in soil or non-availability of zinc to plants.

First report: It was first reported from rice growing areas of 'Tarai Region' from Uttaranchal (India) by **Y. L. Nene.**

Symptoms:

- The disease appears 10-15 days after transplanting.
- Leaves of diseased plant show chlorosis at the base.
- Large number of small brown or bronze spots appear on the lamina.
- The spots get coalesce to form bigger spots and the entire leaf turns bronze coloured and dries.
- Plant shows stunting. The root growth is restricted and main root turn brown. The finer roots are destroyed. In severe cases plants fail to grow further and produce ears.
- Sometimes plants recover naturally and some of them produce ears with few grain.

Management:

- Two sprays of a mixture of zinc sulphate ($ZnSo_4$) 5 kg + 2.5 kg of slaked lime in 1000 litre of water per hectare.
  1^{st} spry given as soon as the symptoms of deficiency noticed
  2^{nd} spray is given 10 days later.
- Incorporate zinc sulphate ($ZnSo_4$) 25-30 kg/ha at every three years.

…..x…..x…..x…..

14. Tobacco (*Nicotiana tabacum*)

(1) Damping off of Tobacco

Economic importance

It is a common disease occurring in tobacco nurseries. The disease causes extensive damage in tobacco tracts of Andhra Pradesh, Maharashtra and Gujarat. The damage in some areas can be cent per cent, in the absence of protective sprays.

C.O. : *Pythium aphanidermatum*

Class : Oomycetes

Order : Peronosporales

Family : *Pythiaceae*

Distribution: China, Indonesia, Africa, South America, U.S.A., Mediterranean region.

Symptoms:

Disease may appear at any stage in nursery.

The disease occurs in two stages ie. Pre-emergence and post-emergence phase.

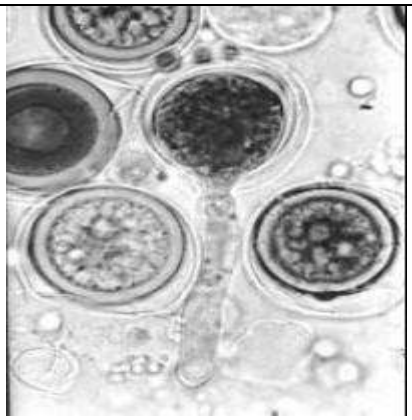
I. Pre-emergence damping-off

- Failure of seedling to emerge after sowing.
- Sprouted seeds when infected, wither before emergence from the soil.
- If the shoot or root emerges it has a dark necrosis areas.

II. Post-emergence damping-off

- It is the most destructive phase.
- Water soaked minute lesions appear on the stem near the soil surface.
- Girdling of stem takes place spreading the infection up and down.
- The infected seedling topples over soil.
- The young seedling in the nursery are killed in patches
- Under favorable conditions the entire seedling killed within 3 to 4 days.
- The affected seedling show shriveling and brown discoloration of the stems near the soils and start rotting.
- A cottony white myceliummay be seen on the tissue.

Sprouted seedlings are infected and wither		The young seedling in the nursery are

before emergence from the soil.		killed in patches
Pre-emergence damping off		**Post-emergence damping off**
		
Oospores		**Stem girdling**

Favourable conditions:

- Overcrowding of seedlings.
- Ill drained nursery beds.
- Heavy shade in nursery.
- High atmospheric humidity 90-100%.
- High soil moisture.
- Low temperature below 24^{o} C & Low soil temperature of about 20^{o} C.

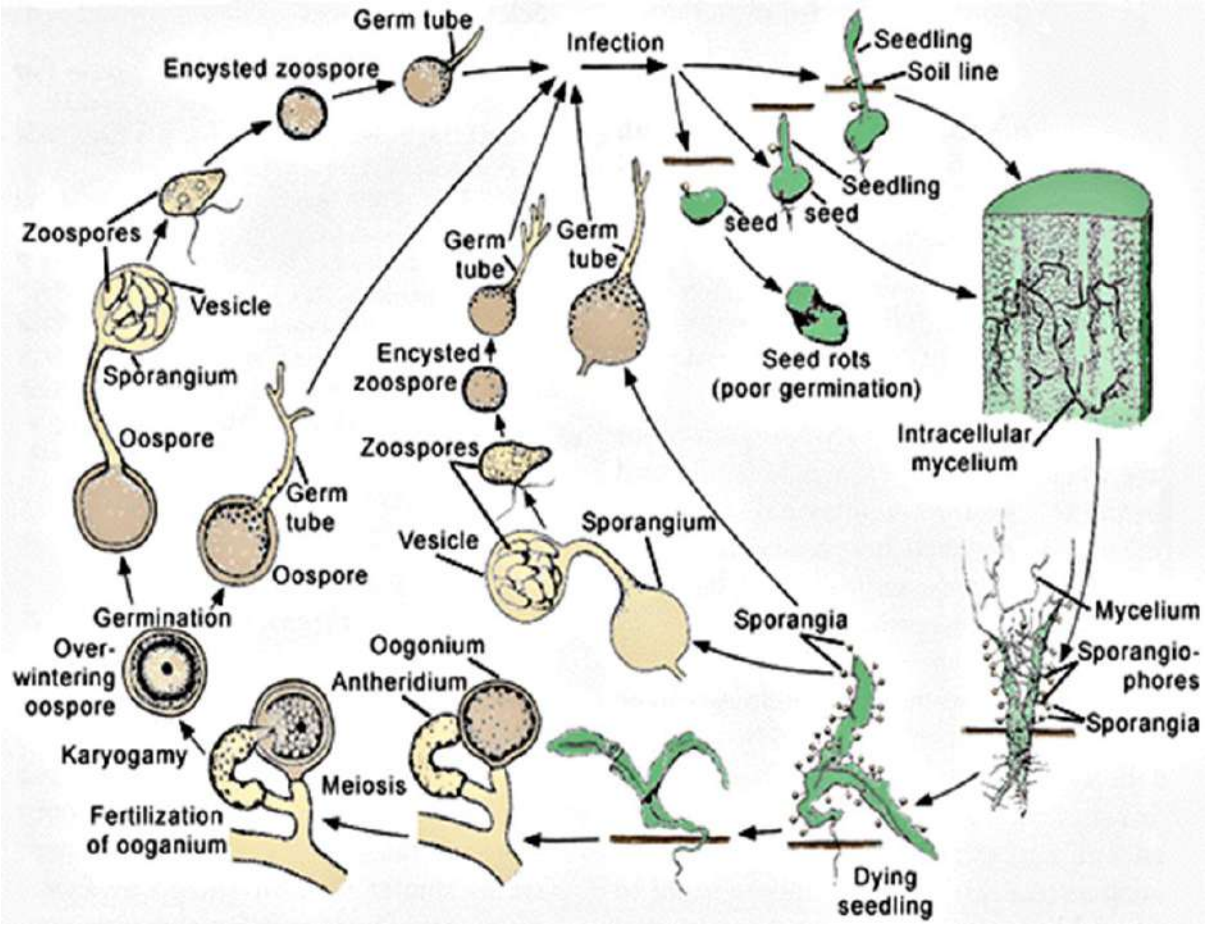
Pathogen:

- ✓ Thick, hyaline, non-septate mycelium.
- ✓ It produced irregularly lobed sporangia, which germinate to produce vesicle containing zoospores.
- ✓ The zoospores are kidney shaped & biflagellate.
- ✓ Zoospores are spherical, light to deep yellow or yellowish brown coloured measuring 17-19 µm in diameter.

Disease cycle:

- ✓ The fungus survives in the soil as oospores and which acts as primary source of infection.

- ✓ The secondary spread is through zoospores disseminated through rain/ irrigation water.
- ✓ The fungus is capable of living for many years in soil, completing its life cycle both saprophytically and as a facultative parasite.



Disease cycle of Damping off of Tobacco

Management:

- ➢ Prepare raised seed beds with adequate drainage facility.
- ➢ Rabbing - slow burning of paddy husk before nursery sowing on seedbeds @ 7 kg/ sq.mt.
- ➢ Drenching the seed bed with 1% B.M. 3-4 lit/m^2 or 0.2% copper oxychloride, two days before sowing.
- ➢ Avoid overcrowding of seedlings (1 to 1.5 g/2.5m^2).

- Drenching with metalaxyl MZ 72% WP 0.2% (8 g/l) @ 3-4 liter/m^2.
- Avoid excess watering to the seedlings.
- Soil solarization in hot summer (May) with 100µ LLDPE transparent plastic films for 15 days, which controls pathogen, weeds, nematodes and other harmful microorganisms.
- Spray nursery beds with 0.2% mancozeb or 0.3% COC at 5 days interval two weeks after sowing or spray 0.2% metalaxyl at 10 days interval commencing after 20 days after germination.

(2) Black shank:

C.O.: ***Phytophthora parasitica* var. *nicotianae***

Class: ***Oomycetes***

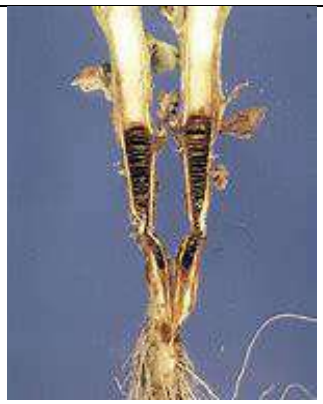
Order: ***Peronosorales***

Family: ***Pythiaceae***

- The disease was first reported from Java in 1893. Now, the disease found in many countries of the world.
- In India, the disease occurs in Gujarat, Karnataka and Tamil Nadu.

Symptoms:

- The pathogen may affect the crop at any stage of its growth.
- The diseased plants show characteristic black lesions on the stem.
- Starting as minute black spots it spreads along the stem causing irregular patches and often girdling it.
- The lesions also spread downward to cover the roots.
- The infected tissues shrink, leaving a depression, and as the disease advances, the stem shrivels and the plant wilts.
- When the stem is split open and examined, the pith region is found to be dried up in disc-like plates.

		
Black lesions on the stem	Split open pith region dried up in disc-like plates.	Blighted leave

- In some cases the pathogen may attack the leaves, producing large water soaked spots, which enlarge to blight the leaves.
- The disease may appear sporadically, affecting a few plants in the field but during rainy and humid weather it spreads rapidly to a majority of the plants in the field.

Favourable condition:

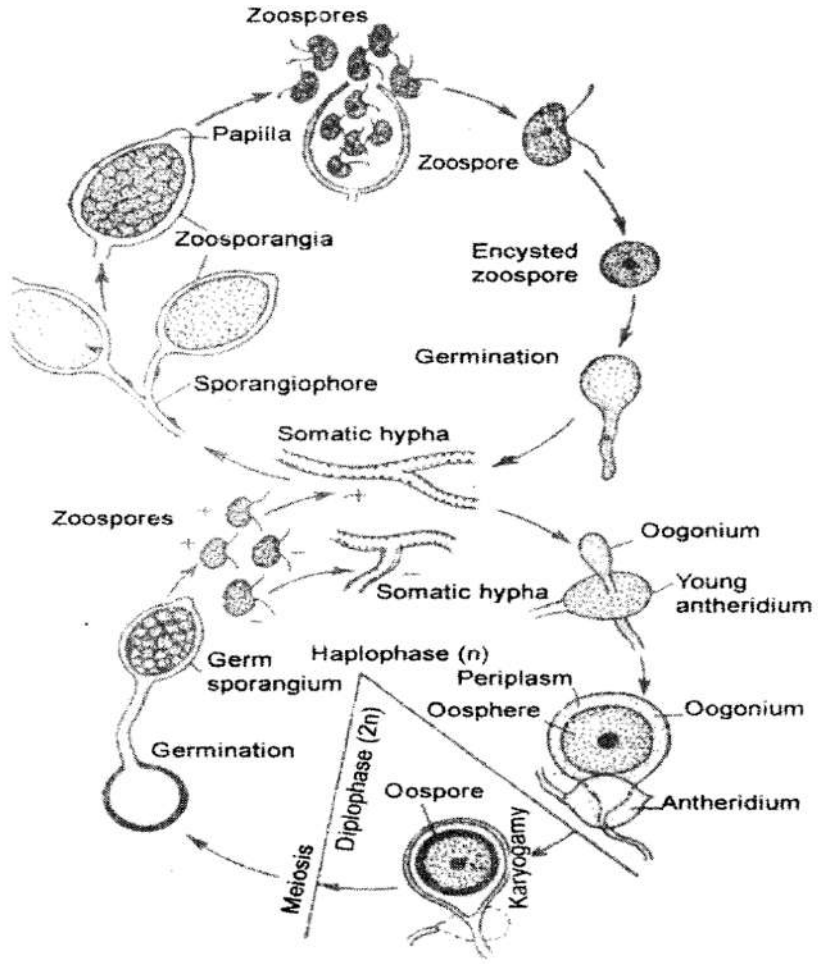
- Frequent rainfall.
- High soil moisture.

The pathogen:

- The pathogen produces hyaline and nonseptate mycelium.
- The sporangia are hyaline, thin walled, ovate or pyriform with papillae develop on the sporangiophores in a sympodial fashion.
- Sporangia germinate to release zoospores which are generally biciliate, kidney shaped measured 11-13 x 8-9 µm.
- The fungus also produce thick walled chlamydospores measuring 27-42 µm in diameter.
- Oospores are thick walled, smooth, globose and light yellow coloured measuring 15-20 µm in diameter.

Disease cycle:

- The pathogen is present in the soil as dormant mycelium, oospores and chlamydospores for more than two years.



- The pathogen lives as a saprophytes on organic waste and infected crop residue in the soil.
- Primary infection is by means of oospores and chlamydospores in the soil.
- Secondary spread is by wind borne sporangia.
- The pathogen in the soil spreads through irrigation water, farm implements and animals.

Management:

- The plant residues and debris must be collected and burnt.
- Drenching the soil with Cheshunt compound (It contains two parts of $CuSO_4$ and 11 parts of Ammonium carbonate) before planting.
- Spray the beds 15 days after sowing with metalaxyl 0.2% or copper oxychloride 0.3% or Bordeaux mixture 0.3% and repeatat fortnightly intervals.

- Provide adequate drainage to prevent water from stagnating in the field.
- Use resistant varieties such as K 20, BSF-1, 2, 3 and 4.

(3) Frog Eye Leaf Spot:

C.O. : *Cercospora nicotianae*

Class : Deuteromycetes

Order :Moniliales

Family :*Dematiaceae*

Economic importance:

- The disease occurs in the seed-beds nursery plant as well as in standing crops in the main fields.
- The severity of disease increased during monsoon season.

Symptoms:

- On lower and more mature leaves brown spots with ash grey centers are observed.
- The spots has a white center, surrounded by grey and brown portions and dark brown to black margin resembling frog's eye at early stages.
- Several spots coalesce to form large necrotic areas, causing the leaf to dry up from margin and wither prematurely.
- Yield and quality reduced greatly
- The disease may occur in the seedlings, leading to withering of leaves and death of seedling.

Elongated conidia	**Greyish spots surrounded by brown border with yellow halo seen on lower leaves**

Favourable conditions:

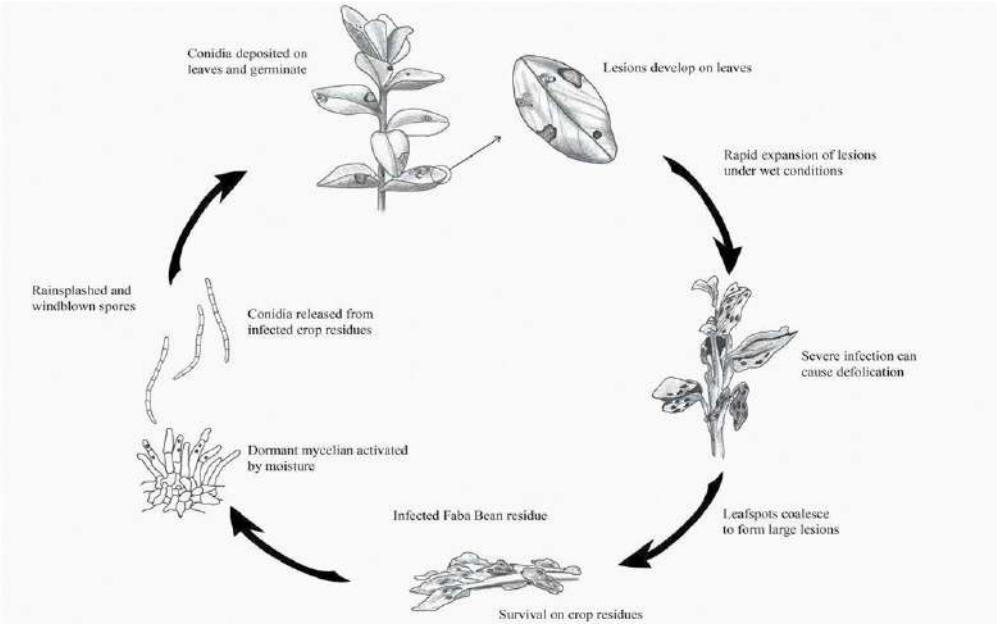
- ✓ Temperature 20°-30° C.
- ✓ High relative humidity 80-90 %.
- ✓ Close spacing and application of frequent irrigation.
- ✓ Excess application of nitrogenous fertilizers.

Pathogen:

- ➢ The mycelium is intercellular and gathered below the epidermis and clusters of conidiophores emerge through stomata.
- ➢ The conidiophores are septate, dark brown at the base and lighter towards the top bearing 2-3 conidia.
- ➢ The conidia are slender, slightly curved, thin walled, hyaline and 2-12 septate.

Disease cycle:

- ➢ The fungus is seed borne and also persists on crop residue in the soil.
- ➢ The primary infection is from seed & soil borne inoculum.
- ➢ The secondary spread is through wind borne conidia.
- ➢ When the infected seedlings are transplanted in field may spread the disease.



Management:

- ➢ Spraying the crop with Bordeaux mixture 0.6% or benomyl 0.1% or thiophanate methyl 0.1% or carbendazim 0.1% and repeat after 15 days.
- ➢ Remove and burn infected plant debris.

- Avoid excess nitrogenous fertilization.
- Adopt optimum spacing, regulate irrigation frequency.

(4) Tobacco Mosaic Virus

C.O.:Tobacco Mosaic Virus (TMV)/*Nicotiana virus*-1or *Marmor tabaci* var*.*

vulgare.

- ✓ Tobacco mosaic is first recognized virus disease in plant world by **Adolf Mayer** in 1882.
- ✓ The virus occurs on many other host plants and over 20 different virus diseases reported on tobacco.

Economic significance:

TMV infects many plants and yield losses in gourd, solanaceous, and leguminous crops can reach 20-70%.

Pathogen:

- It is a rod shaped particle measuring 300 x 18 nm in size.
- The virus is capable of remaining infective when stored dry for over 50 years.
- Thermal death point (TDP) is 90^{o} C for 10 min. and Dilution end point (DEP) is**1: 1,00,000.**

Thermal Death Point:

It is the temperature at which organism loses its infectivity when maintained at that temperature for 10 minutes.

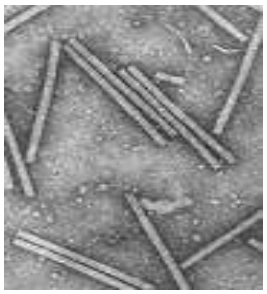
Dilution end point (DEP):

It is the maximum point of dilution which can transmit the disease.

Symptoms:

- Light discoloration along the veins of the youngest leaves.
- Soon the infected leaves develop a characteristic light and dark green patterns showing mosaic symptoms.

- The dark green areas later develop into irregular crumpled swelling or blisters.
- The plant infected early in the season is usually stunted with small, chlorotic, mottled and curled leaves.
- In severe infection, the leaves are narrowed, puckered, thin and malformed.

		
Rod shape virus	**Mosaic symptoms**	**Narrowed, puckered, thin and malformed leaves.**

Favourable condition:

Temperature below 40° C, TMV sustain its stable form.

Disease cycle:

- TMV have no distinct overwintering structure.
- The virus remains viable in the plant debris in the soil.
- The virus has a wide host range affecting nearly 50 plant species belonging to nine different families.
- It is sap transmissible and enters in the host through wounds.
- The virus is not seed-transmitted in tobacco but tomato seed transmit the virus.
- In field the farm workers engaged in topping and clipping operationstransmit the disease.
- The implements used in field also transmit the virus.

Management:

- Removal and destruction of infected plants
- Keep the field weed free which harbor the virus.

- Avoid hand contamination by washing hands with soap and running water before and after handling the plants.
- Prohibit smoking, chewing or snuffing during field operations.
- Spray nursery with leaf extracts of *Bougainvillea* @ 1 lit of extract in 150 liters of water, 2 to 3 times at weekly intervals.
- Follow crop rotation by growing non-host crops for two seasons.
- Grow resistant or tolerant varieties like CTRI special, Jayasree, Virginia, Tobacco 1158, Prabhat, Gautami, Blankat 1, Godavari special, TMV RR-2, 3, 4, 6.

(5) Tobacco Leaf curl

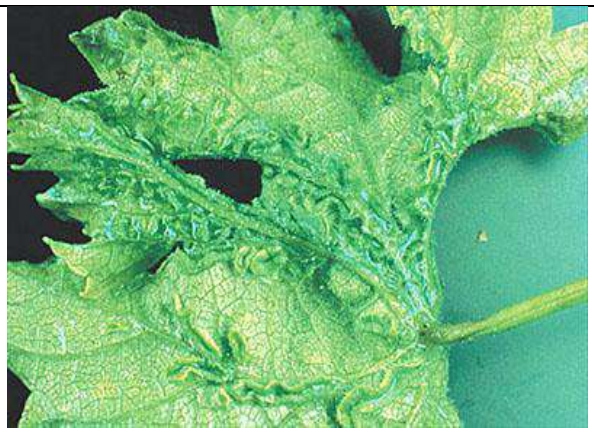
C.O.:*Nicotiana virus* 10 (*Ruga tabaci*)

Economic importance:

This disease is widespread in India and occurs in severe forms affecting up to 15 per cent of plants in Punjab. In South India it is reported only sporadically in some areas.

Symptoms:

- The disease usually appears in the field about 4-6 weeks after transplanting.
- Disease is characterized by the downward curling of young leaves i.e. leaf margins turn downwards.
- Curling of leaves with clearing and thickening of veins,twisting of petioles and puckering of leaves with enations (leaf like out growths) on the lower leaf surface.
- As the disease advances the plant becomes dwarf and most of the leaves are curled.
- Inflorescence is greatly condensed and veins of calyx thickened.

	
Downward curling of young leaves	**Thickening of veins with enations on the lower leaf surface.**

Pathogen:

Tobacco leaf curl virus (TLCV) belongs to *Geminiviridae*, sub-group III, of Geminivirus genus. The virions are geminate, non-enveloped, circular ssDNA genome, spherical measuring 18 nm in diameter.

Disease cycle:

- The virus has wide host range of 63 crop species belonging to 14 families.
- Virus is not seed or sap transmissible.
- The whitefly, *Bemisia tabaci* is the vector responsible for transmission in field.

Host: Tomato, Sunhamp, Zinnia, Petunia, Chilli, Datura, Papaya, Euphorbia,

Ageratum, *Solanum nigrum* etc.

Management:

- Remove and destroy infected plants.
- Rogue out the reservoir weed hosts which harbour the virus and whiteflies.
- Avoid to grow solanaceous crops like tomato near tobacco fields.
- Spray methyl-o-demeton @ 0.2% to control the vectors.

(6) Orobanche/Broom Rape:

C.O.: *Orobanche cernua* var. *Desertorum*

Economic importance:

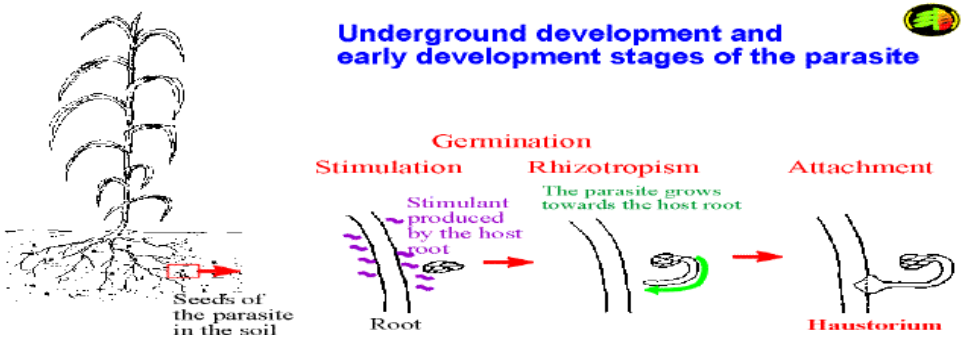
- It is a Phanerogamic plant parasite popularly known as Broomrape.
- It is known by different names in different parts of India such as Tokra in North India, Vakumba in Gujarat and Bambaku in Maharashtra, Pokayilaikalan in Tamil Nadu, Bodu or Malle in Andhra Pradesh.
- It is a holoparasite and draws its nourishment from tobacco by means of haustoria attached to the roots of tobacco.
- In india, the loss in yield and quality of leaf ranging from 10-50%.

Symptoms:

- After 5-6 weeks of transplanting the parasite emerges out near the base of plant.
- Infected plant show stunting, withering and dropping of leaves leading to wilting.
- As many as 50-100 such parasites observe near the base of single tobacco plant.
- Infected plant markedly reduced in size and chlorosis of the leaves is prominent.

Parasite:

This is an annual fleshy flower plant, growing up to 30 cm height with cylindrical stem, pale in colour. The flowers are white and seed are minute. It is complete root parasite.A single broomrape plant can release more than 5,00,000 seeds, which are known to remain viable for decades in the soil. The seeds are extremely small, black, reticulate and ovoid measuring 200-400 µm formed in fruit capsules.



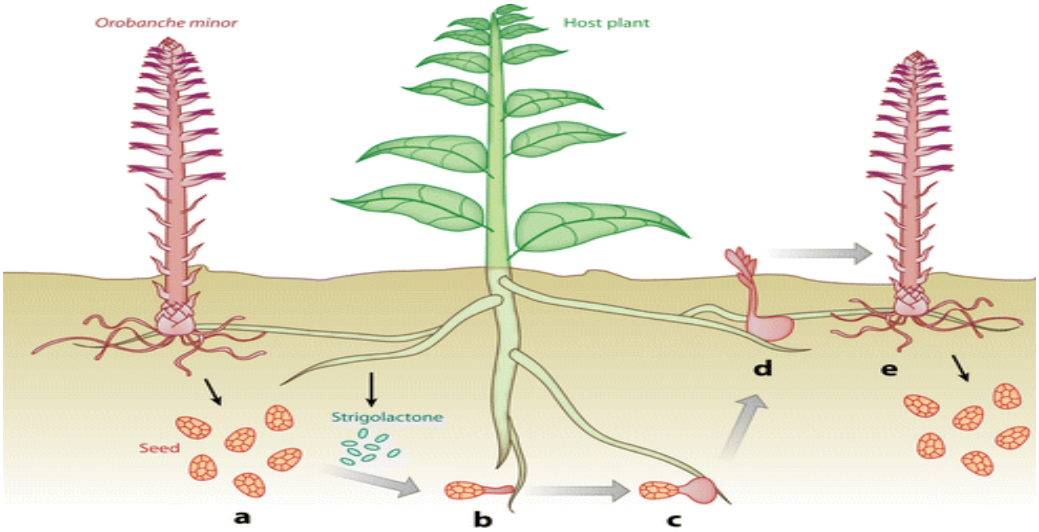
Disease Cycle:

- The seed of parasite remain dormant in the soil for several years.
- The seeds in soil act as primary source of infection.

- The seeds are also disseminated in field through animals, human beings, irrigation water and implements.
- The dormant seeds are stimulated to germinate by the root exudates of tobacco and attaches itself to the roots by forming haustoria.
- The parasite rapidly produces the shoot and flowers when the seeds are set, they shed to the ground & mix with soil.

Collateral Host:

Brinjal, tomato, cauliflower, turnip and other cruciferous plants.



Disease cycle of Broom Rape / Orobanche

Management: -

- Rouge out the tender shoots of the parasite before flowering and seed set.
- Spray the soil with 25% copper sulphate.
- Spray 0.1% Allyl alcohol.
- Apply few drops of kerosene directly on the shoot.
- Grow trap crops like chilli, sorghum, mothbean, cowpea for stimulating seed germination and kill the parasite before transplanting tobacco.

CTRI–Central Tobacco Research Institute, Dist. East Godavari, Rajahmundry, Andhra Pradesh – 533 105.

…..x…..x…..x…..

15. COCONUT (*Cocosnucifera*L.)

1. Bud rot C.O.:*Phytophthorapalmivora*
2. Stem bleeding C.O.:*Ceratocystisparadoxa*
3. Wilt C.O.: *Rhizoctonia solani, Botrydiplodia theobromae*
4. Ganoderma stem rot C.O.: *Ganodermalucidum, G. applanatum*

- Coconut is one of the important oilseeds grown in India.
- The trees come to bear in 4-5 years and continue for about 50-60 years, the peak production period being during 10-40 years after planting.
- Coconut tree suffering from 53different pathogens.

1. Bud rot

C.O.:*Phytophthorapalmivora*

- First reported: In Madras 1906 by Butler who named the causal organism *Phytophthorapalmivora*in 1910.

Distribution:

Brazil, East Africa, British Guiana, Ceylon, Fiji Island, Jamaica, Mexico, Nigeria, Philippines, Trinidad and West Indies.

- Losses: 30-40% palm tree are affected due to this disease in a garden.
- Coconut and toddy palm (*Borassusflabellifer*) suffer badly.
- It also infects Palmyrah&Arecanut. Datepalm is immune to the disease. Bud rot affects the palms at various stages of growth.
- As the name indicates, the ultimate effect is rotting of terminal bud due to infection in or near the bud.

Symptoms

- Severe on young palms. Young leaves and shoots are affected by the disease.
- Yellowish green discoloration of the heart leaf or crown leaf.

- The central expanding leaves turn yellowish green and dry.
- The basal tissues of the leaf rot quickly and can be easily separated from the crown. Dark fruit rots and premature loss young nuts.
- Older leaves develop irregular, water soaked spots which are sunken in nature.
- The fruit rot starts as brown to black elliptical lesion with as grey center, mostly from the stem end.

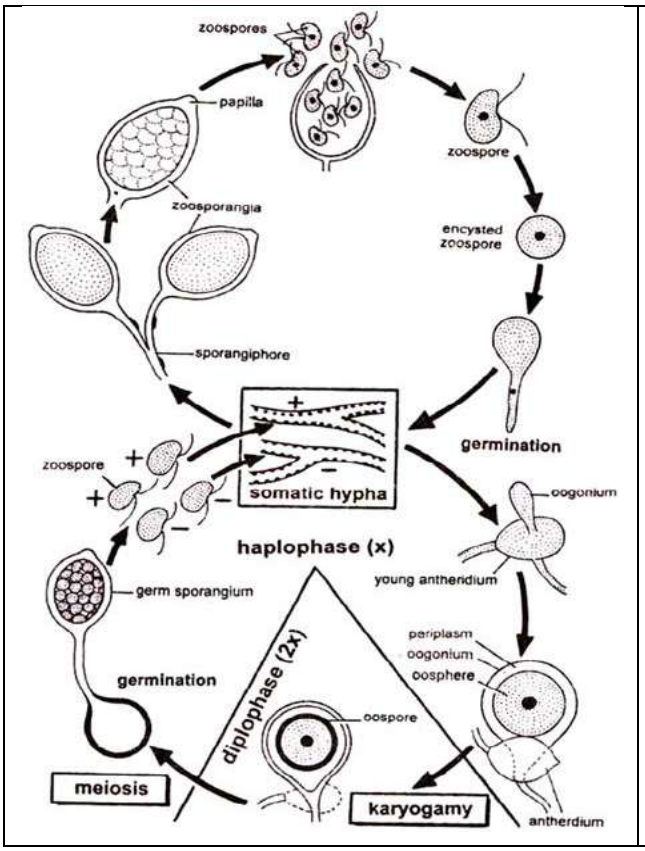
- The withered central shoot can be pulled out very easily from the crown. The central crown may rot and in few months the tree may wilt
- Young nuts fail to mature and fall. In severely affected trees, the entire crown rots and in a few months the entire trees wilt.

Host: Various palms cocao, rubber, castor, safflower and citrus spp.

Pathogen

- The fungus produces intercellur, non-septate, hyaline mycelium.
- Sporangiophores are hyaline and simple or branched occasionally, thin walled, pear shaped with a prominent papillae.
- Sporangia releases reniform, biflagellate zoospores upon germination.
- The fungus also produces thick walled, spherical oospores.
- In addition, thick walled, yellowish brown chlamydospores are also produced.

Disease cycle:



- The pathogen perpetuates in the form of Oospores/chlamydospores in diseased tissues in leaf axils and crown.
- Dormant mycelium oospores or chlamydospores carried over summer months on the host debris. With the onset of monsoon rains, the fungus becomes active producing cottony mycelium that infects tender host tissue.
- The secondary spread takes place through sporangia and zoospores through rain water.
- The rhinoceros beetle (*Orycles rhinoceros*) enhance the disease incidence.

Favourable condition:

- High atmospheric humidity.
- Moisture at the leaf axil level of palm.
- Temp. range of 18-20^{0}C and RH 98-100% in leaf axil of the palm tree are highly conducive for the development of the disease.

Management

- Cutting down diseased trees and burning them.
- Spray 1% Bordeaux mixture or copper oxychloride 50 WP 0.20%.
- Injection of 3.0 g. a. i. of metalaxyl or fosetyl-Al 80% WPon trunk have been found most effective.
- If the disease is detected early remove the infected portions and protect with Bordeaux paste (Tree surgery).
- Spray copper fungicides (B.M @ 1% or COC @ 0.3%) after onset of monsoon to prevent infection.

2. Stem bleeding:

C.O.:*Ceratocystisparadoxa*

Synonyms:*Ceratostomella paradoxa, Ophiostoma paradoxa*

Family: Ceratostomataceae
Order: Sphaeriales
Class: Pyrenimycetes
Division: Ascomycotina
First report: Sri Lanka (1906).
Distribution: India, Malaysia and Philippines. This disease is quite common in South India.
Symptoms:

- On the lower parts of trunk, about 2-3 cm above the soil level, deep reddish brown ooze is coming through the cracks, which dries up to form a black crust.
- The fluid dries up to form black encrustations with brownish orange margins.
- Infection can occur anywhere on the trunk.
- The tissues, except vascular bundles, below infected patch show yellowish brown discoloration and decay.
- Large cavities are formed in diseased stem from which yellowish to whitish fluid oozes out.
- Growth of affected palms is arrested and yield reduced.
- In newly established plants, the infection spreads very rapidly and there is extensive decay of internal tissues.
- In young trees the plant shows wilting within one.

Pathogen:

The fungus produce two types of conidia.

- Macroconidia are spherical and dark green in colour produced on conidiophores, either singly or in chains.
- Microconidia are produced endogeneously inside the long cells of conidiophores and cell ruptures when mature and release the microconidia in long chain.
- The pathogen also produce hyaline perithecia with clavateasci and hyaline ascospores.

Disease cycle:

- The fungus survives in the cracks and cavities in the stem in the form of perithecia and chlamydospores.
- Secondary spread by conidia which is transported by water and insects in the ooze form.

Favourable conditions

- Heavy rainfall followed by drought
- Cracks and insect wounds on stem
- Physiological imbalances
- Poor fertilization and poor maintenance of orchard
- Damage by *Xyleborus, Diocalandra*beetles

Management

- Removing the affected area and cleaning the wound with disinfectant or by using flame.
- Avoid any mechanical injury to the stem.
- Provide proper drainage during rainy season.
- Chisel out affected tissues and dress the wound with hot coal tar or Bordeaux paste.
- Application of Bordeaux paste or mercurial fungicide or hot tar coal on stem.
- To avoid spread of disease on to upper portion of trunk, root feeding with 5% calixin (Tridemorph) may be adopted 3 times a year - April-May, September-October and January-February.
- Along with 50 kg organic manure, apply 5 kg neem cake containing the antagonistic fungi, *Trichoderma* culture to the basin during September.
- Provide adequate irrigation during summer and drainage during rainy season.
- Apply recommended doses of organic manures and chemical fertilizers.

- Coconut stem boring insects like *Xyleborusparvulus, Dicalandrastigmaticolis*should be controlled by applying carbaryl 50% WP on the trunk @ 3g per liter water.

3. WILT

C.O.: *Rhizoctonia solani, Botrydiplodia theobromae*

Loss: It is estimated that about 15 million palm trees affected by the disease and the annual loss amounts to Rs. 300 million.

Symptoms

- Slow wilting of the foliage.
- Sterility of pollen and necrosis of spadix also occur.
- Rotting of roots is an important feature of the disease.
- The quality and quantity of coconuts are adversely affected by the disease.

- In the root rot, the major portion of the root system is destroyed, main and lateral roots start drying from the tip backward.
- The young plants show rapid development of symptoms and succumb within 3-4 years.

Favorable Condition

- The disease is more common in water-logged poorly aerated, heavy soils.
- In sandy and sandy loam soils.
- Water table is high, water lodging cause accumulation of soluble iron, manganese and ammonium nitrogen in the root zone which interferes with uptake of major nutrients.

Management

- The destruction of affected palms in early stage.
- Prevent water lodge condition.
- Improved soil drainages.
- Application 25-50 kg farm yard manure with recommended dose of fertilizers along with 2 kg dolomite or 1 kg lime and 500 g of magnesium sulphate.
- Follow integrated nutrient management apply organic manure. In addition to this Magnesium sulphate also applied along with other fertilizer application.

- Grow green manure crops - cowpea, sun hemp.

4. Ganoderma stem rot

C.O: *Ganodermalucidum, G. applanatum*

- Most serious and destructive disease of coconut.
- Present in the districts of Srikakulum, Visakhapatnam, Vizianagaram, East and West Godavari districts.
- A recurring loss of Rs. 10 lakhs occur every year in A.P. alone.
- The disease is severe in lighter soils and on young palms.

Symptoms

- The most usual symptoms are yellowing, withering and drooping of the outer fronds which remain hanging around the trunk for several months before shedding.
- The fungus infects the roots and spreads upwards killing the entire root system.
- Older leaves droop and wither and remain suspended around the trunk for several months.
- Younger leaves remain green for sometime.
- Tree become barren due to suppression of inflorescence.
- Diseased tree dies slowly and often the stem cracks, giving out a dark brown ooze. The cortical tissues disintegrate and stem turn brown.
- Extensive rotting of roots and peeling of stem tissues. In advanced cases the fungus produces the fruiting structures (**brackets**) along the sides of the basal trunk.
- Diseased tree dies in about two years. The bark turns brittle and often gets peeled off in flakes, leaving open cracks and crevices.
- The internal tissues are discolored and disintegrated, emitting a bad smell.
- Bracket formation at the base of the trunk. *Ganoderma* appears at the base of the trunk. Ultimately the palm dies off.

Disease cycle

- P.I: The fungus is soil borne and survives in the soil for long time.
- Primary infection is through Basidiospores in the soil, which attack the roots.
- Wide host range: Mango, jack, citrus, Arecanut, coffee and tea
- S.I.:Through irrigation/rain water and by root contact (Mostly from March-August)

Management

Preventive measures

- Trenches dug for replanting should be filled with FYM, 5kg neem cake and *Trichoderma viride.*
- Incorporate green manure crops like sunhemp and sesbaniafor moisture retention and multiplication of antagonistic fungi.
- Avoid deep ploughing or digging which are likely to injure roots.
- Arrange separate irrigation to each tree to prevent spread of fungus.
- Apply FYM and neem cake to each tree during June – July months.
- Apply 40 liters of 1% BM in basin of each tree, yearly once.

Curative measures

- Destroy infected palms.
- Apply aureofungin 2g + copper sulphate 1g in 100 ml of water at quarterly intervals.
- Root feeding with tridemorph(6ml in 25 ml of water), 3-4 times in a year, in early stages of infection.
- Control bark eating caterpillar.

16. BANANA (*Musa paradisiaca* L.)

1. **Sigatoka leaf spot**: **C.O:***CercosporamusaeZ*imm.
2. **Bacterial wilt/Moko disease** **C.O:** *Burkholderiasolanacearum*
3. **Panama wilt** **C.O:** *Fusariumoxysporum*f. sp. *cubense*
4. **Bunchy top** **C.O.:** *Musa virus-1*

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1. Sigatoka leaf spot

C.O : Conidial stage: *CercosporamusaeZ*imm.

Perfect stage (Ascospore): *Mycosphaerellamusicola*

Black sigatoka – *Mycosphaerellafijiensis*

Class : Deuteromycetes

Order : Melanconiales

Family :Dematiaceae

Two forms of Sigatoka leaf spots affect banana (1) Yellow and (2) Black. Both diseases are of great importance, but black Sigatoka is more virulent and difficult to control.

- **First Report:**Java-1902 and later in 1913 appeared in epidemic form in Sigatoka valley in Fiji.
- **Distribution**: Guyana, Surinam, Trinidad, Uganda, Costa Rica, Colombia, Jamaica, Mexico, Panama, Venezuela and India.
- **India:** Andhra Pradesh, Assam, Bihar, Gujarat, Karnataka, Kerala, Maharashtra, Tamilnadu and West Bengal.

Symptoms

- The spots are noticed mostly on the older leaves.
- Light yellow or brownish green streaks observed on leaves.
- Streaks become linear, oblong brown to black coloured running parallel to veins.
- Central portion becomes necrotic and grayish surrounded by dark brown band with yellow halos.
- On upper surface of spots fructification of the fungus appear as black specks.
- Spots are mostly seen along the edge of the leaf with defined margin and possess dark brown to black margin. Spots coalesce and whole leaf blade dries up
- Rapid withering, drying and defoliation of leaves.
- Photosynthesis drastically reduced due to defoliation and ethylene content in infected plant is increase due to this disease which results in to premature ripening of banana.
- If the fruit is nearing maturity at the time of heavy infection, the flesh ripens unevenlyand individual bananas appear undersized and their flesh develops a buff pinkish colour, and store poorly.

Pathogen

- Mycelium is hyaline, septate and branched.
- Conidia are elongated, narrow and multiseptate.
- Perithecia are dark brown to black and ostiolate.
- Asci are oblong and clavate (club shaped).
- Ascospores are hyaline, two celled, obtuse (blunt) to ellipsoid.

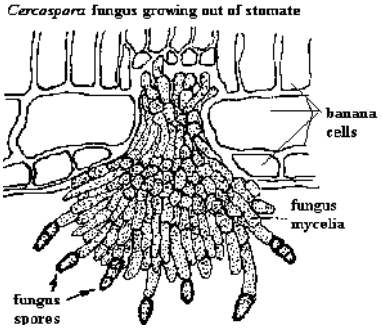
Disease cycle

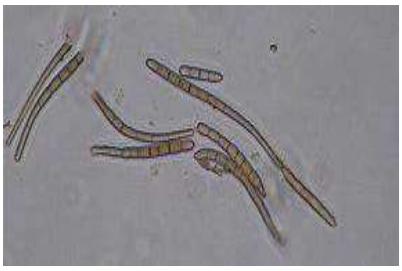
- **Primary source of infection**

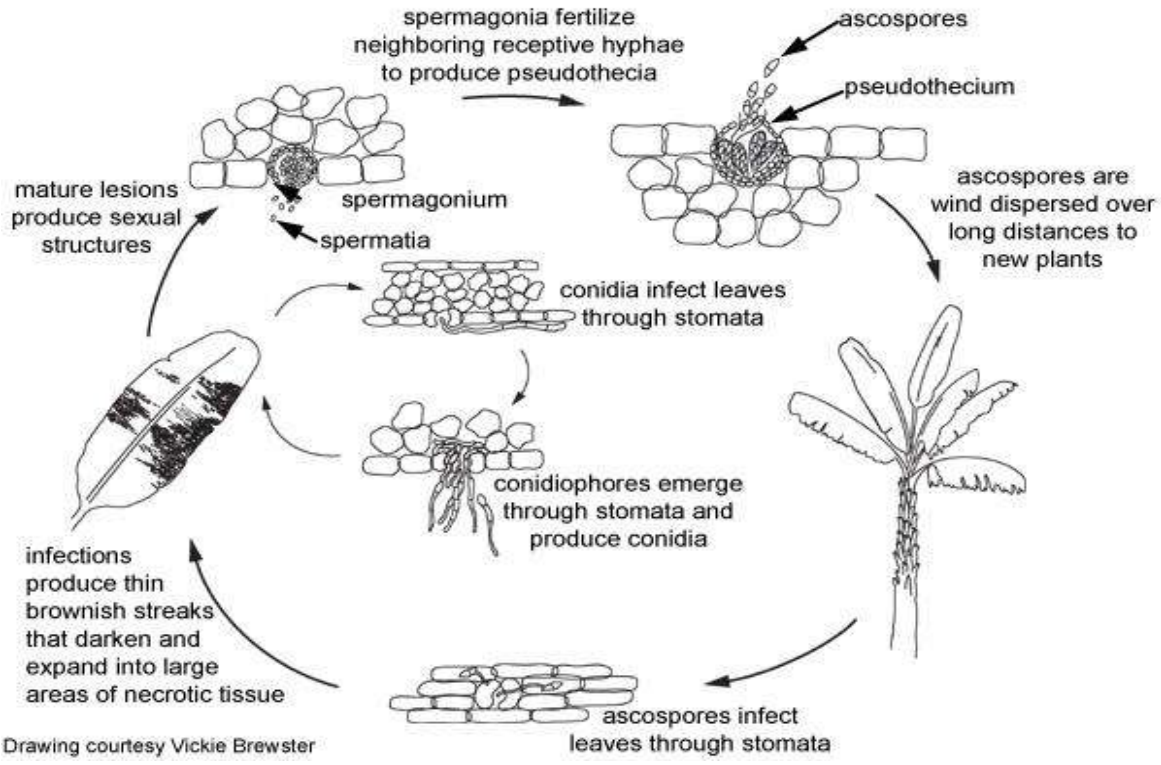
  Survives on dry infected leaves on the field soil and primary infection takes place through ascospores in the infected plant debris.

- **Secondary source of infection**

- Wind borne conidia and ascospores.
- Infection takes place through stomata on the lower surface of young leaves.
- Surface moisture is necessary for release of both conidia and ascospores. Hence the disease is severe in moist weather.







Disease cycle of Sigatoka leaf spot of banana

Favorable condition

- Temperature 23°-25° C. Rainy, wet and humid condition
- Poor- badly drained soils. Shady area, closer spacing, heavy weed, grass cover and frequent irrigation.
- Soils with poor drainage and low fertility favours the disease incidence. Increased number of suckers in a mat promote disease development.

Management

- Remove and destroy affected leaves followed by spraying with BM 1 % + Linseed oil 0.3 %.
- Spray carbendazim 50 WP @ 0.1% (5 g/10 l water) or thiophanate methyl 70 WP @ 0.1% or prochloroz @ 0.1% and mineral oil @ 0.3% or chlorothalonil 75 WP @ 0.2% or mancozeb 75 WP @ 0.2% or propiconazole or hexaconazole @ 5 ml/10 l water along with sticker /spreader teepol or sandovit or triton (1 ml/l).
- Prevent water accumulation around the plant and go for periodical weeding.

2. Moko disease/Bacterial wilt

C.O: *Burkholderiasolanacearum*

First report: ByRorer 1910 from Trinidad

First recorded in Guyana in 1840 in Moko plantain.

In India, first reported from West Bengal in 1968.

Loss: In India fruit yield loss were up to 70%.

Symptoms

- Symptoms start on rapidly growing young plants.
- The youngest three to four leaves turn pale green or yellow and collapse near the junction of lamina and petiole. Most of the leaves collapse within 3-7 days.
- Affected plant show rapid wilting, collapse of leaves, premature ripening of fruits. Stem shows discoloration of vascular strands with wilting and blackening of suckers.
- Vascular discoloration (pale yellow to dark brown or bluish black) is concentrated near the centre of the pseudo stem, becoming less apparent on the periphery.
- Grayish brown or dirty white, slimy bacterial ooze is seen when the pseudo stem of affected plant is cut transversely.
- The reddish brown tinge which is present typically in panama disease is usually absent.
- A firm brown dry rot is found within fruits of infected plants (characteristic symptom). The fruit pulp exhibits a characteristic dark brown discoloration.

Pathogen

- Moko disease is caused by race 2 of *Ralstoniasolanacearum*which infects Musa and Heliconia.

- Cells of bacterium are rod shaped and motile by 1-4 polar flagella (lophotrichus bacteria). They are Gram negative.

Favourable condition:

Warm and moist weather favours disease development.

Disease cycle

The pathogen is soil borne.

The pathogen survives in crop residues left in the field.

Primary source

- Infected suckers and infected material left out in field. The bacteria enter the roots and rhizomes via wounds, caused by nematodes and farm implements.

Secondary Source

- Bacteria can also transmitted through irrigation water and insects.

Management

- Follow strict quarantine and phytosanitary measures.
- Tools used in the interculturing operations must be sterilized.
- Provide better drainage in the field.
- Uproot and destroy infected plant / suckers.
- Use healthy suckers for planting purpose.
- Follow crop rotation with non-host crops.
- Exposure of soil to direct sunlight during hot summer.
- Disinfestation of tools with formaldehyde along with water (1:3).
- Control the flower visiting insects as they are helping in transmission of disease. Fumigation of infected site with Methyl Bromide or chloropicrin.
- Grow resistant varieties like Poovan and Monthan.
- Sucker should be treated with aureofungin (1.2 g/10 l water) + streptocycline (1 g/10 l water) before sowing.
- Spraying of streptocycline (1 g/10 l. water) + copper oxycloride (20 g/10 l. water) effectively control disease.
- Biological control with *Pseudomonas fluorescens*.

3. Panama disease/Panama wilt

C. O.: *Fusariumoxysporum*f. sp. *cubense*

Economic importance:

First reported by Bancroft in Australia in 1874. It was then reported from Panama in 1890. Within a decade the disease had spread to Costa Rica and

subsequent outbreaks occurred in Surinam (1906), Cuba (1908), Trinidad (1909), Jamaica (1911), Honduras (1916) and Guatemala (1919). The disease has since been reported from most banana-producing countries.

Distribution: Asia, Australia, Pacific islands, Africa, North and South America

India: T.N., Karnataka, AP and West Bengal

The popular variety Gros Michel, mostly grown for export quality fruits, was most susceptible and had to be replaced with Giant Cavendish bananas which were resistant.

- Severe on Rasthali variety (Amrutapani), Gros Michel, Monthan,Karpooravalli

Symptoms

- Conspicuous symptoms usually appear on at least 5 months old banana plants, although 2-3 months old plants are also killed under highly favorable conditions.
- Symptoms initially seen in older plants in a mat and on older leaves.
- The earliest symptoms are faint yellow streaks on the petiole of oldest, lower most leaves. Affected leaves show progressive yellowing, break at the petiole and hang down along the pseudostem.
- In the field the disease occurs sporadically and after spreads in concentric rings.
- Young leaves may not dry up but remain erect and they also get affected under severe cases. Varying degrees of longitudinal splitting along pseudo stem are often observed.
- Light yellow to dark brown vascular discoloration of pseudo stem. Usually the discoloration appears first in the outer or oldest leaf sheath and extends up to the pseudo stem
- The fungus grows and blocks the vascular system resulting in wilting of the plant.
- Affected plants give characteristic odor of rotten fish.
- Young suckers also develop the disease and rarely develop external symptoms.
- Affected plants do not produce bunches. Even if produced, fruits are malformed and ripen prematurely or irregularly.
- However the pathogen does not infect the fruits.
- Roots of diseased rhizomes are frequently blackened and decayed.
- The reddish brown tinge which is present typically in panama disease.

Favorable conditions

- Soil conditions like texture and structure, fertility level, temperature, drainage, cultural operation will greatly influence the disease incidence and spread in the field.
- Survival and growth of fungus are generally greater in acidic or light textured soils than in clay or alkaline soils with high calcium content.
- Saturated poorly drained soils have greater incidence.
- Insect injuries and wounds and infestation of nematode, predispose the disease.
- Optimum temperature 20-30º C.

Pathogen:

- Mycelium is septate, hyaline and branched.
- Fungus produce micro, macro conidia and chlamydospores.
- Micro conidia-single celled or rarely one septate hyaline elliptical or oval.
- Macro conidia-sickle shaped hyaline, 3-5 septate and tapering at both the ends.
- Chlamydospores-thick walled, spherical to oval, hyaline to slightly yellowish in colour.

Disease cycle

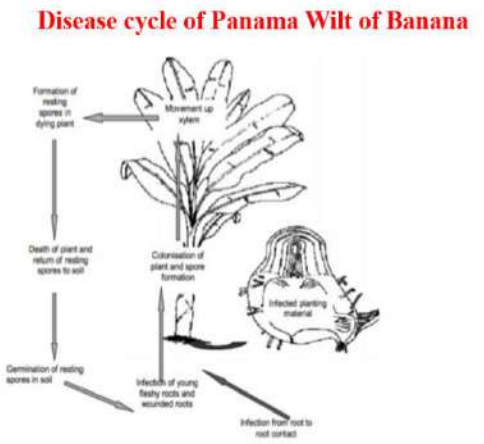
Primary source of Infection:

- The pathogenissoilborne and survive on infectedcropresiduesaprophytically for a long period.
- Primary infection takes place throughinjuredroots.

- The germinatedchlamydospore infect the root and moves towards the rhizome and extensivelydeveloped in vasculartussues and thenprogresstowards the vascular system into the pseudostem and olderleafpetioles.

Secondary source of Infection:

- The secondary infection takes place by macro and micro conidia.
- Adjacent plant root contact spread the disease.
- Flood/irrigation water helps in dispersal of inoculum.



Management

- Add actinomycete culture.
- Application of mercuric chloride (3000 ppm).
- Use of disease free suckers for planting.
- Avoid ill drained soils, and prefer slightly alkaline soils (7-7.5 pH) for cultivation.
- Fallowing for 6 to 24 months or crop rotation with rice.
- Application of lime (1-2 kg/pit) to the infected pits after chopping of the plants parts.
- Dipping of suckers in carbendazim (0.1%) solution before planting.
- Soil drench with 0.1% carbendazim or 0.01% vapam.
- Growing resistant Cavendish varieties, *viz.,* ***Basrai (Vamanakeli), Poovan***(Karpurachakkarakeli), **Moongil.** Dwarf varieties are generally resistant.

4. Bunchy top: C.O.: *Musa virus-1*

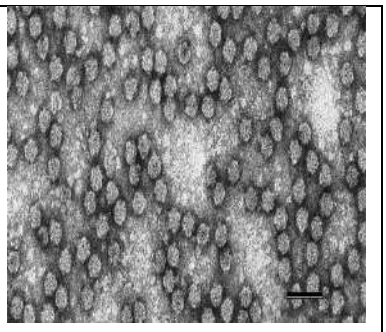
- It is also known as curly top, strangles or cabbage top.
- First reported from Fiji in 1879 in Cavendish varieties

In India First report: 1940 Kerala (India), T.N., Orissa and Assam.

- Around 1940, introduced into India from Srilanka.
- Banana bunchy top virus is a ss DNA virus with single isometric particles measure 20 nm in diameter
- The virus concentration is more present in phloem
- The disease is covered by domestic quarantine regulations. Losses were estimated to be ₹ 4 crores every year and 100% loss occurs if infected suckers are planted.

Symptoms

- Prominent dark green streaks on the petioles and midrib along the leaf veins. Green streaks range from a series of dark green dots to a continuous dark green line. Marginal chlorosis and curling of leaves. Petioles fail to elongate.
- Leaves are reduced in size, chlorotic, stand upright and become brittle and are crowded at the top (Bunchy top). Leaves are typically bunched at the apex forming rosette appearance. Young infected plants are stunted.
- In acute primary infection- fruits are not produced or matured and such plant do not die. Flowers display mottled and streaked discoloration. Plants show marked stunting.



Disease cycle

- Banana bunchy top virus is ass *DNA virus with single isometric particles.*
- The virus is an isometric particle measure 20nm in diameter.
- The virus concentration is more present in phloem

Primary Source of Infection: Through infected plant suckers.

Secondary Source of Infection: By Banana aphid, *Pentalonianigronervosa.*

Reservoir hosts:*Cucumussativus, C. melo, Catharanthusroseus, Gynuraaurantiaca, Colocasia esculenta, Amomum subulatum and Elettaria cardamom*

Transmission

- Banana aphid helps in disease transmission.
- The virus is transmitted to healthy plant by aphid after 1.5 to 2 hrs. feeding.
- The minimum acquisition feeding time for nymphs is 17 hrs.The aphid remain infective for 13 days.

Management

- Destruction of infected plant.
- Selection of healthy suckers for planting. Use of only certified banana suckers for planting.
- Control the aphid by spraying methyl-o-demeton (2 ml/lit).
- Follow strict quarantine measures during the introduction of new material from one state to other state. The new crop should be regularly inspected and the diseased plants destroyed as soon as noticed.

…..x…..x…..x…..

17. PAPAYA (*Carica papaya* L.)

1. Foot rot C.O.: *Pythiumaphanidermatum*(Eds.) Fitz: *Rhizoctoniasolani*Kuhn.

2. **Mosaic** C.O.: Papaya Mosaic Virus (PaPMV), Carica Virus – 1

3. **Leaf curl** C.O.: Tobacco leaf curl virus (TLCV)

4. **Ring spot** C.O.: Papaya Ring Spot Virus (PRSV)

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1. Foot rot

C.O. : *Pythiumaphanidermatum*(Eds.)

Class : Oomycetes

Order : Peronosporales

Family :Pythiaceae

C.O. : *Rhizoctoniasolani*Kuhn.

Distribution:

- Africa, Cuba, Hawaii, Sri-Lanka and India.

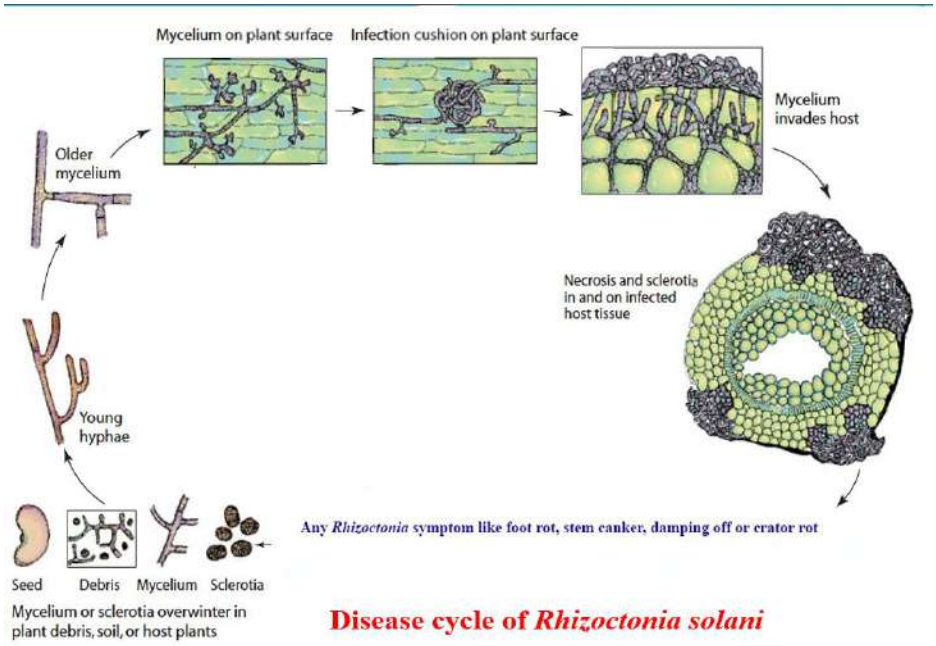
- The disease often appears in rainy season from June – August.
- The severity of the disease depends directly on the temperature and rainfall.

Symptoms

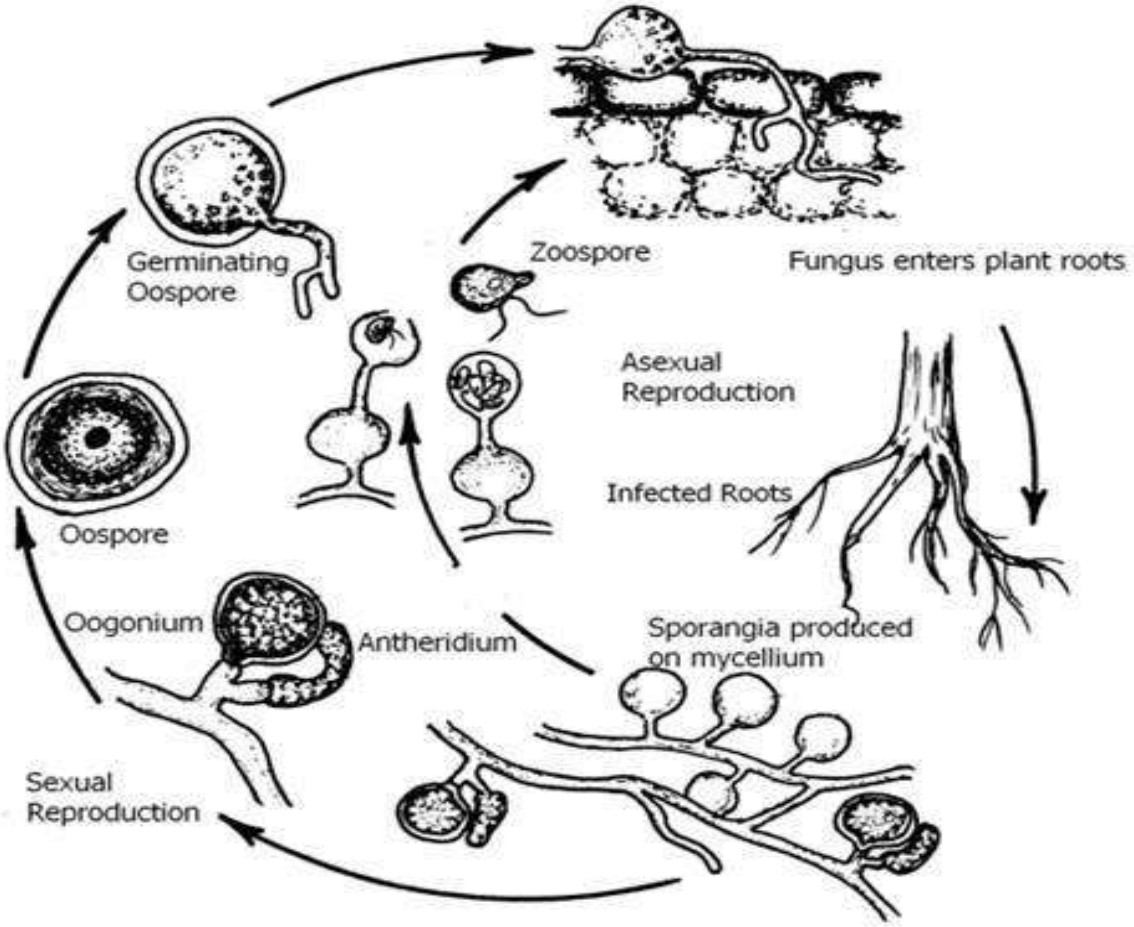
- Disease may lead to complete failure of crop when it appears in the early stages of growth.
- Water soaked patches appear on stem at collar region or immediately at the soil line. These patches enlarge and girdle the base of stem.
- Diseased tissues turn dark brown or black & red. Terminal leaves turn yellow, drop & wilt. Fruits shriveled and drop prematurely
- Disintegration of parenchymatous tissues at the base of stem takes place.
- The entire plant topples over the ground.
- *The internal tissues dry up and give a honeycomb appearance.*
- Rotting may spread above and below on the stem and down to the roots.
- The roots deteriorate and may be destroyed.
- The typical stem rot symptoms are common in 2-3 years old plants but younger plants have also been seen drying due to early infection.
- A damping off of papaya seedling in the nurseries is also common.
- Seedling grown in such nurseries carry the disease and when transplanted, develop symptoms.

Disease cycle

- The fungi survive in the form of oospore (*Pythium*) and sclerotia (*Rhizoctonia*) in the soil.
- This may serve as primary source of inoculum.
- The infected seedlings raised in infected soil carry the disease to the fiel
- Secondary spread takes place through sporangia and zoospores.



Disease cycle of *Rhizoctonia solani*



Disease cycle of *Pythiumaphanidermatum*

Favorable condition

- One week old seedlings are more susceptible than one year old trees.
- Stem rot caused by *P. aphanidermatum* is commonly noticed in 2 to 3 years old trees.
- The disease appears during rainy season and severity increase with the intensity of rainfall optimum temp. 36º C is favorable for disease development.
- *Rhizoctoniasolani* is severe during dry & hot weather 30º C - 35 º C temperature.
- Water logging increase the chance of disease development.

Management

- Seed treatment with Captan 50 SD or Chlorothalonil 75 WP (kavach) @ 4 g/kg of seed.
- Seedlings should be raised on well-drained nursery area.
- Diseased seedlings should be uprooted & destroyed.
- Drenching the tree basin with B. M. @ 1% or Captan 50 WP @ 0.2 % or copper- oxycholoride 50 WP@ 0.25 % or Metalaxyl 25 WP @ 0.1 % or Tridemorph 80 EC @ 0.04 % reduces the incidence of the disease.
- At the time of cultivation care should be taken so that no injury is caused to the basal portion of the stem.
- The crop should be irrigated by adopting the ring method of irrigation so that the water does not come in direct contact with the stem.
- In the case of new plantings, preventing water logging of the soil may control the disease.
- Application of *Trichoderma viride* (15 g/plant) mixed in well-decomposed FYM should be applied around the root zone of the plants at the time of planting.
- In the case of disease attack in existing crops, the rotted portion of the plant should be scraped and Copperoxychloride or Bordeaux paste should be applied.

2. Mosaic

C.O.: Papaya Mosaic Virus (PapMV),Carica Virus – 1

- ***Papaya mosaic virus* (PapMV)** is a plant pathogenic virus in the genus *Potexvirus* and the family *Alphaflexiviridae*.
- PapMV is a filamentous, flexuous rod, 530 nm in length.

Distribution:

- East Africa, Rhodesia, West Indies, Cuba, Hawaii, Brazil, Bolivia, U.S. A., Peru, Venezuela & India
- **India**: Andhra Pradesh., Bihar, Karnataka, Madhya Pradesh., Orissa, Punjab, Rajasthan, Uttar Pradesh, West Bengal and Gujarat.
- **First Report**: Kapoor and Verma from Mumbai & Pune 1947.
- **Fruit yield loss**: In severe cases 90 % yield loss.

Symptoms

- Disease may appear at any stage of crop growth but most serious on young plants.
- Typical mosaic symptoms showing chlorosis with dark green blisters on leaves.
- Top young leaves of diseased plant are much reduced in size and show blister like patches of dark green tissue, alternating with yellowish green lamina and puckering.
- *Mottling and puckering of leaves, especially the young ones.*
- The lamina is reduced and malformed and are often modified into tendril like structures **(shoestrings).**
- Decline & marked reduction in growth of diseased plants (within 30-40 days).
- Older leaves fall down and a small tuft of younger leaves is left at the top with upright position.
- The stem, petiole and fruits develop elongated water soaked areas showing concentric or circular rings.
- Fruits develop innumerable circular, on diseased fruits circular water soaked lesions with central solid spot appears. Fruits are deformed elongated and reduced in size and show mosaic patches. No reduction in the flow of latex.

Virus

- Virus particles are filamentous 530 nm long.
- Thermal inactivation point 50-53º C, dilution end point 10^{-3} to 10^{-4}.

Mode of sprea and survival

- Not transmitted through seed of infected fruits.
- The virus is transmitted by sap, grafting and several aphids – *Aphis gossypii,Aphis medicaginis,Myzuspersicae,* *Aphis malvae, Rhopalosiphummaidis, Microsiphumsolonifolii.*
- Most of the cultivated species of *Carica papaya* are susceptible: *C. candamarcensis*and *C. microcarpa*
- **Other hosts:** *Cucumissativus, Cucurbitapepo, C. maxima, Citrullusvilgaris, Luffaacutangula, Lagenariasiceraria,* Safflower and some ornamental plants.

Management:

- Use healthy seedlings for planting.
- Roughing of infected plant & destroying them.
- Spraying of systemic insecticide for checking spread of vectors, monocrotophos @ 0.05 % or methyl-o- demeton @ 0.02 %.
- *Caricacaulifloras*is resistant. It can be utilized in breeding programme.
- Weekly sprays with 1% groundnut oil also help in preventing the infection.

3. Leaf curl

C.O.: Tobacco leaf curl virus (TLCV)

- Prevalent in several parts of India.
- PLCV is in the family *Geminiviridae*.
- It is not transmitted mechanically.
- Papaya leaf curl was first reported in Tamil Nadu in 1939 by Thomas.

Distribution:- Bihar, Delhi, Karnataka, Kerala and Andhra Pradesh

Symptoms

- The disease is characterized by severe curling, crinkling and distortions of the leaves accompanied by *vein clearing and reduction in leaf lamina.*
- *The leaf margins are rolled downward and inward in the form of inverted cap.*
- Veins thickened and turn dark green.
- Leaves become leathery and brittle and petioles are twisted.
- Affected plants bear only a few flowers and fruits.
- The plant become stunted and leaves get defoliated.

Mode of spread

- The disease is transmitted through white fly *Bemisiatabaci* and not sap transmissible.
- **P.I:** Infected plant parts
- **S.I:** Whitefly, ***Bamesiatabaci***and grafting

Host: Tobacco, tomato, sun hemp, goose berry, chilli, hollyhock, zinnia and several other weeds.

Management

- Infected plants should be destroyed from the nursery.
- In orchard, the infected plants roughed and destroyed.
- Spray triazophos/imidacloprid0.05%or dimethoate 0.03% for controlling vectors.
- Soil application of Carbofuran (1 kg a.i./ha) at the time of sowing for controlling vector.

4. Ring spot

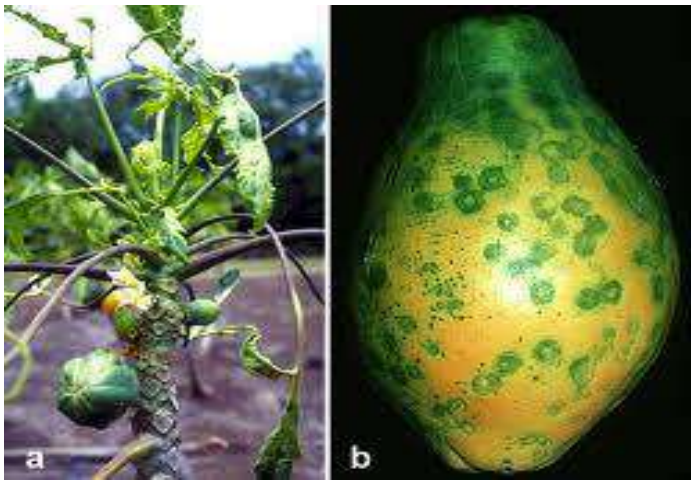
C.O.: Papaya Ring Spot Virus (PRSV)

- The disease was first reported in 1949.
- The term papaya ring spot (PRS) was first coined by Jensen in 1949 to describe a papaya disease in Hawaii.
- PRSV belongs to the genus *Potyvirus*, a large and economically important group of plant infecting viruses in the family *Potyviridae.*

Distribution:- China, France, India, Germany, Italy, Mexico, Taiwan and USA.

Symptoms

- Disease is characterised by vein clearing and puckering.
- The margins and distal parts of young leaves roll downwards and inwards.
- The plant get stunted. Infected plant initially shows chlorosis on youngest leaves followed by vein clearing, rugosity and prominent mottling of laminae.
- Malformation and reduction of the lamina which may become extremely filliform.
- On the stem of young plants, mosaic or mottle symptoms with dark green spot and oily or water soaked streaks.
- Fruits are smaller, showing typical circular and concentric rings.
- Diseased fruits contain 40% lower sugar.
- Latex quality of diseased plants is poor.



Virus

- ✓ The virus particles are rod shaped, length 760-800 nm with 12 nm width, TDP: 54-60^{0} C, DEP: 10^{-3}

Mode of spread and survival

- Sap transmissible.
- Among insect vectors *Aphis gossypii* and *Myzuspersicae* are the most efficient vectors.
- It is also transmitted by *Aphis craccivora*and *Rhopalosiphummaidis.*
- The virus is transmitted in a non-persistant manner.
- It is neither soil-borne nor seed-borne.

Management

- Early detection of infected plants and prompt removal can check the spread of the disease.
- Aphids can be controlled by application of Carbofuran (1 kg a.i./ha) in the nursery bed at the time of sowing seeds followed by 2-3 foliar sprays of Phosphamidon (0.05%) at an interval of 10 days starting from 15-20 days after sowing.
- Cross protection: Cross protection is the phenomenon whereby plants that are systemically infected with a mild strain of a virus are protected against the effects of infection by a more virulent related strain. Cross protection was practiced quite extensively in Taiwan and to a very limited extent in Hawaii both in the 1980.
- Grow genetically modified (GM) papaya. It has been produced and commercialized in Hawaii to successfully control PRS. Two transgenic cultivars rainbow and SunUp are in cultivation there.
- Spraying of neem seed oil also found effective in controlling the vector.

…..x…..x…..x…..

18. POMEGRANATE (*Punicagranatum*L.)

1. Bacterial blight C.O.: *Xanthomonasaxonopodispv. punicae*
2. Cercospora leaf and fruit spot C.O.: *Cercospora punicae*
3. Wilt C.O.:*Ceratocystisfimbriata*
4. Root knot C.O.: *Meloidogyne incognita*

- Pomegranate (*Punicagranatum*L.) is grown in tropical and subtropical regions of the world.
- The total area under cultivation of pomegranate in India is 107.00 thousand ha and production is around 743.00 thousand tons.
- Maharashtra is the leading producer of pomegranate followed by Karnataka, Andhra Pradesh, Gujarat and Tamil Nadu.
- Ganesh, Bhagwa, Ruby, Arkata and Mridula are the different varieties of pomegranates produced in Maharashtra.

- In India, pomegranate is commercially cultivated in Solapur, Sangli, Nasik, Ahmednagar, Pune, Dhule, Aurangabad, Satara, Osmanabad and Latur districts of Maharashtra; Bijapur, Belgaum and Bagalkot districts of Karnataka and to a smaller extent in Gujarat.

1. Bacterial blight of pomegranate

C.O: *Xanthomonasaxonopodispv. punicae*

Economic Importance

- Bacterial blight of pomegranate was first reported in India from Delhi in 1952 and was of minor economic importance until 1998.
- Presently, the disease occurs widely and outbreaks have been recorded in all major pomegranate-growing states including Maharashtra, Karnataka, and Andhra Pradesh.
- Heavy losses caused by bacterial blight were recorded in Hanumangarh district of Rajasthan in 2009.
- The disease was prevalent only in India, but recently, it was also reported from South Africa in 2010.

Symptoms

- The affected leaves showed one to several small irregular water soaked spots, which later turn brown to black in colour with water soaked margin.
- The bacteria infect through wounds and stomatal opening and cause water socked lesions within 2-3 days.

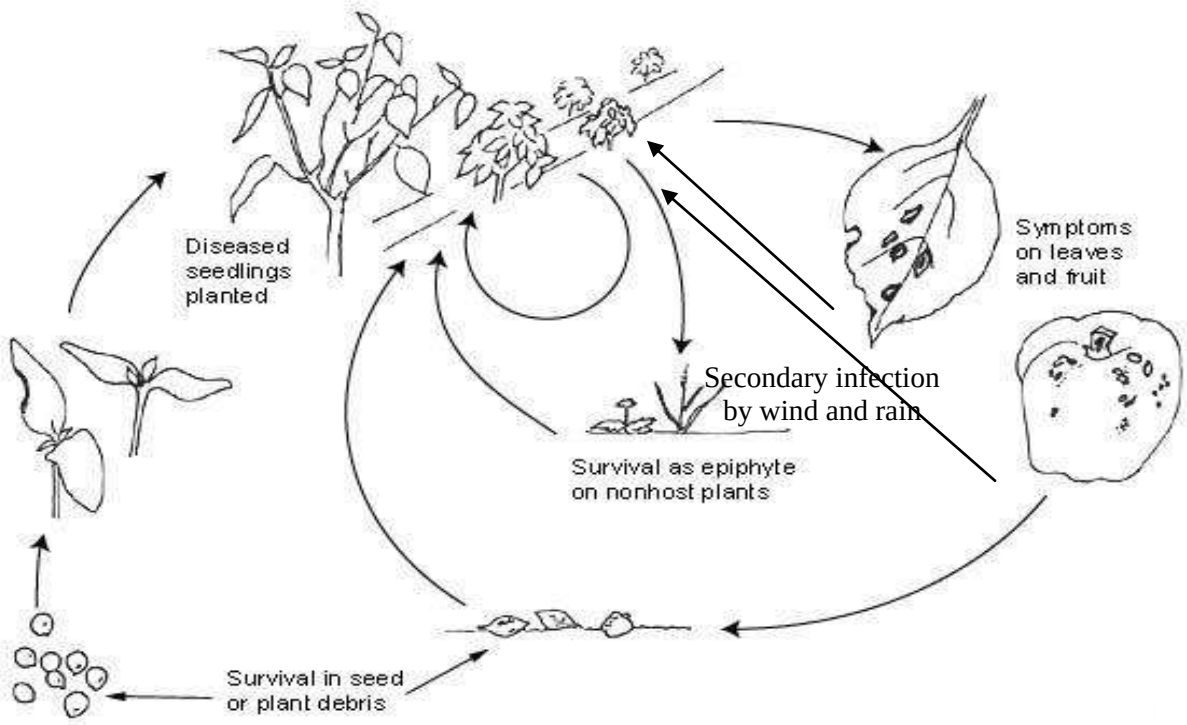
Symptoms on leaves and fruits

- Small, deep red spots of 2-5 mm diameter with indefinite margins on leaf blade.
- The leaves are often distorted and malformed, dry off prematurely and fall down.

- The bacterium also attacks stem, branches and fruits.
- On the stem, the disease starts as brown to black spots around the nodes.
- Girdling and cracking of nodes. Branches break down.
- Brown to black spots on the pericarp of fruit with **L or Y shaped cracks.**
- Flowers and fruit shows the dark brown, irregular, slightly raised and oily spots.
- Plant remains stunted and sickly appearance.

Disease Cycle:

- The bacteria can spread by air borne cells.
- The bacteria can survive in soil for over 120 days and cause primary infection on new flush.
- Primary source of Infection: Infected cuttings
- Secondary source of Infection : Wind splashed rains
- The bacteria infect through wounds and stomatal openings.



Favorable Condition

- Disease buildup is rapid from July to September.
- Severity increases during June and July and reaches a maximum in September and October and then declines.
- High temperatures and low humidity favor disease development.
- Optimal temperature for growth of bacterium is 30°C.

- Thermal death point is about 52°C.

Management:

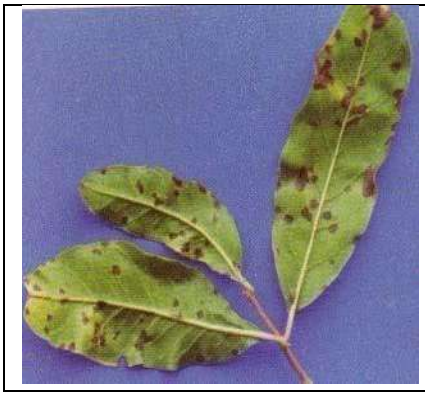
- At disease initiation spray carbendazim 1% or Bordeaux mixture 1% or copper oxychloride 50 WP @ 0.2% 40 gram/10 liter of water, three to four sprays at 15 days interval or spray 0.5 g Streptomycin Sulphate + 2.5 g Copper oxy chloride per liter of water.
- Remove the infected twigs, branches and apply Bordeaux paste. Wide row spacing.
- Selection of disease free seedlings for fresh planting. Pruning affected branches, fruits regularly and burning and clean cultivation and strict sanitation in orchard.

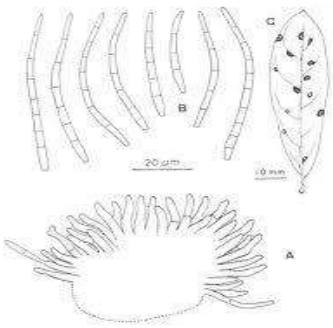
2. Cercospora leaf and fruit spot

C.O.: ***Cercospora punicae***

Symptoms:

- Light zonate brown spots appear on the leaves and fruits.
- Black and elliptic spots appear on the twigs.
- The affected areas in the twigs become flattened and depressed with raised edge.
- Such infected twigs dry up. In severe cases the whole plant dies.
- Leaf spots are minute, brown with yellow halo. Spots are scattered, circular or irregular and become dark brown with age.
- Spots on lower side are sunken with clusters of spore bearing structures hence grayish in color.
- Minute, circular, black spots appear on sepals of the flower.
- The affected fruits also showed small irregular black spots, which later on coalesce, into big spots.





Disease Cycle

The pathogen perpetuate on fallen leaves, flowers, fruits and branches.

Primary Source of Infection: Diseased plant debris

Secondary sources of Infection: Wind borne conidia

Management:

- The diseased fruits should be collected and destroyed.
- Two to three spray of Mancozeb 75 WP or Captan 50 WP at 15 days interval @ 2.5 g in one liter of water after fruit formation gives good control of the disease.
- Application of Thiophanate- methyl 0.1% or Chlorothalonil 0.2% also control the disease.
- Remove the infected twigs, branches and apply Bordeaux paste.

3. Wilt

C.O.: *Ceratocystisfimbriata* Ellis and Halsted

Also, fungal pathogens ***Fusariumoxysporum***Schltdl. and ***Verticilliumdahliae***Kleb. 1913, have been identified as causal agents of pomegranate wilt.

Distribution: Maharashtra, Karnataka and Andhra Pradesh

Symptoms:

- Affected branches show yellow leaves, followed by wilting and drooping of foliage of one or more branches of the plant.
- The entire tree eventually dies off within few weeks to display complete wilting.
- Wilt symptoms can at times spontaneously appear causing immediate senescing of the entire plants' foliage at once.
- Infected plants reveal dried foliage and fruits attached to the branches for many months.
- The xylem area becomes dark reddish-brown to purple deep-brown or black staining.
- Vertical stem cracking in some infections, splitting of root and stem bark and lower branches.
- Cross and vertical sections of infected plant parts generally reveal dark greyish-brown streaks in vascular and adjoining cortex tissues.
- The fungus can spread between adjacent trees and at times randomly across different locations in the orchard.

Pathogen:

- The fungal mycelium is septate.
- The fungus produced endoconidia, aleurioconidia and long-necked perithecia.

- Endoconidia were hyaline, cylindrical and formed endogenously.
- Aleurioconidia were thick-walled ellipsoidal, pyriform or obpyriform, truncate at the base, golden-brown and borne singly or in chains and were intercalarv, lateral or terminal.
- Perithecia were blackish to brown-blackish in colour, globose to subglobose with a characteristic long neck.
- Ascospores were hyaline, ovate to galeate.

Epidemiology:

- The disease is more prevalent in deep heavy soil with high moisture content.
- Temperature 18-30° C and frequent rains.
- Plantation at close spacing.
- Application of excessive irrigation and fertilizers.

Disease cycle:

- Primary source of inoculum: *Ceratocystisfimbriata* and *F. oxysporum*, are soil borne and survive in soil through their thick walled conidia, alleurioconidia and chlamydospores, respectively.
- Secondary source of inoculum: The fungus is easily dispersed as mycelium, conidia, aleurioconidia or ascopores.
- The spread of pathogen takes place through infected seedlings, irrigation and rain water, root contact, shot hole borer (*Xyleborusfornicatus*), implements, pruning and budding tools.
- 'Wilt' disease attacks both wounded and intact healthy roots.
- After entering the host the disease progresses through the xylem in water-conducting cells causing rapid wilting of the plant and broad dark discolouration of the vasicular system.

Management:

- Eradication of wilt infected plants.
- Avoid root contact among adjacent plants by proper spacing.
- Improve drainage facilities and aeration through weeding.
- Pruning of infected shoots and branches.
- Soil drenching with carbendazim 0.2% or propiconazole 0.1% + boric acid 0.5% + phosphoric acid 0.5%.
- Manage shot hole borer by chorpyriphos 0.2%

- Soil sterilization with formalin 0.2% prior to replanting.
- Soil application of neem cake, karanj cake, mahua cake or castor cake when nematode infestation associated with wilt.
- Soil application of *Bacillus subtilis*.

4. Root knot:

C.O.: ***Meloidogyne incognita***

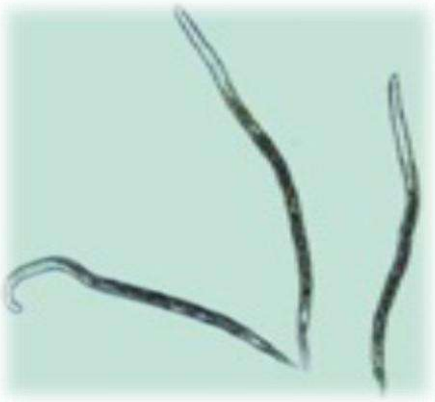
InMaharashtra 32% yield losses attributed in identified hotspots.

Above ground symptoms:

- The above-ground parts of the diseased plants exhibit symptoms typical of mineral deficiency or drought injury due to the slow debility of affected roots to function properly for nutrient, water uptake and translocation even when adequate fertilizers and moisture are present in the fields.
- The other symptoms may include dieback, yellowing, wilting and premature shedding of the foliage with severe stunting, depending upon the initial nematode population in the field.
- Chlorosis of the foliage lowers the quality of the crop resulting in severe losses.
- Poor emergence and death of young seedlings may occur in heavily infested soil but death of grown up plants is rarely seen unless some other co-inhabiting pathogenic fungi or bacteria is involved in the development of disease complex.

Below ground symptoms:

- The below-ground symptoms include galls or knots on the roots or tubers of numerous crops.
- These galls vary in size from pin-head to large size which in case of heavy infection may coalesce to form large secondary galls.
- The size of galls also depends upon the host plants and nematode species.



Management:

- Deep summer ploughing of nursery/main field before establishing/ transplanting of seedlings.
- Crop rotation in the main field before transplanting.
- Use solarized soil for filling the polythene bags or solarization of nursery soil for 2-3 weeks by white transparent polythene sheet (100 gauges=25.4 µ) during the month of May before establishing the grafts from mother plants into polythene bags or nursery.
- Application of FYM/compost at 20-25 t/ha or non-edible oil cakes at 2 t/ha in the nursery as well as in the main field at transplanting and at 'Bahar' (blossom) every year.
- Inter-cropping with sunhemp, tagetes, mustard, fenugreek, onion, garlic etc. during rest period.
- Application of bioagents (*Trichoderma viride*, *Paecilomyceslilacinus*, *Pseudomonas fluorescens*etc.) at 10 to 20 g/m^2 nursery or at 5 kg with 100 kg FYM/compost/ha in the main field at transplanting as well as at 'Bahar' (blossom) every year.
- Soil application of carbofuran 3G or phorate 10G at 2 kg a.i./ha in the nursery as well as at transplanting in the main field and at 4 kg a.i./ha at 'Bahar' (blossom) every year.

…..x…..x…..x…..

19. Coffee (*Coffea Arabica* L.)

1. Rust

C.O.: ***Hemileiavastatrix***Berk and Br.

- The disease was first reported from Ceylon in 1868.
- Occurs in epidemics form in Srilanka in 1867-1893.
- In western hemisphere coffee rust was first reported in Brazil in 1970.
- In India it is found in Kerala, Karnataka and Madhya Pradesh.

Distribution: - Java, Sumatra, Fiji and Ceylon.

Symptoms

- The fungus confines itself mostly to the leaf blade, rarely occurring on the berries.
- The disease manifests itself in the form of small yellowish spots, mostly on the under surface of the leaf.
- The spots get enlarged (10-15 mm) and turn orange brown and powdery masses of spores are seen on the surface.
- Characteristic chlorotic lesion can be seen on the upper surface.
- As the disease advances the orange red colour become white.
- The centers of the spots eventually dry and turn brown, while the margins of the lesions continue to expand and produce uredospore.
- Infection slowly progresses upward in the tree and host tissue becomes dark brown, then black and dries up.
- The infected leaves drop prematurely, leaving long expanses of twigs devoid of leaves.
- The damage to the plant is severe when it affects the young flush, causing defoliation and considerable reduction in yield

Pathogen

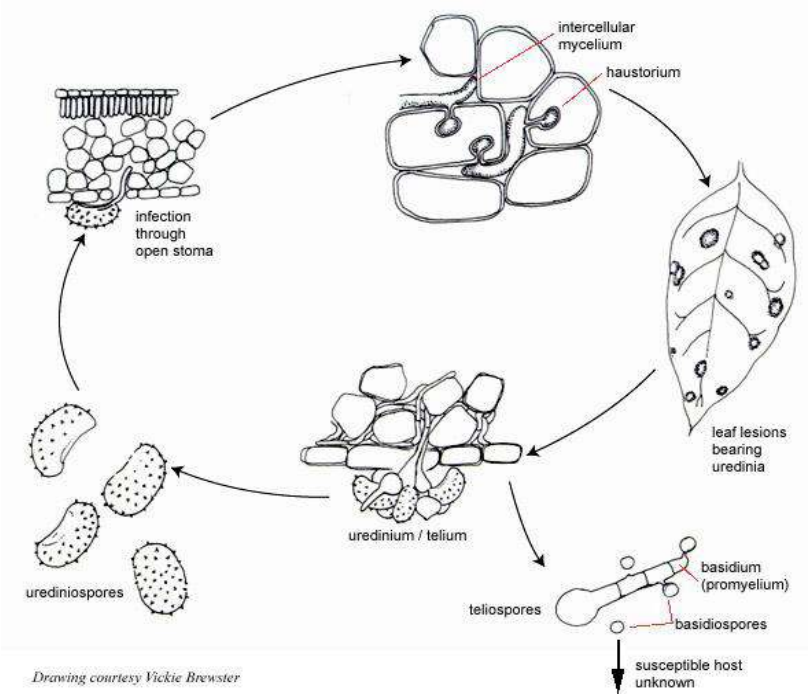
- The fungus is mostly intercellular drawing food from cells through haustoria.
- It produces uredospores and teleutospores on club-shaped erumpent stalks rising through the stomata.
- Uredospores are reniform, with an orange segment like appearance, the convex side echinulate and the lower or concave side smooth.
- Telial stage develops in the same spot producing turnip like, hyaline thick walled teleutospores.

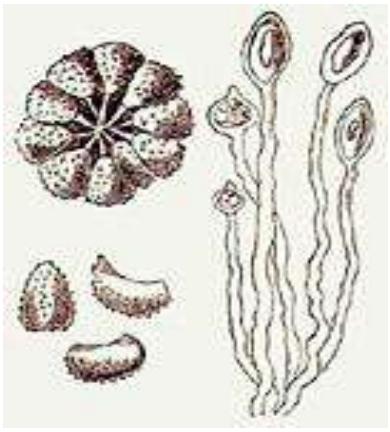
Disease cycle

- It is autoecious rust. The pycnial and aecial stages of the rust are not known and it is believed the fungus multiplies and spreads through the uredial stage and survives in off season through the uredial stage.
- Basidiospore is not able to cause infection in host. Teliospores are non-functional and vestigial, and the sexual life cycle is completed by the uredospores.

P.I: Survive as mycelium or uredospores in infected leaves.

S.I: Uredospores dispersed by wind and water.





Favorable condition

- Rainy weather with mist of dew and moderate temperature favors the disease.
- Uredospores germinate only in free moisture. Thick canopy of shade prevents the ready spread of the spores and thus secondary spread of the disease. Severe from July to December.

Management

- Spraying buses once before monsoon with BM 0.5% OR copper oxychloride 50 WP 0.25% and subsequently 2-3 times during monsoon is effective.
- Triadimefon 25 WP at 0.05% found very effective in controlling the disease.
- Grow resistant varieties- ‘Coorg’ and ‘Kebts’.

- Sanitation of the plantation is essential. Fallen leaves should be composted or destroyed.

…..x…..x…..x…..

20. TEA (*Camellia sinensis*L.)

1. Blister blight C.O.: *Exobasidiumvexans*

It was first reported from Assam, 1986.

Symptoms

- Small pale or pinkish spots appear on leaves. Spots are round in shape.
- On the upper surface of the leaf, spots become light green in colour, depressed into a shallow cavity while underside bulges corresponding covering white growth of fungus on lower surface of leaves.
- In later stages the blister turns dark brown and shrinks to flattened patch.
- Young leaves are circular shaped which becomes elongated along the midrib. Distortion of the leaves occurs.
- On stem, spots without blisters are formed. The fungus eventually penetrates and damages the stem. The leaves and buds withers.
- When many spots coursers, curling of leaves will occur. When it spreads to young succulent stems affected portion are withered. The leaf yield is reduced.

Disease cycle

- The fungus completes life cycle in 11 to 28 days.
- Conidia and basidiospores formed on same blister.
- Spores are air-borne.
- The fungus perpetuates in pre-existing infected bushes.

Favourable condition

- Disease is favoured by cool day with wet or humid condition.
- Succulent growth developing after pruning is susceptible to infection.
- Cool and moist weather favour the disease.
- Relative humidity > 83% for 7 to 10 days favours disease.
- Temperature above 35° C inhibit the disease.

- Bushes in low, moist and shady localities suffer more.
- Pruned trees with new flush is highly susceptible.

Management

- Pruning of affected leaves and shoots of the plant.
- Spray B.M. 1 % after pruning.
- Seedlings should be protected in nursery by weekly sprays of COC @ 0.3%.
- Mancozeb, Tridemorph, Triadimefon offers good disease control under field conditions.

…..x…..x…..x…..

21. BRINJAL

1. Phomopsis blight and fruit rot C.O.: *Phomopsisvexans*
2. Little leaf C.O: Phytoplasma (MLO'S) Mycoplasma like organism

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1. Phomopsis blight and fruit rot

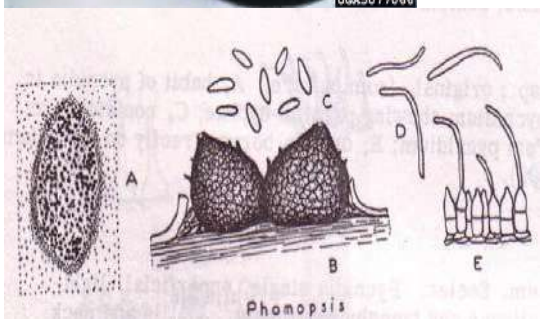
C.O.: ***Phomopsisvexans***

Perfect Stage: *Diaporthevexans*

- The disease is severe in tropical and sub-tropical areas of the world.
- In India this disease was *first reported from Gujarat* in 1914

Symptoms:

- The first phase of the disease is a blight on young seedlings.
- The stem is girdled near the soil level and the infected plant topples over and dies. Brown, round or oval spots develop on the leaf becoming irregular in shape on petiole and stem blighting the affected portions of the plant.
- The plants are attacked at all stages of growth, producing damping-off symptoms in nurseries and collar rot on young plants
- On leaves, circular to irregular, clearly defined grayish brown spots having light centers appear. The diseased leaves become yellowish in colour and may drop off. Several black pycnidia can be seen on older spots.
- The lesions on stem are dark brown, round to oval and have grayish centers where pycnidia develop.



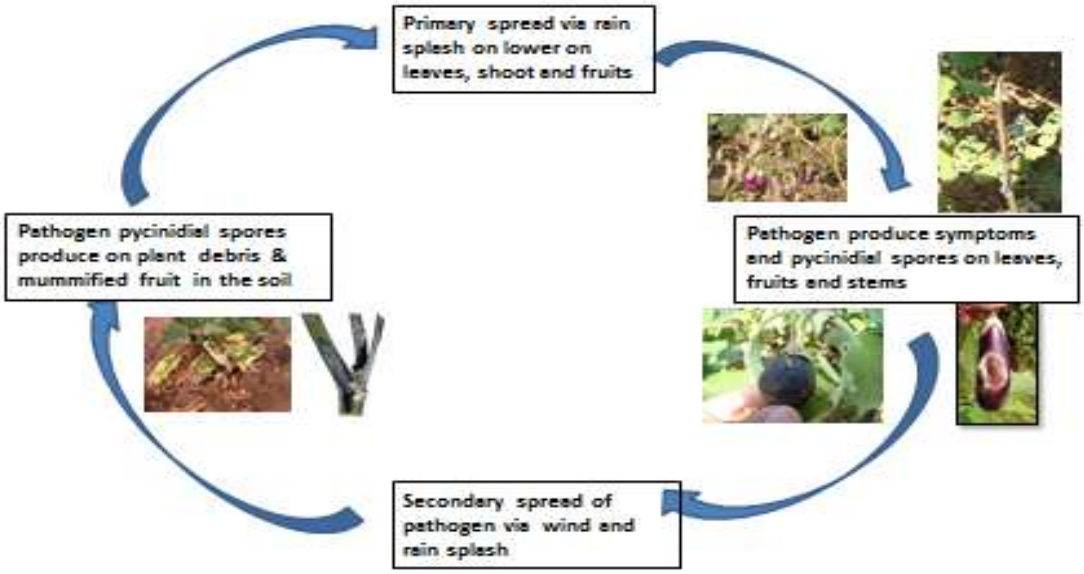
- At the base of the stem, the fungus causes characteristic constrictions leading to canker development and toppling of plants.
- On fruits, small pale sunken spots appear which on enlargement cover entire fruit surface. These spots become watery leading to soft rot phase of the disease.
- A large number of dot like pycnidia also develop on such spots.
- The infection of fruit through calyx leads to development of dry rot and fruits appear black and mummified.

Disease cycle:

- The fungus is seed and soil borne. The pathogen perpetuates in soil living on affected host tissues.
- The pathogen also found infecting seed (Seed borne).

Primary sources of Infection: Pathogen is seed borne and also survives in plant debris as mycelium and pycnidia.

Secondary sources of infection: Conidia dispersed through rain splashes, irrigation water, agricultural tools and insects.



Favorable condition:

- Temperature 30 – 32^0 C
- Wet weather for spread of disease, while, infection take place at 29^0 C and for disease development 26^0 C.

Management:

- Seed dipping in hot water at 50^0 C for 30 min.
- Spray difolatan 75 WP 0.2% or captan 75 WP 0.2% in nursery and field at 7 – 10 days interval
- Practicing crop rotation and summer ploughings helps in reducing initial inoculum.
- Collecting the diseased plants and destroying them
- Spraying zineb 0.2% or BM 0.8% found effective or spray twice with thiophanate methyl or carbendazim @ 0.1% at 20 days interval.
- Use disease free seed for sowing.

2. Little leaf

C.O:**Phytoplasma (MLO'S) Mycoplasma like organism**

Distribution: India, Sri Lanka

First Report: Coimbatore (T.N.)

Loss : 99 % fruit yield

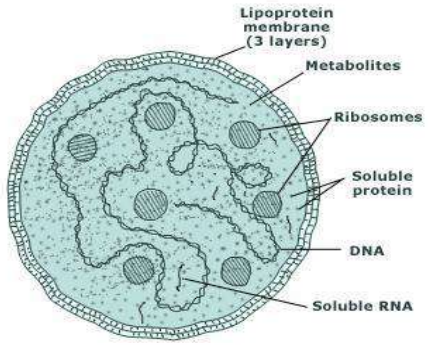
Economic Importance:

This disease of brinjal was reported from India in 1938 and as far as known it occurs only in India and Sri Lanka. In almost all the states of the country it has

become a serious problem facing brinjal cultivation. The yield loss is hundred per cent in the diseased plants.

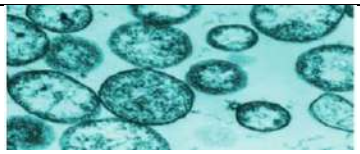
Pathogen:

The disease is caused by phytoplasma having ovoid to spherical body which is concentrated in the phloem sieve tubes.

Phytoplasmaare specialized bacteria that are obligate parasites of plant phloem tissue and transmitting insects (Vector). They were discovered by scientists in 1967 and were named mycoplasma-like organisms or **MLOs**. They cannot be cultured *in vitro* in cell-free media. They are characterized by their lack of a cell wall, normally with a diameter less than 1 micro meter, and their very small genomes phytoplasma *lacks a cell wall and instead is bound by a triple-layered membrane.*	 **Typical Cell of Mycoplasma like organism**

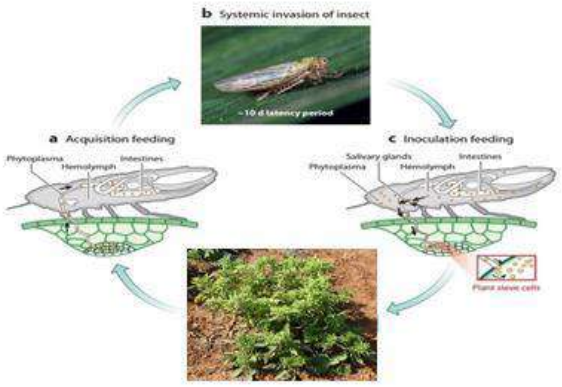
Symptoms

- Diseased plant show extreme reduction in size of their leaves.
- Such leaves are narrow, soft, smooth and yellow.
- Newly formed leaves are much shorter.
- Internodes of the stem are also shortened.
- Axillary buds get enlarged but their petioles and leaves remain shortened. This giving the plant a bushy appearance.
- Mostly there is no flowering but if flowers are formed they remain green.
- Fruiting is rare, if any fruit is formed, it becomes hard, tough and fails to mature. Seeds from infected fruits have low germination.



Spread:

- The pathogen perenneates in weed hosts. The disease is a sap transmissible disease. **Leaf Hopper** - *Hishimonasphycitis.* **Jassid-** *Emposcadevastans*
- The varieties Pusa Purple Long and Selection T are highly susceptible.
- **Collateral hosts:** *Datura fastuosa, D. stramonium, Vinca rosea*, chilli, tomato and tobacco, *Argemone mexicana* (pila datura, Guj- Darudi).



Management

- Eradication of solanaceous weed hosts.
- Destruction of diseased brinjal plants.
- Spray Malathion 50 EC @ 0.05 % @ 30 ml/ 10 liter water or Methyl -o-demeton or Dimethoate @ 20 ml/ 10 liter water.
- Mycoplasmas are reported to be suppressed by tetracycline but field application of this method has not yet been recommended.
- Seedling dip in tetracycline (10-50 ppm). Soil application of phorate @ 10 kg/ha after 10-15 days of transplanting.

…..x…..x…..x…..

22. TOMATO

1. Early blight C.O.: *Alternariasolani*
2. Late blight C.O. :*Phytophthorainfestans*
3. Buck eye rot C.O.: *Phytophthoranicotianae*
4. Leaf curl: Tobacco leaf curl virus (TLCV)
5. Tomato spotted wilt – Tomato Spotted Wilt Virus (TSWV)

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1. Early blight

C.O.: ***Alternariasolani***

Phylum:-Deuteromycotina
Order:-Moniliales
Family:-Dematiaceae

Distribution:

England, Columbia, Bulgaria, Florida, South Africa, India, Australia and United States.

Losses: Datar and Mayee (1981) reported 78 % loss in fruit yield.

Symptoms:

- The disease is prevalent in nursery as well as in main field after transplanting.
- On infected leaves circular to angular, dark brown to black spots with characteristic concentric ring are observed.
- The spot coalesce and cause premature drying of leaves.
- Lowest leaves are attacked first and the disease progresses upwards.
- Several spots coalesces to from large patches, resulting in leaf blight.
- Dark spots are also found at the base of stem near ground and the stem is gradually girdled.

- Spots are also found on the stem at the juncture of the side branches which are easily broken by wind.
- On the fruit- dark brown sunken spots are found at the calyx end and sometimes shedding of immature fruits are also occur.
- Brown spots are also seen on calyx.
- The fungus cause damping off of seedling in nursery beds and collar rot of growing plants.

Favorable condition:

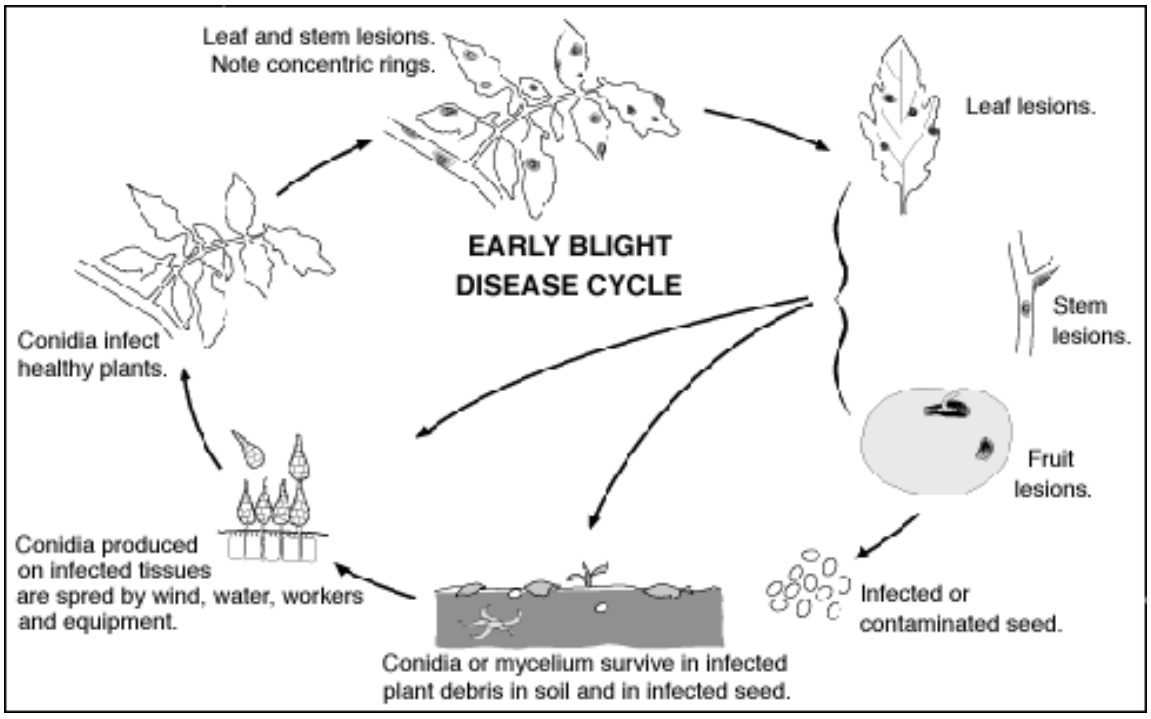
- Conidial germination 28^0 - 30^0 C.
- Older leaves are more susceptible.
- High soil moisture

- High atmospheric humidity-90-95 %
- Plants affected by water stress are more vulnerable for infection.
- Dry warm weather alternating with the intermittent rains

Host: *Capsicum annum* (Chilli), *Lycopersicon esculentum* (Tomato), *Solanum tuberosum* (Potato)

Disease Cycle:

- The pathogen can survive for 3 years in dried infected plant debris under dry condition. Mycelium and conidia survive in soil on disease plant debris.
- It is also seed borne.
- The secondary spread takes place through wind borne conidia and also transmitted by water and insect.



Management:

- Use disease free seed for sowing.
- Seed soaking in thiram 0.2% at 30^0 C for 24 hrs.
- Seed treatment with thiram 2 g / kg seed.
- Spraying of Mancozeb 75 WP 0.2 % @ 27 gram/10 liter water or Chlorothalonil 0.2% 75WP @ 27 gram/ 10 liter water from nursery stage.
- Follow crop rotation.
- Field sanitation.
- Dead haulms should be raked together and burnt immediately after harvest.

2. Late blight

C.O. :*Phytophthorainfestans*

Phylum:-*Oomycotina*

Order:-*Peronosporales*

Family:-*Pythiaceae*

- Late blight is a serious problem affecting cultivation of tomato the world over.
- This disease is reported from some parts of North India, particularly from the hill stations where potato is commonly affected by this fungus and in part of Karnataka

Distribution:

- N. America, Mexico, S. America Europe, West Indies, Africa , India and Australia
- **India:** First Reported by **Butler** in 1903. **Losses:** 95% yield losses were reported.

Symptoms:

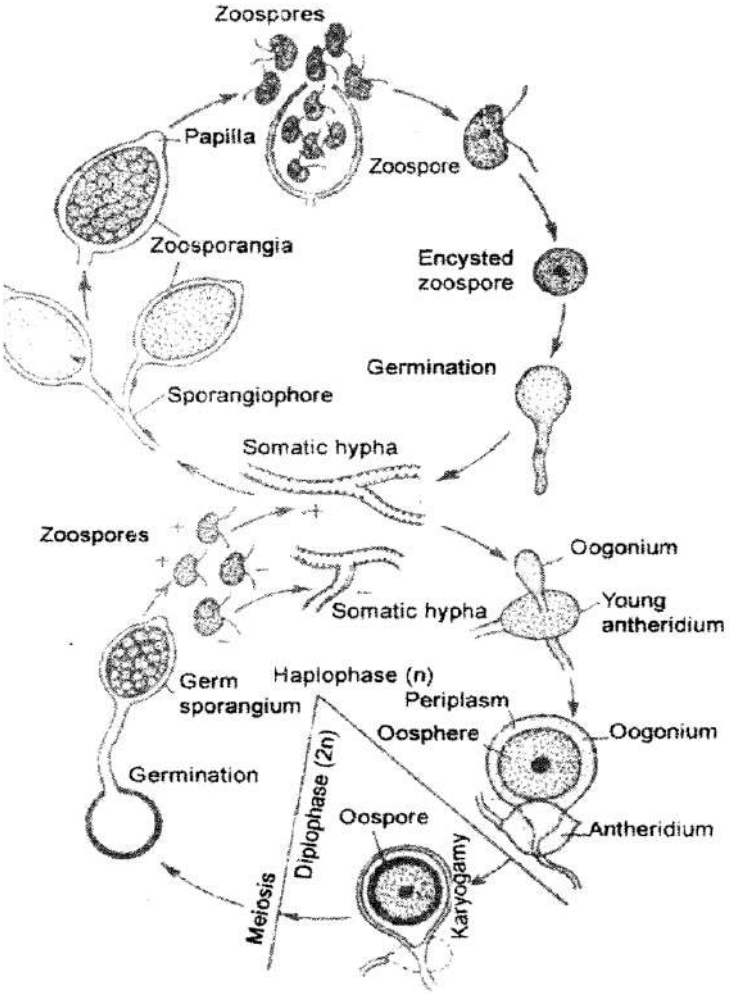
- Leaves, stem and the fruits are attacked by the pathogen.
- Water soaked brown to purple black lesions usually develop on leaflets, rachis, petioles and stems of the infected plants.

- On leaves pale green, irregular spots appears on the tips and margins.
- The spots enlarge rapidly under moist weather showing necrosis of central tissues turning dark brown to black.
- On lower surface of the leaves, a white cottony mycelial growth of the fungus appears. Brown streaks also develops on stem.
- On green tomato fruits, olivaceous greasy spots observed. The spots gradually cover over entire fruits. The fruits become brown and shriveled.
- Blight is followed by soft rot resulting in fruit disintegration.

Host: Potato, Datura, Brinjal

Disease Cycle:

- The pathogen survives in diseased crop debris under wet condition.
- The pathogen also survives on different host plants.
- The secondary spread takes place through sporangium and zoospore with irrigation water.



Favourable Condition:

Rainfed crops, Rainy season, Heavy dew formation, High RH (90-95 %)

BLITECAST System: To forecast late blight disease of tomato in USA.

Management:

- Field sanitation and destruction of diseased plant materials.
- Do not plant tomato plants near potato or old tomato crop.
- Sprinkler irrigation should be avoided

- Spray Mancozeb 75 WP 0.2 % or zineb 75 WP 0.2 % at 3 to 7 days interval or Spray (8% metalaxyl + 64% mancozeb) or (cymoxanil 8% + mancozeb 64%).
- Three sprays of Mancozeb 75 WP 0.2 % or Zineb 75 WP 0.2 %, Carbendazim 50 WP 0.2 % or Benomyl 50 WP 0.05% at fortnightly interval.
- Remove infected plant debris.
- Avoid heavy doses of nitrogenous fertilizers and dense population.
- Irrigation should be given at regular intervals.Avoid water stress or stagnation.

3. Buck eye rot

C.O.: *Phytophthoranicotianae*

- The fungus causing this disease is a destructive pathogen and is found in all parts of the world where moisture is plentiful and temperatures are favourable for its development.
- In tomato buckeye rot of fruits is very conspicuous.
- The fungus can attack beans, brinjal, chilli, onion, potato, pumpkin and maize.

Symptoms

- Spots on tomato fruits are marked with pale brown, concentric rings resembling slightly the markings on a buckeye nut.
- These spots may be small or they may cover a major portion of the fruit surface.
- There is no rotting of the skin but internal flesh may be discoloured up to the core.
- When green fruits are attacked they show brownish circular spots at the blossom end, shrink and are mummified.
- Due to inroad of secondary rot causing organisms the older fruits soon decompose.
- Concentric rings are not formed when the fungus attacks fruits of brinjal and chilli.
- Leaves may be attacked but leaf blight is not a common symptom.

Favourable condition:

- Buckeye rot of tomato fruits does not occur in dry weather.
- Infection occurs only when the host cells are gorged with water.
- Fruits in contact with soil which has been saturated with water for about 12 hours readily catch infection.
- The fungus attacks a plant slightly at a temperature as low as 13°C or as high as 35°C.
- The optimum is in the range of 24° to 30°C although the fungus can grow at temperatures above 35°C.
- In the root rot phase of the disease tomato roots appear to compensate for loss of roots at shallow depths by extracting more water from deeper in the soil profile. Salinity stress predisposes plants to attack of Phytophthora.

Pathogen:

- The hyphae of the fungus are tough, irregular in width but without marked hyphal swellings.
- Sporangiophores are thinner than vegetative hyphae, irregularly or sympodially branched, the sympodia being close in moist air.
- The sporangia are papillate, occasionally have more than one apex, and broadly ovoid, ellipsoid, obpyriform to spherical.
- The apex is not noticeably narrowed. Antheridia and Oogonia are readily formed when opposite strains of the fungus are grown together in culture.
- On maturity oogonia become rough, thick walled, yellow-brown.
- Oospores are aplerotic. The fungus produces chlamydospores abundantly.

Disease cycle

- The fungus sporangia or chlamydospores can be moved in furrow irrigation water and initiate fruit rot.
- The pathogen is soil-borne through its oospores and chlamydospores.
- Pepper, eggplant and tomato seedlings get infected in the nursery beds and carry the infection to the main field without showing any symptoms.
- However, this infection is not the cause of fruit rot which occurs when fruits come in contact with infested soil.
- After infection, spores are produced on fruits and disseminated by wind, water and other agencies to other fruits.

Management:

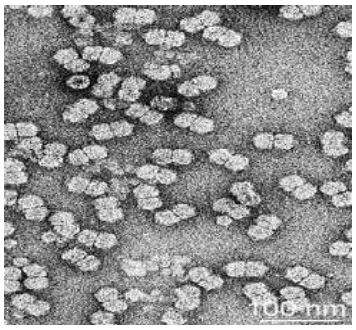
- Crop rotation, destruction of weeds, good soil drainage, selection of light soil, seed treatment, and spraying in the nursery beds as well as in the main field are important for managing buckeye rot.
- Sequential spray of captafol, mancozeb (0.25 per cent) and copper oxychloride.
- Staking of plants to prevent contact between leaves or fruits and soil and removal of lower leaves and fruits up to 30 cm helps in prevention of initiation of the diseases and its spread.

4. Leaf curl: Tobacco leaf curl virus (TLCV)

(Nicotiana Virus – 10) (Gemini Group Virus)

Symptoms:

- The disease is characterized by severe stunting of the plants with downward rolling and crinkling of the leaves.
- Curling, puckering, blistering, and smalling of the leaves.
- The plants are stunted due to shorting of internode. The newly formed leaves show chlorosis. The older, curled leaves become leathery and brittle.
- On the under surface of the leaves, there is leafy out growths called '*enations*' followed by mottling and vein clearing with complete of partial sterility.
- The disease plant produce more lateral branches resulting in bushy growth.



Mode of spread and survival:

- It is neither seed nor sap transmissible. But seeds from fresh fruits having infection may have the virus on the seed coat.
- The virus is transmitted by **white fly (***Bemisiatabaci*) and grafting. Minimum acquisition and inoculation feeding period is 30 min. After acquisition the insect requires another 6 hrs of incubation period to become viruliferous.

Favorable condition:

Dry hot season with low humidity

Host: Sesame, shoe flower, sun hemp, tobacco, zinnia, papaya, lady's finger and eggplant.

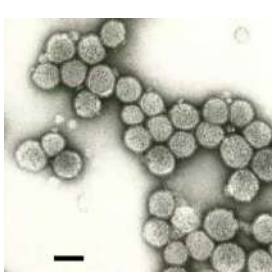
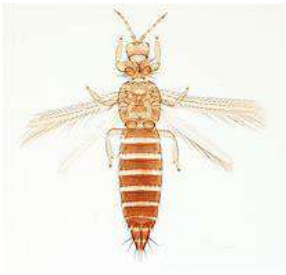
Management:

- Treat the nursery beds with carbofuran @ 1 kg a.i/ha at the time of sowing.
- Give two to three sprays of Triazophos 0.04 % (10 ml/10 liter) or (Imidacloprid 70 % WP 2.5 gram/ 10 liter water) or Dimethoate 0.05% or Metasystox 0.02% at 10 days interval.
- Grow border or barrier crops – Maize or Sorghum or Pearl millet.
- Wild tomato, *Lycopersiconperuvianum* has high degree of resistance, this can be used as resistant source in breeding programme.

5. Tomato spotted wilt – Tomato Spotted Wilt Virus (TSWV)

Symptoms

- Symptoms vary among hosts and in a single host species.
- **Stunting** is a common symptom of TSWV infection.
- **Chlorotic or necrotic rings** form on the leaves of many infected hosts.
- **Pale red or yellow areas with concentric circular marking** in the normal red skin of ripe tomato are formed. Discoloration of seed. Thickening of veins and **bronzing** of young leaves.

- Growing tips may die-back and terminal branches may be streaked.
- Affected plants may have a one sided growth habit or may be entirely stunted and have drooping leaves, suggesting a wilt.

Mode of spread and Survival

- **Thrips**, *Frankliniella schultzii*, *Scirtothrips dorsalis.*
- Adult thrips transmit the disease, only when the larvae acquire the virus from infected plants.
- **Collateral hosts:** *Acanthospermumhispidum, Aster sp., Boerhaaviadiffusa, Chrysanthemum sp., Cleome gynandra*, cowpea, *Dahlia variabilis*, eggplant, French bean, Gerbera sp., groundnut, *Lagascamollis*, lettuce, marigold, pea, pepper, pineapple, potato, *Trianthemaportulacastrum*, water melon and *Zinnia elegans*.

Management

- Removal and destruction of infected plants and weed hosts
- Vector control with Dimethoate (0.05%) or Methyl-o-demeton (0.02%) at 10 days interval.
- Growing *Crotolariajuncea*as a barrier crop reduces vector migration.
- Spraying of sorghum or coconut leaf extract.
- Treat the nursery beds with Carbofuran @ 1 kg a.i/ha at the time of sowing.

…..x…..x…..x…..

23. BHENDI (OKRA): *Abelmoschusesculentus*(L.) Moench

1. Yellow vein mosaic C.O.: Bhendi Yellow Vein Mosaic Virus (BYVMV)
2. Powdery mildew C.O.: *Erysiphaecichoracearum*
3. Root-knot C.O.: *Meloidogynejavanica*, *Meloidogyne incognita*

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1. Yellow vein mosaic

C.O. : Bhendi Yellow Vein Mosaic Virus (BYVMV)

Most serious disease all over India and it is main limiting factor in bhindi cultivation

First report: Kulkarni from India in 1924.

Yield loss : 35 days after germination up to 94%.

Symptoms:

- The disease is characterized by yellowing and vein clearing of the entire network of veins in leaf blade and veins become thick.
- Young infected leaves turn yellow and reduced in size.
- In severe cases the chlorosis may extend to interveinal areas and may result complete yellowing of leaves.

- Plant is very much stunted. Infection starts at any stage of crop growth.
- Flowering of plant restricted. Fruits are fibrous, smaller, malformed, yellowish green in color and tough.

Mode of spread and survival:

- The virus transmission takes place through **white fly- *Bemisiatabaci.***
- The virus is not seed, sap or pollen transmitted.
- The vector require 15 to 20 min to acquire the virus from infected plants with 6-8 hrs. Incubation.
- White flies fed for 12 hrs can remain viruliferous for the entire life.
- **Host**- Weed-*Ageratum, Althea rosea, Croton sparsiflorus, Hibiscus spp.*

Favorable condition:

- Disease incidence is more during March to June when temperature is high and Relative Humidity is low.
- This condition favors white fly multiplication, which helps in faster spread of disease.
- Continuous cropping provides inoculum throughout the year.

Management:

- Rogue out the diseased plant and destroy them.
- Eradication of weed hosts.
- Crop rotation with non-host crops.
- Use resistant var. Guj. Hybrid Bhinda-1, PusaSavani, Punjab-7, ArkaAbhay, ArkaAnamika and *ParbhaniKranti*.
- Application of Furadan 1.5 kg/ha in furrow at the time of sowing.
- Seed treatment with imidacloprid also recommended for control of insect vector.
- Give two to three sprays of Triazophos 0.04 % (10 ml/10 liter) or (Imidacloprid 70 % WP 2.5 gram/ 10 liter water) or Dimethoate 0.05% or Metasystox 0.02% at 10 days interval.
- Soil application of carbofuran 3 G @ 1.5 kg/ha at the time ofsowing followed by 4-5 foliar sprays of Dimethoate @ 0.05 % or Methyl-o-demeton@ 0.02 % or Monocrotophos @ 0.05 % at 10 days interval.
- Sowing 4-5 lines of sorghum, bajara or maize all around the okra plot at least 60 days prior to okra sowing and followed with 3-4 sprays of Dimethoate @ 0.05% or Methyl-O-Demeton @ 0.02 % at 10 days interval.

2. Powdery mildew

C.O.: *Erysiphecichoracearum*

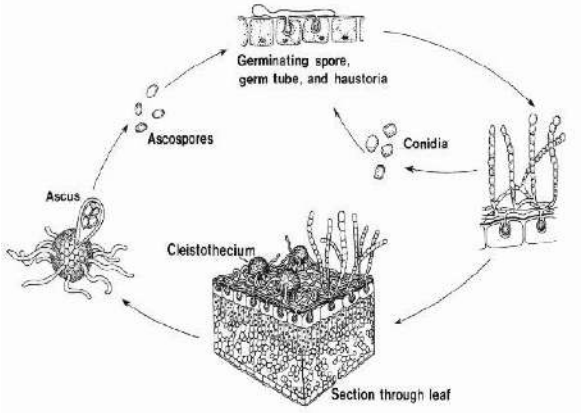
Symptoms:

- The leaves show the presence of white or grayish patches of powdery fungal growth on the upper surface of leaves.
- The entire leaf surface get covered with powdery growth.
- The lower surface may also show infection.
- In severe infection the affected leaves dry up and fall off pre-maturely.
- Early infection causes more effect on plant growth and yield.

Favorable condition:

- Dry weather condition favors powdery mildew.
- Disease observed during September to December.
- Favorable temperature for disease development is 15-30^0 C.

Disease cycle:



- Fungus perpetuate in Cleistothecial stage as well as the collateral hosts plays important role in providing inoculum.
- Secondary spread through wind borne conidia.

Management:

- Application of wettableSulphur 80 WP 0.2% or 300 mesh Sulphur dust at 25 kg/ha thrice at 20 days interval. First spray immediately after appearance of disease

- Spraying with Carbendazim 50 WP 0.05% or Dinocap 48% EC (10ml/ 10 lit. water) is also effective.

3. Root-knot:

C.O: *Meloidogynejavanica, Meloidogyne incognita*

Symptoms:

(A) Above Ground symptoms:

- ❑ Plant shows unthrifty development and stunted growth.
- ❑ Stunting, yellowing of the leaves and poor fruiting.

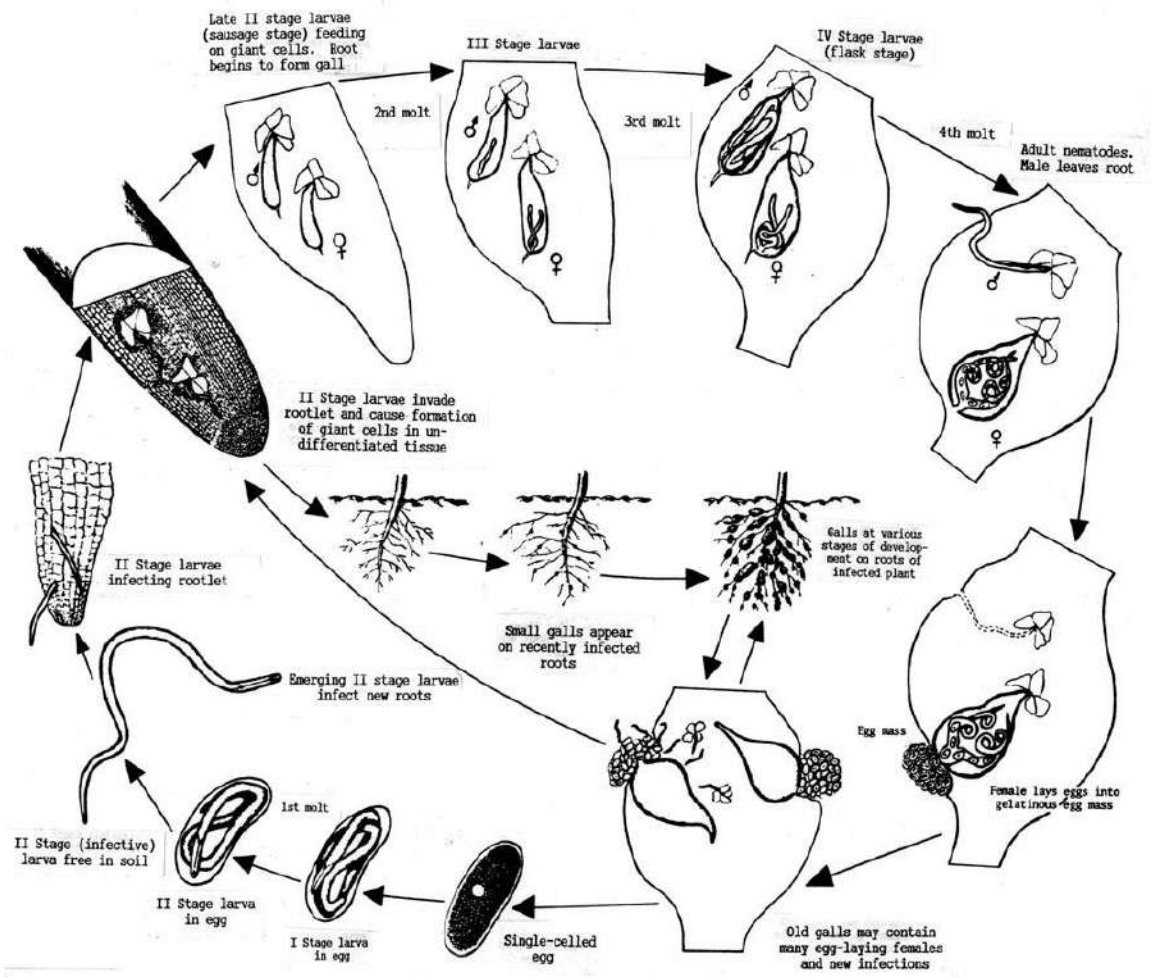
(B) Below Ground symptoms:

- ❑ Formation of knot or gall on root. The main and lateral root bears spherical to elongated various size of gall. Internodes become shortened. Decaying of tissue also observed.
- ❑ Other soil borne pathogens like *Fusarium*, *Macrophomina*may be involved in wilting giving complex phenomenon

Disease cycle

- ❑ The root knot nematodes are sedentary parasites of underground plant parts. The females are present in root galls. They can survive in soil and plant debris.
- ❑ Second stage larvae penetrate the tips of the root of the plant. After the entrance in to the host tissue, the larvae immediately lose mobility and establish a parasitic relationship with the host.
- ❑ Infection takes place through II stage larvae (J_2) Larvae move in soil in film of water. Disease is very common in light textured soil.
- ❑ In the roots the larvae undergo three molts to become adults and become spherical. About 30 days after penetration, the female lays eggs into a gelatinous sac which. The size of the egg sac may be larger than the female body. A single female can

lay 2800-3000 eggs in gelatinous matrix. The eggs hatch to become free living second stage larvae after completion of one molting while inside the shell.



❑

Management:

- Incorporate DD (Dichloropropane - Dichloropropene), DBCP (Dibromochloro-propane), Chloropicrin, Phorate 10 % G and Carbofuran 3 % G nematicides in the soil before planting.
- Follow crop rotation with cereals. Apply non-edible oil cakes 2.5 -3 t /ha10 days prior to sowing. Remove weed hosts. Summer deep ploughing.
- Soil solarization with 100 gauge LLDPE film during hot summer for 15 days.

…..x…..x…..x…..

21. CLUSTER BEAN:*Cyamopsistetragonoloba*(L.) Taub.

1. Bacterial blight C.O.: ***Xanthomonasaxonopodis*pv. *cyamopsidis***

2.Powdery mildew C.O.: ***Leveillulataurica***

3.Bean common mosaic C.O.: ***Bean common mosaic virus (ss RNA)***

---

1. Bacterial blight

C.O.: *Xanthomonasaxonopodis*pv. *cyamopsidis*

This was first reported in 1958 in India from Anand by Dr. M. K. Patel

Distribution:

Bihar, Delhi, Haryana, Punjab, Rajasthan, U.P, Gujarat and Maharashtra.

Symptoms

- Small, circular water soaked spots are formed on leaf.
- Such spots gradually enlarged and become angular and turn brown to black, which covered large area of the leaf and form blighted symptoms.
- On lower surface of leaves oily and olive coloured spots observed.
- Fully developed spots become black.
- The bacteria spread through veins, on leaf, hence the vein are become blank.
- Spots coalesce to form necrotic patches surrounded by chlorotic halo.
- Infection from blighted leaves spreads to stem through petiole. Diseased stem get cracked.
- Shiny layer also appear on affected area. Infected pods show heavy black spotting and seeds become shrivelled.

Bacterium

Gram negative, rod shaped, singly or in pairs, capsulated, motile with single polar flagellum

Disease cycle

- The bacterium is seed-borne and it spreads to new area by infected seeds.
- The bacterium enters in to host through wounds, stomata, hydathodes.
- The spread takes place through rain splash with wind.

Favourable condition

- High rainfall with more no of rainy days. Higher doses of N_2 doses from 20 to 40 kg/ha. Younger plants are more susceptible.

Management

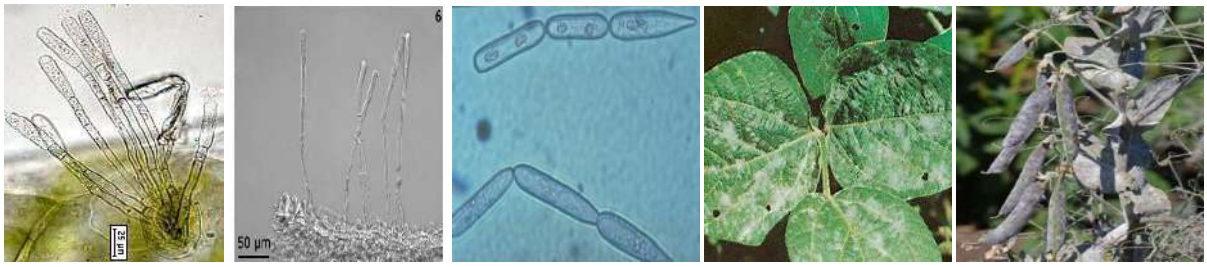
- Treat the seeds with hot water at 56^0 C for 10 min. to eradicate the seed borne infection.
- Use disease free seeds from summer grown crop.
- Before sowing seed, dip in streptocycline solution (250 ppm) (2.5 gram/ 10 litre water) for about 30 minutes.
- Spraying of streptocycline (1.5 g) + Copper oxychloride 50 % WP (40 g) at 12-15 days intervals after initiation of disease.
- Use resistant varieties: G-85, G-102, G-126 and G-255 which are highly resistant.

2. Powdery mildew

C.O.: ***Leveillulataurica***

Symptoms:

The disease is characterised by the appearance of a greyish powdery growth on the leaves and young pods followed by premature drying and defoliation of leaves the seeds become shrivelled and light in weight.



Disease cycle

- The pathogen survives in Cleistothecia on dried infected leaves which provides the primary source of inoculum.
- The secondary spread takes place through wind borne conidia.

Management

- Spray wettable sulphur 80 WP @ 0.2% or Dinocap 48 % EC 11 ml in 10 litre water after appearance of the disease.
- Dusting with 300 mesh sulphur dust @ 25-30 kg/ha after appearance of the disease.

3. Bean common mosaic

C.O: ***Bean common mosaic virus (ss RNA)***

Symptoms:

- Diseased leaves become rough, and show blisters on the leaf lamina.

Survival and spread

- Seed, sap, graft and aphid transmissible.
- **Infected seeds** are primary means of spread.
- *Aphis craccivora*, *Macrosiphumpisi*, *Aphis gossypi*, *A. medicaginis*, *Myzuspersicae*and *Brevicorynebrassicae*transmit the virus in a non-persistent manner.

Management

- Use of disease free seed.
- Vector control with insecticides.

…..x…..x…..x…..

25. BEANS

1. Anthracnose

C.O.: *Colletotrichumlindemuthianum*

Economic Importance

- ✓ The disease is more severe in temperate and subtropical mountainous regions of the world with *cool and wet climate*.
- ✓ The incidence of this disease has been reported to range from 5.0 to 65.0 per cent in different localities of Himachal Pradesh, resulting in considerable yield losses in certain years.

Symptoms

- All the above ground parts are affected at any stage of crop growth. However, the characteristic symptoms appear on pods.
- On cotyledons spots are sunken dark brown or black with pink spore mass.
- Seedling infection results in collapse of seedling.

- Spots on leaves appear on lower side and are black. Later these may also appear on upper surface.
- When the infection is severe, the affected plants wither off.
- *Black, sunken, circular spots of varying sizes appear on pods with bright red, yellow or orange margins.*
- The center of these spots later turns grey or pink due to sporulation of the pathogen, especially in wet weather.
- The border of these spots appear raised.
- In severely infected pods, brown to light chocolate coloured sunken cankers may also develop on seed coat.

Disease cycle

- P.I: Through **seed** and collateral hosts. The pathogen perpetuate in infected seeds and in infected plant debris forming Acervuli.
- S. I: Conidia by splashing rain water or air borne conidia.
- **Host range:** All vegetable beans and cowpea, mungbean, blackgram etc.

Favourable condition

- A relative humidity 92 per cent and above is essential for infection, the optimum being close to 100 per cent.
- The fungus requires about 10 mm of rain to establish initial infection.
- The optimum temperature for disease development ranges from 18 to 27^{o}C with maximum intensity at 21^{o}C and is markedly reduced at 13^{o}C.
- The movement of insects, animals and man may spread conidia particularly when foliage is moist.

Management

- Follow 2-3 years of crop rotation.

- Use disease free seed for planting.
- Seed treatment with Carbendazim or Benomyl @ 2 g/ kg seed (0.2%).
- Use resistant cultivars/ lines like KRC 1, KRC 17, EC 42960 and EC 57080.
- Spray Mancozeb 70 % WP (0.25%) or combination of Mancozeb (0.2%) and Carbendazim (0.05%) or Tricyclazole (0.03%) and repeat at 10 -14 days interval.

2. Bacterial blight

C.O.: *Xanthomonascampestris*pv, *phaseoli*

Significance:

- Like halo blight, this seed borne bacterial disease is a very important and widespread problem on bean.
- The disease is damaging in humid environments and is found in most bean production areas.
- This disease is actually caused by two very closely related, but distinct bacterial pathogens.

Symptoms:

- Early symptoms are water-soaked, angular leaf lesions.
- The young lesions expand and coalesce into larger lesions that are gray-brown and irregular in shape.
- Leaf spots are usually surrounded by a narrow yellow halo.
- Pods develop water-soaked, round to oval spots on sides of pods.
- Pod lesions later turn brown to red-brown. Bacterial ooze may be exuded from pod lesions.

Pathogen:

- This pathogen is an aerobic, Gram-negative, rod-shaped bacterium.
- Strains of this pathogen are restricted to hosts in the Fabaceae family.
- This pathogen is seedborne.

Disease cycle

- Infected seed is an important source of inoculum.
- Weed hosts may be important in some regions.
- In temperate climates, the pathogen may survive in crop residue and provide another source of inoculum.
- The pathogen is spread by splashing water from rain or sprinkler irrigation.
- Disease development is most pronounced at warmer temperatures of28-32° C with high humidity.

Management:

- Use disease free seeds.
- Crop rotation with non host crops.
- Spray strepatocycline 1 g + copper oxychloride 40g/10 litre of water.
- Choose resistant cultivars.
- Irrigate with furrow or drip irrigation, and avoid the use of overhead sprinklers.
- Eliminate volunteer and other hosts.

……x……x…..x…..

26. Colocasia (*Colocasiaantiquorum*)

1. Phytophthora blight:

C.O.: *Phytophthoracolocasiae*Racib.

Blight is the most serious disease of colocasia in India. In addition to *Colocasiaantiquorum*it occurs on other species of the genus as well as on *Amorphophalluscampandulatus*(elephant foot yam) also. The blight causes destruction of leaves thus reducing yield of corms and also causes rotting of fleshy parts in storage.

Symptoms

- The disease first appears in August-September. The earliest symptoms are the appearance of **small, dark, roundish spots on the leaf.**
- These spots rapidly enlarge concentrically and become circular, oval or irregular. Gradually, a very large part of the lamina gets coleased.
- Often, drops of a yellow liquid oozes out of the affected area.
- Later, the spots dry and affected portions drop out leaving shot holes in the leaf.
- The periphery of the spots zonate variable in different shades of brown, green and yellow, and may contain a delicate haze of sporangial growth of the fungus.
- In the case of infection of petioles the damage is often associated with presence of another parasite-a species of *Colletotrichum.*
- The rotting may affect the inflorescence also.
- In unusually wet weather the corms may rot completely.

Pathogen:

- The mycelium of *Phytophthoracolocasiae*Racib. iscoenocytic, intercellular in freshly attacked tissues and intracellular when rotting has advanced.
- The haustoria are slender, long and unbranched.
- Sporangiophores are formed on the surface of the leaf, unbranched, very slender and extremely narrow at the tip.
- They bear single, pear-shaped or elongated sporangia. As many as 12 reniform biflagellate zoospores are formed in each sporangium.
- Direct germination of the sporangium by a germ tube also occurs.
- Thick-walled, round and hyaline chlamydospores are also formed.
- The antheridium is amphigynous.
- The oospores are spherical, and lie free in the oogonium.

- The oogonia are yellowish and almost spherical.
- The antheridia persist at the base of the oogonia for a considerable time after the oospores are formed.

Disease cycle:

- The pathogen perpetuates through oospores in diseased leaf debris in soil and through infected corms used for planting.
- The fungus has poor competitive ability and does not survive in freestate in soil for long.
- Infected leaves buried deep in soil do not yield the fungus after5 days.
- Maximum sporangial production occurs at 21°C when the relative humidity is 100 per cent.
- Relative humidity lower than 90 per cent totally inhibits sporulation of the fungus on the host.
- Viability of zoospores is lost rapidly at relative humidities less than 90 per cent.
- Germination of sporangia by zoospores is optimum at 20° and 21°C. The zoospores germinate within 30 minutes after release.

Management:

- Selection of disease-free seeds.
- Sanitation of the field.
- Spray Bordeaux mixture (2:2:50), copper oxychloride @ 2.5 kg/ha or dithane Z-78 @ 2 kg/ha. ormetalaxyl 25 WP @ 500g/ha. at 10-15 days interval.
- Stickers like rosin (2 kg/500 lit.) or Sandovit should be mixed with the fungicides.
- Many bacteria including actinomycetes and some fungi present as leaf surface microflora are antagonistic to the pathogen and to check the disease on leaves.
- Use of resistant varieties. Following varieties are reported immune or resistant to various degrees:
  - Immune: Poonam Pat
  - Resistant: Sakin V
  - Moderately resistant: Ahina, Bhadia

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THEORY NOTE

B. Sc (Agril.)

Course No. Pl.Path.6.5

DISEASES OF FIELD AND HORTICULTURAL CROPS AND THEIR MANAGEMENT-II (CREDIT 2+1)

Semester-VI

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(1) DISEASES OF POTATO (*Solanum tuberosum* L.)

1. Late blight		
Causal organism	:	*Phytophthora infestans* (Mont.) deBary
Economic importance	:	The likely home of the disease is Mexico, where it is endemic on wild native species allied to the cultivated potato and somehow it has passed to the cultivated species of potatoes. First reported from Andes mountains of South America. **In India**, the disease was first introduced into the hilly regions of the Nilgiris during 1870-1880. Soon after, it was reported form Darjeeling in the Himalayas with the introduction of European potatoes. After 1912, the disease gradually made its way from the hilly regions into the plains. Since 1943, the disease appears regularly in almost all the plains of Northern India. In South India, the disease is prevalent in the potato growing regions of Karnataka and Tamil Nadu. The late blight disease of potatoes is the most devastating disease of potatoes in the world. It is most destructive, however, in areas with **frequent cool, moist weather**. Zones of high late blight severity include the northern United States and the east coast of Canada, western Europe, central and southern China, southeastern Brazil, and the tropical highlands. Late blight is also very destructive to tomatoes and some other members of the family *Solanaceae*. Usually infection starts on six weeks old plants. Late blight may kill the foliage and stems of potato and tomato plants at any time during the growing season. It also attacks potato tubers and tomato fruits in the field, which rot either in the field or while in storage. Late blight may cause total destruction of all plants in a field within a week or two when weather is cool and wet. Even when losses in the field are small, potatoes may become infected during harvest and may rot in storage. The **historical aspects** of late blight of potatoes in relation to the Irish famine and the establishment of *Phytophthora infestans* as the cause of late blight is mention as under:
Potato blight and the **Irish famine**: *a deadly mix of ignorance and politics*	:	**The Irish potato famine of 1845-1846:** In about 1800, the potato, which was introduced in Europe from South and Central America around 1570 a.d., was a well-established crop in Ireland. After strong objections against adopting it because (1) it was new and not mentioned in the Bible, (2) it was produced in the ground and, therefore, was unclean, and (3) because parts of it were poisonous, the potato was nevertheless adopted and its cultivation spread rapidly. Adoption of potato cultivation came as a result of it producing much more edible food per unit of land than grain crops, mostly wheat and rye, grown

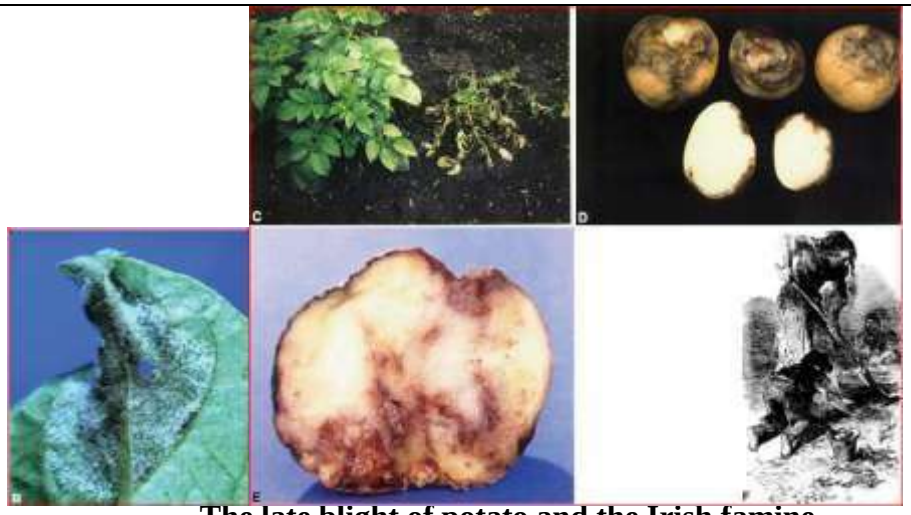
		until then. It was adopted also because the ground protected it from the pests and diseases that destroyed above-ground crops and from destruction by the soldiers sent by absentee English landlords to collect overdue land rents. At that time, most Irish farmers were extremely poor, owned no land, and lived in small windowless, one-room huts. The farmers rented land from absentee English landlords who lived in England, and planted grain and other crops. The yields were poor and, in any case, large portions of them had to be used for paying the exorbitant rent so as to avoid eviction. The Irish farmers also kept small plots of land, usually as small as a quarter of an acre and basically survived the winter with the food they produced on that land. Potato production was greatly favored by the cool, wet climate of Ireland, and the farmers began growing and eating potatoes to the exclusion of other crops and food-stuffs. Irish farmers, therefore, became dependent on potatoes for their sustenance and survival. Lacking proper warehouses, the farmers stored their potato tubers for the winter in shallow ditches in the ground. Periodically, they would open up part of the ditch and remove as many potatoes as they thought they would need for the next few weeks. The potatoes grew well for many years, free of any serious problems. In the early 1840s, potato crops began to fail to varying extents in several areas of Europe and Ireland. Most of the growing season of 1845 in Ireland was quite favorable for the growth of potato plants and for the formation of tubers. Everything looked as though there would be an excellent yield of potatoes everywhere that year. Then, the weather over northern Europe and Ireland became cloudy, wetter, and cooler and stayed that way for several weeks. The potato crop, which until then looked so promising, began to show blighted leaves and shoots, and whole potato plants became blighted and died. In just a few weeks, the potato fields in northern Europe and in Ireland became masses of blighted and rotting vegetation. The farmers were surprised and worried, especially when they noticed that many of the potatoes still in the ground were rotten and others had rotting areas on their surface. They did what they could to dig up the healthy-looking potatoes from the affected fields and put them in the ditches to hold them through the winter. The farmer's worry became horror when later in the fall and winter they began opening the ditches and looking for the potatoes they had put in them at harvest. Alas, instead of potatoes they found only masses of rotting tubers, totally unfit for consumption by humans or animals. The dependence of Irish farmers on potatoes alone meant that they had nothing else

		to eat - and neither did any of their neighbors. Hunger was quickly followed by starvation, which resulted in the death of many Irish. The famine was exacerbated by the political situation between England and Ireland. The British refused to intervene and help the starving Irish with food for several months after the blight destroyed the potatoes. Eventually, by February of the next year (1846), food, in the form of corn from the United States, began to be imported and made available to the starving poor who paid for it by working on various government construction projects. Unfortunately, the weather in 1846 was again cool and wet, favoring the potato blight, which again spread into and destroyed the potato plants and tubers. Hunger, dysentery, and typhus spread among the farmers again, and more of the survivors emigrated to North America. It is estimated that one and a half million Irish died from hunger, and about as many left Ireland, emigrating mostly to the United States of America. The cause of the destruction of the potato plants and of the rotting of the potato tubers was, of course, unknown and a mystery to all. The farmers and other simple folk believed it to have been brought about by "the little people," by the devil himself whom they tried to exorcise and chase away by sprinkling holy water in the fields, by locomotives traveling the countryside at devilish speeds of up to 20 miles per hour and discharging electricity harmful to crops they went by, or to have been sent by God as punishment for some unspecified sin they had committed. The more educated doctors and clergy were so convinced of the truth of the theory of spontaneous generation that even when they saw the mildew fungus growth on affected leaves and on some stems and tubers, they thought that this growth was produced by the dying plant as a result of the rotting rather than the cause of the death and rotting of the plant. Some of the educated people, however, began to have second thoughts about the situation. **Dr. J. Lindley**, a professor of botany in London, proposed incorrectly that the plants, during the rains, over absorbed water through their roots and because they could not get rid of the excess water, their tissues became swollen and rotted. The Reverend **Dr. Miles Berkeley**, however, noticed that the mold covering potato plants about to rot was a fungus (oomycete) similar but not identical to a fungus he observed on a sick onion. The fungus on potato, however, was identical to a fungus recovered from sick potato plants in northern Europe. Berkeley concluded that this fungus was the cause of the potato blight, but when he proposed it in a letter to a newspaper, it was considered as an incredible and bizarre theory unsupported by facts. The puzzle of what caused blight of potato continued unanswered for 16 years after the

Louis Pasteur
(1822 – 1895)

Heinrich Anton deBary
(1831 – 1888)

1845 destruction of potatoes by the blight. Finally, in 1861, **Anton deBary** did a simple experiment that proved that the potato blight was caused by a fungus. DeBary simply planted two sets of healthy potatoes, one of which he dusted with spores of the fungus collected from blighted potato plants.When the tubers germinated and began to produce potato lants, the healthy tubers produced healthy plants, whereas the healthy tubers dusted with the spores of the fungus produced plants that became blighted and died. No matter how many times deBary repeated the experiment, only tubers treated with the fungus became infected and produced plants that became infected. Therefore, the fungus, which, we know now, is an oomycete was named *Phytophthora infestans* ("infectious plant destroyer" from phyto = plant, phthora = destruction, infestans = infectious), was the cause of the potato blight. DeBary also showed that the fungus did not just reappear from nowhere the following growing season but instead survived the winter in partially infected potato tubers in the field or storage. In the spring, the fungus infected young plants coming from these partially rotten tubers, produced new spores on these plants, and the spores then spread to other cultivated potato plants that were infected and killed. With this experiment, deBary actually disproved the **theory of spontaneous generation**, which stated that microorganisms are produced spontaneously by dying and dead plants and animals, and ushered in the **germ theory of disease.** The honor for this proof, however, is reserved for **Louis Pasteur**, who proved the theories while working with bacteria at about the same time, 1861–1863, that deBary published his work with the potato blight fungus.



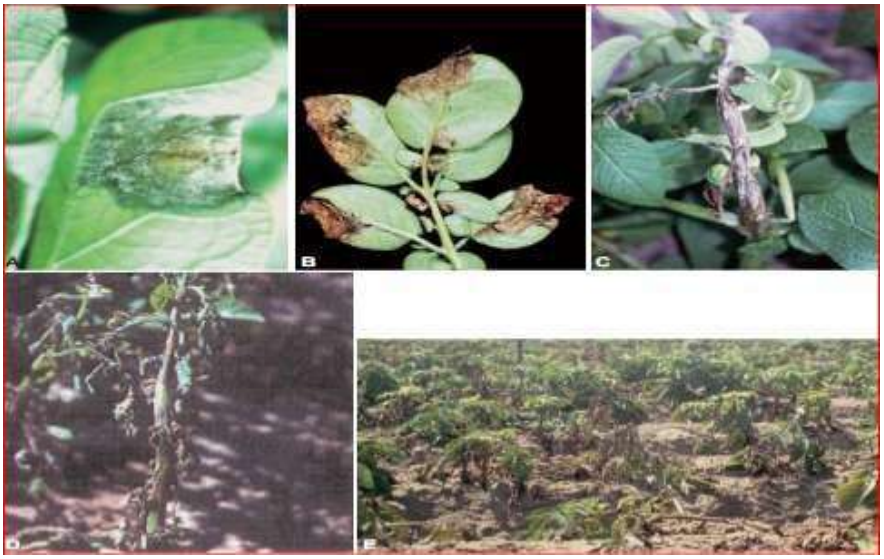
The late blight of potato and the Irish famine

(B) A young lesion on a potato leaf covered with sporangiophores and sporangiospores of the fungus (oomycete). (C) A potato plant killed completely by the blight (right) next to a healthy-looking resistant plant (left). (D) External and internal appearance of potato tubers infected with the late blight disease. The oomycete is still found near the surface. (E) Advanced invasion and rotting of potato tuber infected with late blight. (F) A period drawing of a family digging for potatoes to avoid starvation during the Irish famine (Illustrated London News, 1849).

	:	□ The devastating epidemics of late blight of potato in northern Europe, particularly Ireland, in the 1840s not only dramatized the effect of plant diseases on human suffering and survival, but also greatly stimulated interest in their causes and control. □ In 1861, **deBary** finally established experimentally beyond criticism that a fungus (*Ph. infestans*) was the cause of the plant disease known as late blight of potato, a disease that closely resembles the downy mildews.
Symptoms	:	□ Symptoms appear at first as water-soaked spots, usually at the edges of the lower leaves. □ In moist weather, the spots enlarge rapidly and form brown, blighted areas with indefinite borders. □ A zone of white, downy mildew growth 3 to 5 mm wide appears at the border of the lesions on the undersides of the leaves. □ Soon entire leaves are infected, die, and become limp. □ Under continuously wet conditions, all tender, above ground parts of the plants blight and rot away, giving off a characteristic odor. □ Entire potato plants and plants in entire fields may become blighted and die in a few days or a few weeks. □ In dry weather, the activities of the pathogen are slowed or stopped. Existing lesions stop enlarging, turn black, curl, and wither, and no oomycete appears on the underside of the leaves. □ When the weather becomes moist, again the oomycetes resumes its activities and the disease once again develops rapidly. □ Affected tubers at first show purplish or brownish blotches consisting of water-soaked, dark, somewhat reddish brown tissue that extends 5 to 15 mm into the flesh of the tuber. □ Later the affected areas become firm and dry and somewhat sunken. Such lesions may be small or may involve almost the entire surface of the tuber without spreading deeper into the tuber interior. □ The rot, however, continues to develop after the tubers are harvested. □ Infected tubers may be subsequently covered with sporangiophores and spores of the pathogen or become invaded by secondary fungi and bacteria, causing soft rots and giving the rotting potatoes a putrid, offensive odor.

Figure 1A. A late-blight lesion on a potato leaflet, as seen from the underside of the leaf. Note the thin, delicate white growth of the pathogen on the underside along the perimeter of the lesion.

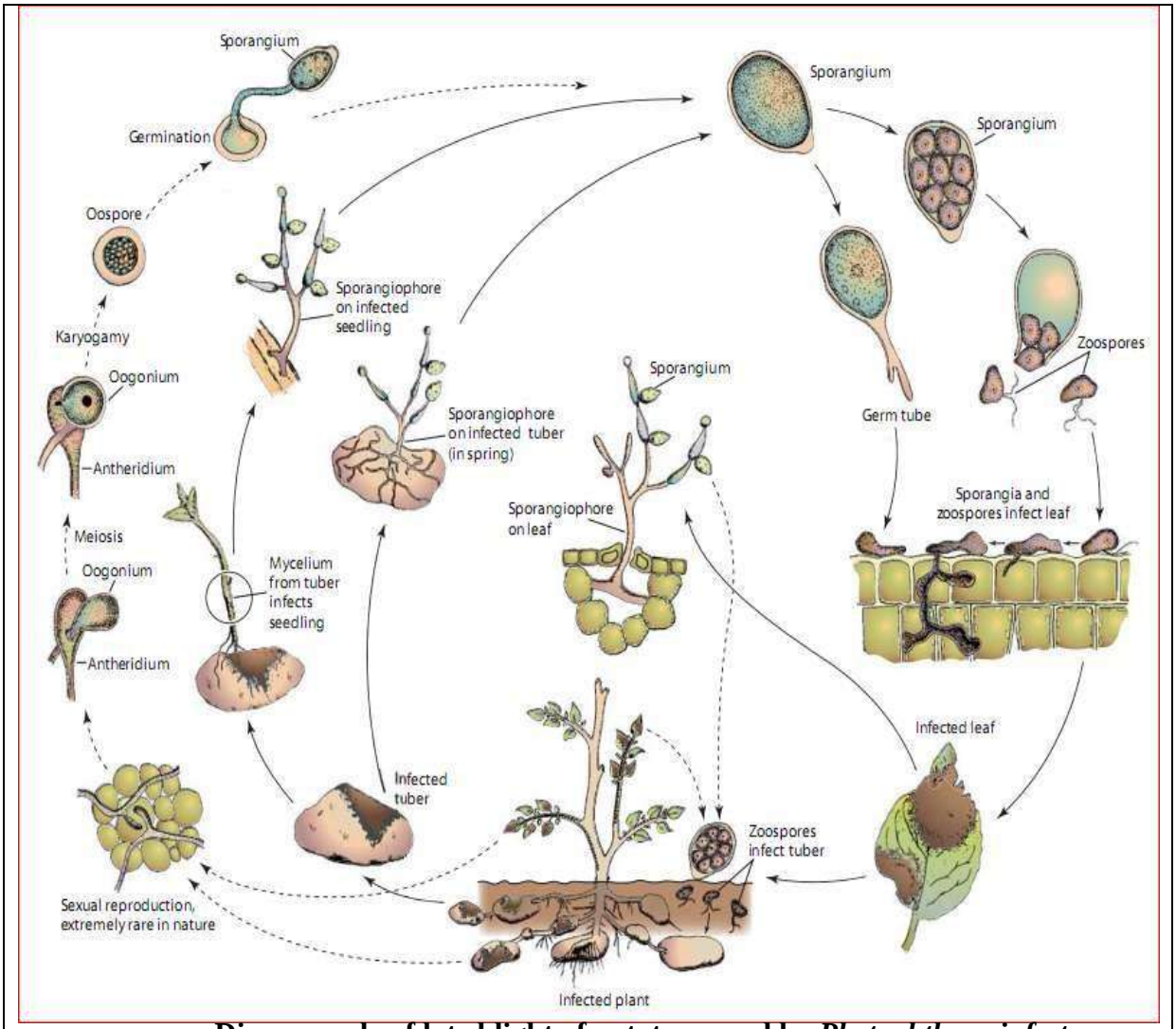
Potato late blight



Stages in potato late blight caused by *Phytophthora infestans*

(A) Single leaf lesion with sporangiophores and sporangia. (B) Blight lesions on many leaflets. (C) Necrosis of stem. (D) Death and collapse of shoots and stem of a potato plant. (E) Death and collapse of blighted plants in the field.

The Pathogen: *Phytophthora infestans*	:	The mycelium produces branched sporangiophores that produce lemon-shaped sporangia at their tips. At the places where sporangia are produced, sporangiophores form swellings that are characteristic for this oomycete. Sporangia germinate almost entirely by releasing three to eight zoospores at temperatures up to 12 or 15°C, whereas above 15°C sporangia may germinate directly by producing a germ tube. The oomycete requires two mating types for sexual reproduction. Until the late 1980s, only one mating type was present in countries outside Mexico. Since then, however, both mating types have become widely distributed in most countries and, as a result, new strains of the pathogen have appeared. Some of the new strains are much more aggressive than the old ones and quickly replace them. When the two mating types grow adjacently, the female hypha grows through the young antheridium (= male reproductive cell) and develops into a globose oogonium (= female reproductive cell) above the antheridium. The antheridium then fertilizes the oogonium, which develops into a thick-walled and hardy oospore. Oospores germinate by means of a germ tube that produces a sporangium, although at times the germ tube grows directly into the mycelium.
Survival and spread	:	**PSI:** Infected tubers containing dormant mycelium and oospores in soil. The fungus can also survive on collateral hosts like tomato. **SSI:** Sporangia and zoospores through wind and irrigation water.
Favourable conditions	:	□ Late blight pathogen *P. infestans* is most serious in the northern latitudes; in the subtropics it is serious only during the winter. □ Cool moist conditions □ Relative humidity >90% and with suitable temperature 12-24^{0}C **Dutch rules for predicting disease epidemic** □ Night temperature below the dew point at least for 4 hours □ Minimum temperature 10°C or slightly above □ Clouds on next day □ Rainfall during the next 24 hrs at least 0.1 mm Forecasting system for late blight disease: BLITECAST (**Ref.: Krause, R. A**., Massie, L. B., and Hyre, R. A. (1975). *BLITECAST, a computerized forecast of potato late blight. Plant Dis. Rep., 59: 95-98*).

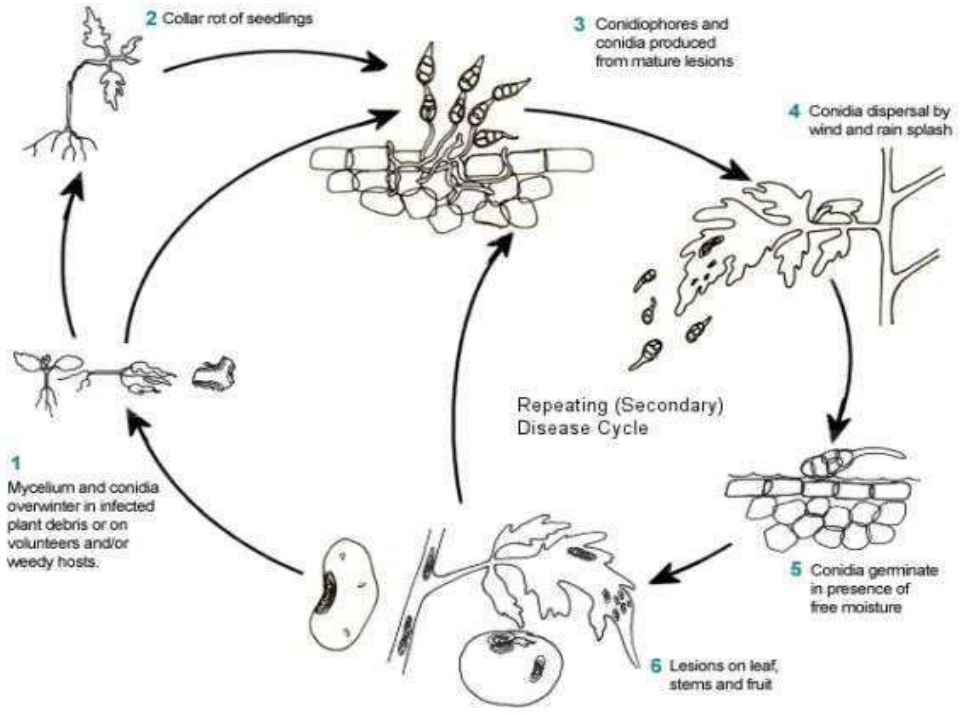


Disease cycle of late blight of potato caused by *Phytophthora infestans*

Reproduction	:	**Sexual reproduction occur in specific location** □ If both mating types (A1 and A2) come into contact with one another, sexual reproduction may occur. □ A nucleus from the antheridium enters the oogonium. Following karyogamy (the fusion of two nuclei), a thick-walled, diploid oospore is formed. □ Only mating type A1 was present in potato-growing areas outside Mexico, so sexual reproduction did not play a significant role in the disease cycle. □ Now, mating type A2 has migrated to most potato and tomato growing regions of the world, and sexual reproduction is believed to occur in some areas.
Management	:	□ *Regulatory measures* restricting movement of infected tubers specially for seed purpose □ Select healthy tubers for planting □ Delayed harvesting □ High ridging to about 10-15 cm height reduces the infection □ Avoid injuries to potato at harvesting time □ Grow resistant varieties such as Kufri Jyothi, Kufri

		Badshah, Kufri Jeevan, Kufri Sherpa, Kufri Naveen, Kufri Moti, Kufri Kuber, Kufri Kishan, Kufri Alankar *Cultural practices:* □ Weed free potato fields □ Tubers should be planted on high ridges. High ridges will cover the tubers thoroughly with soil and thus prevent growing out of the fungus from mother tuber and also infection in daughter tubers. □ Mulching between rows □ Avoid contact between foliage and tubers during harvesting □ Delay in harvesting *Biological Control:* □ Seed treatment and foliar application of *Bacillus cereus*, *Pseudomonas fluorescens* reduces disease severity *Prophylactic measures* □ Copper oxychloride 0.3% or mancozeb 0.25% or chlorothalonil 0.2% or B.M. 1% □ Metalaxyl + mancozeb 0.2% can be applied at 7 to 10 days intervals in the hills and 10 to 15 days intervals in plains □ Dip sprouted tubers in 0.2% Metalaxyl 72 WP for 30 min.
2. Early blight		
Causal organism	:	*Alternaria solani* (Ell. & Mart.) Jones & Grout
Economic importance	:	In India, it is found on potato crops grown in the hills as well as in the plains. Since the disease appears early in the season, it is known as early blight, in contrast to *Phytophthora infestans* which appears late in the season, to cause blighting of the foliage.
Symptoms	:	□ Disease occurs early in the season on three weeks old plants □ Dark brown necrotic spots with yellow halo develop on older leaves □ Spots characteristically show zonations or concentric rings that give a target board look □ Chlorosis and necrosis extend beyond the infected tissue because of alternaric acid toxin produced by the pathogen □ Under favourable conditions, spots become hard and shots holes may develop □ Severer infection may result defoliation and stem rotting

Survival and spread	:	**PSI:** Conidia in infected debris, and collateral host *Solanum nigrum* and tomato **SSI:** Wind dispersed and rain splashed conidia



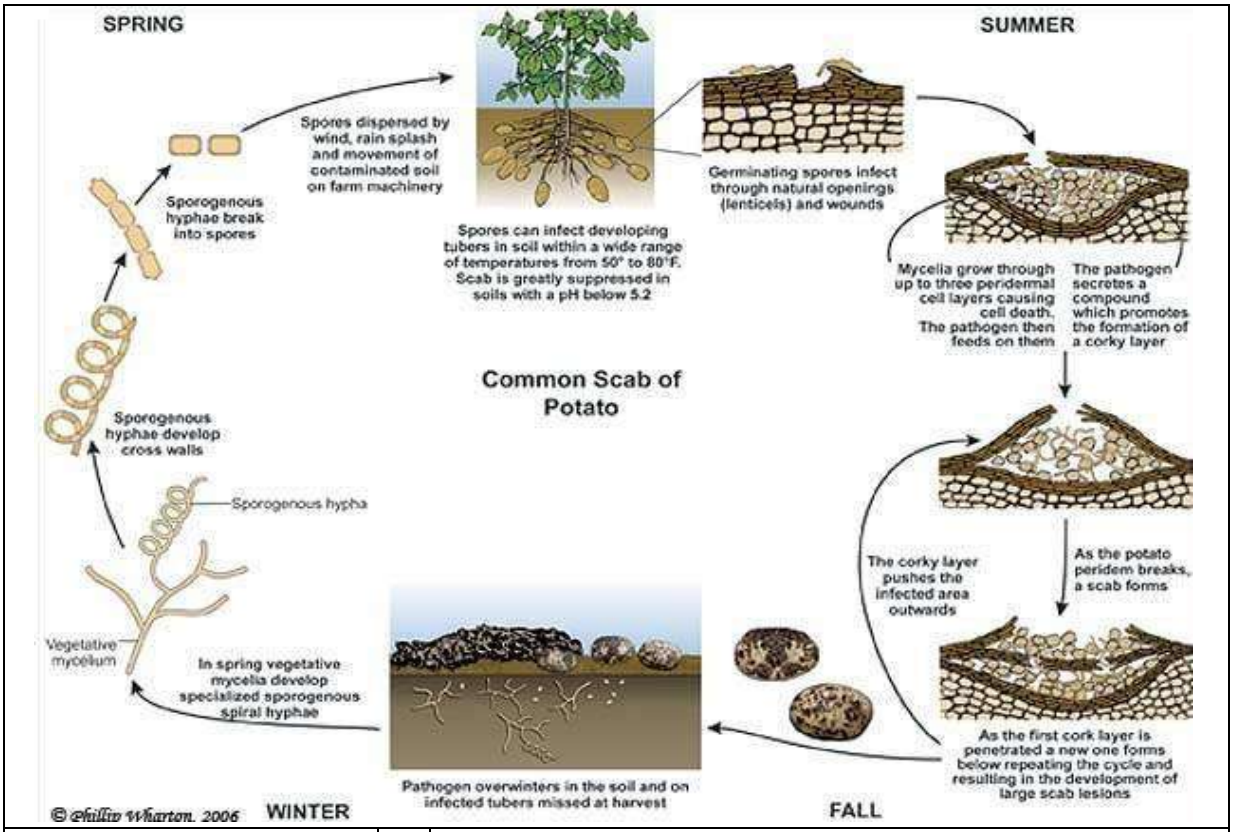
Favourable conditions	:	□ Previous crop either potato or tomato □ Temperature 13 to 26^{0}C □ RH: >80% for prolonged periods □ Infrequent rains □ Shorter photoperiods Forecasting system for early blight in tomato: **FAST** – Forecast for *A. solani* in Tomato

Management	:	□ Grow resistant varieties such as Kufri Naveen, Kufri Sindhuri, Kufri Jeevan, □ Resistant sources: *Solanum andegena Cultural practices*: □ Crop rotation, not involving potato or tomato for two years □ Select healthy tubers for planting □ Removing and burning infected plant debris and eradicating weed hosts. □ Maintain optimum growing conditions including proper fertilization, irrigation and management of other pests. □ Grow later maturing, longer season varieties. □ Avoiding irrigation in cool and cloudy weather. *Chemical*: □ Spraying Zineb 0.25% or Mancozeb 0.25% or Propineb 0.2% or Captan 0.2% or Copper oxychloride 0.25% or Chlorothalonil 0.2% two to three times at 7 to 14 days interval.

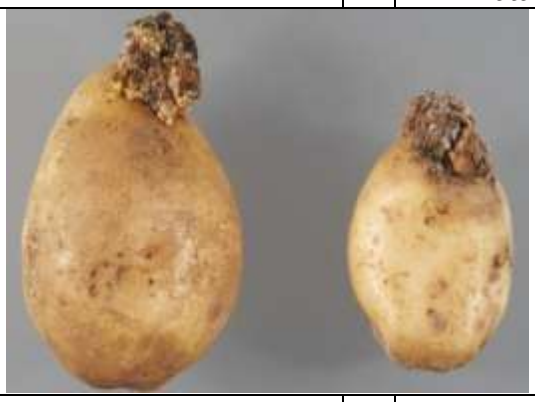
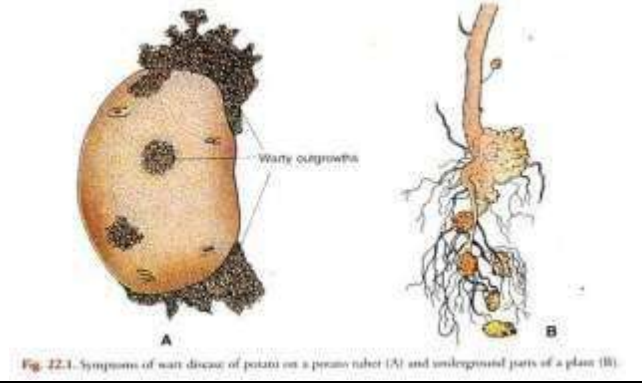
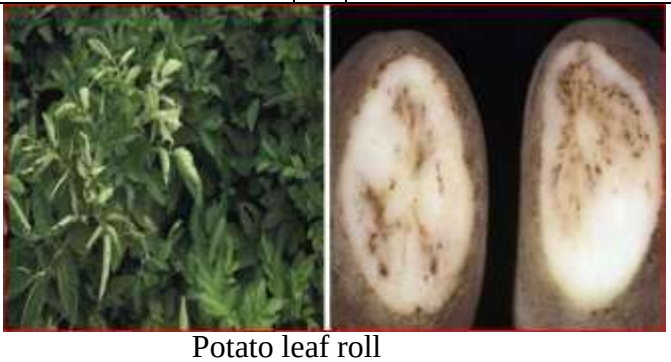
3. Brown rot/ bacterial wilt		
Causal organism	:	*Ralstonia solanacearum* (race 1) *= Pseudomonas solanacearum* *= Burkholderia solanacearum* Smith
Economic importance	:	Different synonyms (Name) Ghera / Utkha: Uttar Pradesh Bangle blight or Bangoli: Maharashtra Bangadi: Gujarat First bacterial disease reported in India First recorded in 1891 from Pune district of Maharashtra Also referred as bacterial wilt or ring disease or bangle disease Occurs in Deccan and Central plateau, Assam, West Bengal, Orissa, hills of Uttar Pradesh and Nilgiris Bacterium infects other solanaceous crops like egg plant and tomato causing wilt
Symptoms	:	□ Wilting, stunting and yellowing of foliage followed by collapse of entire plant □ Browning of xylem in vascular bundles □ Stems, petioles of the lower leaves and roots become brown and on cutting of infected materials slimy mass of bacteria oozes out □ Brown ring is formed in the tubers due to discolouration of vascular bundles (ring disease) □ Formation of pockets or cavities around vascular bundles in the pith and cortex □ Infected tubers may rot at harvest or during storage

Survival and spread	:	**PSI:** Bacterial cells survive in diseased and contaminated tubers and in soil **SSI:** Bacterial cells spread through contaminated soil and irrigation water
Favourable conditions	:	□ High soil temperature (25-35^{0}C) □ Soil moisture >50% □ Previous crop in the same field either egg plant or tomato
Some fact about *Burkholderia solanacearum*	:	□ Brown rot of potato caused by *Ralstonia solanacearum* race 3 biovar 2 is among the most serious disease of potato worldwide, which is responsible for an estimated $950 million in losses each year. □ Race 3 biovar 2 is cold tolerant and classified as a quarantine pathogen. □ In addition, this race/biovar has been listed as a Select Agent in the Agriculture Bioterrorism Act of 2002 and is considered to have potential to be developed as a bioterrorist weapon.

Management	:	□ Crop rotation with maize or soybean for 3 yrs □ Use disease free tubers for sowing □ Deep summer ploughing □ Treat seed tubers with 0.02% streptocycline for 30 min giving 4 mm deep sharp cut □ Disinfestations of cutting knives with sodium hypochlorite (10%) or mercuric chloride
4. Common scab		
Causal organism	:	*Streptomyces scabies*
Economic importance	:	Important in Punjab and Himachal Pradesh
Symptoms	:	□ The bacterium enters through unsuberized lenticels and causes infection of tubers. □ Produces a toxin called thaxtomin which is of significance in pathogenesis □ Two types of scabs are produced: shallow scab and deep lesions scab □ In shallow scab, tubers shallow superficial roughened areas often slightly sunken below the plane of healthy skin. Small brownish and slightly raised spots on tubers may develop □ In deep lesion scab, lesions may be as deep as 3 mm or more and darker than shallow lesions. These lesions enlarge, coalesce and become corky □ Infected tubers will become unfit for consumption and lose their market value
Survival and spread	:	**PSI:** Conidia or spores formed by fragmentation of mycelium and mycelium in seed tubers and soil **SSI:** Wind dispersed, rain splashed and irrigation water borne conidia or spores



Favourable conditions	:	□ Soil pH (5.2-8.0). Increasing pH favours disease development □ Soil temperature of 20-22^{0}C □ Low soil moisture
Management	:	□ Use of disease free tubers □ Crop rotation with wheat-oat or potato-onion-maize (4 yrs) □ Deep ploughing in summer followed by green manuring with green gram or sunhemp in *kharif*. □ Dipping of seed tubers in 3% boric acid for 30 minutes before planting □ Maintaining soil pH at about 5.2 by addition of sulphur □ Biocontrol with *Bacillus subtilis* □ Crop rotation with legume crops. □ Addition of organic matters to enhance activity of Bio-control agent. □ Regular irrigation to maintain the soil moisture. □ Avoid use of acidic fertilizers like CAN (calcium ammonium nitrate)
5. Wart		
Causal organism	:	*Synchytrium endobioticum* (Schilb.) Perc.
Economic importance	:	Losses seen in temperate regions In India, the disease is endemic to Darjeeling hills and is under domestic quarantine
Symptoms	:	□ All underground parts except roots and base of the stem show symptoms □ Buds on underground stems, stolons and tubers are the

		parts from which abnormal outgrowth starts and lumps of warts that resemble cock's comb develop □ On tubers warts are more typical and conspicuous, distorted and branched, sometimes covering the entire tuber surface and may grow bigger than the tuber itself □ As the crop matures, warts become black and rot and may emit smell because of putrefaction due to attack of secondary saprophytic organisms □ Also attacks other solanaceous crops like tomato and Datura but warts are not produced
  Fig. 22.1. Symptoms of wart disease of potato on a potato tuber (A) and underground parts of a plant (B)		
Survival and spread	:	**PSI:** Thick walled resting spores in soil and infected tubers (viable in soil for 40 years) **SSI:** Water borne and autonomous zoospores
Favourable conditions	:	□ High soil moisture □ Optimum temperature of 21^0C (12 to 28°C) □ Slightly acidic to neutral pH
Management	:	□ Strict quarantine measures and use of pathogen free seed tubers □ Grow resistant varieties such as Kufri Jyoti, Kufri Jeevan, Kufri Muthu and Kufri Kanchan
6. Leaf roll		
Causal organism	:	*Potato leaf roll virus* (PLRV)
Economic importance	:	Genus: Luteovirus with spherical particles having ssRNA
Symptoms	:	□ Upward and inward rolling of leaves, which have a stiff leathery texture □ Leaves show mild or diffused mottling □ Leaves become soft and droop □ Plants stunted and have a stiff upright growth □ Phloem necrosis of tubers in some varieties
 Potato leaf roll Potato aphid		

Survival and spread	:	**PSI:** Virus particles in infected tubers **SSI:** Virus particles transmitted by aphids, *Myzus persicae*
Management	:	□ Disease free seed tubers for planting □ Rouging of diseased plants □ Aphid control through raising barrier crops and spraying systemic insecticides

7. Black Scurf of Potato

Rhizoctonia solani (Black scurf and stem canker in potato)

- This disease occurrence is common both in hills and plains.
- There are two phases of disease -

1. Stem Canker phase: The sprouts are killed before they emerge and this delays the germination resulting in loss of yield. The canker may cause wilting of the plants also.
2. Black Scurf phase: Appearance of black crust on the tubers which reduces their acceptability to the consumers.

Management:
Disease development do not possible in low soil PH

1. The seed tubers should be treated before sowing.
2. Soil drenching. Combination of seed and soil treatment gives the best control of the disease.

MINOR DISEASES		**1. Potato spindle tuber**
Causal organism	:	*Potato spindle tuber viroid* (PSTVd)
Economic importance	:	Occurs in Canada, USA, Russia and South Africa Reported from Shimla hills in India
Symptoms	:	□ Plants appear erect, spindly and dwarfed □ Leaves small, erect and leaflets dark green □ Tubers elongated with cylindrical; middle and tapering ends □ Infected tubers are more smooth than normal tubers and have more tender skin and flesh □ Tuber eyes are numerous and more conspicuous and shallower
Survival and spread	:	**PSI:** Virus particles in infected tubers and mechanically through tuber cutting knives from infected to healthy tubers **SSI:** Virus particles transmitted by pollen and contaminated mouth parts of beetles, fleas and bugs

Management	:	□ Use of PSTVd free potato seed tubers □ Disinfestation of cutting knives by dipping them on sodium hypochlorite solution

Management	:	□ Crop rotation with cereals □ Healthy seed tubers should be planted after treating with Streptocycline 100 ppm and $CuSO_4$ 40 ppm for 30 min. □ Crop should be planted in well drained areas and at sufficient distances to allow adequate ventilation □ Avoid wounding of plants during inter-cultivation and tubers at harvest, transport and storage □ Remove all debris from warehouses and disinfect the walls with formaldehyde or copper sulphate □ Products to be stored should be dry, and the humidity and temperatures of warehouses should be kept low □ Control flies in field and storage

2. Mosaic

Potato mosaic virus is divided into three categories. Symptoms of the different mosaic virus of potatoes may be similar, so the actual type usually can't be identified by symptoms alone and is often just referred to as mosaic virus in potato. Still, it is important to diagnosis can be made by mosaic patterns on foliage, stunting, leaf deformities and tuber malformations. The three types of recognized mosaic virus in potato are Latent (Potato virus X), Mild (Potato virus A), Common mosaic (Potato virus Y).

Symptoms:
Latent mosaic, or Potato virus X, may produce no visible symptoms depending upon the strain but yields of infected tubers may be reduced. Other strains of Latent mosaic show light leaf crinkling. When combined with Potato virus A or Y, crinkling or browning of leaves may also be present. In an infection of Potato virus A (mild mosaic), the plants have light crinkling, as well as mild yellow mottling. Leaf margins may be wavy and appear rough with sunken veins. Severity of symptoms depends on the strain, cultivar and weather conditions. Potato virus Y (Rugose mosaic) is the most severe of the viruses. Signs include mottling or yellowing of leaflets and crinkling that is sometimes accompanied by leaf drop. Underside leaf veins often have necrotic areas showing as black streaking. Plants may be stunted. High temperatures exacerbate the severity of the symptoms. Again, symptoms vary hugely with both potato cultivar and virus strain.

Both viruses A and Y are carried in tubers but are also transmitted by several species of aphids. All of these viruses overwinter in potato tubers.

Black Heart

Black heart of potato

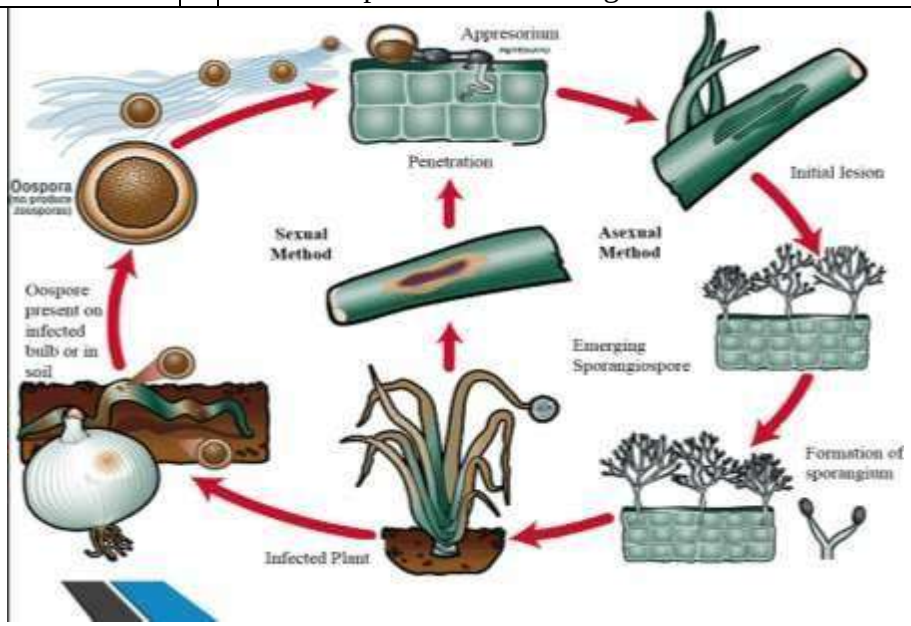
- Important physiological disorder, storage, transit and market disease caused by poor oxygen relation leading to dark necrotic cavities.
- Also occur when the temperature goes above 32°C during growth and maturation of tuber and also during transit.

- Symptoms **first visible at the center** of the tuber.
- Center of the affected tissue is **dark grey to black** in an irregular pattern indicating necrosis.
- Darkened areas are **firm** but on exposure to room become soft .
- Advance stage, affected **tissue dry out, shrinks** with the **formation of cavity** in the center.
- Symptoms on reaching the surface appear as **dark sunken patches.**

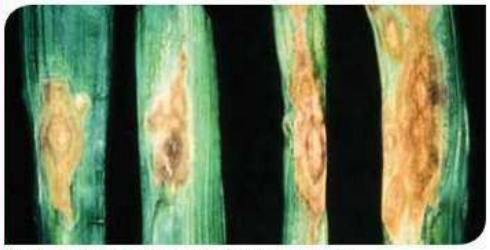
MANAGEMENT
Timely and proper irrigation and flooding.
Application of calcium rich fertilizer like gypsum and limestone.
Maintain moderate temperature during cold storage.
Maintain adequate ventilation during transport, storage to avoid asphyxiation.

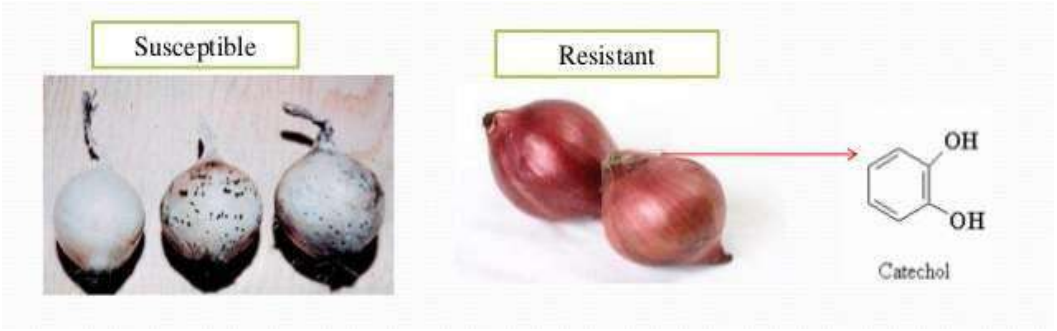
Avoid harvesting at extremely heat around 30 c and excessively low temperature around 0 c.
Do not delay harvest i.e. timely harvest.
Plant variety resistant to heat stress e.g.. Atlantic variety is must susceptible while Harley Blackwell is most resistant.

(2)DISEASES OF ONION (*Allium cepa* L.)

<table>
<tr><th colspan="3">1. Downy mildew</th></tr>
<tr><td>Causal organism</td><td>:</td><td>Peronospora destructor (Berk.) Casp. ex Berk.</td></tr>
<tr><td>Symptoms</td><td>:</td><td>□ The disease can rapidly destroy the foliage and hprevent the formation, ripening and storage of onion bulbs.
□ The symptoms vary with the type of infection. In systemic infection, the plants remain stunted, become distorted and become pale green incolour.
□ In humid atmosphere, the downy growth of the fungus develops over the entire leaf surface.
□ In local infections on the leaf, oval to cylindrical spots are formed on the leaves.
□ The spots may contain alternating green and chlorotic zones.
□ Older leaves are affected first and the infection spreads tthe sheath and inner leaves.</td></tr>
<tr><td colspan="3"> </td></tr>
<tr><td>Survival and spread</td><td>:</td><td>PSI: Oospores present in infected debris and bulb.
SSI: Zoospores with rain/irrigation water</td></tr>
<tr><td colspan="3"> 
</td></tr>
<tr><td>Favourable conditions</td><td>:</td><td>□ The fungus requires cool moist nights and moderate warm days for best development.
□ Heavy dew or heavy rains cloudy weather.
□ The conidia are produced in humid atmosphere at 4-25^{0}C with optimum at 13^{0}C.</td></tr>
</table>

Management	:	□ A regular fungicide spray program based on climatic conditions can reduce crop losses. □ Avoid planting onion sets that are contaminated with the fungus. □ A 3-4 year rotation out of onions in areas where the disease is present can help reduce losses. □ Bulbs used for propagation should be obtained from healthy fields. □ Field should be well drained. □ Spray Zineb 75 WP or Mancozeb 75 WP @ 0.25% after 20 days of planting and repeated at 10-12 days interval.
2. Purple blotch		
Causal organism	:	*Alternaria porri* (Ellis) Cif. (syn. *Macrosporium porri Ellis*)
Economic importance	:	
Symptoms	:	□ Minute whitish dots on the leaves with irregular chlorotic areas on tip portions □ Circular to oblong, concentric velvety rings appear in the chlorotic area □ Sometimes yellow halo is seen around lesions □ Spots coalesce and leaves die from tip backwards □ Diseased leaves break at point of infection and hang down □ Infection also seen on outer scales of bulb, seed stalk and neck □ Severely infected crop dries up □ Bulbs become dry and papery
 Purple blotch Thrips		
Survival and spread	:	**PSI:** Conidia in infected plant debris or seed bulbs **SSI:** Wind borne conidia
Favourable conditions	:	□ Warm humid weather with rains or dew □ Thrips infestation
Management	:	□ Select healthy bulbs for planting and bulb treatment with mancozeb or thiram 0.25% solution □ Seed treatment with thiram 3 g/kg of seed □ Spray mancozeb 0.2% or chlorothalonil 0.2% □ Manage thrips by insecticides
3. Stemphylium leaf Blight		
Causal organism	:	*Stemphylium vesicarium*

Symptoms	:	□ Initial infections on the leaves and leaf sheaths are small, light yellow to brown and water-soaked. □ As the lesions expand, they coalesce causing extensive blighting of the leaves. □ Typically, lesions are found in higher numbers on the side of leaves facing the prevailing wind. □ The centres of lesions turn brown to tan, then dark de brown and finally black as the fungus sporulates. sometimes fruiting bodies called perithecia may appear in infected tissue as small, black, pinhead-like raised bodies. □ Symptoms of stemphylium leaf blight are very similar to those of purple blotch, which often results in misidentification.
 Dark brown to black sporulation on leaves. Dark brown to black sporulation on senescing leaves.		
Favourable conditions	:	□ Extended periods of leaf wetness from dew formation, rainfall or overhead irrigation during bulb formation and development can result in severe leaf blighting. □ Bulb size can be greatly reduced due to loss of foliage. □ Infection is usually limited to leaves and does not extend down to the scales of the bulb.
Management	:	□ Chemical control with fungicides is effective in reducing disease development. □ Long-term rotation with unrelated crops may reduce losses. □ In addition, good field drainage and reduced plant density may lessen disease severity.
4. Smudge		
Causal organism	:	*Colletotrichum circinans*
Symptoms	:	□ Chiefly a disease of scales of bulb. Red scaled onions are usually resistant to the smudge due to the presence of protocatechuic acid and catechol □ Damping-off in seed bed under wet and warm conditions □ Disease appears at all stages and also during storage and transportation □ Sub-cuticular, dark green to almost black smudge appear on the bulb, neck or green leaves that are clinging to bulb after digging □ Circular lesions with concentric rings of dark stromata and mycelium appear on leaves □ Small, sunken and yellow lesions on inner scales □ Pinkish mass of fungal growth on lesions under humid conditions



J. C. Walker (1929) reported that **Protocatechuic acid and Catechol** in red scaled onions are resistant to *Colletotrichum circinans*

Survival and spread	:	**PSI:** Stromata in infected plant debris or soil **SSI:** Wind borne conidia
Favourable conditions	:	□ Wet soils with a temperature of 26^0C
Management	:	□ Resistant varieties: Nasik red, Pusa Ratna, Pusa Red □ Protection of bulbs from rains after harvest □ Bulbs should be dried properly before storage by hot air at 37-48^0C □ Spray Zineb or Captan 0.2% before harvest of crop

5. Botrytis Neck and Bulb Rot•

CO: *Botrytis aclada*, *Botrytis allii*, *Botrytis porri*

Symptoms and Signs

Botrytis neck and bulb rot is a disease of both onion and garlic, as well as leek and shallot.

In onions, Botrytis bulb rot generally appears during storage, although infection originates in the field. Initial symptoms usually begin at the neck, where affected tissue softens, becomes water-soaked, and turns brown. In a humid atmosphere, a gray and feltlike growth (where spores are produced) appears on rotting scales, and mycelia may develop between scales. Dark-brown-to-black sclerotia (the resting bodies of the pathogen) may eventually develop in the neck or between scales.

In garlic, symptoms appear either in the field towards the end of the season, or during storage. Plants infected in the field may be stunted, with dead and dying outer leaves. Affected tissue is initially water-soaked, but later turns dry and necrotic. Sclerotia form in the neck or adhere to the rotten outer scales of the bulb.

In both onion and garlic, initial infections may not be noticeable, and symptoms may develop only when leaves senesce and become necrotic.

Comments on the Disease

Botrytis allii and *Botrytis aclada* cause this disease in onion, whereas *B. porri* causes it in garlic. These fungi persist on dead onion and garlic tissue and persist as sclerotia in the soil for long periods. The sclerotia germinate in moist weather and produce airborne conidia (spores), which land on tissue, germinate, and infect when conditions are favorable.

The greatest incidence of infection occurs in persistent cool (50° to 75°F) and moist weather. Infections are favored by excessive use of nitrogen fertilizer, applications of nitrogen after bulb initiation, and poor drying of the tops and curing of the necks.

The fungi are associated with garlic and onions wherever they are grown and are common colonizers of senescent tissue. Infection can also occur through wound sites on the bulb.

Management

Cultural control (especially methods that reduce crop injury) is the primary way to avoid damage from Botrytis neck and bulb rot. Healthy onions that are stored properly and harvested at the right time are seldom affected. To prevent infection:

- Harvest onions and garlic only when the crop is mature and necks are well cured.
- Minimize damage to bulbs from insects and diseases during the growing season.
- Avoid excessive or late applications of nitrogen fertilizer.
- Avoid late-season irrigation to allow the foliage and neck to dry before harvest. The neck tissue must be well cured before the crop is stored.
- Handle the crop to minimize bruising or wounding.
- Store bulbs at temperatures of 41°F or below.
- Minimize relative humidity and promote good air circulation during storage.
- Maintain stable temperatures when moving bulbs into and out of storage, as well as shipping to markets. Wide fluctuations in temperature can cause condensation on the bulbs, which favors the development of the disease.

(3) DISEASES OF CUMIN (*Cuminum cyminum* Linn.)

1. Blight/ Alternaria blight		
Causal organism	:	*Alternaria burnsii* Uppal, Patel & Kamat
Economic importance	:	□ It is one of the important cash crops of Gujarat and Rajasthan. In Gujarat, its cultivation is mainly confined to North Gujarat and Saurashtra regions. Mehasana, Patan, Banaskantha, Sabarkantha, Surendranagar, Ahmedabad and Rajkot are important cumin growing districts of Gujarat. While, major cumin growing areas in Rajasthan are Jalore, Barmer, Nagaur, Jodhopur, Pali, Ajmer and Tonk districts. □ The climatic conditions of Gujarat and Rajasthan are more favourable for cumin cultivation, these two states contributes more than 80% in National area and production. □ Alternaria blight caused by *Alternaria burnsii* (Uppal, Patel & Kamat) is most devastating disease in major cumin growing areas in Gujarat and Rajasthan. The volatile oil *i.e.* cuminol content of seeds is reduced due to the disease. □ Humid and cloudy weather favour the disease development. The disease initiation takes place in 30-35 days old crop at flowering stage and spread quickly and assumes the epiphytotic condition resulting in complete failure of the crop. Under such conditions, the crop needs to be protected with application of fungicides at 8 to 10 days intervals starting from initiation of the disease. □ The market value of cumin depends much on seed quality and therefore, besides the yield, the farmers are very much concerned with the quality of the produce. □ Quality of the seed and the yield are affected badly even with the mild intensity of blight. The repeated sprays of fungicides though control the disease; yet it leaves residues in the produce. The residues of fungicides in seeds are of export concern of traders. Moreover, repeated sprays of the fungicides also increase the cost of crop production.
History	:	□ Alternaria blight of cumin was first reported from Bombay Presidency by Uppal (1930) and the causal organism was identified as *Alternaria burnsii* Uppal, Patel & Kamat from Kheda district of Gujarat by Uppal *et al.* (1938). □ The seed borne nature of pathogen was observed by Uppal *et al.* (1938) and Patel and Desai (1971).

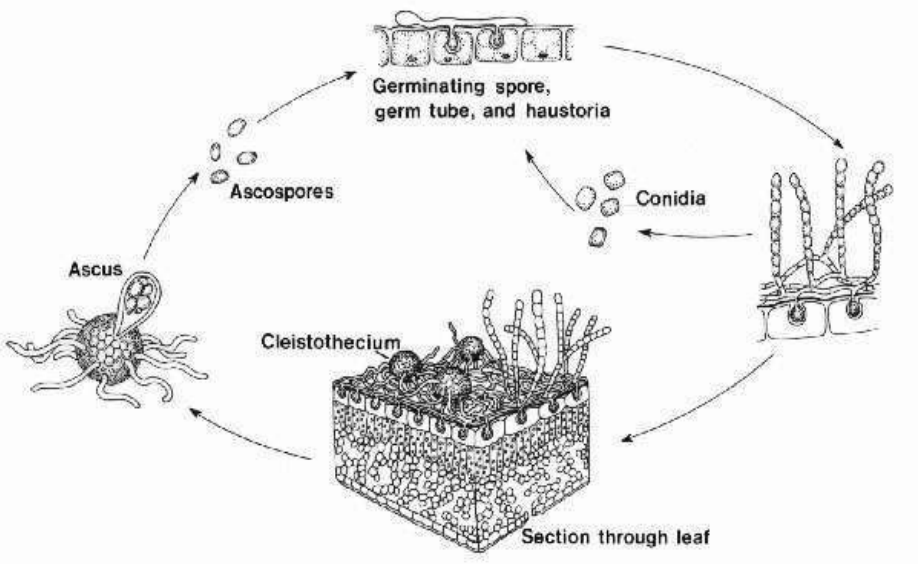
Symptoms	:	□ The disease appears at flowering at about 30-35 days old plants in the form of small necrotic spots on the aerial parts, *viz*., leaves, inflorescence, seeds, *etc*. which enlarge, coalesce and turn purple brown to black. □ Under favourable environmental conditions stem and flowers are also infected. In such infected plants seed formation is adversely affected, when the developing seeds remain small, shrivelled, lightweight and blackish in

		colour. □ The diseased crops show the burn appearance from the distance. □ The infection spreads rapidly in plants resulting in heavy yield losses particularly during cloudy weather or when plants remain wet due to heavy due formation. □ In severe diseased plants, no seed formation take place, however if formed they are shrivelled, dark coloured, light in weight and non-viable.
 Blight symptoms (a) on inflorescence (b) on seeds, (c) infected crop in field (d) disease at harvest, (e, f) heavily infected field of cumin		
Disease cycle	:	□ The pathogen is seed borne and survives on the crop debris in soil. □ Survival of the pathogen on seed during storage was 100% during the month of April and May, which decreased to 70 % during October, November under laboratory condition at 25°C temperature and 40-50% RH. Whereas, in infected crop debris in soil, its survival was 40-60% during October, November.

		□ Infected seeds are the primary source of inoculum to an extent of 10-32%. □ The fungus may remain viable for 10 months in seed and the crop debris. □ Secondary source of inoculum are conidia, which spread through wind. □ The environmental conditions *viz*., relative humidity of about 90 per cent and temperatures of 23-28^{0}C favour the pathogen as well as the development of the disease. □ The plants become susceptible only after the flowering stage and if there is rain or heavy dew formation after infection, the disease spreads quickly.
Favourable conditions	:	□ Conidial germination of the pathogen was found maximum at 26-27^{0}C. □ Plants at flowering stage and onwards are infected quickly. □ The pathogen survives mainly in seeds and the infected seeds play an important role in the transmission of the pathogen. □ Rain, persistent cloudy weather, cool humid conditions with >85% humidity and 23-28°C temperature favour disease development. □ Early sown crop particularly during October has greater severity of the disease. December sown crops found to be less disease incidence; but has also less seed yield. □ The incidence of Alternaria blight increased with the increase in the duration of dewfall, leaf wetness, relative humidity, congenial temperature and number of rainy days. □ Maximum temperature between 29^0-35.5^{0}C, minimum temperature 9.6^0-19.7^{0}C, average relative humidity of >60% in the morning and >28% in afternoon, wind speed 2.1-4.8 km/hr and 8-10.4 bright sunshine hours were favourable for disease development.
Management	:	***Cultural practices*** □ Collect and destroy the infected plant debris. □ Follow crop rotation and select disease free field for cumin cultivation. □ Timely sowing of the crop during November. □ Give light irrigation to crop during daytime and avoid water logging in field. □ Balance use of chemical fertilizers *i.e.* apply 30 kg N + 30kg P_2O_5 /ha as basal and 30 kg N/ha as top dressing at 30 DAS for obtaining higher yield. □ Use blight tolerant lines/varieties *viz*; MC-43, Gujarat cumin-1, RZ-19, Gujarat cumin-2, Gujarat cumin-3 and Gujarat cumin-4. ***Use of Non-chemical / chemical methods i.e. botanicals and bioagents for the disease management*** □ Use of bioagents and the fungicides have been found to reduce the fungicides load / reduce pesticide in the crop. □ *Trichoderma harzianum* when applied in the soil grown on

		sorghum seeds (24 g/ 6 m^2) resulted least blight disease. Similarly, when 10% spore suspension of *T. harzianum* use as seed treatment and as spray at flowering of the crop. □ Seed treatment with thiram or carbendazim @ 3-5 g/kg seed to control seed borne pathogen and treatment of 100 g *Trichoderma* formulations with one kg seed. □ Soil application of *Trichoderma viride* @ 2.5 kg/ha in 100 kg FYM + seed treatment with Carbendazim @ 1 g/kg seed followed by two sprays of difenconazole @ 0.05% at 35 and 50 days after sowing was found better to manage wilt and blight of cumin. □ Two sprays of aqueous extracts (10%) of *C. procera*, Azadirachta seed kernel extract and Azadirachta leaf suppressed Alternaria blight and enhanced seed yield in cumin under field condition. □ The disease could be managed satisfactorily with four sprays of mancozeb, 0.25% as well as two sprays at an interval of 10 days with mancozeb + two sprays of *Trichoderma harzianum*, starting at 30 days after germination. Two sprays of mancozeb, 0.25% followed by two sprays of *Pseudomonas fluorescens*. □ Prophylactic spray of chlorothalonil followed by two sprays of azoxystrobin or propiconazole. □ Seed treatment with thiram @ 5 g/kg seed followed by six sprays of propineb 70 WP, 0.2% or metiram 70 WG, 0.2% or propiconazole 25 EC, 0.1% starting from disease initiation and at seven days intervals.
2. Wilt		
Causal organism	:	*Fusarium oxysporum* f. sp. *Cumini*
Economic importance	:	□ In India, this disease occurs in Gujarat and Rajasthan states. □ Crop loss of about 5-25% have been reported but, under severe condition losses may go as high as 70%.
Symptoms	:	□ The plants are attacked at all stages of crop growth, but the disease usually appears in patches in the field when the crop is about one month old. □ The affected plants turn yellow, wither and show characteristic wilt symptoms and dry up. □ In the initial stage, top of the branches droop down and gradually infected plant dry-off. □ The roots of diseased plants become dark brown. □ On splitting the roots, vascular bundles appears brown coloured indicating the presence of fungal mycelium. □ The disease spreads through the affected soil and in severe cases the whole plant wilts and the stems shrivels. □ The affected seeds may carry the inoculum to the next season.

Survival and spread	:	**PSI:** Pathogen is mainly soil and seed borne, survives as chlamydospores **SSI:** Through macro and micro conidia through farm implements and irrigation water.
Favourable conditions	:	□ Soil temperature 25-30^{0}C □ Mono-cropping
Management	:	□ Use healthy seeds obtained from disease free crop. □ Treat the seeds with Carbendazim 50 WP □ Deep ploughing during summer. □ Soil solarization □ Crop rotation with cereals. □ Use FYM/organic matter to enhance antagonistic activity of microbes. □ Use of bioagents like as fungal bio-agents: *Trichoderma viride* or *Trichoderma harzianum* and bacterial bio-agent *Pseudomonas fluorescens* as seed dresser and soil application (5.0 kg/ha). □ Grow resistant variety like Gujarat Cumin-3, Gujarat Cumin-4. □ Removal of infected plant from field.
3. Powdery mildew		
Causal organism	:	*Erysiphe polygoni*
Economic importance	:	The losses due to this disease ranged from 10-15%, if the disease appears late. But, if it appears during flowering stage, the losses may be 50%.
Symptoms	:	□ The infection takes place at any stage of plant growth, but more severe at flowering stage. □ Small, white or grayish spots appear on lower leaves. □ They gradually enlarge, coalesce and cover the entire surface with whitish mycelial growth and conidia of the fungus. □ The infection rapidly spreads to stem, flowers and seeds. □ Under warm and moist condition, the spread of the disease is very rapid and entire plant becomes affected and dries off. In case of severe attack, the leaves/plants show ashy white appearance. □ Seed setting is also adversely affected. The seed from infected plants are small and light in weight. Late sown

		crop is severely affected. Early infection prohibits seed formulation.
		
Disease cycle	:	□ The fungus is ectoparasitic, spreading on the surface of host and sending haustoria into epidermal cells to draw out nutrients. □ The Cleistothecia occur either in cluster or scattered on host tissues. □ Primary infection is through Cleistothesia (Ascospore). □ It also survives on collateral host from which it provides

		primary source of infection. □ Secondary infection: Through air borne conidia.
Favourable conditions	:	□ Soil temperature 20-22^{0}C, Low relative humidity < 60%, Cloudy weather.
Management	:	□ Late sowing should be avoided. □ Spray wettable sulphur 80 WP @ 0.2% (25 g/10 liter water) or Dinocap 48 EC @ 0.025% from the start of the disease. □ Carbendazim 50 WP 0.05% (10g /10 liter water) or Hexaconazole 5 EC 0.005% (10 ml /10 liter) also effectively controls the disease. □ Dusting of sulphur powder (300 mesh) @ 15-20 kg/ha during early morning. (*It is not recommended to spray dust sulphur in high temperature*).

(4)DISEASES OF CORIANDER (*Coriandrum sativum* L.)

1. Stem gall		
Causal organism	:	*Protomyces macrosporus* Unger
Economic importance	:	The crop yield is reduced by a number of fungal diseases of which stem gall disease caused by *Protomyces macrosporus* Unger, is the most destructive and versatile disease. The disease is prevalent in all coriander-growing areas of India and is considered as a limiting factor for successful cultivation of the crop. The pathogen is seed as well as soil borne and causes up to 15% damage to seed yield. Stem gall cause severe yield losses and deteriorate the quality of seed.
The Pathogen	:	This disease is caused by *Protomyces macrosporous* Unger. The mycelium of the fungus is found only in the tumors although the resting spores of the fungus cause systemic infection. The hyphae are intercellular, septate, and broad. Scattered cells in the hyphae swell and form ellipsoidal or globose bodies, which later develop into chlamydospores. As they mature, they become surrounded by thick, hyaline, three-layered walls and attain a diameter of 50–60 µm. The chlamydospores function as the resting spores of the fungus. These resting chlamydospores germinate in water by rupturing outer walls (exospores) and form sporangia and spores.
Scientific classification (Kirk *et al.*, 2008)	:	Kingdom : Fungi Phylum : Ascomycota Subphylum : Taphrinomycotina Class : Taphrinomycetes Order : Taphrinales Family : Protomycetaceae Genus : *Protomyces* Species : *macrospores*
Symptoms	:	□ Stem gall is a common and widespread disease of coriander causing heavy losses, especially when associated with wilt. □ The symptoms of the disease first appear as gall like appearances on the lower part of stem, which gradually extends upwards to flower and seeds. □ The tumors are at first glossy but later rupture and become rough. The swellings are usually elongated (9–12 mm) and 3–5 mm in diameter. The sizes of the swellings vary according to the dimensions of the parts infected. □ These galls are caused by hypertrophy and hyperplasia. Shape of coriander seeds change due to effect of the disease. □ The diseased seeds are hypertrophied depending upon the stage of infection, ultimately lowering the crop yield and quality. □ In the presence of excessive soil moisture, especially under shaded conditions, when the stem fails to harden and remain succulent, the tumors are numerous.

(a) (b)

Stem gall of coriander. (a) Close-up of diseased plant showing galls on stem and leaves of coriander; (b) hypertrophied inflorescence and floral parts of diseased plant.

Disease cycle and epidemiology	:	□ Disease appears in excess of soil moisture and shaded condition. The pathogen mainly survives in seed as well as on soil. □ The disease appears continuously in every year in the field of coriander grown on high pH soil, moisture, and adverse ecological and adaptic factors. Soil and infected seed material serve as a source of inoculum causing heavy loss. □ The disease is soil borne and the inocula present in the soil are the source of primary infection. Pathogen may survive in soil as resting spore for several years. Relatively high soil moisture and soil temperature are favourable for the infection.
Management	:	□ Use disease free seeds, field sanitation and crop rotation are helpful in reducing the primary source of inoculum. □ Diseased plant parts should be destroyed and seeds bearing galls should not be used. □ Grow disease resistant varieties/ lines/ Cvs. *viz*., UD-20, UD-125, UD-317, UD-749, Rlr-480, genotype K. Selection (Narendra Dhaniya-2), Pant Haritima, PH-7, COR-2, COR-17, Dania-8, DH-13, DH-M-4, DH-19-M-11-2, JD-1, G-5365-91, and Rcr-41. □ To keep the disease under check use balanced dose of fertilizers. The applications of Potassium and Nitrogen fertilizers reduce the disease intensity, while Phosphorus fertilizer increases the disease intensity in the absence of Nitrogen. □ Sowing the crop between Oct. 16 and Nov. 16 to minimize losses due to disease. □ Effective control by combined seed (250 g/ 100 kg seed) and soil (20 kg/ha) treatment with Thiram. □ Thiram or carbendazim as seed dressing or seed dressing + foliar spray reduces the disease intensity and increases seed yield. □ Hexaconazole as seed treatment (0.2%) and foliar spray

		after 40, 60 and 75 DAS (0.2%) is effective for management of stem gall disease of coriander. □ Seed treatment with *Trichoderma viride* @ 4 g/ kg seed + soil treatment with *T. viride* @ 2 kg/ ha recommended for management of stem gall of coriander.
2. Powdery mildew		
Causal organism	:	*Erysiphe polygoni* DC
Economic importance	:	Powdery mildew is one of the most destructive diseases of coriander in India leading to production of small shrivelled seeds, thereby reducing the yield and quality. The incidence of the disease has increased in recent years due to changing production practices, especially because of the use of high yielding, late maturing varieties in new environments.
The Pathogen	:	The disease is caused by *Erysiphe polygoni* DC is an obligate parasite. The mycelium is generally fine, persistent, rarely thick. The conidiophores arise vertically from the superficial hyphae on the host surface. Conidia are formed singly or in short chains. They are ellipsoid or ovate with vacuolated cytoplasm. Sometimes late in the season, but not always on coriander leaves in the field, the cleistothecia appear sharp, black minute bodies scattered in the mycelial web. The cleistothecium is provided with a number of myceloid appendages. Usually four to eight asci are formed in each cleistothecium. Each ascus contains three to five ascospores.
Scientific classification (Kirk *et al.*, 2008)	:	Kingdom : Fungi Phylum : Ascomycota Subphylum : Pezizomycotina Class : Leotiomycetes Order : Erysiphales Family : Erysiphaceae Genus : *Erysiphe* Species : *polygoni*
Symptoms	:	□ It appears as small, white, powdery patches on young parts of stems, leaves and buds, which increases in size, and coalesce to cover entire area of leaf surface. □ Affected leaves are reduced in size and distorted. Premature sterility is also common. In severe cases, the umbels dry up. Seed formation may not take place in affected plants due to this disease. □ Small, white spots appear on leaf surface and later cover the entire surface. □ Powdery growth can be seen on petiole and umbels. □ Under severe infection, plants present an ashy white appearance.

Powdery mildew of coriander

Disease cycle and Epidemiology	:	The disease is favored by dry weather conditions can cause significant yield reduction. The fungus can survive in dead plant debris in the form of cleistothecia, which serve as primary source of inoculum for the next season. Otherwise, the fungus overwinters in conidial stage on different hosts in the area, which cause primary infection. The ripe conidia fall-off quickly and dispersed long distances by wind. Conidial germination is best in between 20-24^{0}C. A fairly dry soil and heavy application of nitrogenous fertilizers tend to increase the incidence of the disease.
Management	:	□ Remove the disease infected plants from the field. □ Collect and destruct the infected plant debris from the field. □ Apply balanced doses of fertilizers and early sowing reduces the overall disease severity. □ Coriander varieties/ lines *viz*., Cor-31 (DH 220) showed least infection to the disease and can be used in breeding programme for developing resistant variety. □ Early planting coupled with application of dinocap reduced the powdery mildew disease severity on coriander. □ Spray application of hexaconazole (0.1%), propiconazole (0.1%) and wettable sulphur (0.3%). □ Application of carbendazim as soil drench (0.1%) and spray (0.1%) at 60 days after sowing or spray of wettable sulphur (0.2%) at 60 days after sowing. □ Plant extracts *viz*., *Andrographis paniculata* Nees. and *Azadirachta indica* A. Juss. reduces the disease severity and increases the yield. □ Apply conidial suspension of hyperparasite *Ampelomyces quisqualis* to reduce the severity of disease.
3. Wilt		
Causal organism	:	*Fusarium oxysporum* f. sp. *corianderii* Kulkarni, Nikam & Joshi
Economic importance	:	The occurrence of the disease in India has been reported from Madhya Pradesh, Rajasthan and Tamil Nadu. The pathogen associated with wilt disease is soil-borne in nature, which causes 60 per cent yield losses.
Symptoms	:	□ The disease can easily be recognized in the field by drooping of the terminal portions, followed by withering

		and drying up of leaves, eventually resulting in death □ Discoloration of vascular system of the root is observed. Partial wilting is also found. In partially wilted plants, growth is arrested. □ The leaves become pinkish yellow to yellow. Sterility is often noticed in such plants. Seeds, if formed are immature and light in weight. □ Severe infection in the early stage results in total failure of the crop. □ In affected plants lower leaves turn yellow and later dry. □ Later, the affected plants show wilt symptom. □ The roots of affected plants shows vascular discoloration.
The pathogen	:	The disease is caused by *Fusarium oxysporum* f. sp. *corianderii* Kulkarni, Nikam & Joshi. The mycelium is abundant, fluffy and cottony white to light pink. Both micro and macro conidia are produced. The microconidia are oval to elliptical while macroconidia are septate and sickle shaped measuring 10.06-7.90 x 2.7-1.6 to 70.62-99.4 x 2.97-1.98 µm in size. Later in the season chlamydospores are also produced.
Scientific classification (Kirk *et al.*, 2008)	:	Kingdom : Fungi Phylum : Ascomycota Subphylum : Pezizomycotina Class : Sordariomycetes Order : Hypocreales Family : Nectriaceae Genus : *Fusarium* Species : *oxysporum* f. sp. *Corianderii*
Disease cycle and Epidemiology	:	The fungus is soil borne and overwinters in the form of chlamydospores. In favourable weather conditions, chlamydospores germinate and cause infection. Micro and macroconidia are produced which cause secondary infection. Optimum temperature and pH for the disease is 28^0C and 5.8 to 6.9, respectively. Infection increased with the increasing soil moisture content but the population of the pathogen was highest at 52 per cent moisture holding capacity of the soil.
Management	:	The combination of biocontrol agents with fungicides as seed and soil applications could be very effective method for wilt disease. □ Follow long crop rotations. □ Collect and burn the infected plant debris and deep plough during the summer months. □ To reduce the disease incidence, addition of oilcakes in the soil and raising of soil pH to 8.2. □ Coriander varieties/ lines *viz*., MP 5365 and UD 38 showed least infection to the disease and can be used in breeding programme for developing resistant varieties. □ Seed treatment with carbendazim @ 3 g/ kg of seeds. □ Soil application of *Trichoderma harzianum* or *Pseudomonas fluorescens* @ 2.5 kg + 2.5 t FYM/ ha + spray of carbendazim (0.1%) or mancozeb (0.25%) at 45

		DAS. □ Soil application of 2.5 kg of *Trichoderma viride* along with 50 kg of FYM before sowing. □ Seed treatment with *Trichoderma viride* @ 4 g/kg of seeds.

4. Rhizoctonia Crown and Root Rot

Cause The fungus, *Rhizoctonia solani* (sexual stage: *Thanatephorus cucumeris*), has been found to cause root rot and crown disease on coriander . This fungus also causes damping-off of a wide range of plant species. It overwinters in soil or plant debris and can survive many years as sclerotia or chlamydospores.

Symptoms Leaves turn yellow and then brown and dry as foliage dies. Plants wilt and may die. A brown rot of tissues occurs in the taproot and crown portion.

Cultural control

- Practice a 3-year rotation with non-host crops (grains).

(5)DISEASES OF MANGO (*Mangifera indica* L.)

1. Powdery mildew		
Causal organism	:	*Erysiphe cichoracearum* DC. *Oidium asteris - punicei* Peck [anamorph] *Oidium mangiferae* Berthet.
Economic importance	:	The disease is worldwide in distribution. Reported from India, Pakistan, Ceylon and South Africa. In India, the disease is particularly destructive in U.P. Maharashtra and Karnataka severe particularly during the months of December-March, *i.e.* cooler months.
Symptoms	:	□ The disease is easily recognized by whitish or grayish powdery growth on the inflorescence and tender leaves □ Generally, the infection starts from the inflorescence and spreads downwards covering the floral axis, tender leaves and stem. Infected leaves become twisted, curled and fall □ Infected floral parts are severely damaged and drop off. If the fruits are set, they do not grow in size and may drop before attaining pea size. Fruits are sometimes malformed, discolored due to severe mildew attack □ Because of poor fruits set and heavy flower and fruits drop, the loss due to the disease may be as high as 70-80%
Survival and spread	:	**PSI:** Dormant mycelium and conidia in infected plant debris **SSI:** Wind borne Oidium type conidia
Favourable conditions	:	Disease spread is favored by warm humid weather with cool Nights
Management	:	□ Two preventive sprays with wettable sulphur 0.3% before flowers open and after fruit set □ Dusting twice or thrice with fine sulphur □ Spraying with Karathane 0.1% before flowering and after fruit set (peanut stage). □ Resistant varieties: Neelum, Zardalu, Banglora, Torapari-khurd and Janardhan pasand
2. Anthracnose		
Causal organism	:	*Colletotrichum gloeosporioides* (Penz.) Penz & Sacc. in Penz. *Glomerella cingulata* (Stoneman) Spauld. & Schrenk [teleomorph] *Colletotrichum acutatum* J.H. Simmonds
Economic importance	:	Reported from several districts of Punjab in 1939. Now prevalent in all mango – growing tracts of the country. The

		disease is a problem both in the field and storage. Neelum and Banglora are more susceptible.
Symptoms	:	□ The fungus produces leaf spots, blossom blight, wither tip, twigs blight and fruit rot □ On leaves circular, light yellow brown spots are produced initially which soon enlarge with dark brown to black margins. If infection is severe individual spots coalesce leading to twisting of foliage and premature defoliation □ The disease spreads rapidly during rainy season and covers the tender twigs causing them to wither □ On inflorescence stalks and flowers minute spots are produced which increase in size and cause flower drop – the symptom hence is called blossom blight □ Sunken spots appear on fruits near the stem end as small brown areas that enlarge rapidly and become black. In some cases, the areas involved are in the form of streaks running down from the stem end. Fruit pulp beneath the spots become hard followed by cracking and decay at ripening □ The decay is confined to the skin of the fruit except in late stages where it penetrates the flesh in shallow areas. Infected fruits may also drop off prematurely □ Latent infection is carried from the field and develops further which causes rotting in the storage
Survival and spread	:	**PSI:** Saprophytic mycelium and conidia in infected plant debris **SSI:** Wind borne and rain splashed conidia
Favourable conditions	:	□ Temperature 25^0C and RH 95-97% □ Injuries to fruits at harvest and in storage
Management	:	□ Avoid over-crowding in orchards □ Tree sanitation is important. Diseased twigs are to be pruned and burnt along with fallen leaves and fruits □ Spray carbendazim or thiophanate methyl (0.1%) or Chlorothalonil (0.2%) at 14 days intervals from fruit set until harvest □ Before storage, treat the fruits with hot water (50-55°C) for

		15 minutes or dip in Benomyl solution (500 ppm) or Thiobendazole (1000 ppm) for 5 minutes or expose them to ammonia and sulphur dioxide gases.
3. Malformation		
Causal organism	:	*Fusarium moniliforme* var. *subglutinans*
Economic importance	:	This is one of the severe diseases of mango and is important in North India. It appears in and around Hyderabad and Medak Districts In A.P. this was first noticed in Aragonda village in 1971 In India, it is known to occur in U.P., Maharashtra, Haryana, Bihar, Punjab and A.P. particularly severe in U.P. causing much damage. Coastal areas are free from the disease.
Symptoms	:	There are two types of symptoms namely vegetative malformation and floral malformation because of hypertrophic effect of Gibberellic acid produced by the pathogen. ***Vegetative malformation*** □ There is a proliferation of infected tissue. The affected plants develop excessive vegetative branches which are of limited growth, swollen and have short internodes □ These dwarf branches are of various sizes, which are often produced on the top of young seedlings giving a bunchy top appearance. The auxiliary buds of dwarf branches are unusually enlarged □ Vegetative malformation is more pronounced in young seedlings and seedling trees than in grafted plants ***Floral malformation*** □ The flowering panicles instead of coming out as a normal one turns into compact bunch of hard flowers. Individual flower is greatly enlarged and has a large disc □ Based on compactness of the inflorescence three types of malformations have been described □ Light malformation– light compaction of inflorescence but bears fruits □ Medium malformation– moderately compact inflorescence with discontinuous growth and no fruit set □ Heavy malformation– entire inflorescence is converted into a compact mass of sterile flowers. □ Inflorescence continues to grow with flowers having hypertrophied bracts. The percentage of bisexual flowers in malformed panicles is very low □ The malformed heads dry up in black masses and persist on the trees for a long time □ Affected inflorescences generally are aborted and bear no fruit

Mango malformation Eriophyid mite

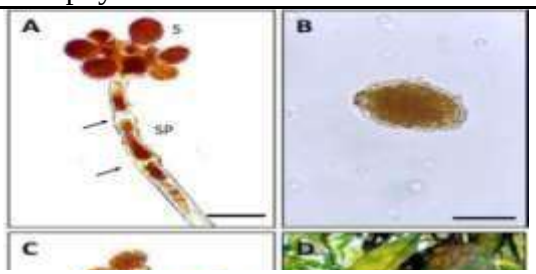
Survival and spread	:	**PSI:** Saprophytic mycelium and chlamydospores in infected plant debris **SSI:** Wind borne and mite disseminated micro and macro conidia
Favourable conditions	:	□ Prevalence of eriophyid mite □ Misty wet and cool weather
Management	:	□ Use of disease free planting material and prophylactic spray of insecticides and fungicides □ Pruning of diseased parts along with basal 15-20 cm apparently healthy portions □ Spray Carbendazim (0.1%). □ Spray napthelene acetic acid (NAA) 200 ppm during first week of October

4. Dieback		
Causal organism	:	*Botryodiploidia theobromae* *Lasiodiplodia theobromae* (Pat.) Griffon & Maubl. *= Botryosphaeria theobromae* Pat.
Economic importance	:	This disease is prevalent in all mango-growing states in India. In U.P. 30-40% of road this disease affects side and other plantation.
Symptoms	:	□ The disease is characterized by dying back of twigs from tip downwards particularly in older leaves. □ It is giving an appearance of scorching by fire followed by complete defoliation. Barks are discolored and darkened at certain distances from tip. □ Such dark patches are generally seen on young green twigs. When the dark lesion increase in size, dying of young twigs begin at the base affecting leaf mid ribs extending along the veins. □ The upper leaves use their healthy green color and gradually turn brown accompanied by upward rolling of leaf margin. In, advanced stage, such leaves shriveled, fall off in a month are more, leaving the shriveled twigs. □ Internal browning in the wood tissue is observed on longitudinal splitting along the long axis. Cracks appear on branches, which exude gum. In fruits, the pericarp darkens near base of the pedicel. □ The affected area enlarges to form a circular, black patch, which under humid atmosphere extends rapidly and turns the whole fruit completely black within 2/3days. The pulp becomes brown and softer
Survival and spread	:	The fungus is a wound parasite. Dead twigs and bark of the trees harbor the fungus. The spores are spread through rain splashes.

Favourable conditions	:	□ High temperature during summer predisposes the trees to the disease. □ Relative humidity of about 80%, max. &min. temperature of 31.5^{0}C and 25.9^{0}C respectively and rains favour the disease development. □ It causes great damage and when mango grafts are kept in
		humid propagation shed.
Management	:	□ Fruits should be harvested on clear dry days. □ Injuries should be avoided on fruits at all stages handling. □ Dipping of mangoes in 6% solution of borax at 43^{0}C for min. gives effective control. □ Carbendazim 0.1% or Thiophanate-methyl 0.1% chlorothalonil 0.2% spraying in the field before harvesting gives effective control
MINOR DISEASES 1. Loranthus (Giant or true leafy mistletoes)		
Scientific name	:	*Loranthus longiflorus / Dendrophthoe falcata*
Economic importance	:	Loranthus is a partial stem parasite. Loranthus seeds are disseminated by birds on to the stems of the host.
Symptoms	:	□ Loranthus seeds do not require a host germination stimulus and will readily germinate □ The seedling radicle is negatively phototrophic and hgrows towards a dark surface (often the host branch) □ The first attachment structure formed is called a holdfast. The haustorium from the holdfast eventually connects to the host xylem and removes water and mineral nutrients from the host □ When in contact with the host cambium, the loranthus haustorium induces formation of additional wood that enlarges into fluted columns □ Loranthus produces long attractive tubular flowers which attract birds that disperse seeds
Management	:	□ Pruning of affected branches and burning them before the parasite flowers and sets seeds □ Bird scaring

2. Red rust / Algal leaf spot		
Causal organism	:	*Cephaleuros virescens* Kunze

Economic importance	:	In India, it has been observed in Bihar, Karnataka and U.P. The disease appeared in an epidemic form in orchards of Tarai in 1956
Symptoms	:	□ The disease is characterized by red rusty spots on leaves and young twigs □ The spots are initially circular, slightly elevated and later coalesce and become irregular □ The upper surface of the spot consists of numerous unbranched sterile or fertile filaments of the pathogen which project through the cuticle □ Initially the spots are greenish-grey in colour but later on the surface bears reddish-brown appearance because the algal cells are impregnated with pigment which masks the normal green chlorophyll



BLACK TIP OR NECROSIS OF MANGO

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- Occurred in UP, Punjab, Bihar and W. Bengal
- Not occurred in South India
- Prevalent near brick links
- Affect till 90% fruits

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- **Occurred in UP, Punjab, Bihar and W. Bengal**
- **Not occurred in South India**
- **Prevalent near brick links**
- **Affect till 90% fruits**

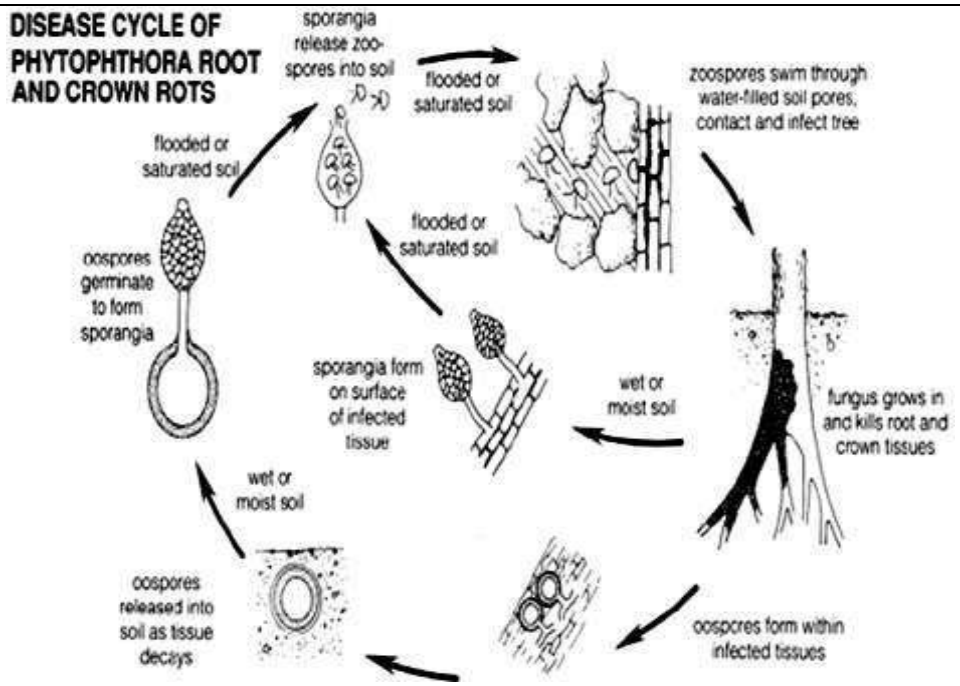
- Appear after 6-8 weeks old fruits or at ripening stage
- Necrosis on tip of fruits
- Tip become black & flat
- Inner portion of tip become soft, rottened & secretion of dark brown liquid

- Spray Borax @ 3-4 kg/500 lit. water.
  I&II Spray – flowering stage
  III Spray – fruiting stage
- No brink links should be near garden
- Chimney should be 15-18 Meter height
- Distance of Planting of new garden trees
  East West - 1.6 Km
  North south – 0.8 Km
- Covering of mango plants with cellophane bags rescue from brick links smoke.

(6)DISEASES OF CITRUS (*Citrus aurantifolia*)

1. Gummosis/ Foot rot		
Causal organism	:	*Phytophthora citrophthora, Phytophthora nicotianae* var. *parasitica, Phytophthora palmivora*
Economic importance	:	Gummosis is widespread in Punjab and Assam. Lemons are more susceptible than grapefruit and rough lemons. In South India, it is common in sweet orange.
Symptoms	:	□ Disease starts as water soaked patches on basal portions of the stem near ground level. □ Patches turn dark staining of bark brown. □ Infection progresses into the wood. □ Infected bark dries, shrinks, cracks, and shreds lengthwise in to vertical strips. □ Reddish brown gum exudes from the bark of infected trunk. □ Gum exudation is considerable in sweet oranges, but relatively little in grapefruit. □ Infection extends to crown roots. □ Affected collar region is girdled and finally the infected tree dies. □ Prior to death, the tree usually blossoms heavily and dies before the fruits mature.

Survival and spread	:	**Mode of disease spread:** □ Use of susceptible root stock □ Flood irrigation and flat bed system □ Retention of water for longer period in beds □ Prolonged period of wet weather □ Low budding □ Repeated use of same land for nursery raising □ Location of nurseries near old orchards □ Regular contaminating through soil and irrigation water are the predisposing factors for Phytophthora diseases. **PSI:** Oospores or as dormant mycelium **SSI:** Sporangia and zoospores spread by splashing rain water and irrigation water



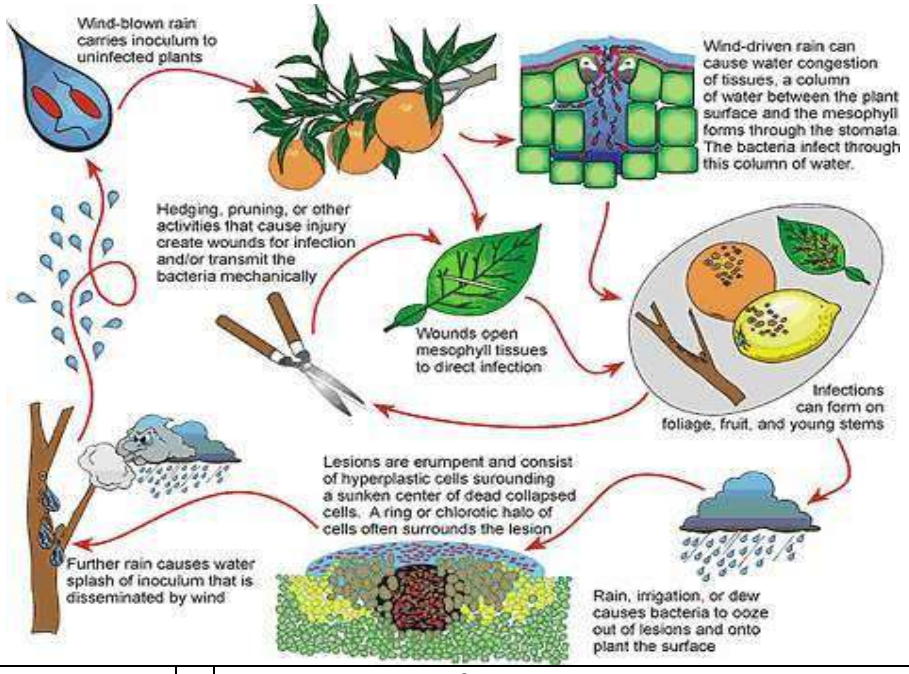
Favourable conditions	:	□ Prolonged direct contact of trunk with water, as in flood irrigation and water □ Logging predisposes trees to disease □ Incidence is more in black soils than in light soils □ High water table leads to high incidence □ The disease is severe in high rainfall areas □ Low budded grafts are mostly affected
Management	:	*Preventive measures:* □ Selection of proper site with adequate drainage. □ Selection high budded grafts (30 to 45 cm or above) □ Following Double ring method of irrigation by providing an inner ring about 45 cm around the tree trunk to prevent direct moistening of trunk. □ Avoid injuries to crown roots or base of stem during cultural operations. □ Use resistant sour orange or trifoliate orange rootstocks for propagating popular/commercial varieties. □ Painting Bordeaux paste or with ZnSO4, CuSO4, lime (5:1:4) to a height of about 60 cm above the ground level at least once a year. *Curative measures:* □ Scrape/chisel out the diseased portion. □ Protect the cut surface with Bordeaux paste followed by spraying with Fosetyl-Al 0.2%. □ Soil drenching with 0.2% Metalaxyl. □ Apply *Trichoderma viride* multiplied on neem cake. □ Stem pasting of Metalaxyl 68 WP (50 g/L) + drenching of Fenamidone 10% + Mancozeb 50% WG @ 0.2% (10 L/tree) twice *i.e.* first at onset of monsoon and second at one month of first application (AAU Recommendation-2018).

2. Canker		
Causal organism	:	*Xanthomonas citri* subsp. *citri* (=syns. *X. axonopodis* pv. *citri*; *X. campestris* pv. *citri*)
Characteristics of pathogen	:	It is rod shaped, Gram negative and aerobic in nature. It is monotrichous bearing polar flagella. Pathogen is known to produce xanthomonadin compound, which gives yellow colour to colonies on agar medium.
Economic importance	:	In India, citrus canker is endemic and occurs in all the citrus growing areas. It is reported from Punjab, Tamil Nadu, Andhra Pradesh, Karnataka, Rajasthan, Madhya Pradesh, Assam, and Uttar Pradesh. Acid lime is highly susceptible than other citrus fruits. Total destruction of citrus orchards was done for eradication of canker in Florida state of USA.
Symptoms	:	□ Canker appears on leaves, petioles, twigs, branches, fruit stalks, fruits and thorns. When it is severe, trunk and roots are also affected. □ However, the symptoms are most conspicuous on leaves, twigs and fruits. On leaves minute water soaked round spots develop which enlarge slightly and turn brown, eruptive and corky. □ On acid lime and sweet orange leaves spot measure about 2 to 3 mm in diameter and are surrounded by a characteristic yellow halo. □ Due to severe infections of the leaves, there may be defoliation, and severe infections of the twig and stem may cause dieback symptoms. □ The plants also remain stunted and fruit yields are reduced considerably. □ Canker lesions on the fruit are not surrounded by yellow halo. Several lesions on fruit coalesce to form a patch. The crater-like appearance is marked on fruits than on leaves. □ The market value of the fruits is considerably reduced bythe canker spots, though such infections are mostly confined to the fruit skin.

Symptoms on (A) leaves, (B) stem, (C) fruit, and (D) fruit close-up

Citrus leaf miner (*Phyllocnistis citrella*)

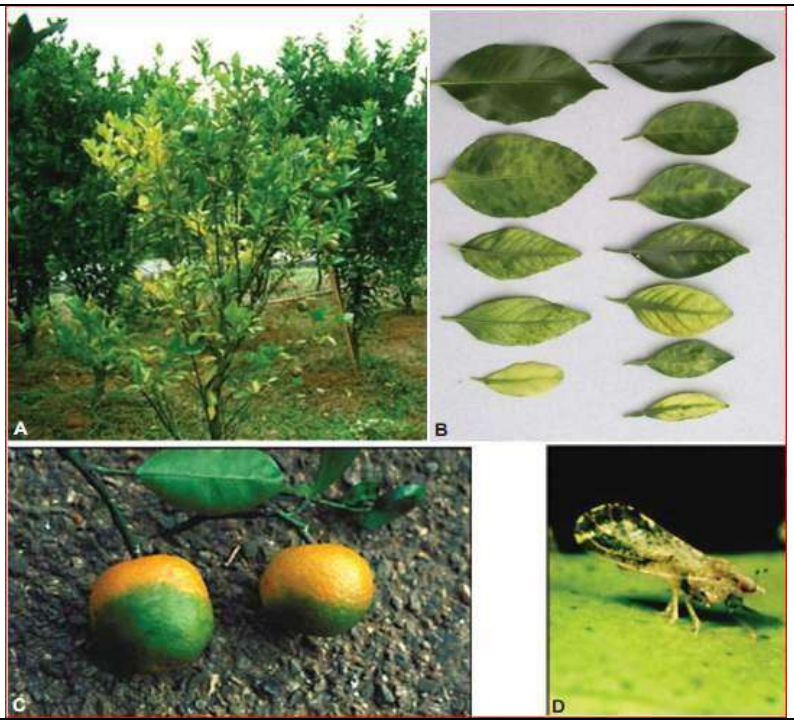
Citrus leaf miner (*Phyllocnistis citrella*)

Survival and spread	:	**PSI:** Bacteria survive in cankerous leaves, twigs and branches (leaves-5 months. Twigs-76 months). □ Previously infected plant and its parts, seedlings, fallen leaves and fruits **SSI:** Bacteria oozing from infected plant parts. □ Natural openings (stomata) and wounds. Injuries caused by leaf miner (*Phyllocnistis citrella*) serve as entry point for bacteria. □ Through wind splashed rains, leaf miner, equipments, etc. Long distance dissemination through diseased planting material.
Disease cycle: 		
Favourable conditions	:	□ Free moisture for 20 minutes at 20-30°C and high relative humidity (>85%) favour initiation of disease. □ Abundant citrus leaf miner population.
Management	:	□ Select healthy seedlings and grafts for planting in new areas. □ Sanitation: collect and destroy infected leaves and twigs. □ Thorough inspection of nursery and orchards quarantines and on-site burning of infected/pruned debris before monsoon and spray 1% Bordeaux mixture at periodic intervals. □ Periodical spraying of Streptocycline (1 g) + Copper oxychloride (30 g) in 10 litres of water at fortnightly interval in nurseries and at fortnightly interval in orchards during rainy season. □ Use canker tolerant varieties like 'Tenali selection' and 'Balaji'. □ Spray Spinosad 45 SC @ 3 ml/ 10 L or Imidacloprid 17.8 SL @ 4 ml/ 10 L of water for controlling leaf miners when young flush is produced.

MINOR DISEASES	**1. Citrus Tristeza Virus (CTV)/ quick decline/ stem pitting**	
Causal organism	:	*Citrus Tristeza Virus* (CTV) Genus *Closterovirus*
Economic importance	:	This disease was first reported in *Citrus aurantifolia* and *C. sinensis* from Italy and Florida in the U.S.A. In India, tristeza is prevalent in Andhra Pradesh, Tamil Nadu, Karnataka, Madhya Pradesh, Maharashtra, Punjab, Bihar, West Bengal and Sikkim.
		It was observed in South Africa in 1899 as a disease of sweet orange. The name 'Tristeza' was coined by Moreira in Brazil, a Portuguese word meaning the "Sad disease/ sadness". The disease is also known as quick decline, seedling yellows and dieback. The disease named as quick decline because the disease resulting in host death just days after the first symptoms are noticed. Now the is widespread in Argentina, Australia, Brazil, India, Indonesia, Java, Malaysia, Pakistan, Panama, Sri Lanka, Thailand, USA and Venezuela. It affects all citrus types but sweet orange, grapefruit and lime are more attacked. Tristeza symptoms consisting of a quick or chronic tree decline are particularly common and severe on trees propagated on sour orange rootstocks. Kagzi lime and Nasnaran are indicator plants for CTV detection.
Symptoms	:	□ The virus exists in nature in numerous forms (strains) hence induce different kinds of symptoms. □ Tristeza affected trees look chlorotic and sickly in the early stages. Gradually the leaves drop and the defoliated twigs show dieback. The declining trees die gradually but sometimes apparently, healthy trees die suddenly. □ Vein clearing or vein flecking (elongated translucent area) in young leaves of acid lime is visible when viewed against light. □ Characteristic stem pittings are formed on infected trees. □ In sweet orange, the specific symptom of tristeza is honeycombing, a fine pitting of inner face of bark in the portion of trunk below the bud union. In acid lime, vermiform or linear pits appear in the woody cylinder. □ Tristeza infected citrus trees on sour orange rootstocks cause phloem necrosis at the graft union. □ Diseased trees usually blossom heavily. Trees with stem pitting are stunted and set less fruits. The fruits are of smaller size and of poor quality. As the fruits develop, the tree wilts partly or completely. □ Grapefruit and acid lime are susceptible irrespective of rootstock.

<table>
<tr><td colspan="3"> </td></tr>
<tr><td colspan="3">CTV induced quick decline of sweet orange CTV induced declined mosambi plant</td></tr>
<tr><td colspan="3"> </td></tr>
<tr><td colspan="3">CTV induced mosambi leaves showing chlorosis
of mid veins and lateral veins and leaf yellowing Black and brown Aphid-vector of CTV</td></tr>
<tr><td>Survival and spread</td><td>:</td><td>PSI: Infected bud woods and nursery seedlings.
SSI: Brown aphid Toxoptera citricida in semi-persistent manner. It is also transmitted by the parasitic dodder (Cuscuta reflexa).</td></tr>
<tr><td>Favourable conditions</td><td>:</td><td>□ The optimum temperatures for virus infection and multiplication are 20^0C and 25^0C, respectively.</td></tr>
<tr><td>Management</td><td>:</td><td>□ Strict quarantine measures to be enforced. Use certified bud wood free of CTV to prevent primary (vertical) spread of disease.
□ Remove all diseased trees as and when the disease is noticed. Fresh plantings to be taken with virus free materials on tolerant rootstocks.
□ For sweet orange and mandarin, avoid susceptible rootstocks.
□ For Andhra Pradesh, Maharashtra and Karnataka, Rangapur lime is recommended as a rootstock resistant to Tristeza. For the Punjab region, Jattikhatti, Cleopatra mandarin and sweet orange are recommended as resistant rootstocks.
□ For acid lime, use seedling pre-immunized with mild strain of tristeza virus (Cross protection).
□ Periodic sprays of insecticides like dimethoate or methyl- o-demeton or triazophos @ 2 ml/L to control aphids and reduce the secondary (horizontal) spread of the disease in the orchard.
□ The production of virus-free plantlets by shoot-tip grafting.</td></tr>
<tr><td></td><td></td><td>Cross protection: “Protection of plant tissues infected with one strain of a virus from severe effect of other strain of the same virus”.</td></tr>
</table>

2. Greening or Huanglongbin (HLB)		
Causal organism	:	*Candidatus liberobacter asiaticus* – in Asia *Candidatus liberobacter africanus* – in Africa *Candidatus liberobacter americanus* – in America (Fastidious phloem limited bacterium) Obligate Gram-negative bacterium.
Economic importance	:	Greening disease is known to occur in Andhra Pradesh, Karnataka, Punjab, Uttar Pradesh, Himachal Pradesh, Rajasthan, Maharashtra, Jammu, Bihar, Bengal and Sikkim. This disease affects almost all citrus varieties irrespective of rootstock but more severe on sweet orange than on acid lime, mandarin and grapefruit. In India Mosambi sweet orange and Darjeeling orange (*Citrus reticulata*) are good indicator plants for greening.
Symptoms	:	□ Affected trees are stunted with pronounced leaf and fruit drop. Varied chlorotic patterns on leaves are noticed which are persistent and cannot be corrected by mineral nutrient sprays. □ Reduction in leaf size is common. Many affected leaves show small circular green islands within the chlorotic areas. Heavy leaf fall occurs with the onset of summer. Often new flush may come out with leaves that are short, upright and chlorotic having green veins or with green blotches on the leaves. □ Twig dieback occurs. Some branches in a tree exhibit severe symptoms whereas others in the same tree are apparently normal. □ Fruits show reduction in size, lopsided in growth and oblique (curved) columella. □ The rind surface exposed to sun appears yellow whereas the remaining portion remains dull green. □ Diseased fruits are valueless owing to small size, distortion, low in juice and soluble solids, high in acid and insipid taste. □ Seeds are poorly developed, dark colored and aborted.



(A) Citrus tree affected by yellow shoot and citrus greening, (B) Leaves of greening-infected orange and lemon trees showing progressive symptoms of the disease, (C) Oranges showing delayed and abnormal coloration due to citrus greening, (D) Citrus psylla, one of the important vectors of citrus greening.

Survival and spread	:	**PSI:** The bacterial cells spread through bud woods and grafts. **SSI:** The pathogen is spread by vegetative propagation and by two psyllid insects. The Asian strain is spread primarily by *Diaphorina citri*, whereas the primary vector for the African strain is *Trioza erytreae*, but both insect vectors can transmit either strain of the bacterium.

Management	:	□ Select certified disease-free seedlings. Use pathogen free bud wood for propagation. □ Raise virus free plants through shoot tip grafting. □ Removal of infected trees as soon as they are detected. □ Regulatory (quarantine) measures should be strengthened to limit movement, sale and use of infected bud wood or nursery stock. □ Strict control of nurseries through registered disease-free certification scheme is essential to prevent the spread of disease. □ Since the disease also spreads through the vector, citrus psylla, suitable insecticides e.g., monocrotophos @ 0.7 ml/litre or quinalphos @ 1.0 ml/litre of water should be sprayed to control its spread. □ Spraying should be done at bud burst stage of when infestation is noticed on sprouts in June-July, January-February and October-November. □ Although tetracyclines (500 ppm) have been reported to suppress greening symptoms when applied as trunk injection, complete elimination of the pathogen is not possible by this method.

(7)DISEASES OF APPLE (*Malus domestica*)

1. Scab		
Causal organism	:	*Venturia inaequalis* (anamorph: *Spilocaea pomi*)
Economic importance	:	✍ First reported from Sweden (1819) ✍ First reported on Ambri variety in Kashmir valley (1935) ✍ In India, the disease was first reported in the Kashmir valley in 1935 by P. Nath on an indigenous apple cultivar 'Ambri'. ✍ However, in 1973, the existence of the elite cultivars, such as Red Delicious, came into jeopardy by the disease in epidemic form in the valley. ✍ During 1977, scab was also detected in Himachal Pradesh in an orchard and in a few localized pockets of Kullu, Mandi and Chamba districts. ✍ Apple scab fungus *Venturia*, are **hemibiotrophs**, *i.e*., they must pass part of their lives on the host as **parasites** and part on dead tissues of the same host on the ground as **saprophytes** in order to complete their life cycle in nature. ✍ **The ascopores are unequally bi-celled hence the name *inaequalis.*** ✍ The most adverse effect of the disease is the reduction of quality of fruits. ✍ In India, the first epidemic of scab was observed in Kashmir valley in 1973 and this ruined the apple crop worth Rs. 54 lakhs in a single season. ✍ In India, varieties of Delicious group are highly susceptible. ✍ Losses due to scab result from pre-mature defoliation of the tree, weakening of tree, quantitative loss in fruit yield, low market value of fruits due to malformation and scab and increased cost of cultivation and disposal of fruits. ✍ Apple scab exists worldwide but is more severe in areas with cool, moist springs and summers. ✍ In the United States it is most serious in the north central and northeastern states. Similar scab diseases affect pears (*V. pyrina*) and hawthorns (*V. inaequalis* sp.f. *pyracanthae*). ✍ Scab is the most important disease of apples. Its primary effect is reduction of the quality of infected fruit. ✍ Scab also reduces fruit size or results in premature fruit drop, defoliation, and poor fruit bud development for the next year, and it reduces the length of time infected fruit can be kept in storage. ✍ Losses from apple scab may be 70% or more of the total fruit value.

		☞ In most apple-producing areas, no marketable fruit can be harvested if scab control measures are not taken.
Symptoms	:	☞ Scab infections usually noticed on leaves and fruits ☞ Affected leaves become twisted or puckered and have black, circular spots on their upper surface ☞ On the under surface of leaves, the spots are **velvety** and may coalesce to cover the whole leaf surface ☞ Severely affected leaves may turn yellow and drop ☞ Scab can also infect flower stems and cause flowers to drop ☞ Scabby spots on fruit begin as sooty, gray-black lesions and may have a white or red halo ☞ The lesions later become sunken and tan and may have spores around their margins ☞ Infected fruit become distorted and may crack, allowing entry of secondary organisms ☞ Pulp beneath scabby lesion may rot ☞ Infection spreads and causes damage in transport and storage also ☞ Severely affected young fruit may drop
Survival and spread	:	**PSI:** Ascospores formed in pseudothecia in leaf litter **SSI:** Wind borne conidia

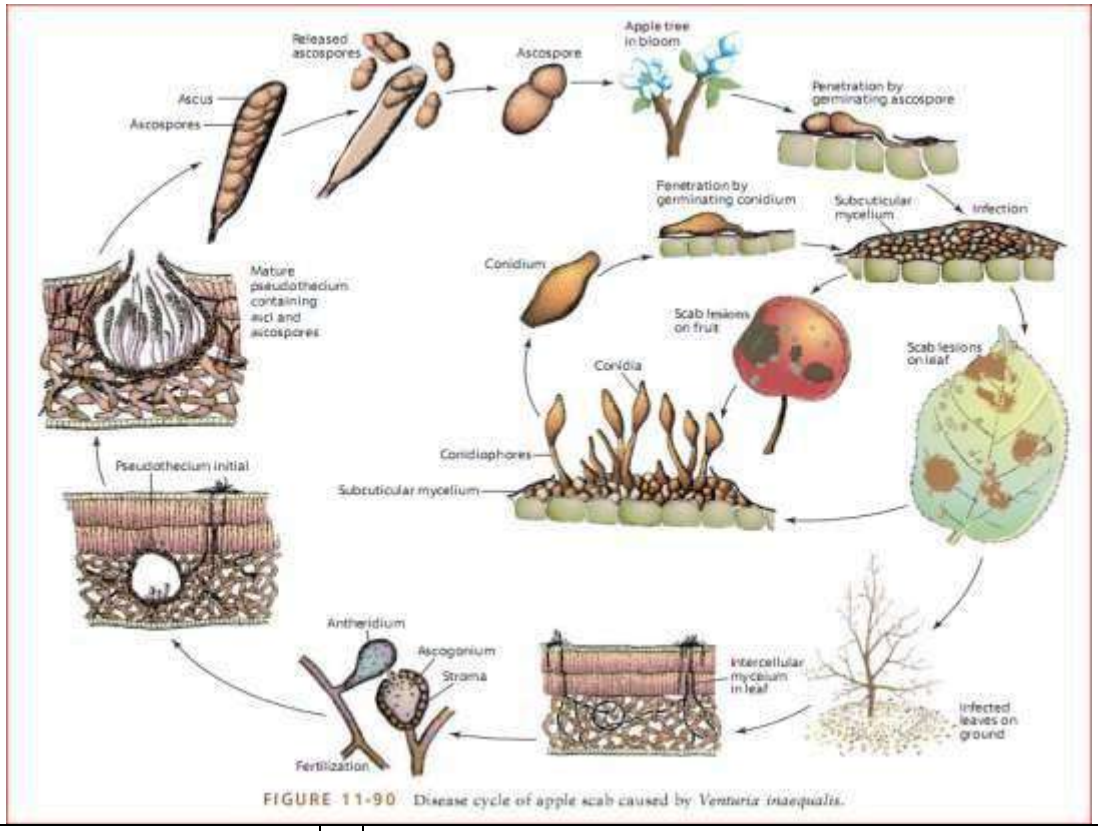


FIGURE 11-90 Disease cycle of apple scab caused by *Venturia inaequalis*.

The Pathogen: *Venturia inaequalis*		☞ The mycelium in living tissues is located only between the cuticle and the epidermal cells. ☞ There, it produces short, erect, brownish conidiophores that give rise to several, one- or two-celled, Spilocaea-type conidia of rather characteristic shape. ☞ In dead leaves, the mycelium grows through the leaf tissues and produces ascogonia and antheridia; following fertilization, pseudothecia form. ☞ The latter are dark brown to black with a slight beak and an opening. Each pseudothecium contains 50 to 100 asci, each with eight ascospores consisting of **two cells of unequal size.**

Management	:	☞ Clean cultivation ☞ Spraying **urea** 5% on leaf litter and 2% on twigs as nitrogen inhibits **pseudothecial formation** and thereby reduces primary inoculum production ☞ **Resistant varieties**: Emira, red free, Ambstraking, Ambroyal, Ambrich and Ambred ☞ Spray **dodine** 0.25% or captan 0.2% at short intervals after petal fall ☞ Single application of difolaton 0.3% at green bud stage followed by captan 0.2% at petal fall

Spray schedule:

Spray No.	Fungicide/ 100 litre water	Tree stage
I	Captafol (300 g) or mancozeb (400 g) or captan (300-400 g) or dodine (150 g) or diathianon (75-100 g)	Silver tip to green tip
II	Repeat mancozeb (300 g) or captan (200-300 g) or diathianon (50 g), if captafol not used in 1st spray	Pink bud to early bloom (10 days after 1st spray) Petal fall (two weeks after 2nd spray)
III	Carbendazim (50 g) or dodine (100 g) or thiophanate methyl (50 g)	Fruit set (10 days after 3rd spray)
IV	Carbendazim or thiophanate methyl (25 g) + mancozeb (250 g) or mancozeb (300 g) or captan (200-300 g) or diathianon (50 g) or dodine (75-100 g)	Fruit development (two weeks after 4th spray)
V	Repeat captan or diathianon or manacozeb or dodine (75-100 g)	Fruit development
VI	Repeat captan or diathianon or mancozeb	Pre-harvest (2-3 weeks before harvest)
VII	Captafol (150 g) or captan (200-300 g) or diathianon (50 g) or mancozeb (250-300 g)	Before leaf fall (7-10 days before general leaf fall)
VIII	Urea (5 kg) or Ankur (5-7 g)	

Mill's rules:

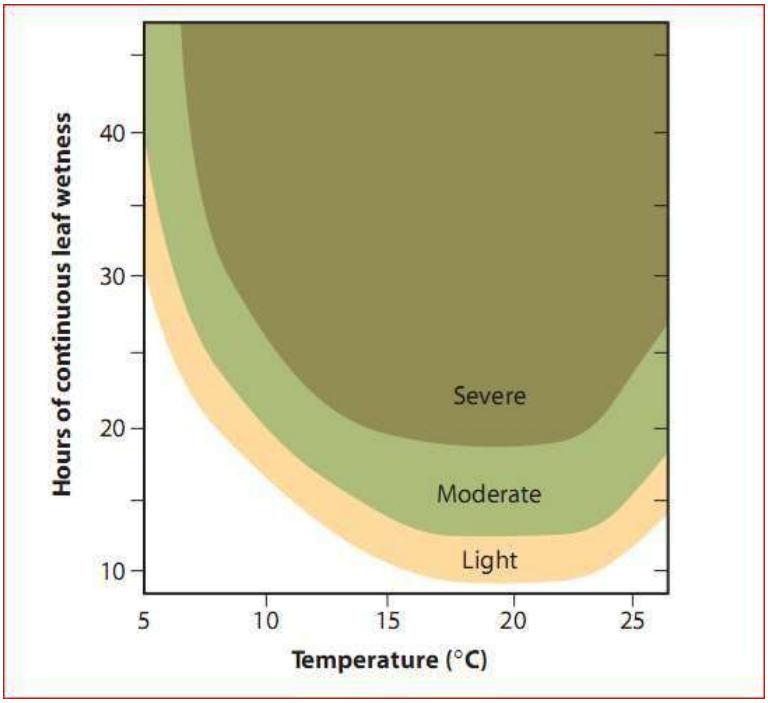
- **Mill's rules** are used to **predict Apple scab** disease.
- From studies of the apple scab disease, Mills in 1944 a table listing the duration of rain required at each temperature for apple buds, leaves, and fruit developed it to become infected by the ever-present apple scab fungus.
- He and others then could use this information to predict whether infection would take place and whether, therefore, control measures (fungicides) should be applied.
- In many fungi, moisture also affects the liberation of spores from the sporophores, which, as in apple scab, can occur only in the presence of moisture.
- The number of infection cycles per season of many fungal diseases is closely correlated with the number of rainfalls per season, particularly of rainfalls that are of sufficient duration to allow establishment of new infections.
- Thus in apple scab, for example, continuous wetting of the leaves, fruit, and so on for at least 9 hours is required for any infection to take place even at the optimum range (18 to 23°C) of temperature for the pathogen.
- At lower or higher temperatures, the minimum wetting period required is higher, e.g., 14 hours at 10°C and 28 hours at 6°C.
- Similar conditions are required for the initiation and development of infections in many other diseases.
- If the length of the wetting period is less than the minimum required for the particular temperature, the pathogen fails to establish itself in the host and fails to produce disease.

Forecasts Based on Amounts of Initial and Secondary Inoculum

- In apple scab (caused by the fungus *Venturia inaequalis*), the amount of initial inoculum (ascospores) is usually large and is released over a period of 1 to 2 months following bud

break.

- Infections from the primary inoculum must be prevented with well-timed fungicide applications during blossoming, early leafing, and fruit development; otherwise, the entire crop is likely to be lost.
- After primary infections, however, secondary inoculum (conidia) is produced, which multiplies itself manyfold with each succeeding generation.
- The pathogen can infect wet leaf or fruit surfaces at a range of temperatures from 6 to 28°C.



Relationship of temperature and duration of leaf wetness to the occurrence and severity of apple scab caused by the fungus *Venturia inaequalis*. [From **Mills (1944).** *Cornell Ext. Bull.* 630.]

Table: Wetting period (in hours) required for apple scab infection at different air temperatures, and time required for development of conidia by lesions at different air temperatures. W.D. Mills, Cornell University, as modified by A.L. Jones, Michigan State University

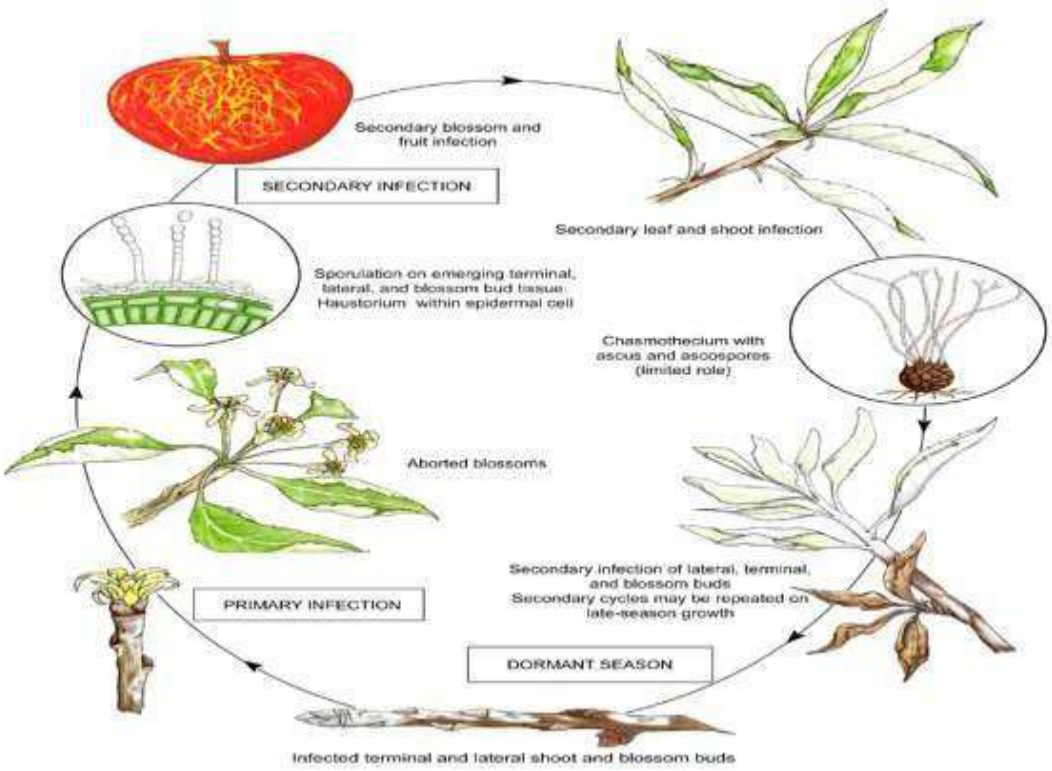
Wetting period (HOURS)					
Average Temperature (F)	Average Temperature (C)	Light Infection	Moderate Infection	Heavy Infection	Incubation Period (days)
78	25.6	13	17	26	...
77	25.0	11	14	21	...
76	24.4	9.5	12	19	...
63-75	17.2-23.9	9	12	18	9
62	16.7	9	12	19	10
61	16.1	9	13	20	10
60	15.6	9.5	13	20	11
59	15.0	10	13	21	12
58	14.4	10	14	21	12
57	13.9	10	14	22	13
56	13.3	11	15	22	13
55	12.8	11	16	24	14
54	12.2	11.5	16	24	14
53	11.7	12	17	25	15
52	11.1	12	18	26	15
51	10.6	13	18	27	16
50	10.0	14	19	29	16
49	9.4	14.5	20	30	17
48	8.9	15	20	30	17
47	8.3	17	23	35	17
46	7.8	19	25	38	17
45	7.2	20	27	41	17
44	6.6	22	30	45	17
43	6.1	25	34	51	17
42	5.5	30	40	60	17

2. Powdery mildew

Causal organism	:	*Podosphaera leucotricha* (I. S: *Oidium* spp)

Economic importance	:	☞ The disease is present in all apple-growing regions of the world and is common in apple orchards of northern India. ☞ Damage to nursery plants is more than the adult ones during April-June. ☞ Sometimes the disease causes considerable damage to leaves and young shoots resulting into reduction of fruit yield. ☞ Besides apple, the pathogen also attacks peach, quince and pear.

Symptoms	:	☞ Appear soon after the buds develop into new leaves and shoots ☞ Small patches of white or grey powdery masses on under surface of leaves ☞ Leaves grow longer and narrower than normal leaves and the margin is curled ☞ Twigs covered with powdery mass ☞ Affected fruits remain small and deformed and tend to develop roughened surface ☞ In nursery plants, formation of wood is affected
Survival and spread	:	**PSI:** Dormant mycelium in diseased vegetative buds and fruits **SSI:** Wind borne conidia



Management	:	Sanitation of orchard Pre-bloom spray of lime sulphur (1:60) Spray dinocap @ 0.15% or wettable sulphur Resistant varieties: Maharaja chunth and Golden Chinese (apple cultivars), Yantarka Altaskya, Dolgoe (Crab apple cultivars)

3. Fire blight		
Causal organism	:	*Erwinia amylovora*
Economic importance	:	✍ *Erwinia amylovora* is a native pathogen of wild, rosaceous hosts in eastern North America. ✍ It was the first bacterium proven to be a pathogen of plants. Today, fire blight is an important disease of apples and pears in many parts of the world. ✍ The first report of fire blight as a disease of apple and pear occurred in 1780, in the Hudson Valley of New York. ✍ *E. amylovora* is also one of the first plant pathogens to be associated with an insect vector.
Symptoms	:	✍ All above ground tissues including blossoms, fruits, shoots, branches, and in the rootstock near the graft union on the lower trunk are affected ✍ **Blossom** symptoms are first observed 1-2 weeks after petal fall. The floral receptacle, ovary, and peduncles become water soaked and dull, grayish green in appearance. Later these tissues shrivel and turn brown to black giving a burnt appearance ✍ Tips of shoots may wilt rapidly. Leaves on diseased shoots often show blackening along the midrib and veins, before becoming fully necrotic ✍ Numerous diseased shoots give a tree burnt and blighted appearance ✍ Bark on younger branches becomes darkened and water-soaked. At advanced stages, cracks will develop in the bark, and the surface will be sunken slightly ✍ Wood under the bark will show streaked discolorations. Similar symptoms can be observed in infected apple rootstocks ✍ Infected small immature fruit becomes water soaked, then turn brown, shrivel, turn black and cling to the tree for several months after infection ✍ Droplets of milky coloured, sticky ooze may appear on infected parts which usually turns amber coloured on exposure to air

Survival and spread	:	**PSI:** The bacteria overwinter at the margins of the cankers and possibly in buds and apparently healthy woody tissue **SSI:** Insects such as bees, flies and ants spread the bacteria to the flowers

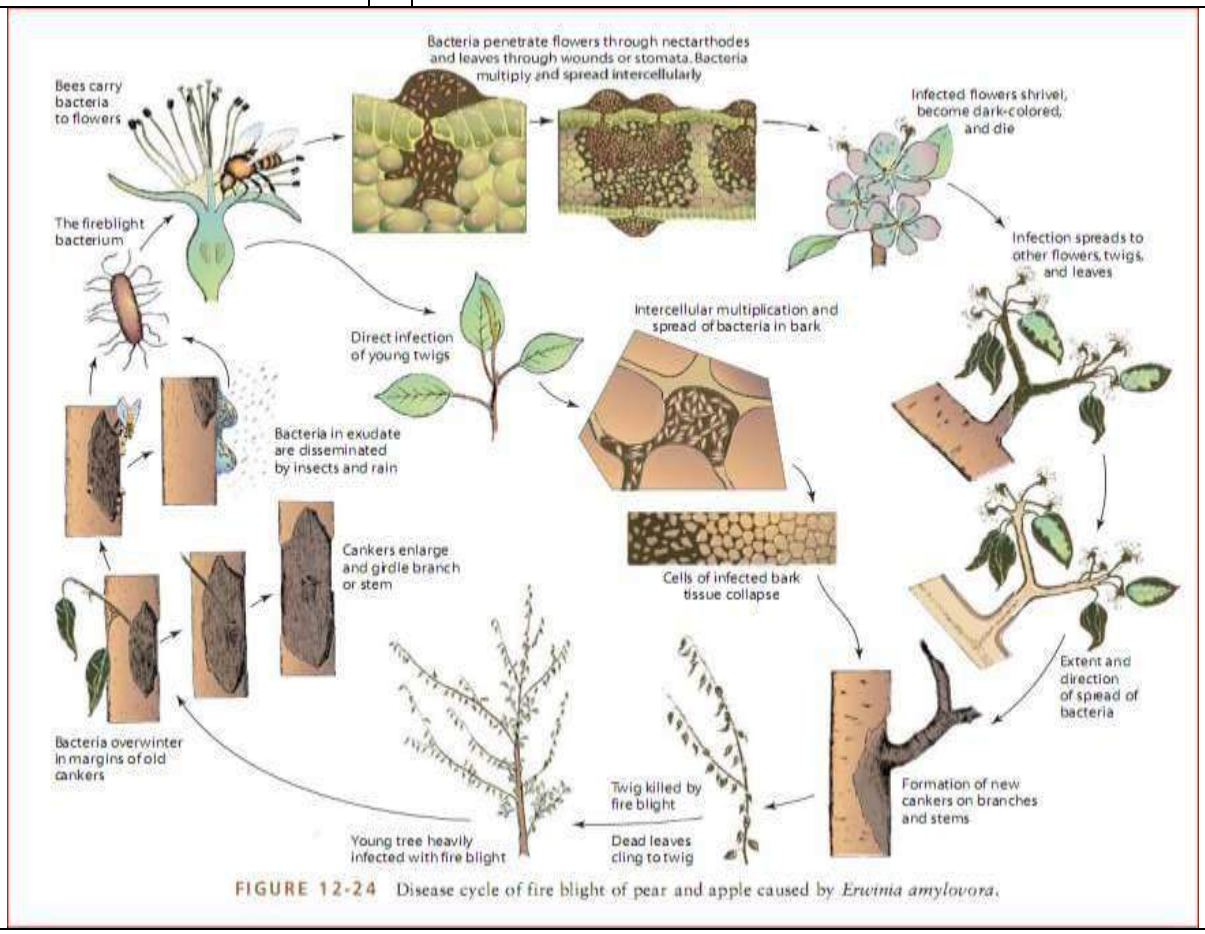


FIGURE 12-24 Disease cycle of fire blight of pear and apple caused by *Erwinia amylovora*.

Management	:	☞ During winter prune out and burn blighted twigs, branches and cankers, and even whole trees, at about 30 cm below the point of visible infection ☞ Disinfect the tools after each cut with 1% sodium hypochlorite ☞ Insect control ☞ Resistant varieties ☞ Dormant sprays with Bordeaux mixture ☞ Bordeaux mixture + streptomycin sulphate is effective as blossom spray ☞ Alternate spray of streptomycin sulphate and kasumin is required to suppress the pathogen

(8) DISEASES OF GRAPEVINE (*Vitis vinifera*)

1. Powdery mildew		
Causal organism	:	*Uncinula necator* (I.S: *Oidium tuckeri*)
Economic importance	:	✍ Losses in yield of fruits may be up to 40-60%. ✍ In addition to loss of yield, infected berries tend to have higher acid content than healthy fruits and are unsuitable for wine making.
Symptoms 	:	✍ The disease attacks vines at any stage of growth. All the aerial parts of the plant are attacked. Cluster and berry infections usually appear first ✍ Floral infection results in shedding of flowers and poor fruit set ✍ Early berry infection results in shedding of affected berries ✍ Powdery growth is visible on older berries and the infection results in the **cracking of skin of the berries**. Often infected berries develop a net-like pattern of scar tissues ✍ Powdery growth mostly on the upper surface of the leaves ✍ Malformation and discolouration of affected leaves. Leaf lesions appear late and cause lesser damage than cluster or berry infection ✍ Infection of stem leads to dark brown discolouration
Survival and spread	:	**PSI:** Through **dormant mycelium** and **conidia** present in the infected shoots and buds **SSI:** Through air-borne **conidia**
Favourable conditions	:	✍ Cool dry weather ✍ Maximum temperature in the range of 27-31^{o}C with R.H. up to 91% favour disease incidence (November and December)
Management	:	✍ Clean cultivation of vines or removal and destruction of all diseased parts ✍ Dustings of vines with 300 mesh sulphur (1st when new shoots are 2 weeks old, 2nd prior to blossoming, 3rd when the fruits are half ripe) ✍ Prophylactic spray with B.M. 1% or lime sulphur at dormant stage delays development of disease by decreasing initial inoculum ✍ Spray wettable sulphur @ 0.3% or dinocap or tridemorph @ 0.1% ✍ Oxythioquinox (Morestan) @ 0.03% sprayed at 4 days interval starting from last week of December to 1st week of March ✍ Grow resistant varieties like **Chholth Red, Chholth white, Skibba Red, Skibba White**
2. Downy mildew		

Causal organism	:	*Plasmopara viticola*
Economic importance	:	Since 1875, this disease caused heavy losses to wine industry in France It led to discovery of Bordeaux Mixture by Prof. Millardet in 1885
Symptoms	:	Symptoms appear on all aerial and tender parts of the vine. Symptoms are more pronounced on leaves, young shoots and immature berries Irregular, yellowish, translucent spots limited by veins appear on upper surface of leaves Correspondingly on the lower surface dirty white, downy growth of fungus appears Affected leaves become yellow and brown and dried due to necrosis Premature defoliation Dwarfing of tender shoots Infected leaves, shoots and tendrils are covered by whitish growth of the fungus White growth of fungus on berries, which subsequently becomes leathery and shrivels. Infected berries turn hard, bluish green and then brown Later infection of berries results in soft rot symptoms. Normally, the fully grown or maturing berries do not contact fresh infection as stomata turn non-functional No cracking of the skin of the berries
Survival and spread	:	**PSI:** Oospores present in the infected leaves, shoots and berries. Also as dormant mycelium in infected twigs **SSI:** Sporangia or zoospores by wind, rain and insects

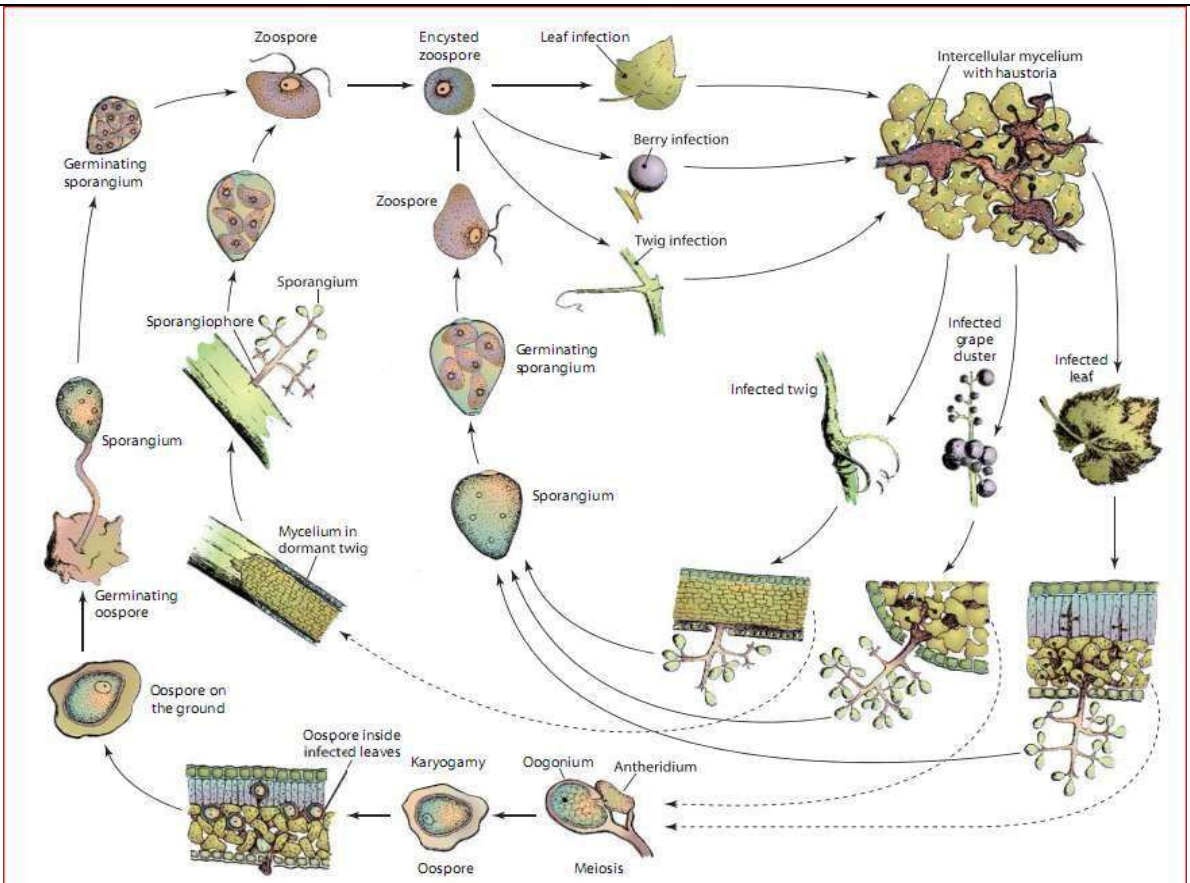
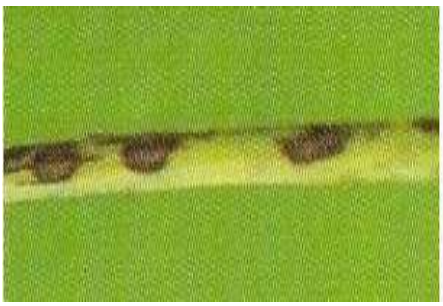


FIGURE 11-32 Disease cycle of downy mildew of grapes caused by *Plasmopara viticola*.

Favourable conditions	:	Optimum temperature : 20-22°C Relative humidity : 80-100% High soil moisture
Management	:	Field sanitation by collection and burning of fallen leaves and twigs Vine should be kept high above ground to allow circulation of air by proper spacing Pruning (April-May & September-October) and burning of infected twigs **Targeted pruning to improve air circulation slows down disease progression** Grow resistant varieties like Amber Queen, Cardinal, Champa, Champion, Dogridge and **Red Sultana** The disease can be effectively managed by giving 3-5 prophylactic sprays with Bordeaux mixture 1% or fosetyl-Al (Aliette) 0.2% or curative spray with metalaxyl + mancozeb (Ridomil MZ 72) 0.2% or Azoxystrobin 0.1% Spray schedule with Bordeaux mixture 1% and other chemicals 1. Immediately after pruning of vines 2. When new flush is formed (3-4 weeks after pruning) 3. Before buds open 4. When bunches or berries are formed 5. During shoot growth

3. Anthracnose / Birds eye disease		
Causal organism	:	*Elsinoe ampelina* (I.S: *Gloeosporium ampelophagum* or *Sphaceloma ampelina*)
Economic importance	:	It is especially serious on new sprouts during rainy season. Among various foliar diseases of grapevine in India, anthracnose has longest spell spread over from June to October
Symptoms	:	Visible on leaves, stem, tendrils and berries Young shoots and fruits are more susceptible than leaves Circular, greyish black spots or red spots with yellow halo appear on young leaves Later the centre of the spot becomes grey, sunken and fall off resulting in a symptom called 'shot hole' Black, sunken lesions appear on young shoots Cankerous lesions on older shoots. Girdling and death of shoots may occur Infection on the stalk of bunches and berries result in the shedding of bunches and berries **Sunken spots with ashy grey centre and dark margin** appear on fruits (**bird's eye** symptom). In warm and wet weather, pinkish spore mass develops in the centre of spots **Mummification** and shedding of **berries**
		
Survival and spread	:	**PSI:** Survives as dormant mycelium in the infected stem-cankers **SSI:** Wind borne and rain splashed conidia
Favourable conditions	:	Warm wet weather with continuous drizzle of rain and windy weather for 2-3 days Low lying and ill drained soils Anab-e-Shahi variety is susceptible.
Management	:	Removal of infected twigs Selection of cuttings from disease free areas and dipping them in 3% $FeSO_4$ solution for 30 minutes before planting. Spraying Bordeaux mixture 1% or CoC @ 0.25% or **carbendazim** @ 0.1%. Varieties like Bangalore blue, **Golden Muscat**, Golden queen and Isabella are resistant

(9) DISEASES OF GUAVA (*Psidium guajava*)

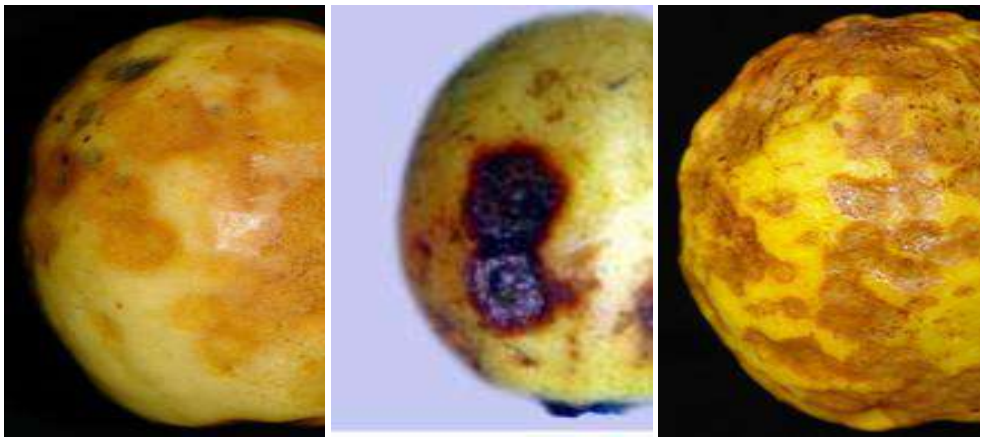
1. Wilt		
Causal organism	:	*Fusaium oxysporum* f.sp. *psidii*
Economic importance	:	✍ It was first reported in 1935 from Allahabad. ✍ Area under guava cultivation in A.P was reduced to half due to the disease.
Symptoms	:	✍ The disease can be categorized into slow wilt and sudden wilt. In **slow wilt**, plant takes several months or even a year, to wilt after the appearance of initial symptoms and in **sudden wilt**, infected plant wilts in a month ✍ Loss of turgidity in a branch or branches of one side of the tree is the first external symptom. Leaves in tis branch wither turn pale and droop before finally dropping-off ✍ Symptoms gradually appear on other branches also ✍ Infected and withered branches do not recover ✍ A few plants may show partial wilting, which is a common symptom of wilt in guava ✍ As all the branches have withered the tree wilts and dies ✍ The finer roots show black streaks, which appear prominent on removal of bark. The roots also show rotting at the basal region and the bark is easily detachable from the cortex ✍ Light brown discoloration is noticed in vascular bundles
Survival and spread	:	**PSI: Chlamydospores** in soil **SSI: Macro and micro conidia** through irrigation water
Favourable conditions	:	✍ **pH 6.0** is optimum for disease development. Both pH 4.0 and 8.0 reduces the disease. ✍ Disease is more in loamy alluvial soils than in heavy soils ✍ Infected guava plants start showing sign of wilting right with the onset of rainy season in August with

		maximum number dying in September and October. **The disease is more prominent during rainy season.** **Further, the disease incidence increases in post-monsoon period** The nematode, ***Helicotylenchus dihystera*** aggravates the disease
Management	:	***Cultural*** **:** Layers or grafts for planting in new orchards should be procured from disease free areas Proper sanitation of orchard Wilted plants should be uprooted, burnt and a trench of 1.0-1.5 m should be dug around the tree trunk. Treat the pits with Bordeaux mixture and cover the pit for a few days before planting new plants While planting damage to the root of layers or grafts should be avoided Maintain proper tree vigour by timely and adequate manuring, inter-culture and irrigation Application of oil cakes like neem cake, mahuva cake, kusum cake supplemented with urea Judicious amendments of Nitrogen and Zinc ***Host Plant Resistance*****:** Resistant varieties: **Apple guava** and **Bhuvanagiri** *Psidium* species, *Psidium cattleianum var. lucidum* and *Syzigium cumini* (Jamun) are resistant to wilt ***Biological*** **:** *Aspergillus niger*, *Trichoderma viride*, *Trichoderma harzianum* and *Penicillium citrinum* can used as biocontrol agents ***Chemical*** **:** Drench the basins of trees in orchards with disease incidence with 0.2% benomyl or carbendazim, four times in a year

2.Anthracnose: *Gloeosporium psidii*

Symptoms:

- The most characteristic symptoms appear during the rainy season as small pin-head sized spots on the unripe fruits.
- They gradually enlarge to form sunken and circular, dark brown to black spots.
- The infected area of the unripe fruits becomes harder and corky.
- Acervuli are formed on fruit stalks.
- The pathogen remains dormant for about three months in the young infected fruits.
- In moist weather, acervuli appear as black dots scattered throughout the dead parts of the twigs.
- The conidia are spread by wind or rain.



Initial symptom **Circular black spots** **Corky fruit**

Management:

- Spraying the trees with Copper oxychloride 0.2% before the onset of monsoon reduces the disease incidence.

Emerging Nematode Problem in Guava

The **guava root**-knot **nematode** (Meloidogyne enterolobii) is considered to be the most damaging **root**-knot **nematode** in the world because of its wide host range, aggressiveness, and ability to overcome the resistance that has been developed against **root**-knot **nematodes** in many crops.

Management and Disease cycle same as Root knot of Vegetable(Pl.Path.5.4)

(10) DISEASES OF CHILLI (*Capsicum annuum*)

<table>
<tr><th colspan="3">1. Anthracnose/Die-back / fruit rot</th></tr>
<tr><td>Causal organism</td><td>:</td><td>Colletotrichum capsici (Teleomorph: Glomerella cingulata)</td></tr>
<tr><td>Economic importance</td><td>:</td><td>Severe in Guntur and Krishna districts</td></tr>
<tr><td>Symptoms</td><td>:</td><td>Dieback Symptoms:
Disease is more in December - October in transplanted crop
Small, circular to irregular, brownish black scattered spots appear on leaves
Severely infected leaves defoliate
Infection of growing tips leads to necrosis of branches from tip backwards
Necrotic tissues appear grayish white with black dot like acervuli in the center
Shedding of flowers due to the infection at pedicel and tips of branches
Fruit symptoms:
Ripe fruits are more vulnerable to attack than green ones
Small, circular, yellowish to pinkish sunken spots appear on fruits
Spots increase along fruit length attaining elliptical shape
Severe infection result in the shrivelling and drying of fruits.
Tissues around lesions will be bleached and turn white or greyish in colour and lose their pungency
On the surface of the lesions minute black dot like fruiting bodies called ‘acervuli’ develop in concentric rings and fruits appear straw coloured
The affected fruits may fall off subsequently. The seeds produced in severely infected fruits are discoloured and covered with mycelial mat</td></tr>
<tr><td colspan="3">
Die-back phase

Fruit rot phase</td></tr>
<tr><td>Survival and spread</td><td>:</td><td>PSI: Mycelium and conidia in acervuli in infected seeds and diseased crop debris</td></tr>
</table>

		SSI: Conidia dispersed by rain splash and wind
Favourable conditions	:	Temp, 28^{o}C with RH more than 97% Humid weather with rainfall at frequent intervals Intercropping with **turmeric** which is another host of the fungus
Management	:	Seed treatment with captan or thiram 3-4 g/kg Collect **seeds** only **form fruits without infection** **Avoid intercropping** with susceptible **turmeric** cultivars Collect and destroy all infected plant parts Removal and destruction of Solanaceous weed hosts and infected plant debris Spray three times with captan 1.5% or mancozeb @ 0.25% just before flowering, at fruit formation stage and 15 days after second spray Resistant varieties: **G3, G4, B61, Lorai,** etc
2. Leaf curl		
Causal organism	:	*Nicotiana virus*-10 *Tobacco leaf curl virus*
Symptoms	:	Young leaves become folded upward showing a boat like structures Leaf curling spreads from the center of the plant to other older leaves Reduction in leaf size Internodes become shortened Plants are stunted and remain unproductive Leaves become pale yellow in colour Thickening and swelling of veins observed The infected plant shows bushy and stunting appearance Fruits formed on infected plant are small and deformed
Virus transmission	:	The virus is not seed or sap transmissible. In nature it is transmitted by white fly-*Bemisia tabaci*
Management	:	Infected plants should be rouged out and destroyed Spray methyl-o-demeton 0.025% or dimethoate 0.05% at 10 days interval Remove the various collateral hosts from nearby vicinity to reduce the source of inoculum Spraying should be stopped at least 20 days before picking of fruits

3. Wilt: Causal organism: *Fusarium oxysporum f.sp.capsici*
Family: Tuberculariaceae
Order: Tuberculariales
Class: Hyphomycetes
Sub.Division: Deuteromycotina

Fusarium wilt is one of the important disease occurring in both temperate and warmer areas. The degree of loss caused by the pathogen varies depending upon host cultivar, race of the pathogen and environmental conditions.

Symptoms:

1. On the young seedlings initially, water soaked areas developed at the collar region and a brown sunken lesion which soon appeared as girdled resulting in seedling collapse.
2. On adult plant initially, slight drooping of leaves which led to drying of leaves starting from lower ones extended from root to stem region and plants exhibited wilting symptom.

Disease cycle:

Primary source of inoculum: Chlamydospores, Soil, Seed.

Secondary source of inoculum: Micro conidia, Macro conidia, water

Epidemiology:

- Fusarium is a soilborne fungus. Once a field is infested, the pathogen may survive in the soil for many years. The fungus can be transported by farm equipment, drainage water, wind, or animals, including humans. The fungus is seed and soil borne.
- Warmer and drier climates (>25o C) favor disease and also when crop rotations are not practiced.
- Management as as other Fusarium wilt

(11) DISEASES OF CUCURBITS

1. Downy mildew		
Causal organism	:	*Pseudoperonospora cubensis*
Host range	:	Musk melon, Sponge gourd and Bitter gourd etc.
Symptoms	:	Yellow, angular spots restricted by veins resembling mosaic mottling appear on upper surface of leaves The corresponding lower surface of these spots shows a purplish downy growth in moist weather The spots turn necrotic with age The diseased leaves become yellow and fall down Diseased plants get stunted and die Fruits produced may not mature and have a poor taste
Survival and spread	:	**PSI: Oospores** in soil and sporangia from perennial collateral weed hosts in the vicinity **SSI:** Wind borne and rain splashed **conidia (sporangia)** or autonomous **zoospores**
Favourable conditions	:	Relative humidity >90% High soil moisture Frequent rains
Management	:	Destruction of cucurbitaceous weeds around field Spray metalaxyl + mancozeb (ridomil) 0.2% or copper oxychloride 0.3%
2. Powdery mildew		
Causal organism	:	*(1) Erysiphe cichoracearum* *(2) Sphaerotheca fuligena*
Host range:	:	Pumpkins, bottle gourd, *Coccinia*, cucumber, ridge gourd Bitter gourd is less affected
Symptoms	:	Whitish or **dirty grey, powdery growth** on foliage, stems and young growing parts The superficial growth ultimately covers the entire leaf area The diseased areas turn brown and dry leading to premature defoliation and death Fruits remain underdeveloped and are deformed

Survival and spread	:	**PSI: Dormant mycelium** or **cleistothecia** in infected plant debris or conidia from collateral hosts **SSI:** Wind borne **conidia**
Favourable conditions	:	Morning relative humidity > 90% Cool and dry weather
Management	:	Field sanitation Removal and destruction of collateral weed hosts from the vicinity of the field Spray tridemorph 0.1% or dinocap 0.2%
3. Cucumber mosaic		
Causal organism	:	*Cucumber mosaic virus*
Host range	:	Cucumber, Pumpkin, gourds, Cowpea, tomato, chilli, *etc.* *Cucumovirus* with spherical particles having ssRNA
Symptoms	:	Symptoms appear on the youngest and still expanding leaves when infection occurs at 6-8 leaves stage Typical mosaic pattern develops on young leaves Leaves curl downwards and become mottled, distorted, wrinkled and reduced in size Veins appear bunchy because of shortening of internodes When infection occurs at midseason previous growth remains normal and produces normal fruit Fruit set is very less if infection occurs early in crop growth Fruits are often misshapen, mottled, warty and reduced in size

Survival and spread	:	**PSI: Virus particles** on collateral and other weeds, ornamentals or crops Hosts: Banana, clover, corn, passion fruit, safflower, spinach, sugarbeet, wild cucumber, *Commelina communis, C. diffusa, C. nudiflora, Solanum elaegnifolium, Phytolacca* sp., periwinkle, *Gladiolus* sp., *Impatiens* sp. and *Phlox*
		SSI: Virus particles transmitted by aphids (*Aphis craccivora*, *Myzus persicae*) and spotted and striped cucumber beetles
Management	:	Rogue out infected plants Eliminate perennial weed hosts Raise barrier crops Vector control by spraying phosphamidon, 1.5 ml/liter of water

(12) DISEASES OF MARIGOLD (*Tagetes* spp.)

1. Alternaria leaf spot & flower blight/ Inflorescence blight		
Causal organism	:	*Alternaria tagetica, Alternaria zinniae*
Symptoms	:	☞ Spots on leaves, stems, and flowers. ☞ Disease starts on lower leaves and progresses upward. ☞ African marigold varieties are the most susceptible to leaf spots. ☞ The infection appears as minute, small, blackish-brown, circular spots which later enlarge, coalesce to form large irregular spots or blotches, which are light brown to dark brown in colour.

Fig.1. Symptoms of the Alternaria leaf spot and flower blight in the field

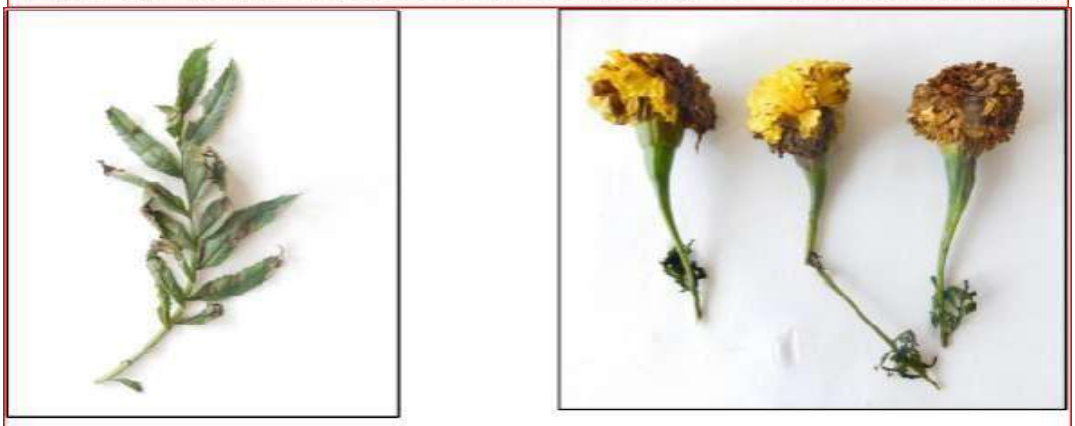


Fig.2. Symptoms of the disease on leaves and flowers

Disease cycle	:	☞ The infected leaves and debris, which contains mycelium and conidia, which acts as a primary source of inoculum. ☞ The secondary spread takes place through the wind borne conidia.
Management	:	☞ The disease is controlled by spraying mancozeb or copper oxychloride 0.2%.

		☞ Disease mostly found in rainy season. ☞ Fungal growth appears on spot as a conidia and conidiophores.
Management	:	☞ Spray chlorothalonil or mancozeb of zineb @ 0.25% at disease initiation and repeat it, if required twice or thrice.

(13) DISEASES OF ROSE (*Rosa* spp.)

1. Powdery mildew		
Causal organism	:	*Sphaerotheca pannosa* var. *rosae*
Symptoms	:	☞ The infected leaves show grayish powdery fungal growth ☞ Rolling of leaf margins with the onset of sprouting season ☞ Affected leaves become curled and distorted ☞ Badly infected flower buds fail to open ☞ Infected floral parts become discoloured, dwarfed and dried
Survival and spread	:	**PSI: Dormant mycelium** or **cleistothecia** in dormant buds and shoots
		SSI: Wind borne **oidium type conidia**

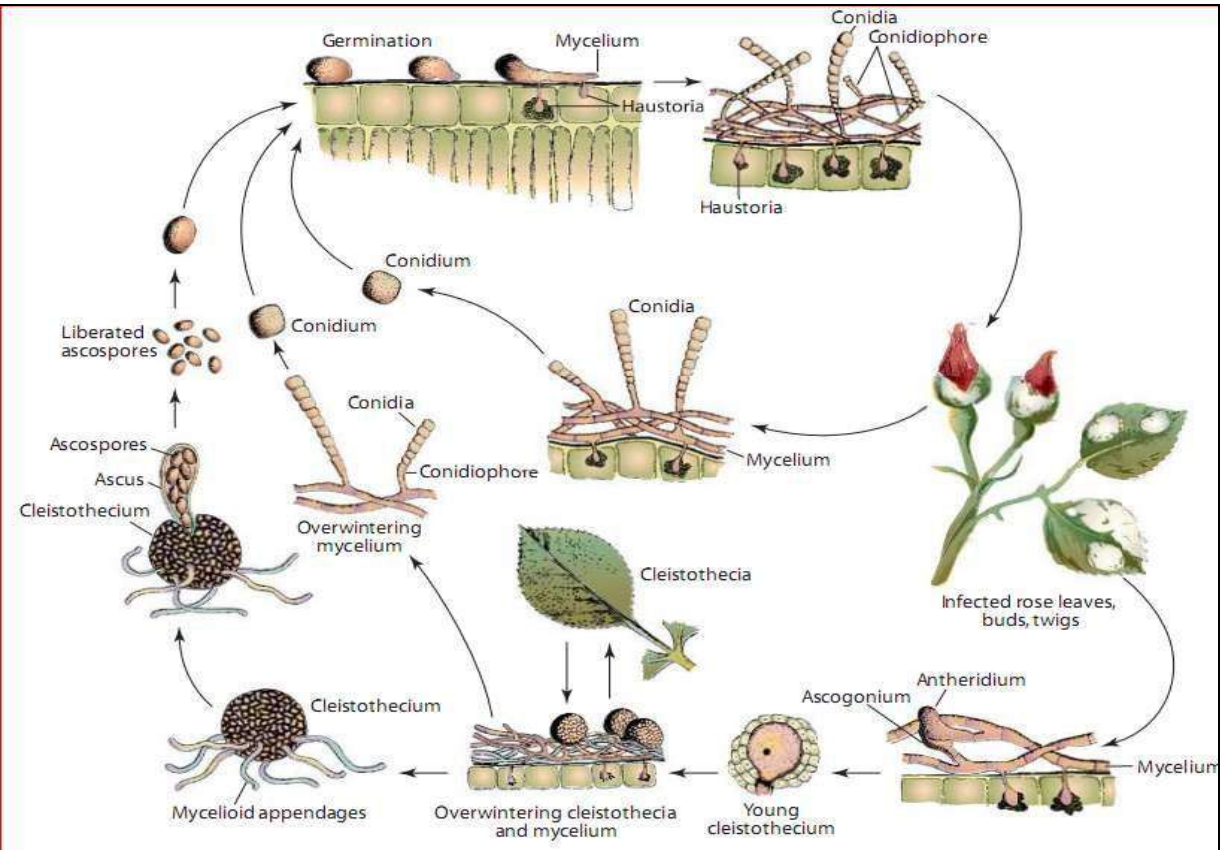


FIGURE 11-50 Disease cycle of powdery mildew of roses caused by *Sphaerotheca pannosa* f. sp. *rosae*.

Management	:	Collect and destroy infected plant debris Four sprays with wettable sulphur 0.3% or carbendazim 0.1% or dinocap 0.1% at 10 days interval Resistant varieties: **Ashwini, Ambika, Angeles, American pride, Surabhi**

2. Black spot

Causal organism	:	*Diplocarpan rosae* (Anamorph*: Marssonina rosae*)
Symptoms	:	Dark brown tar coloured spots with fringed borders Spots coalesce forming large patches Infected leaves turn brown and defoliate Fungus may also attack stems and flowers of rose bushes On stems, infected areas are blackened with blistered appearance dotted with pustules

Survival and spread	:	**PSI: Pycnidia** or **ascospores** in infected plant debris
		SSI: Wind borne **conidia**
Management	:	Affected parts should be collected and destroyed Spray tridemorph 0.1% or benomyl 0.1% at weekly intervals starting with the sprouting of the plants till new foliage appears Shade and extensive irrigation should be avoided Resistant varieties: Bebe bune, Coronado, Grand opera, Sphinx Cultivar, Radiance escapes infection due to waxy surface
3. Die-back		
Causal organism	:	*Diplodia rosarum*
Symptoms	:	Infection starts from the pruned surface of the twigs Infected portion become brown to dark brown or black Infected twig dries from tip downwards Infection spreads from twig to main stem and roots and finally whole plant is killed Older plants and neglected bushes are more frequently attacked
Survival and spread	:	**PSI: Pycnidia** in infected and dead twigs
		SSI: Conidia spread through irrigation water and wind
Management	:	Diseased twigs should be carefully removed and destroyed After pruning the cut end should be protected with Chaubattia paste Spray difolatan 0.2% or mancozeb 0.2% or chlorothalonil 0.2% in early September and late October Resistant varieties: **Blue moon, Red gold, Summer queen** etc.

(14) DISEASES OF FENNEL (*Foeniculum vulgare*)

1. Ramularia Blight		
Causal organism	:	*Ramularia foeniculi*
Symptoms	:	☞ Symptoms first appear on the lower and older leaves in the month of January, as a minute, angular ashy-brown, necrotic spots. ☞ Later these spots become large and are covered with grayish white erumpent growth. ☞ At later stage, linear and rectangular spots cover the entire stem, peduncles and fruits. In case of severe attack, whole plant turns to brown colour, resulting in drying up of plant.

Disease cycle	:	☞ Pathogen survives on infected plant debris in the soil. ☞ Pathogen infects lower part first and then above plant parts. It spreads through wind and rain splash.
Favourable conditions	:	☞ Disease favours high humidity >80%
Management	:	☞ At the initial stage, spray 0.25 % Mancozeb (25 g/ 10 liter water) to control disease. ☞ Subsequently, 2-3 sprayings should be given at an interval of 10 to 15 days. ☞ Seed treatment of thiram 3 g/ kg seed. ☞ Select healthy seed for sowing.
2. Collar /stem rot		
Causal organism	:	*Sclerotinia sclerotiorum*
Symptoms	:	☞ This disease appears in those plots where water stagnation near the collar of plant is more. ☞ Collar portion of the plant start decaying and the plants turn to yellow colour & die later on.

Disease cycle	:	Infection usually occurs through injuries caused during transplanting. **Primary**: Dormant mycelium and sclerotia survive in the infected crop debris, seeds and volunteer plants. **Secondary** spread to nearby plants by rain splash or water.
Favourable conditions	:	High humidity, high soil moisture and temperature. Crowded seedlings.
Management	:	Drench 1% Bordeaux mixture or spray copper oxychloride @ 0.25% or any copper fungicides to

		control the disease. ✍ Avoid water stagnation in the field.
3. Sugary secretion		
Causal organism	:	It is a physiological disorder
Symptoms	:	✍ It is a physiological disorder, due to which flowers secrete sugary substance; attracting saprophytic fungi. ✍ Quality and production of crop is affected. ✍ Sugary secretion spreads on flower parts, stem etc. ✍ The mold and other fungus develop on plants causing black and gummy appearance. ✍ The sugary secretion attracts aphids too. Combined effect of above gives poor appearance of disease.
Management	:	✍ Stop irrigation and manuring. ✍ Spray dimethoate, 0.03% solution or phosphamidone, 0.05 % or any systemic pesticide.

(15) DISEASES OF CRUCIFERS

1. Club root of crucifers or Finger and toe disease		
Causal organism	:	*Plasmodiophora brassicae*
Economic importance	:	Fairly severe in hilly regions on cabbage, cauliflower and other crucifers Once infected, fields become unfit for cultivation of crucifers for several years
Symptoms	:	Infected plants have pale green to yellowish leaves initially. Later, infected plants show wilting in the middle of hot, sunny days, recovering during the night Stunting of above ground parts and reduction in size of heads Characteristic symptoms become apparent in advanced stage of root infection as **spindle like, spherical, knobby, or club-shaped swellings** The swellings may be few, or they coalesce and cover the entire root system
Survival and spread	:	**PSI:** Soil borne resting spores, which survive for longer periods in soil (10 years)
		SSI: Autonomous zoospores, also carried through irrigation water or by root contact
Favourable conditions	:	It occur at a temperature range of 12-27^{o}C High soil moisture Neutral to acidic soils 5-7.0 pH
Management	:	Use of seedlings from disease free fields Plant cabbage and other susceptible cruciferous crops in well drained fields that have a pH slightly above neutral (usually about pH 7.2) Add hydrated lime to soil to increase pH to 7.2 (6 weeks before planting @ 2.5 t/ha) Avoid excess irrigation Treat the soil of seedbed areas with chloropicrin, methyl bromide or vapam two weeks before sowing.

2. Alternaria leaf spot		
Causal organism	:	*Alternaria brassicola, A.brassicae, A.raphani*
	:	The disease is common on cabbage and cauliflower
Symptoms	:	Symptoms usually on older leaves Spots are small, dark coloured They enlarge, soon become circular Under humid conditions groups of conidiophores will be formed in the spot Spots develop concentric rings Finally the spots coalesce leading to blighting of leaves Shot holes may be formed due to falling off of infected spot tissue The fungus is seed borne and cause shriveling of seeds and poor germination Linear spots also appear on petioles, stems and pods
Survival and spread	:	**PSI:** Dormant mycelium or conidia on seed or in infected plant debris
		SSI: Wind borne conidia
Favourable conditions	:	Relative humidity >80% Less vigorous plants Late in the season
Management	:	Hot water treatment at 50^{O}C for 30min Seed treatment with thiram or mancozeb Foliar spray with mancozeb 0.25% or oxychloride 0.2% twice copper
3. Black rot		
Causal organism	:	*Xanthomonas campestris* pv. *campestris*
	:	Serious on cabbage, cauliflower, knol-khol and raddish
Symptoms	:	First appear as chlorotic or yellow (angular) areas near the leaf margins Yellow area extends to veins and midrib forming characteristic '**V**' shaped chlorotic spots which later turn black Veins and veinlets turn brown and finally black Vascular blackening extend beyond affected veins to midrib, petiole and stem In advanced stages, infection may reach the roots system and blackening of vascular bundles occurs. Bacterial ooze can also be seen on affected parts
		If the infection is early, the plants wilt and die If the infection is late plants succumb to soft rot and die
Survival and spread	:	**PSI:** Bacterial cells **internally seed borne** and soil borne
		SSI: Bacterial cells dispersed through irrigation water and rain splashes
Favourable conditions	:	Relative humidity >90% High soil moisture Frequent rains

Management	:	☙ Hot water treatment at 50^0C for 30 min, for killing seed borne inoculum followed by a 30 min dip in streptocycline 100 ppm ☙ Spray agrimycin or streptocycline 100 ppm at transplanting, curd formation and pod formation ☙ Crop rotation for 2-3 years with non cruciferous crops ☙ Drenching seed bed with antibiotic solution in nursery beds ☙ **Resistant varieties:** **Cabbage**: Cabaret, Defender, Gladiator, Pusa Muktha **Cauliflower**: Pusa ice, Pusa snow ball-K-I-F,

Comparison:

Features	**Powdery mildew**	**Downy mildew**
Description	A powdery growth on the plant surface, often on leaves	A downy growth with angular spots on a plant surface. The color of spores is yellow to light brown and growth of the spores is restricted by vein.
Types of symptoms	Necrosis	Chlorosis
Favourable climatic conditions	Cold weather or cool dry weather	Monsoon or cool wet weather
Leaf part affected	Mostly the powdery growth appears on the both sides of leaves	Mostly downy growth appears on the lower sides of the leaves
Type of mildew	True mildew	False mildew
Mycelium	Septate	Aseptate
Sexual spore	Ascospore enclosed in cleistothecium	Oospore
Sexual organs	Antheridium and Ascogonium	Antheridium and Oogonium
Fungal growth composition	Conidiophore and conidia	Sporangiophore and sporangia
Sporangiophore branching	Absent	Present
Genera	*Erysiphe, Uncinula, Podosphaera, Phyllactinia, Leveillula*	*Perenospora, Bremia*

Features	**Smut**	**Rust**
Perfect spore	Intercalary	Terminal
Sporidial number	Indefinite	Definite (4)
Mechanism of discharge	Passive	Active
Basidiocarp	Rare	Absent
Alternation of generation	Indistinct	Distinct
Parasitism	Facultative	Obligate (Biotrophs)
Clamp connection	Common	Rare
Sex organs	Absent	Specialized
Heteroecism	Absent	Common
Polymorphism	Absent	Distinct

(Three types of wheat rust)

Leaf rust Stem rust Stripe rust

(16) Wheat

Black or stem rust

C.O.: *Puccinia graminis tritici*

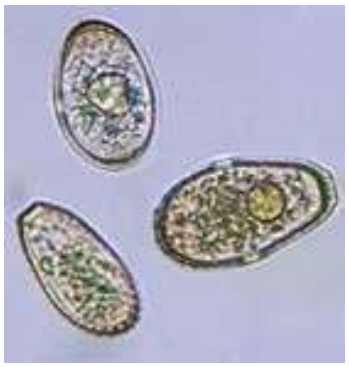
Economic importance

- The most important and destructive disease throughout the world where ever wheat is grown. The rust epidemics of 1946-47 in M.P., Maharashtra, Rajasthan and U.P destroyed over two million tonnes of grain. In 1956-57, rust was severe in W.B., Bihar and Eastern parts of U.P. causing heavy damage and rendered the grain in some tracts unfit to harvest.
- In India, though black stem rust is prevalent in all parts of the country it normally appears in epidemic form only in central, southern and eastern parts of the country where high temperatures prevailed during crop season.
- In Northern India, the disease usually appears during March when the crop is reaching maturity causing only a limited loss to the grain yield, whereas, in Southern parts, it appears during Nov to Dec causing severe losses. Barley is also susceptible to this rust.
- The fungus is obligate parasite living only on host plant.
- In India, 26 races (biotypes) have been reported.
- Races 11, 15C, 34-4 & 122 are most predominant and virulent.
- New Race from Uganda reported recently- **Ug99**

Symptoms

- The first symptom of rust infection is **flecking** of leaves, leaf sheaths, culms and floral structures.
- These flecks soon develop as oblong, **reddish brown uredo-pustules**, frequently merging into one another, finally bursting to expose a mass of brown uredospores.
- When large number of uredosori burst and release their spores, the entire leaf blade and other affected parts will have a brownish appearance even from a distance.
- Later in the season, **teleutosori** are produced. They are conspicuous, linear or oblong, dark brown to black, and often merging with one another, to cause linear patches of black lesions, which account for the name black rust. On maturity, the teleutosori burst open, exposing masses of dark brown teleutospores.
- In the transitional stage, there is a mosaic of brown and black masses of spores on the affected tissues, which dry up prematurely.
- Moreover, in the case of severe infections the diseased plants are stunted and produce small spikes and shrivelled grains, or no grain at all.

Stem rust



Uredospores

Teliospores

Promycelium

Pathogen

- Black stem rust is heteroecious full cycle rust.
- It requires more than one host species to complete its life cycle. The uredial and telial stages occur on wheat, barley and some grasses and the pycnial and aecial stages on the species of *Berberis* (Barbery) and *Mahonia*, the alternate hosts.
- The uredospores are brown, oval shaped, thick walled and marked with thin short spines and borne singly on stalks. The teleutospores are dark or chestnut brown, two celled, germinating by producing thin walled, hyaline four-celled promycelium (basidium).
- The fungus is highly specialized and has number of physiological races (over 250). Races 11, 15c, 34-A and 122 are most predominant appearing in virulent form in wheat growing tracts of India.

Disease cycle

- Primary infection is mainly through barberry, *i.e. Barberis vulgaris*. These barberry plants play a role in USA, Europe and Australia, where as in India they are not known to play any role in the perpetuation of the fungus.
- The source of inoculum for black rust comes from south, *i.e.,* Nilgiri and Pulney hills. In plains of North India during summer months, the uredospores cannot survive because of the high temperatures.
- The possibilities of the fungus surviving on ratoon tillers or self-sown wheat plants, late and off season wheat crops and certain grasses growing in cool areas particularly in the foothills of Himalayas in the North, the Nilgiris and Pulney hills in the South appear to be great.
- The grasses, *viz*., *Briza minor, Bromus patula, Brachipodium sylvaticum* and *Avena fatua*, harbor the fungus in the off-season. It is believed that the fungus over summers on the wheat plants and grasses in the hilly areas and spreads to the plains in the main wheat crop season. In the central Nepal, the wheat crop sown in August and harvested in December, January becomes infected by *P. graminis tritici* from October. This may be a source of inoculum for the main crop sown in the plains, which becomes infected from February each year.

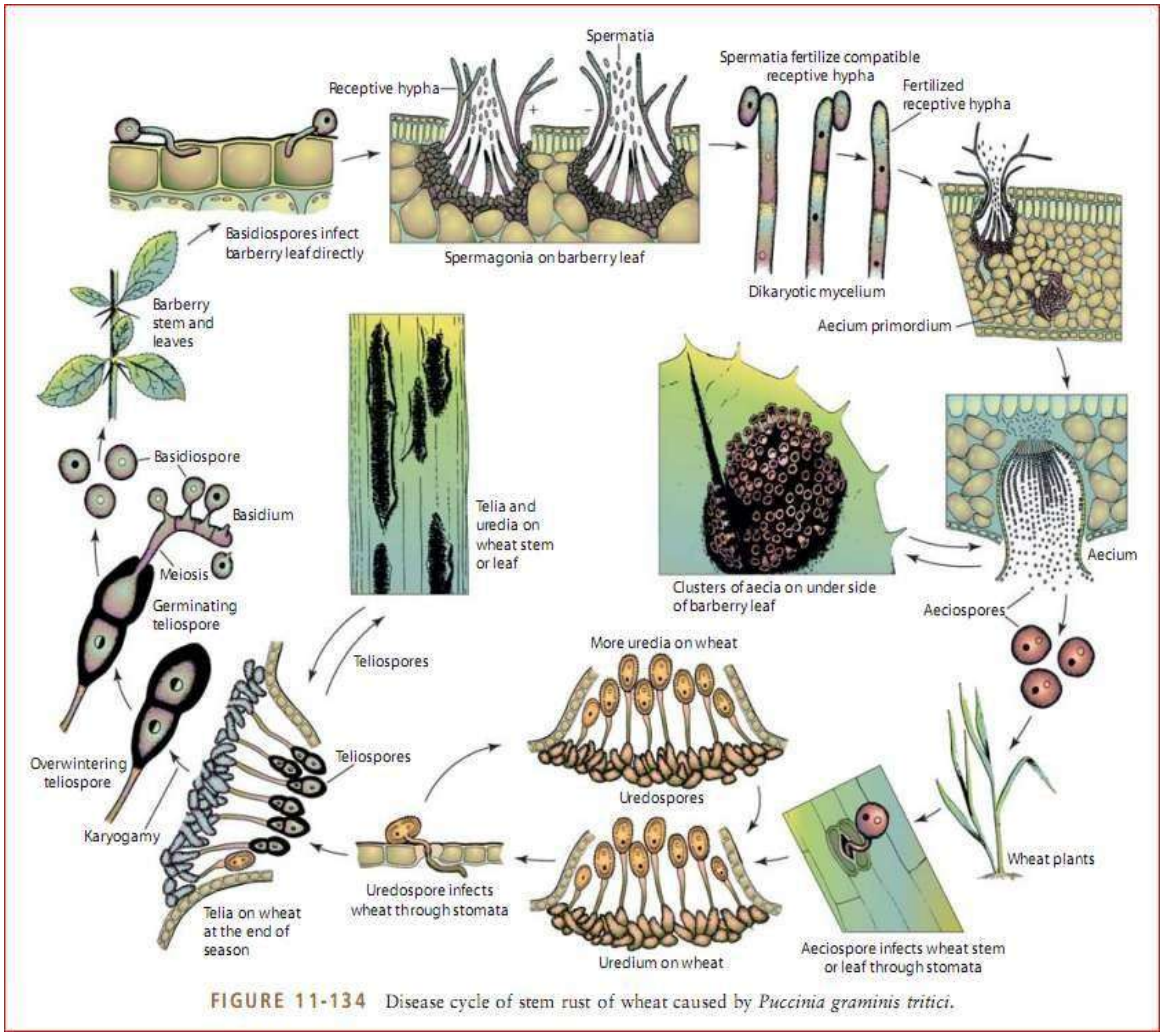


FIGURE 11-134 Disease cycle of stem rust of wheat caused by *Puccinia graminis tritici*.

Uredospores:

- Brown, oval shaped, thick walled marked with thin short spines.
- They borne singly on stalk and germinate in water by germ tubes.
- Upon infection, the fungus produced uredosori, which release the uredospores.
  - As the disease advance, same mycelium in host produces teleutospores either in uredosori or in teleutosori.

Teleutospores:

- Dark, chestnut brown, two celled, thick walled, smooth, rounded or pointed at the apex.
  - They germinate by producing a long, four-celled promycelium called basidium on which four basidiospores are produced.
- Germinate by germ tube and infect the alternate host barberry.
- Later on produced pycnia mostly on upper side of leaf surface.

Pycnia:

- Flask shaped with narrow opening produce numerous pycniosporophores.
- At the tip of pycniosporophores small thin walled pycniospore (spermatia) is produced.
  - Pycniospore push out and mixed with honey like fluid which attract insects and that transfer to receptive hyphae projecting from pycnia.
  - Both fuse leading to fertilization and diplodization called spermatization followed by formation of aecial primordia on lower leaf surface.

Aecia:

- Cup like aecia arise from diploid aecial primordia.
- Form the base of aecium numerous aeciospores are produced in chain.
    - They are capable of infecting host wheat, barley and grasses leading to production of uredosori; the repeating stage.
    - The possibilities of the fungus to survive on self sown wheat, certain grasses growing in cool regions particularly at altitudes upto 2000 mt. in Himalaya or in Nilgiris Hills.

Management

- Eradication of self sown wheat plants and weed hosts
- Adjusting sowing date, to escape the crop from disease
    - Grow resistant varieties like Kalyanasona, Sonalika, Choti Lerma, Lerma Rojo, Safed Lerma, NP 700 and NP 800.
- Avoid late sowing
- Balanced application of nitrogenous fertilizers
    - Seed dressing with oxycarboxin (Plantavax) @ 0.1% followed by two sprays with the same chemical
    - Spray twice or thrice with zineb @ 0.25% or mancozeb @ 0.25% or oxycarboxin @ 0.1%, at 15 days interval.

1. Leaf, brown or orange rust

C.O.: *Puccinia recondita*

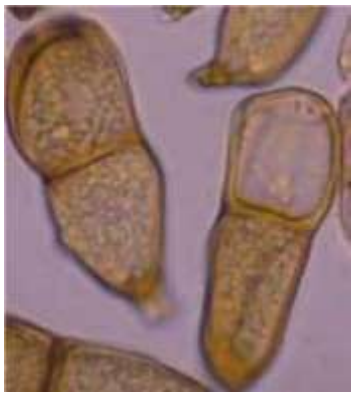
Economic importance

- In India, it is the most common rust in the northern and eastern parts. In Punjab, Bihar and UP, it causes more damage than stem rust.
- In South India, it is found in the crops grown both in the hills and in the plains. 10% yield losses are reported.

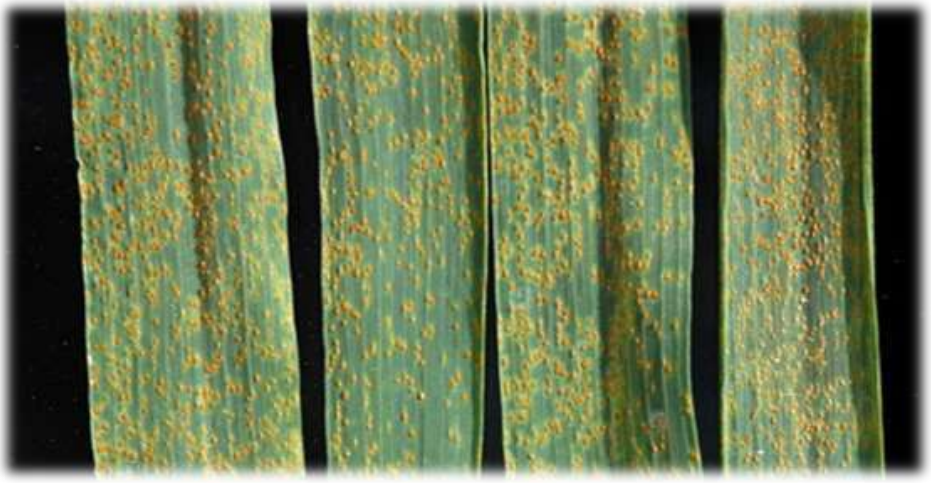
Symptoms

- The first symptom of the disease is the appearance of minute, round, **orange sori**, irregularly distributed on the leaves, **rarely on the leaf sheath and stem**.
- The sori turn brown with maturity. As the disease advances, the telial stage may be found in the same pustule. The telia are small, oval to linear, black and covered by the epidermis. The telia are also found on the leaf sheath. Severe rusting of leaves causes reduction in yield.

Brown rust



Teliospores



Pathogen

- The fungus, *Puccinia recondita*, is **heteroecious**. The uredial and telial stages appear on wheat and some other grasses and **aecial and pycnial stages on species of *Thalictrum*.**
- In India, the role of *Thalictrum javanicum* and *T. flavum* as alternate hosts has not been precisely determined. In Russia, *Isopyrum fumaroides* is known to act as a natural alternate host.
- The uredospores are brown, spherical and minutely echinulate with 7-10 germ pores. Telia are rare, but when formed are found mostly on the lower surface of the leaf and do not rupture. Teleutospores are smooth, oblong, thick walled and brown with a rounded and a prominent thickened apex.

Disease cycle

- Alternate host, species of *Thalictrum*, helps the fungus to over-summer in other countries. The role of *Thalictrum* is not clear in India.
- In early January, the rust gets well established in the foothills of Himalayas and also in the plains of Tamil Nadu and Karnataka in the South. The first build up of inoculum takes place in the plains of Karnataka and moves northwards to Maharashtra and Madhya Pradesh.

- The inoculum from the foot hills of Bihar and UP moves to the Northern plains. Therefore the brown rust appears slightly later in the Western hills of North India. The rust population of the north and the south moves in opposite directions, finally merging into each other, and causes serious disease in the wheat growing states.

Management

- Same as stem rust

2. Yellow or stripe rust

C.O.: *Puccinia striformis*

Economic importance

- It is confined to the cooler parts particularly the foothills of Himalayas, Punjab, Himachal Pradesh, Haryana, U.P., and parts of Rajasthan and Bihar.
- It is totally absent from South India except in Nilgiris and Pulney hills. It appears every year, but the damage is seen only in occasional years.
- Sonara-64 is susceptible to yellow rust.

Symptoms

- The uredosori appear as **bright yellow pustules** chiefly on the leaves. But in severe infections they may be seen on leaf sheaths also.
- The sori are elongated and are arranged in **linear rows** between the veins of the leaf and hence it is referred as stripe rust. The sori are mostly sub-epidermal and are remained covered by the epidermal layer and break only at the time of crop maturity.
- The teleutosori appear late in the season and are also arranged in linear rows. They are compact, elongated, and black which remain sub-epidermal. They do not break through epidermis for a long time remaining as black crust.

Yellow rust

Teliospores

Pathogen

- Uredospores are yellow, spherical to oval with a spiny wall.
- The teleutospores are dark brown, two celled, thick walled and **flattened at the top**. The teleutosori are filled with numerous unicellular, brown lengthy paraphyses.

Disease cycle

- The fungus overwinters in its uredial stage in England and other countries. Its persistence in India is not known. It may overwinter on volunteer wheat plants at an altitude of about 1500 to 1800 meters in the Himalayas.
- The uredospores germinate after a period of dormancy and form a source of inoculum for early sown wheat crop. In U.P., early sown crop is severely infected by the fungus than the late sown crop.
- Some weeds like *Agropyron semicostatum, Bromus catharaticus, Bromus japonicus* and *Hordeum murinum* also serve as primary source of inoculum. Secondary infection is by wind borne uredospores.
- There are about 40 races in the world including 13, 14, 19, 20, 24 and 31 A which are widespread in India.

Management

- Same as stem rust

3. Loose smut

C.O.: *Ustilago nuda tritici*

Economic importance

- Loose smut is one of the major diseases on wheat. There was loose smut epidemic during 1970-75 in Punjab, Haryana and Western U.P.
- In Sonalika, the incidence was 5 to 6%. Incidence is more in North than in

Southern parts of India. Countrywide loss is about 23% in yield.

Symptoms

- The symptoms are evident only at the time of emergence of the panicle from boot leaf.

  All the spikelets in a panicle transform into a **mass of black powdery spores**.
- The infected panicle emerges two days earlier than healthy and the spores are covered with the silvery membrane. This thin membrane gets ruptured exposing the mass of black spores. The spores are easily blown by wind leaving the bare rachis.

Pathogen

- Chlamydospores of the fungus are pale, olive brown, spherical to oval in shape. These smut spores germinate and produce promycelium or sporidium.
- The promycelial cells fuse and give rise to germ tubes that enter the ovary through the stigma and become established in the embryo remaining dormant until seed germination.

Disease cycle

- It is **internally** and **externally seed borne** and is systemic.
- The fungus is carried over in the seed as dormant mycelium. When the planted seed germinates the mycelium becomes active. It grows along with the plant and when the panicle is produced the mycelium reaches the ovaries and transforms the ovaries into a mass of black smut spores.
- Secondary spread occurs through wind borne smut spores. The sporidia infect the healthy flowers. The mycelium enters the ovary and remains in the seed as dormant mycelium.

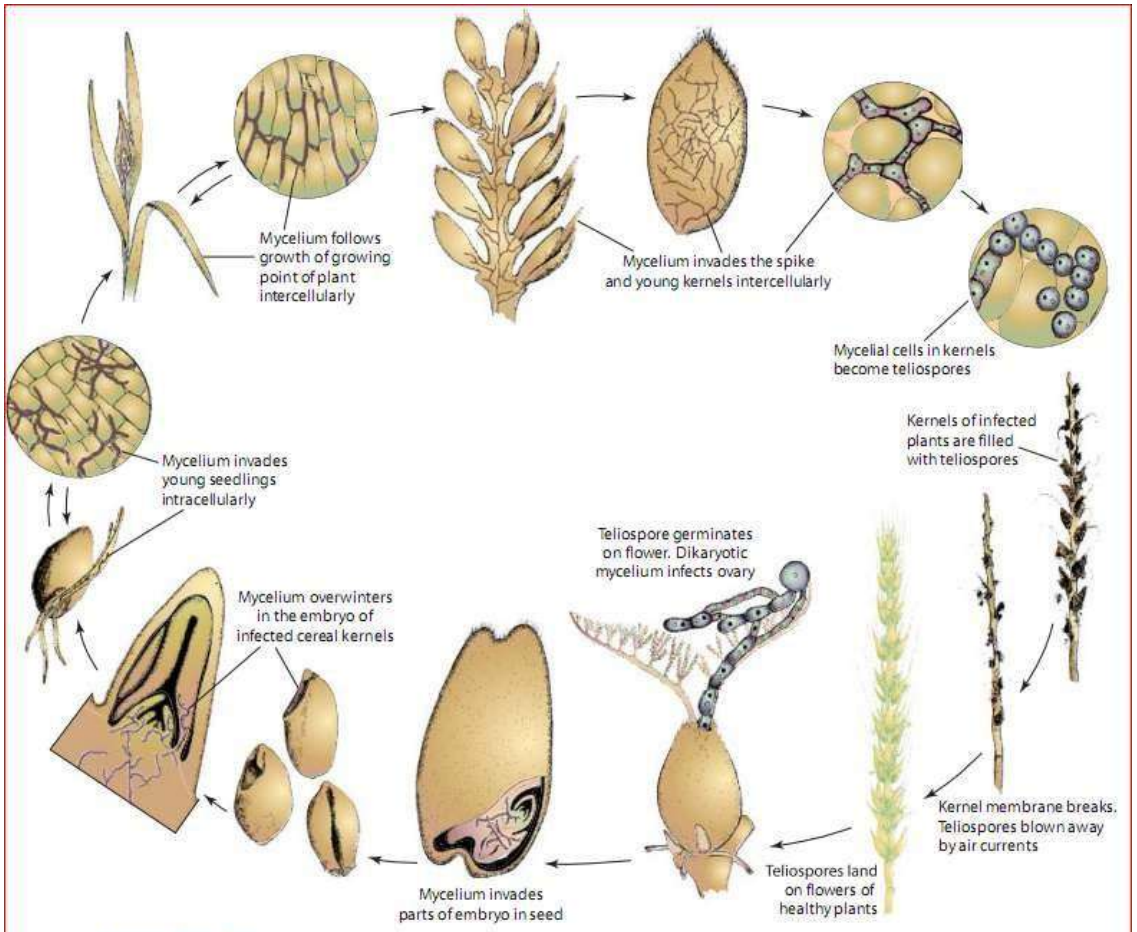


FIGURE 11-147 Disease cycle of loose smuts of barley and wheat caused by *Ustilago nuda* and *U. tritici*.

Management

- Grow resistant varieties Kalyanasona, PV 18, WG 307 and HD 450.
- ***Hot water treatment*** (**Jensen**, 1908): Soak the seed in cold water for 4 hours and then immerse the seed in hot water at a temperature of 132 ^{0}F or 52^{0}C for about 10 minutes. Dry the seed in shade before sowing.
- ***Solar seed treatment*** (**Luthra and Sattar**, 1934): Soak the seed in water for 4 hours (8 AM to 12 Noon) and expose the seed to the hot sun for 4 to 5 hours (from 12 Noon to 5 PM) on cement or rocky surface. This can be practiced in the areas where the summer temperatures are high (42-44^{0}C)
- ***Anaerobic seed treatment*** (USA): Soak the seeds for 2-4 hours in water between 6070^{0}F and keep the moist seeds in airtight containers for 65-70 hours and there after dry the seed.
- Seed treatment with systemic chemicals like carboxin (vitavax) @ 0.2% or Benlate @ 0.2%

Karnal Bunt

There are three bunts:

Stinking bunt: *Tilletia caries*

Dwarf / Hill bunt: *Tilletia foetida*

Karnal bunt: *Neovossia indica* (formerly *Tilletia indica*)

- Karnal bunt disease was first reported in India from **Karnal** (Haryana) by **Mitra in 1931**, named after the town Karnal of India, affects wheat and triticale (a hybrid of wheat and rye) on which it causes a covered smut.
- Karnal bunt is caused by the fungus *Neovossia indica* (formerly *Tilletia indica*).
- Karnal bunt is also known as **partial bunt** because only a portion of the wheat kernels is affected. The disease has now been reported from several other countries in Asia, in the early 1980s from Mexico, and, since 1996, from a few places in the south-western United States.
- A great effort was launched in the United States to determine the extent of the area to which the Karnal bunt fungus had spread.
- The disease was less severe till 1970's, however it assumed greater importance in early seventies with the adoption of high yielding, semi dwarf nutrient responsive varieties. The disease appeared in epidemic form in different parts of India in 1976, 1979, 1981- 83 and 1986.

Symptoms:

- The infection is usually confined to a **few grains** in the spike with irregular arrangement.
- In some cases the infection may spread to only a part of the grains. In severe cases, the grain is reduced to black shiny sac of teliospores. As the grains mature the outer glumes spread and the inner glumes expand, exposing the bunted grains.
- The bunt balls are first enclosed by the pericarp but when it bursts the masses of bunt spores are exposed.
- The bunt affected plants emits a foul smell which is mainly due to the presence of

Trimethyl amine.

Bunted grains

Teliospore

Allantoid sporidia

Teliospore of *Tilletia indica*

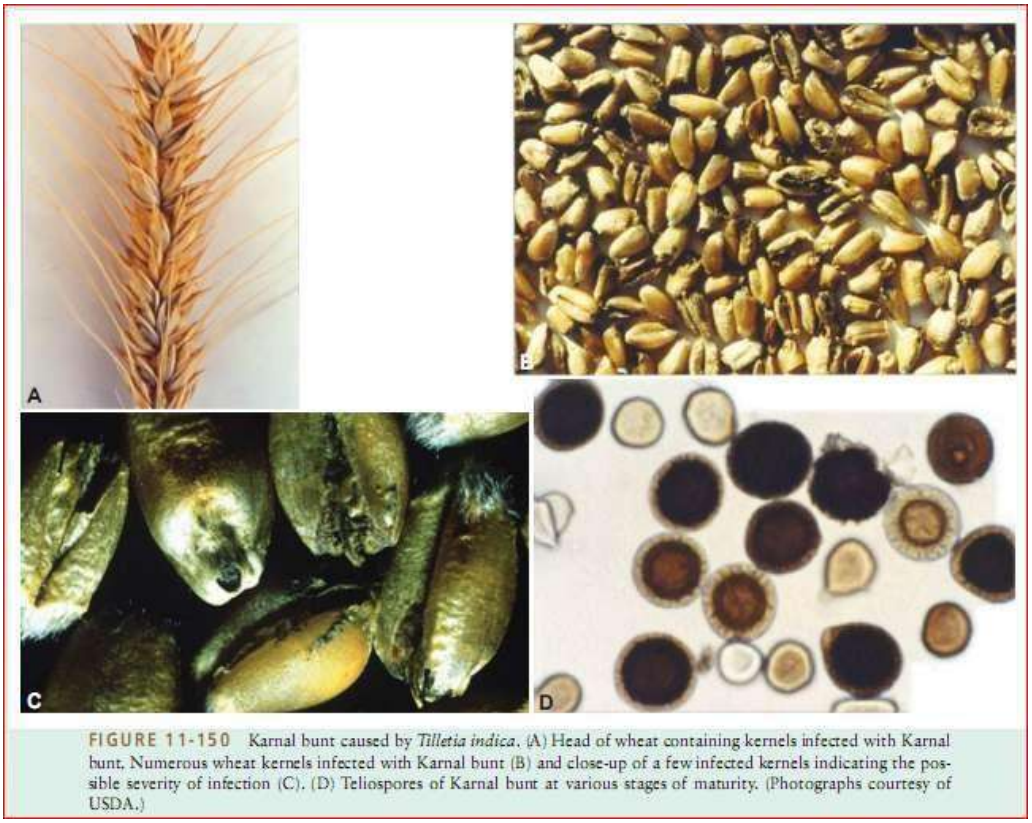


FIGURE 11-150 Karnal bunt caused by *Tilletia indica*. (A) Head of wheat containing kernels infected with Karnal bunt, Numerous wheat kernels infected with Karnal bunt (B) and close-up of a few infected kernels indicating the possible severity of infection (C). (D) Teliospores of Karnal bunt at various stages of maturity. (Photographs courtesy of USDA.)

Pathogen

- Teliospores are smooth walled measuring 22-49µ in diameter and require a long resting period. Teliospores germinate and produce a large number (60-120) of **needle shaped** primary sporidia on a short stout basidium.
- Later, sickle shaped (**rimordial**) secondary sporidia are produced which help in the dispersal of Karnal bunt.

Disease cycle

- The teliospores in soil germinate producing primary sporidia. The sporidia become air borne and deposit on leaves of host plants. Under high humid conditions, they produce a secondary crop of secondary sporidia (allantoids).
- If boot emergence stage coincides with drizzle, the secondary sporidia get washed down to sheath. The sporidia germinate on glumes to enter epidermal cells to penetrate ovary. The sporidial germ tubes penetrate stomata in rachis, glumes, lemma and palea.
- The disease progresses systemically to other florets within an infected spikelet. The infection mostly starts from the embryonal end and spreads **along the grain suture**.
- The hyphae grow through the base of the glumes into sub-ovarian tissue and enter pericarp through funiculus. Hyphae of the pathogen proliferate, remain restricted to pericarp and produce teliospores terminally. In severe cases, grain is reduced to black shiny sack of teliospores.
- The embryo and endosperm are not colonized. The pericarp ruptures during threshing and teliospores deposit in soil and adhere to the surface of the seed.
    - **PSI**: Smut spore germinate in the soil producing a large number of needle shaped primary sporidia (rimordial) and sickle shape secondary sporidia.
    - **SSI**: Air borne secondary sporidia infect ovary.

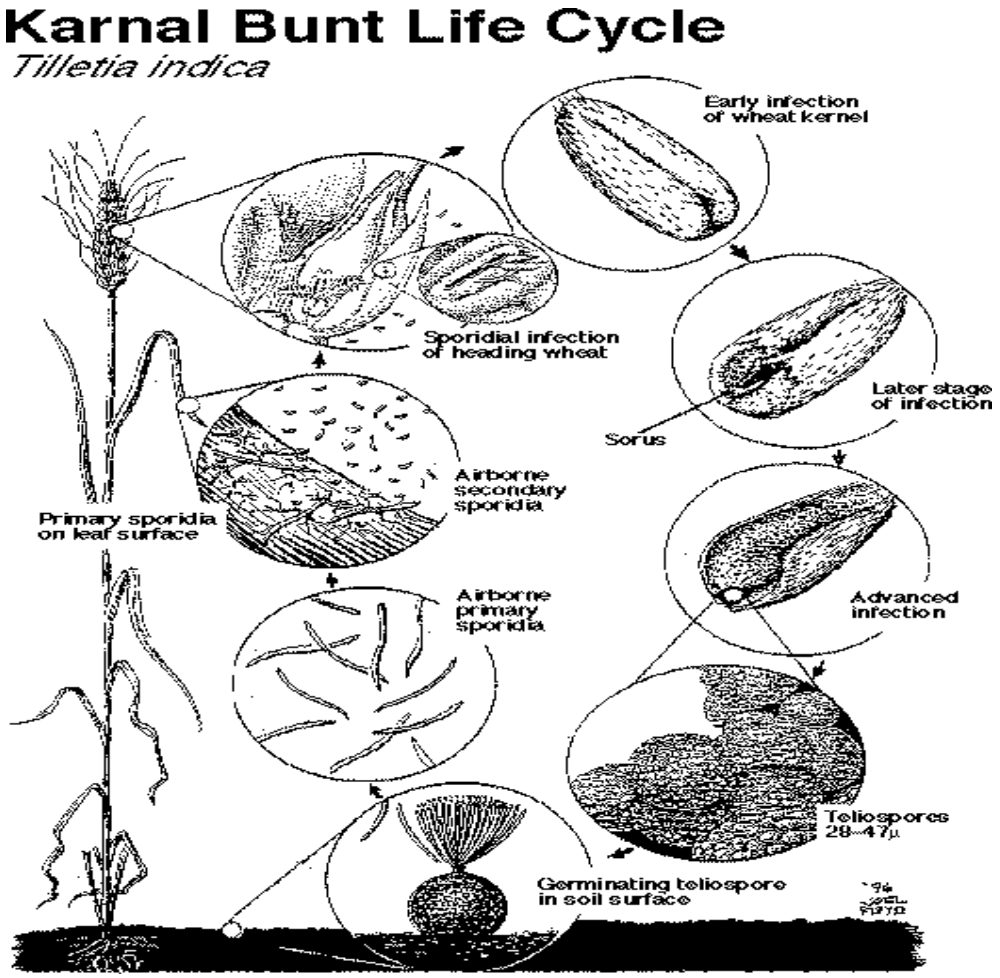
Favourable conditions

- Moderate temperatures (19-23^{0}C)
- High humidity (>70%) and cloudiness or rainfall during anthesis favours disease development in susceptible host varieties.

Management

- Grow tolerant varieties, viz., WL 1562, HD 2281, etc. Use resistant sources like wild species of *Aegilops* and *Triticum*, HD 2329, HD 29 and HD 20 for breeding programme.
- Follow strict quarantine measures
- Use disease free seed for sowing

- Judicious application of nitrogenous fertilizers
- Adjust date of sowing
- Intercropping with Gram or Lentil
- Seed treatment with carboxin, thiophanate methyl, triadimephon @ 2 g/kg of seeds.
- Spray with carbendazim @ 0.1% or carboxin @ 0.2% or mancozeb @ 0.25%



Three bunt of wheat

Character	Rough-spored bunt/ stinking smut	Smooth spored bunt	Karnal bunt
Pathogen	*Tilletia caries*	*Tilletia foetida*	*Neovossia indica*
Spores	Reticulate, rough walled, measuring 15-20 µ in diameter	Reticulate, smooth walled, measuring 15-25 µ in diameter	Smooth walled, measuring 22-49 µ in diameter
Germination	No resting period. Produce primary sporidia, which unite to form H shaped structures	No resting period. Produce primary sporidia, which unite to form H shaped structures	Requires a long resting period. Sporidia produce in large numbers (60-120) on short, stout basidium. The primary sporidia are needle shaped and flexible, like *T. caries* and *T. foetida*. The secondary sporidia are sickle shaped.
Spread	Externally seed borne, usually infecting all the spikelets in an ear	Externally seed borne, usually infecting all the spikelets in an ear	Soil borne and air borne, usually affecting only a few spikelets in an ear
Physiological races	Heterothallic with 20 races found	Heterothallic with 10 races found	No races reported and sexuality not studied

MINOR DISEASES:

Economic importance:

Alternaria blight/ black point

C.O.: *Alternaria triticina*

Reported by **Prasad** and **Prabhu** in 1962 from India. It is prevalent in parts of Maharashtra, Bihar, West Bengal and UP. Seedlings are not prone to infection.

Symptoms

- The disease appears in the field when the crop is 7-8 weeks old.
- Reddish brown oval spots appear on young seedlings with bright yellow margin.
- In severe cases, several spots coalesce to cause drying of leaves. The young leaves are not usually infected.
- Heavily infected fields display a burnt appearance even from a distance. In some varieties reduction in grain yield is as high as 90% if the infection takes place at or before the boot leaf stage.

Pathogen

- Fungus produces light brown coloured multicellular conidia, with 1-10 transverse septa and 1-5 longitudinal septa, singly or in chains (2-4).

Disease cycle

- Pathogen over summers in plant debris and soil. Primary spread is by **externally** and **internally** seed-borne conidia.
- Secondary infection is mainly through wind-borne conidia.

Favourable conditions

- Temperature of 25 ^{0}C and high relative humidity favours the disease.

Management

- Soak the seeds in water for 4 hrs followed by 10 min. dip in hot water at 52^{0}C.
- Grow resistant varieties like Co.25, Sonalika, Arnautka, E6160 and K7340.
- Spray the crop with mancozeb @ 0.25% or zineb @ 0.25%

Ear Cockle/ Tundu disease/ yellow slime disease

C.O.: *Anguina tritici* (Nematode) + *Corynebacterium tritici* or *Clavibacter tritici* (Bacteria)

Economic importance

- This disease has been reported from Punjab, Haryana, U.P. and Rajasthan
- First reported and discovered by Needham, 1743.

Symptoms

- The tundu disease is characterized by the twisting of the stem, distortion of the ear head and rotting of the spikelets with a **profuse oozing of yellow liquid** from the affected tissues.
- The ooze contains masses of bacterial cells.
- The nematode alone causes winkling, twisting and various other distortion of the leaves, stem and produce small round galls on the leaves.
- The infected plants are shorter and thicker than healthy plants. In the distorted earheads dark galls are found in place of kernels.
- When the bacterium is associated with the nematode, the disease symptoms are intensified at the flowering stage and yellow ear rot sets in due to combined action of the nematode and bacterium.
- The earhead becomes chaffy and the kernels are replaced by dark nematode galls which also contain the bacterium.
- The infected plants produce more tillers than the healthy ones.
- Another interesting feature is the **early emergence of ears** in the nematode infected plants which is about 30 to 40 days earlier than the healthy ones.

Pathogen

- *Corynebacterium* is rod shaped, Gram positive and is motile by single polar flagellum.

Disease cycle

- The disease starts from the seeds contaminated with the nematode galls.
- When such contaminated seeds are sown in the field, they absorb moisture from the soil and the larvae (juveniles) escape from the galls and climb upon the young wheat plants.
- At the time of flowering, the nematodes enter the floral parts and form galls in the ovaries. When once the nematode is inside the tissues of the ovary, the bacterium becomes active and causes rotting.
- The yellow ooze coming out of the rotting earhead provides the inoculum for the secondary spread of the disease which is favoured by wind and rain.
- The nematode probably functions as a vector transporting the bacterium to otherwise inaccessible meristematic regions of the host.
- The nematodes secrete some substances in the presence of the host bacterium,

which can remain viable for at least 5 years in the galls of *A. tritici*.

- The nematode galls are reported to remain in the soil for 20 years or more and the bacterium can also survive for the same period inside the nematode gall.

Management

- Sow gall free seeds.
- Seed soaking in brine solution (14.5 kg salt / 450 lit of water) to separate out the galls.
- Wheat, barley or oat should not be sown in the infested soil.
- Spray the crop with streptocycline, 1g in 10 liters of water.

(17) DISEASES OF SUGARCANE

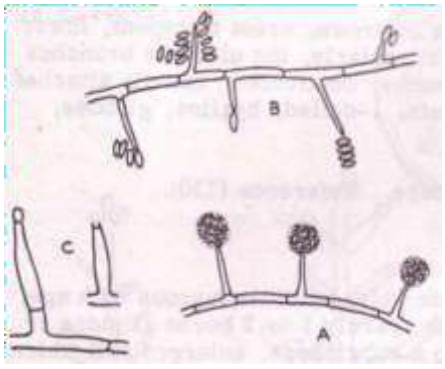
1. Wilt

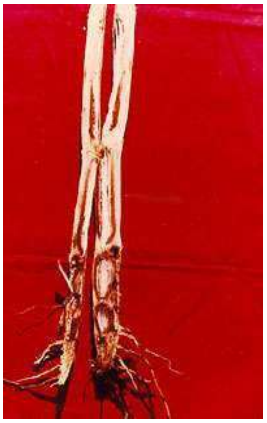
C.O. : *Fusarium moniliforme* var. *subglutina*

It was first reported by Butler and Khun (1913) from India. In Gujarat it was first reported during 1985 in highly susceptible variety COC-671. The yield loss is 10-80%.

Symptoms:

- Typical wilt symptoms appear during the monsoon periods.
  - The affected plants, either singly or in small groups displays a conspicuous stunting and unthrifty appearance.
- The yellowing or withering of crown leaves.
- The mid rib of leaves turn yellow while leaf lamina remains green.
  - On splitting open the canes, at early stages of infection, diffused purple or muddy red colour is seen at each node.
- These canes emit a characteristic disagreeable smell.
  - Luxurious fungal growth at nodal portion in cavity may also produced roots may also get blackened.





Favourable conditions:

- Ill drained soils.
- Heavy irrigation.
- Water logging and water stress conditions.
- High soil pH (<7.0)
- More N_2 fertilizers.

- Monoculturing especially with single susceptible genotype.
- Imbalance use of nutrients.
- No crop rotation.
- More ratooning.
- More root borer and nematode damage increases the disease.
- High C: N ratio.
- Pre monsoon drought followed by excessive water.
- Use of diseased setts leads to more infection.

Disease cycle:

- Primary source of inoculum: chlamydospores & infected seed setts (sett borne).
- The fungus can also survive in soil (soil borne) as a saprophyte for 2-3 years.
- Secondary spread of macro, micro conidia is through water or soil contamination.

Management:

- Deep ploughing during summer.
- Selection of healthy setts.
    - Seed treatment with MHAT 54^0C /4 hrs or AST 52^0C / 4 hrs. and then steeping in carbendazim 0.1 % solution for 5-10 minutes.
    - Roughing -Infected clump should be uprooted, and burnt and drench that portion with carbendazim 0.1 % solution.
- Avoid ratooning; do not select the setts from ratoon crop.
- Facilitate the field with proper drainage.
- Avoid water logging or water stress conditions.
- Judicious use of irrigation and fertilizer.
    - Crop rotation-sugarcane-paddy (summer) + paddy (kharif) + Green manuring with Dhaincha (kharif end) – followed by sugarcane.
- Mulching with sugarcane trash.
- Use organic manure- castor, neem, poultry cakes, FYM or Press mud.
- Do not allow water from infected field to healthy fields.
- Resistant variety-C0-8338, CoN- 91 132, CoLk- 8001.
    - Apply *Trichoderma viride* or *T. harzianum* or *T. longibrachyatum* multiplied in press mud @ 8 t/h.
    - Application of carbofuran @ 1.5 kg.a.i./ ha. (50 kg furadan) first 30 days after planting, second at the time of earthing up to manage borer/ nematodes.

2. Red rot

C. O.: *Colletotrichum falcatum*

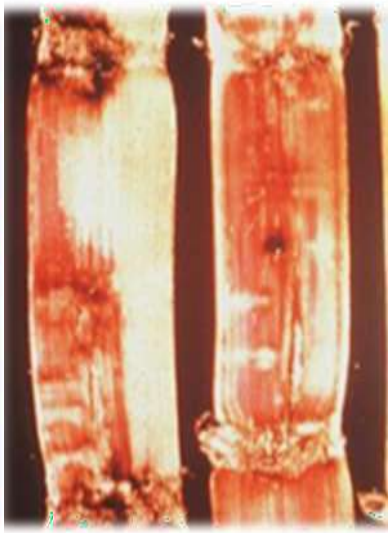
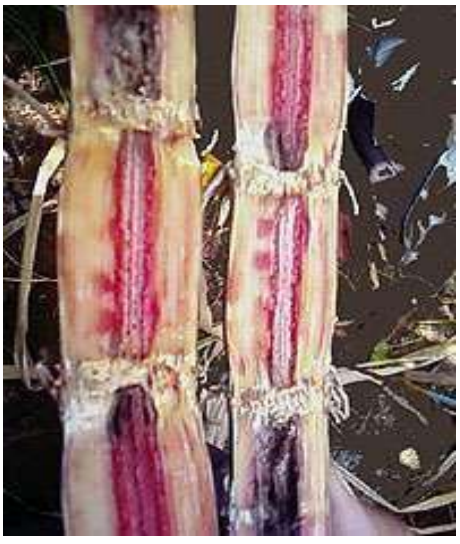
Per. Stage: *Glomerella tucumanensis*

In India first reported by Butler (1906) while in Gujarat it was first reported from South Gujarat during 1993 in. The losses may range from 25-80% and sugar recovery may also reduce. Breeding for resistance work is carried out at Sugarcane Breeding Institute (SBI) at Coimbatore.

Symptoms:

- The first symptoms of red rot in the field are discoloration of the young leaves with reddening of midrib.

- The margins and tips of the leaves wither and the leaves droop.
  - The discoloration and withering continues from tip to the leaf base until the whole crown withers and the plant dies, within 4 to 8 days.
- In a single stool, most of the stalks may wither almost simultaneously.
- The discoloration may also found on cane rind.
  - Ultimately this leads to cane rotting. When a diseased plant is open, it emits a characteristic sour odor.
  - The tissues are reddened through out the basal portion, specially the vascular bundles, which are intensely red.
- As the disease advances the entire stem rots and the central tissues become pithy.
- The internodes may shrink.
  - Often a profuse whitish growth of fungal mycelium may be found in the centre and pithy tissues.
  - In some cases, black, minute bodies, representing the acervuli of the fungus, can be seen. On leaf lamina circular brownish lesion with red margin may also produced in certain variety.
- This disease may also found in association with wilt disease causing severe losses.

Favourable conditions:

- High humidity.
- Water logged conditions.
- Ill drained soil.
- Lack of proper cultural operations.
- Monoculturing and ratooning lead to the epidemics.

Disease cycle:

- The planting material i.e. the setts, may harbor the fungus and thus perpetuate the disease from season to season.
- The fungus may also persist in the soil on diseased clumps dry leaves left behind.
- The primary infection, however appears to be mainly from infected setts.
- In soil the fungus can survive as conidia or chlamydospores up to the six month.
    - Secondary spread in the field may be through irrigation water, raindrops and cultivation tools / implements and wind-borne inoculum.
- Stem Infection may take place through insect borer.
    - Soil borne fungus may also enter the healthy setts through cut ends, and cause early infection of the shoots.

Management:

- Deep ploughing during summer.
    - Selection of healthy setts-preferably from ten months old crops having no disease symptoms.
    - Thermotherapy: - Aerated Steam Treatment (AST) at 50^0C for 2 hrs. and then sett steeping in carbendazim (bavistin 0.2%) solution for 10 minutes. OR Moist Hot Air Treatment (MHAT) at 54 °C for 4 hrs. and above chemical steeping.
    - **Chemotherapy**: when there is no facility of thermotherapy plant, then setts can directly treated with the above chemical solution.
- Avoid water-logging conditions.
    - Judicious use of irrigation and fertilizers. More N_2 fertilizers especially urea (more then 250 kg) and flood irrigation leads to more incidence.
- Follow crop rotation: crop rotation with paddy in south Gujarat.
- Inter crop with coriander, soybean, and mustard found effective.
- Avoid ratooning.
- Do not select the seed from ratoon crop.
- Do not allow water form infected field to the healthy.
- Resistant varieties Co-6806, Co-87263, CON 1132, COLK 8001, Co-8338
    - Incorporate bio-control agents i.e. *Trichoderma viride* or *Gliocladium virens* multiplied in pressmud @ 8 t/ha.

- Apply organic manures: castor cake, neem cake, poultry manures *etc.*
- Green manuring with dhaincha.

3. Whip smut/ culmiculous smut

C.O.: *Ustilago scitaminea*

Among the 102 sugarcane-growing countries, only Australia is free from this disease. In Gujarat, it is a major problem in Saurashtra region, where CO-7527, CO-791 and CO-419.

The fungus mycelium is present in abundance in the infected canes. The fungus produce chlamydospores, they germinate and produce sporidia, which infect the plants.

Symptoms:

- This is most recognizable disease.
  - The affected plants are stunted and central shoot is replaced by a long whip like, dusty black structure, several feet in length.
  - Excessive tillering and thin canes. In early stages, the structure is covered by a thin silvery white film, which rapture, exposing the mass of dense, black dust containing smut spores.
  - The whip like structure, representing the central shoot with its various leaves, may be produced by each one of the shoots arising from the clump.
  - Since the plants are greatly stunted and the black whips stand out prominently in the field, it is easily identified from a distance.

Disease cycle:

- Sugarcane crop has no off-season and the disease easily gets ready hosts.
  - The mode of transmission of the disease through the fungal mycelium contained in the setts taken from diseased clumps.
- The healthy setts can also become infected by soil- borne spores.

- The spores germinate in moist soil and the sporidia enter into the root primodia of the setts, through their germ tubes.

- In a ratoon crop, the fungus left over in the clumps becomes more active.
  - The fungus attacks other graminaceous plants *i.e*. S*accharum spontaneum*.

Favourable conditions:

- Optimum temp. for spore germination is about 25^0 to 30^0C, maximum being 36^0 to 40^0C.
- 100% RH is essential for spore germination.

Management:

- Removal of smutted whips from the infected field.
- Discourage the practices of rationing.
- Avoid planting infected setts: Selection of healthy seed from healthy field.
- Disinfection of sets before planting
- Chemotherapy: carbendazim 0.1% solution-dip setts for 10 min.
- Thermotherapy: Hot Water Treatment (HWT) 55^0 to 60^0 C, for 10 min. dip.
- Aerated Steam treatment (AST) 50^0C / 2 hrs.
- Moist Hot Air Treatment (MHAT) at 54^0C / 4 hrs.
- Crop rotation with non-host crop.
- Roughing before rupturing of silver membrane of whip and destroying them.
- Avoid planting of susceptible varieties and use resistant varieties CO-6806.
- Deep ploughing in summer and flooding the field will destroy soil inoculum.
- Do not take setts from infected field.

4. Grassy Shoot of sugarcane

C.O.: Mycoplasma like organism (MLO's)/ Phytoplasma

It is also known as yellowing or white leaf diseases (albinism) of sugarcane in India, Taiwan and Thailand. This is one the serious disease of sugarcane especially in Maharashtra and was first time reported during 1949. Threshold level for GSD is 15 % infection.

Symptoms:

- The disease shows different types of symptoms at different stages of plant growth and has been described as "Bunchy disease" or "leaf tuft".
- Profuse tillering.
- The most pronounced symptom is the grassy appearance.
- Shoots growing from diseased setts remain dwarf and stunted.
- The leaves are narrow and small, like grass leaves.
  - The canes are thin with short internodes, giving a bunchy or grassy appearance

to the clump.

- The leaves appear yellow and in some cases may be entirely devoid of any pigment (white leaf or albinism).

- In secondary infection, the new leaves and internodes show the above symptoms.
- Leaves exhibit straight, long, white or light green or yellowish streaks.
- The lower nodes produce large numbers of grassy shoots.
- In systemically infected canes the disease appearing in May-June.

Disease cycle:

- Primary source of infection: Sett borne.
  - Secondary source of infection: Transmitted by sap inoculation through aphid Longiunguis *sacchari*, *Melanapsis indosacchari* and *Aphis maidis* vectors.

Other hosts: Jowar, Napier grass, Madras grass.

Management:

- Selection of healthy sets for planting.
  - During early stages of the crop, protect the crop by controlling insect vectors- by spraying systemic insecticide (malathion 0.16 %).
- Infected clumps should be dug out and destroyed.
  - Thermotherapy to setts before planting: - Sett treatment -HWT 50^0C / 2 hrs. or HAT 54^0C / 8 hrs.
  - Chemotherapy to setts: - Tetracycline (250 ppm) applied to seed setts have been found effective to eliminate the organism.
- Use of the resistant variety.

MINOR DISEASES:

Mosaic

C.O.: Sugarcane Mosaic Virus (*Marmor sacchari*) It belongs to Potato Virus Y group.

Reported from almost all sugarcane tracks of India. In India, the loss caused has been estimated in the range of 10-20 %.

Symptoms:

- More commonly, elongated yellowish stripes alternate with the normal green portion of the leaf to give a mosaic appearance.
- In severe infections, the chlorotic area may be greater than the healthy.
  - The mosaic symptoms are more clearly apparent on the younger basal portion than on older leaves.
  - The same symptoms may also be seen on the leaf sheaths and stalks and in highly susceptible varieties, yellow stripes may be seen even on the canes.
  - Sometimes elongated necrotic lesions are produced on the stalks, in which cases stem splitting is common.
- The entire plant may become stunted and chlorotic, easily identified from a distance.

Disease cycle:

- Primary source: It spreads through infected setts.
    - Secondary spread through: aphids, *Rhopalosiphum maidis, Hysteroneura setariae* and by green bug *Toxoptera graminum.* The virus is non-persistent type in its vectors. The infection in plant is systemic.

Other hosts of the virus: sorghum, maize and wild grasses.

Management:

- Use of selected healthy setts for seed.
- Heat therapy (Hot water / Hot Air) is effective against certain strains and can be used.
- Systemic roughing of the infected canes if the incidence is not very high.
- Elimination of the grass hosts.
- Use resistant/ tolerant variety.

5. Ratoon stunting

C.O.: *Clavibacter xyli* pv. *xyli* (Xylem limited fastidious bacteria)

Economic importance

The disease is present throughout the sugarcane growing areas of our country. **CO 419** variety was worst affected in parts of Karnataka where it was withdrawn from cultivation. The disease appears in both plant and ratoon crop, but more pronounced in ratoon crop.

Symptoms

- Diseased clumps usually display **stunted** growth, reduced tillering, thin stalks with shortened internodes and yellowish foliage (**mild chlorosis**).
- When mature canes are split open, vascular bundles appear discoloured.
- In young canes, **pink colour** is seen in the form of minute pinhead like areas near the nodes.
- The disease reduces the length, girth and the number of canes per clump.

Pathogen

- The pathogen is known to present in the xylem cells of infected plants.

- They are small, thin, rod shaped or coryneform.

Disease cycle

- The disease spreads through use of diseased setts.
- The disease also spreads through cane harvesting implements contaminated with the juice of the diseased canes.
- Maize, sorghum, Sudan grass and *Cynodon* are some of the collateral hosts of the pathogen.

Management

- Select the setts from disease free field.
- Remove and burn the clumps showing the disease.
- Sterilization of cutting knives with lysol or any other antiseptic solution.
- Hot air treatment of setts at 54^0C for 8 hours or hot water treatment at 50^0C for 2 hrs or aerated steam treatment at 50^0C for 1 hour.

(18)DISEASES OF MUSTARD

1. White Rust

C.O.: *Albugo crucifearum*/ *Albugo candida*

Obligate parasite causing systematic infection resulting in malformation of the inflorescences.

Symptoms:

- Local as well as systemic infections are noticed.
  - Disease appear on leaves and characterized by the appearance of white creamy raised pustules on the under surface of leaves.
- Corresponding upper surface become yellow.
- Pustules ruptures and released chalky dust of spores (sporangia).
- Systemic infection causes hypertrophy and hyperplasia on inflorescences and pods.
- Affected flowers show malformation.

Sexual cycle:

Primary source:

- The oogonia and antheridia are formed from the mycelium and oospores developed on fertilization of anthredium with oogonium.

- Oospores germinates and forms zoospore.

Asexual cycle:

Secondary spread:

Sporangia produce biflagellate zoospore.

Favourable conditions:

- Oospores survives under dry storage for 20 years.
- Rain coupled with relatively low humidity.

Management:

- Spray metalaxyl (ridomil MZ-72) or mancozeb @ 0.25 % or chlorothalonil (kavach)
  0.2 %

Powdery mildew(MINOR DISEASE)

C.O.: *Erysiphe polygoni/ Erysiphe crucifearum*

Symptoms:

- Appear in the form of dirty white, circular, floury patches on both sides of leaves and pods.
- Leaves surface covered with powdery growth of fungus resulting in drying.
- Affected plant produce small and shriveled seeds.

Favourable conditions:

- Dry weather
- Temp. 25 0C
- Low humidity (65%)

Disease cycle:

- Obligate parasite and mycelium is ectophytic.
  - Primary source: Cleistothecia produce globous asci. Each ascus produces 2 to 6 ascospores
- Secondary spread: conidia -disseminated by wind.

Management:

- Spray Karathane @ 0.07 %
- Spray Wettable sulphur, Sulfex or Thiovet @ 0.3%

Alternaria blight(MINOR DISEASE)

C.O.: *Alternaria brassicae*

Symptoms:

- Irregular lesions surrounded by chlorotic haloes are produced on the leaves and petioles.
- The spots get enlarged to cover the entire leaf blade with girdling the petiole to cause blighting of the leaves.
- Entire foliage gets blighted.
- Seeds get shriveled and content low oil.
- Grain yield is reduced.

Disease cycle:

- Primary source: Infected leaf debris serves the pathogen to perpetuate.
- Secondary spread: Through wind borne conidia.

Favorable condition:

- Cool and dry weather.
- Temperature 10-15^{0}C.
- R.H. 80% & above.

Management:

- Spray chlorothalonil @ 0.25 % or mancozeb 0.2%.
- Remove the infected plant debris and destroy it.

Downy mildew

C.O.: *Peronospora brassicae*

Symproms:

- It is common among young plants, but also appears on mature plants.
    - The disease is characterized by the appearance of purplish-brown spots on the underside of the leaves.
- The spots may remain small or become enlarged.
- The upper surface of the leaf on the lesion is yellow.
    - Sometimes white rust is also found side by side on the same leaf and the symptoms of the two look very much similar from a distance.
- The stem also get infected and swell up with malformed siliques.

Disease cycle:

- Primary source: The pathogen survives in the form of oospores in the soil. Contaminated Seeds also act as source of inoculum. The co-lateral hosts plays important role in perpetuation of pathogen.
- Secondary spread: Through sporangia containing zoospores.

Host: Turnip, radish, cabbage, cauliflower and *Brassica* spp.

Management:

- The weed hosts of the pathogen should be eradicated.
- Crop sanitation- removing and burning the diseased leaves/ shoots etc.
- Deep ploughing in summer.
- Spray 0.8 to 1.0% B.M. or metalaxyl (ridomil MZ) 0.2% after initiation of the disease.

(19)DISEASES OF GRAM/ CHICK PEA/ BENGAL GRAM

1. Ascochyta blight

C.O.: *Ascochyta rabie*

Symptoms:

- Plants are more vulnerable for infection at flowering and podding stage.
- Water soaked lesions on leaves.
- The spots are roundish with brown margin with yellowish grey center.
- Similar spots appear on stem and pod.
- Stem girdles causing plant to wilt.
- Several spots on leaves, pods and stem may coalesce to blight the plant.
- In the center of spot, numerous minute black pycnidia arranged in concentric rings observe on stem and pod.
- Symptoms appear in a sporadic form.

Disease cycle:

- Primary source of inoculum: The pathogen is externally and internally seed borne. The hyphae / pycnidia and perithecia on infected debris also serve as primary source.
- Secondary spread is mainly through air borne inoculum/ pycnidiospores.

Favorable condition:

- Cool, cloudy, wet and rainy weather.
- Young plants are more vulnerable for infection.
- Temperature 25^0- 27^0C for disease development.

Management:

- Hot water seed treatment to remove seed borne inoculum at 50^0C for 5 minutes.
- Seed treatment with captan @ 2-3 gm/kg of seed.
- Spraying of carbendazim @ 0.05 %.

2. Wilt

C.O.: *Fusarium oxysporum* f.sp. *ciceri*

Symptoms:

- Disease can affect crop at any stage of crop growth.
- In seedling stage- infected seedling collapse on ground.
- In grownup plants, leaves turn yellow to light brown colour and drop off prematurely.
- On collar region, necrosis and discoloration can be seen.
- If plant is uprooted, the infected lateral roots shows blackish discoloration and they become weak.
- Transverse section of stem of roots reveal the presence of masses of fungal hyphae.
- The vascular bundle become blackened and discolored.

Favorable condition:

- Temperature 25-30^{0}C.
- Acidic soils.
- Heavy black soils.

Disease cycle:

- Primary source: It is seed and soil borne. The pathogen survives on dead organic matter as saprophyte and on infected debris. It may survive for several years in Chlamydospore stage.
- Secondary spread: The secondary spread in field takes place through macro-micro conidia with irrigation/rain water.

Management:

- Use disease free healthy seeds.
- Resistant varieties: Gujarat Chickpea 2 (GC 2), Gujarat Gram 5 (GG 5), Gujarat Junagadh Gram 6 (GJG 6)
- Treat the seeds with carbendazim @ 3 g /kg seed.
- Add more organic matter to silo for enhancing antagonistic microorganism *i.e. Trichoderma, Pseudomonas, Bacillus* spp. *etc*.
- Follow crop rotation for 6 years.
- Deep ploughing in summer.
- Green manuring in kharif.
- Seed treatment with bio-agent like *Trichoderma* spp. @ 4-6 g /kg seed.

3. Stem and Root rot or dry root rot

C.O.: *Rhizoctonia bataticola*
(Pycnidial stage: *Macrophomina phaseolina*) (Sexual stage: *Thanatephorus cucumeris)*

Symptoms

- The disease generally appears around flowering and podding time in the form of scattered dried plants.
- The seedlings can also get infected. The first symptom of the disease is yellowing of the leaves.
- The affected leaves, petioles and leaflets droop within a day or two. The leaves and stems of the affected plants turn straw coloured and plants wilt within a week.
- The lower portion of the taproot usually remains in the soil when plants are uprooted. The taproot is dark showing signs of rotting and is devoid of most of the lateral and finer roots. Dark minute sclerotial bodies can be seen on the roots exposed or inside the wood.

Pathogen

- The hyphae of the fungus are dark brown, filamentous and septate with constrictions in hyphal branches at the junction with main hypha.
- The sclerotia are brown and irregular in shape. The fungus has its sexual stage, *T. cucumeris*, which produces 2-4 basidiospores in terminal clusters on a celled hypha.

Disease cycle

- The pathogen survives in the soil in infected host debris as sclerotia for several years.
- The secondary spread is through farm implements, irrigation water and rain splash.

Favourable conditions

- Maximum ambient temperatures above 30^0C, minimum above 20^0C, and moisture stress favour disease development.

Management

- Treat the seeds with carbendazim or thiram at 2 g/kg or seed pelleting with *Trichoderma viride* at 4 g/kg or *Pseudomonas fluorescens* @ 10 g/kg of seed.
- Apply farmyard manure at 10 t/ha.
- Grow tolerant genotypes like ICCV 10

MINOR DISEASE: Chick pea stunt virus

C.O.: *Bean leaf roll virus*

This is the most important viral disease of chickpea prevalent in most of the chickpea growing countries.

Symptoms:

- Affected plants in field can be easily spotted out.
- Plant shows stunting with bushy appearance.
- Leaflets become small, yellow, orange or brown.
- Tips and margin of leaflets often become chlorotic.
- The stem and leaves of diseased plants are stuffy and thicker than normal.
- The most characteristic symptom is browning of phloem.
- Severely affected plant dry prematurely.
- Sometimes stunt and Fusarium infection occurs together. In such cases, xylem discoloration due to Fusarium is observed.

Disease cycle:

- The disease is transmitted by aphids *i.e*. *Aphis craccivora* and *Myzus persicae.*

Management:

- Resistant variety: Gujarat Gram 5 (GG 5)
- Adopt closer spacing.
- Sow the crop when aphid activity is low.
- Rouge out infected plants.
- Control sucking pest by spraying systemic insecticides *i.e*. methyl-o-demeton @ 0.025% or dimethoate @ 0.03 %.

(20) DISEASES OF SUNFLOWER.

1. Alternaria leaf spot/ blight

(MINOR DISEASE)

C.O.: *Alternaria helianthi*

- It is the most destructive disease and is widely distributed wherever sunflower is grown.
- It occurs on all the varieties in the winter season and it spreads rapidly during the rainy season.
- This disease has been reported to reduce the seed yield by 27 to 80% and oil yield by 17 to 35%.
- The disease also affects the quality of seeds which adversely affects seed germination and vigour of seedlings.

Symptoms

- The fungus produces brown spots on the leaves, but the spots can also be seen on the stem, sepals and petals.
- The lesions on the leaves are dark brown to black, circular to oval spots, ranging from 0.2 to 0.5 mm in diameter. The spots are often surrounded by a chlorotic zone with necrotic center.
- The spots later enlarge in size with **concentric rings** and become irregular in shape.
- Under high atmospheric humidity, several spots coalesce to show bigger irregular lesions leading to drying and defoliation.
- The disease sometimes cause rotting of flower heads and affects the quality of seeds by reducing the germination percentage.

Pathogen

- The fungus produces cylindrical conidiophores, which are pale grey-yellow coloured, straight or curved, geniculate, simple or branched, septate and bear single conidium.

- Conidia are cylindrical to long ellipsoid, straight or slightly curved, pale grey-yellow to pale brown, 1 to 2 septate with longitudinal septa.

Disease cycle

- The fungus survives on seed, host debris and weed hosts.
- The secondary spread is mainly through windblown conidia.

Favourable conditions

- Rainy weather, cool winter climate and late sown crops are highly susceptible.

Management

- Grow tolerant variety like BSH-1.
- Remove and destroy infected plant debris.
- Rogue out weeds at periodical intervals.
- Sow the crop early in the season (June sowing).
- Spacing of 60x30 cm or 45x30 cm reduces disease build up.
- Treat the seeds with thiram or carbendazim at 2 g/kg.
- Spray twice or thrice with zineb or mancozeb at 0.2% or carbendazim @ 0.1% at 10 days interval starting from first appearance of the disease or 35 DAS.

(21) DISEASES OF SAPOTA

1. Pink leaf spot		
Causal organism	:	*Phaeophloeospora indica*
Economic importance	:	This diseases was first reported from Dharward, Karnataka. It is also called *Phaeophloeospora* leaf spot.
Symptoms	:	Symptoms appear on leaves as small, pinkish to dark brown scattered circular spots visible on both surfaces. Later they coalesce and the affected leaves fall off prematurely.
Mode of spread	:	The pathogen spreads through wind –borne conidia.
Favourable conditions	:	It is severe during Oct- Dec. when humidity is high. The fungus grow best at 25 ^{0}C temperature and more than 90% relative humidity.
Management	:	Cricket Ball, Kirthabarthi and Minusops hexandra (root stock) susceptible to disease such varieties should be avoided for new planting. Spraying 0.2% zineb 75 WP (20 g/ 10 litre water) or
		ziram 75 WP 0.2% (20 g/ 10 litre water) or copper oxychloride 50 WP 0.25% (25 g/ 10 litre water) at monthly interval for control the disease
. Glomerella leaf spot		
Causal organism	:	*Glomerella cingulata*
Symptoms	:	Symptoms appear as yellow irregular spots on the leaves. In severe infection, the leaf turns yellow. Diseased portion turn grey to dark olive. Large no. of fruiting bodies of the fungus seen as minute black structures on the spots.
Pestalotiopsis leaf spot		
Causal organism	:	*Pestalotiopsis versicolor* (Syn: *Pestalotia versicolor*, *P. sapotae* and *P. podocarpi*)
Symptoms	:	Small, reddish brown specks on leaf lamina. Spots enlarge, become circular measuring 1 to 3 mm in diameter. Fully developed spots show grayish centre and dark brown to reddish margin. Minute black fruiting bodies (pycnidia) are seen in the center of spots.
Management	:	The disease is controlled by spraying the leaves with zineb 75 WP 0.2% or copper oxychloride at month is interval.

2. Sooty mould		
Causal organism	:	*Capnodium* sp.
Symptoms	:	It is a fungal disease developed on honeydew-like excretion secreted by aphids and scale insects. The fungus slowly covers the entire leaf area severely affecting the process of photosynthesis. This results in reduced translocation of food to the fruits, which leads to reduction in their size.
Pathogen	:	The mycelium is septate, dark colored and superficial. The fungus is associated with the infestation of scales and mealy bugs.
Management	:	Spray starch solution 5% to remove the fungal growth. Control insects by spraying systemic insecticides.

Root knot nematode

C.O.: *Meloidogyne arenaria, M. hapla, M. incognita, M. javanica*

Symptoms

Above ground:

- Stunting and chlorosis of plant, reduction in leaf size.
- The yield is drastically reduced.
- The plants finally get wilted.

Under ground:

- Severe knotting of roots.
- Infected roots swell at the point of invasion forming knots.

Favourable conditions

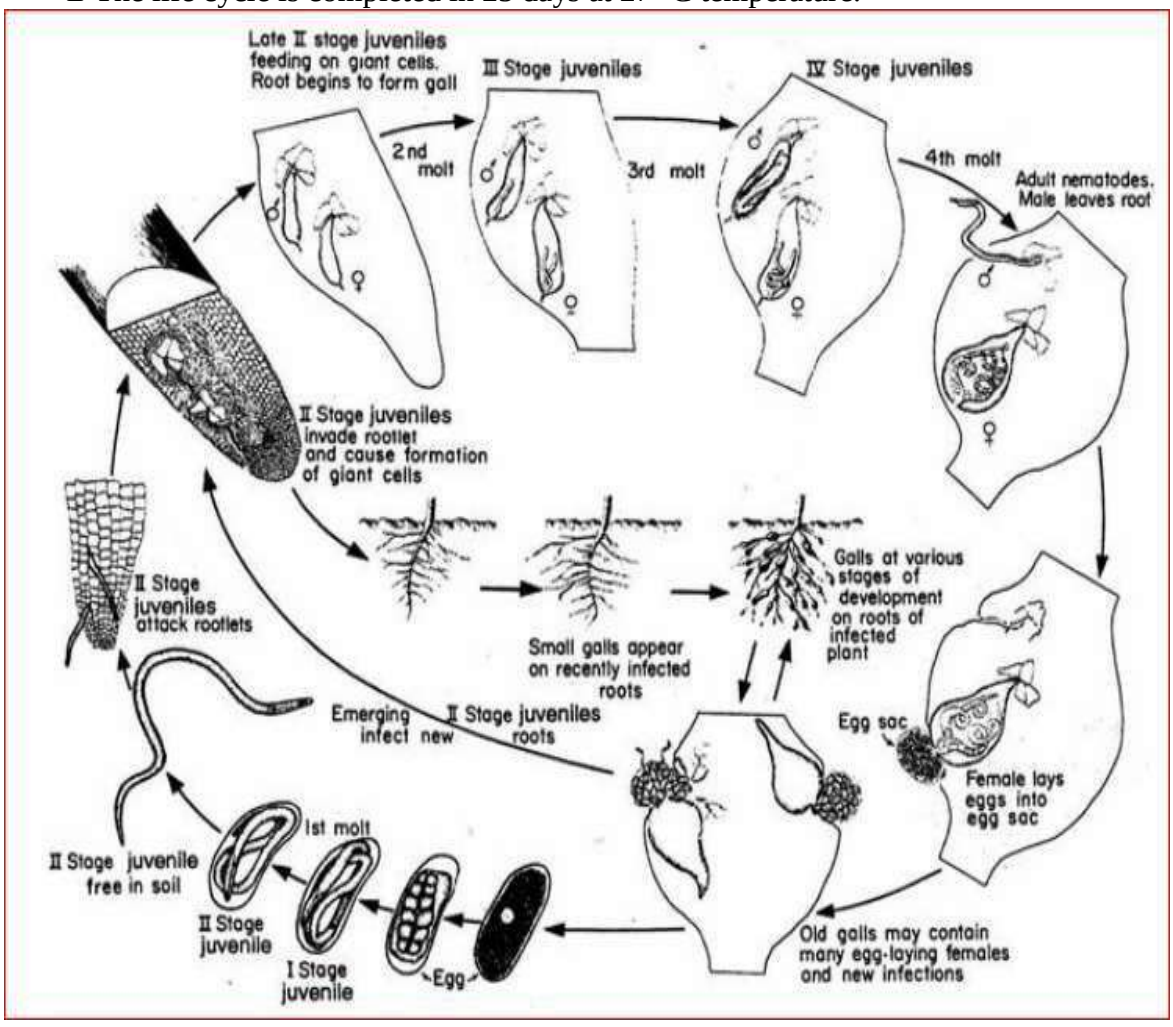
- Warm and hot climate.
- Sandy loam soil.
- Sufficient soil moisture.

Disease cycle

- Each female lays approximately 5000 eggs in gelatinous matrix.
- I and II stage juveniles develop inside the egg.

- **II stage juvenile is only infective stage**.

- In III and IV stage the larvae remain in roots.
- In IV stage male/female differentiation takes place.
- They may follow both sexual / asexual reproduction.
- The life cycle is completed in 25 days at 27^{o} C temperature.



Life/ disease cycle of root knot nematode

Management

- Grow **Anand Bidi Tobacco 10 (ABT 10)** highly resistant variety and **Mosaic Resistant Gujarat Tobacco Hybrid 1 (MRGTH 1)** tolerant variety to root knot nematode (**AAU recommendation**).
- Plant crop during 1st to 3rd week of September (**AAU recommendation**)
- Application of castor or neem cake @ 2 - 3 t /ha.
- Soil solarization with 100 LLDPE transparent plastic films during April- May when the temperature is above 40^0C.
- Rabbing with bajra/paddy husk. 7 kg/m^2.
- Application of Carbofuran 3G 1 kg a.i./ha i.e. furadan 33 kg/ha in open furrows.

(22) DISEASES OF TURMERIC

1. Colletotrichum leaf spot

C.O.: *Colletotrichum capsici Colletotrichum curcumae Colletotrichum gleosporioides*

- This disease is common in many parts of India and in recent years has become very severe, causing much damage to the crop.

Symptoms:

- Oblong brown spots with grey centres are found on leaves.
- The spots are about 4-5 cm in length and 2-3 cm in width.
  - In advanced stages of disease, black dots representing fungal acervuli occur in concentric rings on spot.
- The grey centers become thin and gets teared.
- Severely affected leaves dry and wilt.
- They are surrounded by yellow halos.
  - Indefinite number of spots may be found on a single leaf and as the disease advances; spots enlarge and cover a major portion of leaf blade.

Disease cycle:

- Primary source: The infected rhizomes.
- The secondary spread is by wind, water and other physical and biological agents.
- Besides turmeric, the same fungus is reported to cause leaf spot fruit rot of chilli.
  - If chilli is grown in the nearby fields or used in crop rotation with turmeric, the fungus perpetuates easily, building up inoculum potential for epiphytotic outbreaks.

Favourable condition:

- The disease spreads rapidly during the rainy season.
  - Often the shade from mixed cropping with castor provides favourable conditions for the disease.
- The infection mostly starts during mid of August and advances up to October.
- Relative humidity of 80% and temperatures of 21 – 23^0C favours the primary infection

Management:

- Select seed material from disease free areas.
  - Treat seed material with mancozeb @ 3g/litre of water or carbendazim @ 1 g/litre of water, for 30 minutes and shade dry before sowing.
  - Spray mancozeb @ 2.5 g/litre of water or carbendazim @ 1g/litre; 2-3 sprays at fortnightly intervals.
  - The infected and dried leaves should be collected and burnt in order to reduce the inoculum source in the field.
  - Spraying CoC (blitox) or blue copper at 3 g/l of water was found effective against leaf spot.
- Crop rotations should be followed whenever possible.
- Resistant variety- Voltamitta 8-3, Jaweli, Krishna.
- Cultivate tolerant varieties like Suguna and Sudarshan.

2. Rhizome rot and root rot

C.O.: *Pythium aphanidermatum*
Pythium graminicolum (Andhra Pradesh)
Pythium myriotylum (Assam)

- Ramakrishnan and Soumini (1954) have reported the occurrence of a rhizome and root rot of turmeric from Andhra Pradesh and Madras state of India.

Symptoms:

- The leaves of affected plant exhibit gradual drying along with the margins.
- This ultimately results in complete drying of all leaves.
- The basal portion of the shoot appears watery and soft.
- The root system is very much reduced and its tissues are also affected.
- In advanced stages, the infection spreads to rhizomes, causing decay of tissues.
- The development of rhizomes is poor.
  - The disease may appear in isolated plants or may involve several adjacent clumps resulting in the appearance of diseased patches in the field.

Management:

- Seed material should be selected from disease free areas.
- Avoid water stagnation in the field. Light soil may be preferred and drainage facility to be ensured.
- Grow tolerant varieties like Suguna and Sudarshan.
- Use resistant variety Ca-69 and Shillong (Assam).
- Crop rotation to be followed.
- Deep plough in summer.
- Planting is to be done in ridge and furrow method.
- Remove diseased plants and the soil around plants to be drenched with mancozeb (3 g/lit) or 3 g ridomil MZ
- Spray the crop with mancozeb (2.5 g/lit) or carbendazim (1g/lit) +1ml sandovit.
- Keep rhizomes in 3g metalaxyl or 3g mancozeb mixed in one litre of water for one hour and shade dry before planting.
- Drenching of the fungicides- Bordeaux mixture 1 % or CoC 0.3 %.

(3) Brown leaf spot / Leaf blotch

This is a common disease found throughout India. In many areas of the country almost 100 % plants are found infected during continuous rain in August and September. A few species of *Curcuma* and *Zingiber* are also attacked by the same fungus.

C.O.: *Taphrina maculans* Butler.

Symptoms:

- The spots appear thickly in great numbers, covering both sides of the leaf.
- The attacked leaves bear a reddish brown appearance instead of the normal green.
- These leaves become yellow.
- The spots are small usually 1-2 mm in diameter.
- In severe attack the spots are larger, irregular in shape and coalesced.
- The attacked plants are not killed but excessive spotting and destruction of chloroplasts the functional lamina surface is considerably reduced resulting in indirect effect on the productivity.

Disease cycle:

- ✓ The exact mode of transmission of the fungus is not yet known.
- ✓ How long the ascospores remain viable in plant debris has not yet been ascertained.
- ✓ The possibility that the mycelium reaches the rhizomes to remain there in a dormant state and then give rise to the disease when the rhizomes are sown.
- ✓ It is just possible that the inoculum might be surviving on the dried infected leaves in the soil in the fields from which the asci might be the chief source of the subsequent years for infection.

Favourable climatic conditions:
More favourable during continuous rains in August and September.
Management:

- Field sanitation.
- Spraying with Bordeaux mixture 1 % or copper oxychloride 0.3 %.

Resistant varieties -China, Jaweli, Ca-69 and Shillong cultivars are resistant in Assam

(23) DISEASES OF BER

1. Powdery mildew		
Causal organism	:	*Oidium erysiphoides* f.sp. *zizyphi*
Symptoms	:	White to greyish powdery mass appears on young leaves and fruits Severely affected leaves shrink and defoliate White powdery growth appears on young fruits which later becomes profuse and finally turns brown to dark brown in colour Infected fruits become corky, crack, misshapen, underdeveloped and finally drop prematurely
Survival and spread	:	**PSI: Dormant mycelium and conidia** on infected plants in Bud Wood
Disease cycle	:	The pathogen survives in bud wood of the host plant. The secondary spread is by air-borne conidia
Favourable conditions	:	Cool and dry conditions with morning R.H more than 90%
Management	:	Spray dinocap 0.1% or wettable sulphur 0.2% during first and third weeks of November Two sprays of carbendazim 0.1% at 15 days interval, starting from the time when the fruits are
		followed by dinocap 0.1% spray at 10-15 days interval