Dr. S.S. Patale
(B.Sc. III Year Botany Paper XVI (C) Plant Pathology – Notes)

B.Sc. III Year Botany
Semester –V
Paper XVI (C) Plant Pathology- Theory 45L
Unit-1 Credit-1
Fundamentals of plant pathology:
1. Plant pathology – history, scope, losses due to pathogens, importance and need to study plant pathology (02)
2. Classification of plant diseases on the basis of symptoms and causal organisms – animate and inanimate (03)
3. Plant pathological institutes – IARI (Indian Agricultural Research Institute), ICRISAT(International Crop Research Institute for Semi Arid Tropics) (02)
4. Seed pathology – concept and importance of seed pathology, seed borne pathogens, methods to study seed borne pathogens (03)
5. Study of air borne pathogens: methods and applications (03)
6. Field and laboratory diagnosis of plant disease - Koch's postulates (02)
Unit-2 Credit-2
Plant diseases:
Study of the following diseases with respect to symptoms, causal organism, disease cycle and disease management:
1) Cereals:
a. Black stem rust of wheat (05)
b. Grain smut of jowar
c. Ergot of bajra
2) Pulses:
a. Wilt of pigeon pea (04)
b. Yellow vein mosaic of bean
3) Vegetables:
a. Late blight of potato (05)
b. Little leaf of brinjal
c. Black rot of onion (*Aspergillus*) (04)
4) Oil seeds:
a. Tikka disease of groundnut
b. Damping off of mustard
5) Cash crops:
a. Grassy shoot of sugarcane (06)
b. Downy mildew of grapes
c. Angular leaf spot of cotton
d. Citrus canker
6) Ornamentals:
a. Powdery mildew of rose (02)
7) Weeds:
a. Rust of Euphorbia (02)
8) Trees:
a. *Cercospora* on *Albizzia* fruits (02)

Unit – 1 Fundamentals of plant pathology:

1) Introduction

Plant pathology – History, Scope, losses due to pathogens, importance and Need to Study Plant Pathology:

Plant pathology (also phytopathology) is the scientific study of diseases in plants caused by pathogens (infectious organisms) and environmental conditions (physiological factors). Organisms that cause infectious disease include fungi, bacteria, viruses, viroids, virus-like organisms, phytoplasmas, protozoa, nematodes and parasitic plants.

Plant pathology also involves the study of pathogen identification, disease etiology, disease cycles, economic impact, plant disease epidemiology, plant disease resistance, how plant diseases affect humans and animals, pathosystem genetics, and management of plant diseases.

History of Plant Pathology :

- Greek philosopher 'Theophrostus' was the first to study and write about plant diseases. He also recorded harmful effects of wind, weather to plants. Pathology taken grip from 14th century. Hook 1667 observed spores of rust fungi under compound microscope,
- Leeuwenhook 1675 discovered bacteria, and in Italy, Micheli 1729 described several new genera of fungi with their illustrations. Linnaeus 1753 gave nomenclature.
- After Micheli, Tillet 1755 experimentally proved that wheat seeds dusted with bunt powder gave bunted plants. This established relation between bunt and wheat. Prevost,1807 work on wheat smut and also treated it with $CuSo_4$. Prevost laid foundations for many branches in pathology. He was able to find out cause for the disease.

The late blight of potato in middle of 19th century made havac, it swept over the whole Europe and USA but in Irland it was catastrophic. More than one million people died and about 2 million migrated. This tragic event hastened the realization of importance of plant diseases the causal organism was identified by Anton De Bary (1861 -67).It was Phytophthora infestans.

The Plant pathology further studied by Berkley. Berkley was British mycologist who described Oidium powdery mildew of vine. It is interesting that editor of Garden Chronicle which recorded, miseries of the potato epiphytotic in an inimitable style was John Lindley. De Bary working with Sclerotinia rot of carrots noted the killing of tissues in advance of fungus. Juice from diseased tissue macerated healthy tissue, this ability was lost by boiling. H.M. Ward emphasised the role of weather on the diseases of Coffee rust in Lanka. Kuhn in 1858 wrote first book on plant pathology. He wrote ill his book the fungal role for disease development. Brefeld Germany (1875) developed techniques for inoculation and culturing micro-organisms. Which were followed afterwords by Petri, Koch and others. Parasitic fungi were then grown oh sterile synthetic media.

In 1878 vine orchards in France were attacked severely by downy mildew. Growers used to spray " Poison" (Cu S04 and lime) on vines. Millardet Professor of Botany from Bordeaux was invented Bordeaux mixture (1885).

Here after, that is after 1885 Fungicide era began. Bordeaux mixture saved vines in Europe and it was popular all over world. The discovery of Bordeaux mixture was a great impetus to develop chemical control of disease.

In 1905 Disease resistant plants found controlling diseases. Biffen in U. K. working with rust of wheat showed that resistance in crosses in between resistant and susceptible wheat plants was inherited as a recessive factor.

Pasteur and Koch I876 found all anthrax diseases of plants due to bacteria. Burrill 1878 reported that five blight diseases of pear and apple were by bacteria.

Adolf Mayer in 1870 worked on serious tobacco disease caused by "Mosaic virus" . Ivanowski 1892 filtered the infective juice which contained virus.

Last half 19th century progressed pathology in many directions.

Physiological plant pathology initiated by De-bary and Ward. After 2nd world war biochemical approach was more intense. Role of enzymes and toxins in disease, started by Gaumann, Walker, Dimond, Sadashivan, etc. Who contributed role of enzymes and toxins of fungi.

Genetics of pathogen and host was studied first by Biffin on rust. E. C. Stalkman 1914 contributed valuable work on physilogical roles of fungi. In the year 1904, Blakeslee, gave advance knowledge of sexuality in fungi. Flor 1955 explained host parasite relation in flax rust by gene relationship and the pathogenicity.

Fungicide research has notable names of H. D. Sister, Cox, Ludwig, Luken etc. Scientists Tisdale and Williams 1934 jumped into organic fungicides.

Later on Horsfall 1943 discovered Alkylene bis dithiacarbamates the most widely used fungicide. Emerson 1936 prevented damping off of citrus seedlings, by some antibiotics. L. R. Johnes who studied the effect of soil temperature on cabbage yellows.

Link and Walker 1933 has been noticed presence of proto catechuic acid and catechol industry scales of onion resistant to *Colletotrichum.* Report of phytoalexins by K.O.Miller ,1956 gave importance to antibody like substances in plants. Tissue culture enabled pathologist to grow organs, tissues in free cells in vitro.

Ecology of pathogens realized that root disease was due to relationship between host and pathogen but also with microbial interactions. Waksman 1917 emphasised important role of fungi in soil. Reinking and Mann in 1933 reported some species of *Fusarium* found in soils. In 1935 virology was established by Stanley by isolating TMV in crystalline form.

MLO type of pathogens are recently discovered as causal agents of some diseases. Japanese worker Doieta,1967 located dwarf mulberry potato witches broom etc. they were due to insect like vectors. Now a days pathology started obligations on society. Disease forecasting due to modern techniques is possible.

Scope :

A plant becomes diseased when it is continuously disturbed by some causal agent that results in an abnormal physiological process that disrupts the plant's normal structure, growth, function, or other activities. This interference with one or more of a plant's essential physiological or biochemical systems elicits characteristic pathological conditions or symptoms. Plant Pathology is the scientific study of diseases in plants caused by pathogens and environmental conditions.

Importance of Plant Pathology:

- Plant Pathology has advanced techniques to protect crops from losses due to diseases.
- The science of plant pathology has contributed disease free certified seed production.
- Most of the diseases with known disease cycle can now be avoided by the modification of cultural practices.
- With the knowledge of mode of disease spread, many diseases of economic importance can now be checked, minimized or controlled.
- Crop improvement and varietal resistance have been achieved against many diseases through the joint effort of breeder and plant pathologist.
- Plant pathology has made possible to restrict the spread of plant diseases from one place to other and one country to other through suitable measures and quarantine legislation.

-With the knowledge of studying about plant diseases, various prophylactic measures are adopted for successful management of diseases. These measures are seed treatment in seed borne diseases, soil treatment and crop rotations.

-Diseases can be avoided in cold storage by the application of plant pathological measures as per recommendations made for different diseases for protection of fruits and vegetables in storage.

-Plant pathology has made possible to recognize, discard or utilize toxic substances by producing toxic substances or by competition or by parasitism. Organism exerting such lethal or damaging effect on the other is called antagonist.

-There are huge opportunities for becoming plant pathologist.

Losses due to plant pathology -

Losses caused by plant pathogens have been and remain important constraints, worldwide, on efforts to increase crop production and productivity. With what appears to have been a decade of small improvements in the yield of major food and fiber crops, interest has been renewed on better definition and reduction of losses as a means of increasing crop yields.

Crop loss assessment and management requires a multidisciplinary approach because pathogens (fungi, bacteria, viruses, nematodes) not only interact with each other, but with other biotic and abiotic factors to affect yield. This review addresses both historical and current issues associated with the physiological basis of yield loss in plants, the statistical estimation of field disease and losses, and the use of modern technology and systems modeling in the study of losses at different levels of biological organization.

2) Classification of Plant Diseases

General symptoms are basically belonging to three main categories:

I. Necrosis

II. Hypertrophy and hyperplasia

III. Hypoplasia

I. Necrosis:

It is commonest and most destructive type of effect. The pathogen causes immediate and severe damage to host tissue and obtain their nutrients from cells killed in advance by

secretion of enzymes and toxins. They are thus called necrotrophs. Some pathogen does not kill cells in advance, but they invade and kill host cells.

1) Leaf spot:

Localized lesions on leaves consisting of dead and collapsed cells. They are of various size and shape. If dead tissue shrinks and separates from the healthy tissue, the condition is known as shot hole.

2) Streak or Stripe –

In the form of Elongated, narrow lesions on leaves, usually of brown shade.

3) Blight –

General and extremely rapid and sudden browning and death of leaves, branches, twigs and floral parts or entire seedling/young plant turns brown to black and soon disintegrate.

4) Damping-off –

Soil born pathogen causes fast death and toppling down of young seedlings due to disintegration of stem tissues at ground level.

5) Burn, Scald or Scorch - In succulent organs like fruits, limited area die and turn brown.

6) Rots :

i) Root rot - Decay or disintegration of part on the entire root system.

ii) Stem rot: Disintegration of the stem mostly at the basal region.

iii) Soft rot and dry rot- Maceration and disintegration of fruits, roots, bulbs, tubers, corns, rhizomes and fleshy leaves.

7) Wilting –

Wilting of infected leaves due to plugging of Xylem vessels by fungus or mucilaginous substances is caused by some fungi. Later the whole plant wilts and dies.

8) Die Back –

Death of plant parts, as stem or branches from tip backwards.

9) Canker –

Localized dead area in bark or cortex of woody stems, often appears sunken.

10) Chlorosis –

Discoloration from normal color is common in some cases. The green pigments may be destroyed and the tissue becomes yellow. Caused by Viruses and fungi.

11) Blotch-

Due to infection some fruit develop superficial growth appearing as a blotch area.

12) Scab -

Localized, rough, slightly raised or sunken and cracked lesions on fruit, leaf, tuber etc.

13) White Bristles or Pustules –

White blister-like pustules on leaves or stem which break open at maturity exposing powdery mass of spores.

14) Rust –

Number of small pustules on leaves or stems, usually of a powdery mass of brown rusty color.

15) Smut –

Infected ovaries or galls filled with sooty or charcoal like, dark brown to almost black powdery mass of spores or mycelium of the fungus.

16) Mildew –

Disease areas on leaves stems, blossoms and fruits, covered with whitish mycelium and the fructifications of the fungus. In downy mildew the areas show a fur-like downy growth of sporangiophores and sporangia on the undersurface of leaves, which appear pale yellow, water-soaked area on the upper surface. In powdery mildews, the area is covered with superficial whitish powdery mass of oidia and oidiophores of the pathogen gives a dusty appearance.

II. Hypertrophy and hyperplasia (over development):

In some cases, there is abnormal increase in size of an organ or of entire plant, this is due to increase in size of individual cells of affected tissue is. Hypertrophy or due to increase in number of cells as a result of cell division, i.e. hyperplasia.

1) Elongated internodes:

Some of infected plants develop elongated internodes and become abnormally tall.

2) Galls and tumors:

These are globose, elongated irregular large sized outgrowth formed on attacked part. Smaller galls are warts tubercles

3) Witches' broom:

They are formed by fungi, bacteria and certain viruses as well as insects and mites. They arise basically from stimulation of structures which normally remain dormant as buds. The broom appears as an upright cluster of small shoots, contrasting with horizontal growth habit of normal shoots.

4) Curls:

The leaves are arched, puckered, twisted, curled and distorted.

5) Floral abnormalities:

Some fungi caused the infected inflorescence to enlarge; green and fleshy with stamens converted into leafy structures. The inflorescence becomes distorted.

III. Hypoplasia (under development):

In these instances, there is reduced development of the whole plant, parts of the plant, certain tissue, flowers or fruit, or chlorophyll. In extreme cases the organ or tissue does not develop at all.

1) Chlorosis:

Reduced development of chlorophyll results into various kinds of Chlorosis, mosaic and molting. Chlorosis may take the form of streaking. There may vein clearing or vein banding or a general Chlorosis.

2) Reduction of individual organ:

Individual leaves and Flowers may be reduced in size or altered in shape. Internodes are reduced in dwarf bunt of wheat.

3) Floral abnormalities:

In anther smut of Caryophyllaceae, stamens become sterile One very common and characteristic floral abnormality is Phyllode i.e. transformation of floral parts into green leafy twisted structures.

3) Plant pathological institutes – IARI (Indian Agricultural Research Institute), ICRISAT (International Crop Research Institute for Semi Arid Tropics) (02)

INDIAN AGRICULTURAL RESEARCH INSTITUTE

Indian Agricultural Research Institute was established in the Year 1905 at PUSA, Bihar. It's original name before 1947 was Imperial Agricultural Research Institute. After the Independence the name was changed to Indian Agricultural Research Institute. In the year 1936 due to a massive earth quake it was shifted to Pusa, New Delhi. Since Independence IARI, is a premier institute for Agricultural Research and was instrumental in 1960-70 Green Revolution.

The Indian Agricultural Research Institute (IARI) is the country's premier national Institute for agricultural research, education and extension. It has served the cause of science and society with distinction through first rate research, generation of appropriate technologies and development of human resources. In fact, the **Green Revolution** was born in the fields of IARI and our graduates constitute the core of the quality human resource in India's agricultural research and education. The Institute has all along been adjusting and improving its policies, plans and programmes to effectively respond to the needs and opportunities of the nation. During the fifties, the advancement of scientific disciplines constituted the core programme and provided the base for its fast expansion in the 1960's and 1970's in all its three interactive areas, namely, research, education and extension. Besides basic research, applied and commodity research gained great importance resulting in the development of several popular high yielding varieties of almost all major crops and their associated management technologies, which brought about an unprecedented increase in the national food and agricultural production.

International Crops Research Institute for the Semi-Arid Tropics

The **International Crops Research Institute for the Semi-Arid-Tropics** (ICRISAT) is a non-profit organization in India.

It was founded in 1972 by a consortium of organizations convened by the Ford and the

Rockefeller Foundations. Its charter was signed by the FAO and the UNDP. Since its inception, India, the host country, has granted a special status to ICRISAT as a UN Organization operating in the Indian territory making it eligible for special immunities and tax privileges.

ICRISAT is managed by a full time Director General functioning under the overall guidance of an international Governing Board. The current Director General is William Dar (Philippines). The current chair of the Board is Nigel Poole (UK).

The Agro-Eco Region: The Semi-Arid Tropics

The semi-arid tropics (SAT) region is characterized by highly variable, low-to-medium rainfall and poor soils, further characterized by lack of irrigation. In general, the historical average annual rainfall in the SAT is below 700 mm. In agricultural policy terms, this region is considered to be a less favored area (LFA).

Research strategy

ICRISAT adopts Integrated genetic and natural resources management as its overarching research strategy. The aim is to combine tested methods of crop commodity research with well-established practices in research in natural resources management. The original goal was to use crop improvement research as the basis to improve food availability in drought-prone areas of the tropics. In the last ten years, ICRISAT research, especially in India, China, the Philippines and Vietnam, has tended to emphasize creation and sustenance of rural livelihoods in addition to releasing crop varieties that yield better.

Mandate crops

ICRISAT performs crop improvement research, using conventional as well as methods derived from biotechnology, on the following crops: Chickpea, Pigeonpea, Groundnut Pearl millet Sorghum and Small millets.

Research themes

ICRISAT conducts its research under four themes: Agro-ecosystems development, Harnessing plant biotechnology and bioinformatics, Crop improvement and management, and

Institutions, Markets, policy and Impacts

Genebank

The ICRISAT Genebank serves as a repository for the collection of germplasm of the five mandate crops – sorghum, pearl millet chickpea, pigeonpea and groundnut; and six small millets – finger millet, foxtail millet, little millet, kodo millet, proso millet and barnyard millet. The collection has over 119,700 germplasm accessions assembled from 144 countries. Several landraces now conserved in the ICRISAT genebank have disappeared from their natural habitats in Africa and Asia.

Innovations and Impact of ICRISAT Crop Improvement Research

Most of ICRISAT's crop improvement research is directed at LFAs, At an aggregate level, there is evidence from India that crop improvement research is having favorable productivity and poverty impacts in many LFAs. Based on an econometric analysis of time-series data for three different types of agricultural areas (irrigated,high-potential rainfed, and low-potential rainfed), non-ICRISAT experts found more favorable marginal returns (measured as Indian rupees of agricultural production per additional hectare planted to modern varieties) for crop improvement research in low-potential rainfed areas than in either high-potential rainfed areas or irrigated areas. Moreover, additional crop research investment in low potential rainfed areas lifts more people out of poverty than in the other two types of areas (reference). They found that ICRISAT-improved chickpea varieties have been widely adopted in a poor tribal area in Gujarat, India, with favorable impacts on yields, unit production costs, and net returns per hectare. ICRISAT's package of improved groundnut varieties grown in combination with improved agronomy practices is another example of a commodity-improvement program that has paid off handsomely in an LFA – in this case the semi-arid tropical areas of Central India.

Two major science-based breakthroughs attributed to crop improvement research at ICRISAT relate to Pearl Millet and Pigeonpea. A team of researchers at ICRISAT have released the first-ever, public sector-bred marker-assisted hybrid pearl millet, HHB 67. This was released in India in 2006. It is assessed to have superior agronomic performance and improved tolerance to terminal drought. The first-ever release of a hybrid pigeonpea by ICRISAT

researchers has been reported in 2008.

Virtual Academy for the Semi-Arid Tropics (VASAT)

Virtual Academy for the Semi-Arid Tropics (VASAT)[9] is a strategic coalition for information, communication and capacity building, operating in South Asia (SA) and West and Central Africa (WCA) . VASAT links and mobilizes stakeholders for drought mitigation in the semi-arid tropics. It is an innovative and cost effective medium to educate and support a critical mass of rural women and men spread across vast geographical areas by informing them about drought and desertification.

Information Products and Services

ICRISAT formally adopted an Open Access policy for its research publications in 2009. It is among a small number of agricultural research organizations to do so. As of June 2010, about 3000 publications are available online.

DBT-ICRISAT Platform for Translational Research on Transgenic Crops

The International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) is collaborated with the Department of Biotechnology (DBT), Ministry of Science and Technology, Government of India, to establish a DBT-ICRISAT Platform for Translational Research on Transgenic Crops (PTTC) at ICRISAT's global headquarters at Patancheru, near Hyderabad in India.

According to the Director General of ICRISAT, Dr William Dar, the PTTC will strengthen transgenic research for crop improvement by providing a platform, building synergies among institutions. ICRISAT will continue to harness transgenic research to solve problems that cannot be solved through conventional breeding.

4) Seed pathology – concept and importance of seed pathology, seed borne Pathogens, methods to study seed borne pathogens (03)

SEED PATHOLOGY

Seed borne pathogens present a serious threat to seedling establishment. Close association with seeds facilitates the long-term survival, introduction into new areas and widespread dissemination of pathogens. Under greenhouse conditions, the risks of significant economic losses due to diseases are great because factors including high populations of susceptible plants, high relative humidity, high temperatures and overhead irrigation, promote explosive plant disease development. Under these conditions, the most effective disease management strategy is exclusion which is accomplished by using seed detection assays to screen and eliminate infested seed lots before planting. The following will explore the current state of seed detection technology and include recent advances. A summary of the features of each assay is presented.

Conventional Seed Detection Methods:

Testing seeds for plant pathogens can be a difficult task. Unlike infected vegetative plant tissues, infested seeds can be asymptomatic, making visual detection impossible. Additionally, pathogen populations on seeds may be low, and infested seeds may be no uniformly distributed within a lot. Many detection assays exist for different seed borne pathogens, however, few satisfy

the minimum requirements for adequate seed tests. Ideally, seed assays should be sensitive, specific, rapid, robust, inexpensive and simple to implement and interpret. Seed assays have been developed based on different technologies including visual examination; selective media; seedling grow-out tests and serological techniques. While these tests have been used for many years, some of them have shortcomings that make them less than ideal. Brief descriptions of these assays including their advantages and disadvantages are discussed below.

Visual Examination.

In some cases infected seeds display characteristic symptoms, including discoloration and shriveling. Examples of such seed borne diseases include purple seed stain (*Cercospora kikuchii*) and advanced stages of Phomopsis seed decay (*Phomopsis longicolla*) of soybean (*Glycine max*), and Cylindrocladium black rot (*Cylindrocladium parasiticum*) of peanut (*Arachis hypogeae*). In these cases seedlot infestation can be reduced by using automatic devices that sort seeds based on visual of physical characteristics. These systems usually

display low detection sensitivity. Additionally, seeds infested by fungi, bacteria and viruses may display no macroscopic symptoms, making visual or physical inspection of seeds useless as a detection assay.

Selective Media:

A direct method of testing seeds is by allowing pathogens to grow from them onto appropriate artificial media. This can be done by directly plating surface sterilized seed samples or seed-wash liquid onto artificial media, followed by incubation under adequate conditions. Once a pathogen is isolated it can be identified by its cultural or biochemical characteristics for ex: the production of dark, muriform conidia in the case of *Alternaria* spp. Unfortunately, seeds may be contaminated by saprophytic microorganisms (nonpathogens) that grow as well as, or better than target organisms on nutrient-rich, artificial media. The excessive growth of saprophytic organisms including *Rhizopus* spp., *Penicillium* spp., and yeasts make it impossible to identify pathogens that may be present. The inability to identify the unique characteristics of the target pathogens in the presence of contaminating microorganisms lead to inaccurate assessments of seedlot infestation. To overcome this problem, selective artificial media are developed that use antibiotics, fungicides, selected carbon and nitrogen sources and other inhibitory compounds to retard the growth of nontarget microflora while allowing the pathogen to grow. Many selective and semiselective media have been developed for seedborne fungi and bacteria. Unfortunately, development of such media is time consuming and requires specific knowledge of the nutritional requirements and chemical tolerances of the target organism, relative to the nontarget seed microflora. Employing selective media also requires 2 to 4 d for pathogen growth and the test operator must be familiar with the range of cultural characteristics associated with the pathogen. Finally, while selective media can be applied for certain bacteria and fungi, it cannot be applied for nonculturable obligate parasites, e.g., viruses, nematodes and certain fungi and bacteria.

Serology-based assays:

Serological seed assays rely on antibodies (polyclonal or monoclonal) generated against unique antigens on the surfaces of plant pathogens. Antibodies bind strongly and specifically to their antigens and can subsequently be detected by the enzymatic digestion of substrates or fluorescent tags. Serology based seed tests have several formats including the widely applied enzymelinked immunosorbent assay (ELISA) and immunofluorescence microscopy. Serological assays do not require pure isolations of the pathogen and, hence, are

applicable to biotrophic and necrotrophic seedborne pathogens. Currently serology is the most widely used detection assay for seedborne viruses and it has proven to be sensitive and robust. Serology has also been widely used for the detection of bacterial and fungal plant pathogens, but the unavailability of species-specific antibodies is a limitation. Additionally, the detection thresholds of serology-based assays vary significantly based on the quality of the antibody and the testing format. Finally, with serology-based assays it is possible to detect nonviable pathogens which results in erroneous (falsepositive) interpretation.

Seedling grow-out assay

The seedling grow-out assay is a direct measure of the the seedlot's ability to transmit a disease. To conduct this assay, seedlot samples are planted under greenhouse conditions conducive to disease development and after germination, seedlings are observed for the development of symptoms. Seedling grow-out is one of the most applicable and widely used seed detection assays but for successful implementation, infected seedlings must display obvious and characteristic symptoms. Unfortunately, this is not always the case as some diseases have nondistinct symptoms, e.g., wilting, chlorosis etc. Another drawback of the seedling growout assay is that large seed samples (10,000 to 50,000 seeds in the case of bacterial fruit blotch (*Acidovorax avenae* subsp. *citrulli*) of watermelon (*Citrullus lanatus*) must be tested to statistically ensure that one infested seed can be detected. In addition to losses associated with the destructive testing of expensive seeds, assaying this quantity of seeds requires large areas of greenhouse space and adequate labor for assay set up and evaluation.

The seedling grow-out assay is also time consuming, requiring up to 3 weeks for seedling germination and symptom development. Finally, seed test evaluators must be familiar with the symptoms associated with each disease. This can be difficult since each disease has a range of possible symptoms that are influenced by environmental conditions. Hence, for the seedling grow-out assay, greenhouse conditions must be strictly regulated to ensure consistent results. In large greenhouses this can be a challenge and it can lead to erroneous test results. Also, because of the variations in seedling symptom expression it is often necessary to isolate the pathogen from suspected seedlings for confirmation. These extra steps further prolong the time required to complete the seedling grow-out assay. Residual contamination and cross-contamination between spatially separated seedlots are also issues of concern under greenhouse conditions.

Conventional seed detection assays including visual examination, selective media, serological assays and the seedling grow-out assay have been used extensively, but all have shortcomings

ranging from inefficiency to lack of specificity and sensitivity. Like other fields in which pathogen detection is critical, seed detection assays must be based on new technologies.

5) Study of air borne pathogens: methods and applications (03)

Introduction:

Airborne particles are a major cause of respiratory ailments of humans causing allergies, asthama and pathogenic infections of the respiratory tract. Airborne fungal spores are also important agents of plant disease and the means for dissemination of many common saprophytic fungi.

The term **Air borne disease** refers to any disease that is caused by pathogens and transmitted through the air. Such diseases include many that are of considerable importance both in human and veterinary medicine. The relevant pathogens may be viruses, bacteria, fungi and they may be spread through coughing, sneezing, raising of dust, spraying of liquids or similar activities likely to generate aerosol particles or droplates. Strictly speaking airborne diseases do not include conditions caused simply by air pollution such as dusts and poisons, through their study and prevention may be related. Air borne diseases are caused by pathogens which can ride on either dust particles or small respiratory droplates that can stay suspended in the air and travel distances on air currents.

The pathogen transmitted may be any kind of microbes and they may be spread in aerosols of dust or liquids. Airborne pathogens or allergens often cause inflammation in the nose, throat, sinuses and the lungs. This is caused by the inhalation of these pathogens that affect a person's respiratory system or even rest of the body. Many common infections can spread by air borne transmission at least in some cases, including Anthrex.

Virus diseases:

Chickenpox, Flu, Measles, German measles, Mumps and Smallpox.

Bacterial diseases:

Whooping cough, Meningitis, Diphtheria, Pneumonia and Tuberculosis.

Air Borne fungal diseases:

Vertilicium wilt, Powdery mildew, Anthracnose, Sooty mold and associated pathogens.

Methods for sampling for air borne fungal spores:

1) Culturable air samples:

If the air was sampled by impaction onto a growth media, then the samples are first incubated at suitable temperature followed by counting and identification of resulting colonies. A number of factors may affect the reliability of the colony a group of hyphae(fungal colony) or cells (bacterial colony) which arise from one spore or cells. It's possible to have mixed colonies of different organisms and counts data.

2) Tilak air Sampler:

The sampler was installed at a constant high of 5 feet at center of crop. The cellophane tape which is fixed over the rotating drum inside the sampler. The sampler as kept for 8 days in crop field. After 8 days the cellophane tape was cut into 16 divisions of equal size. Each division represented the day/ night spores composition. After removing the cellophane tape from the drum the case was taken and to avoid rubbing off of the deposits. Mounting the cellophane tape on the slides was done with the help of glycerin jelly because of its best optical property for the visual examination, the slides can observe under the microscope. The qualitative and quantitative analysis of the composition catches were carried out and identification of fungal spores was recorded on the morphological characters by using manuals.

Applications:

The forecasting of plant diseases, the knowledge of weather conditions and epidemiology in relation to plant diseases may be utilized to controlling plant diseases. The host parasites and weather interaction study. To check the disease incidence. Seasonal variations of the pathogens can check with the help of airspora.

Unit- 2

1) Cereals:

a) Black Stem Rust of Wheat

Causal Organism: *Puccinia graminis tritici*

Host: *Triticum aestivum* (Wheat)

The rust usually appears late in the season when the temperature starts rising. It is often not seen until March or even later in northern India when the crop is in earing or maturing stage.

The stem rust is a serious threat to wheat in India. The annual loss from rusts in India was estimated to be about Rs. 40 million (Butler and Hayman, 1906). The losses include reduction in grains, its poor quality and shriveled lighter grains. These effects are due to two factors:

1. Rubbing off of the food by the pathogen for its own use.
2. Excessive transpiration due to numerous slits in the host tissue.

These factors result in lessened metabolism, in partial sterility, reduction in the number of the grain in the head and shriveled grain.

Symptoms:



The rust appears in the form of elongated, reddish brown pustules (uredinia), primarily on the stem, followed by leaf sheaths and leaves. Thus, stem is most severely attacked (stem rust). The uredinia frequently merge with each other and soon burst exposing the brown powder of urediniospores, surrounded by ruptured epidermal fringes.

Another kind of pustules (telia) develops later in the same sorus or independently. Telia are darker and by this time pustules change from brown to almost black colour. These are also most prominent on culms, followed by leaf sheaths and leaves. A number of physiological changes are brought about by rust infection.

Causal organism and disease cycle:

Black or stem rust is caused by *Puccinia graminis tritici*. The hyphae in wheat leaves and stem are intercellular with small round or branched haustoria. From this mycelium

urediniospores develop in uredinia beneath the epidermis. Each urediospore is oval, stalked, brown, body, 25-30 X 17-20 µm single celled with echinulate exospores. There are four germ pores along an equatorial band. As host epidermis ruptures, urediospores disseminated by wind. They germinate on healthy parts of plant causing secondary infection. Thus urediospores propagate the disease in the entire field.

Later in the season, telia begin to develop from the same mycelium in stem, leaf sheaths and leaves. Telia are more frequent on stem. Telia are oblong to linear, dark brown to black exposed through the rifted epidermis. Each teliospore is stalked, two celled with a thick smooth wall. The apex is round or pointed. Spores are of chestnut brown colour, 40-46 X 15-20 µm. each spore has a germ pore, that of upper cell at the apex, and that of the power at one side just below the septum. Teliospores undergo a period of rest for several months. In India, these spores are unable to survive the hot summer temperatures following the harvest of crop. However, if conditions are suitable each cell of teliospore germinates to form a four-celled promycelium. From each promycelium, four haploid basidiospores are formed on sterigmata.

Puccinia graminis tritici is heterocious rust as its basidiospores cannot infect wheat but infect *Berberis* spp which serve as alternate hosts. About 83 species of *Berberis* are known world over to be infected by *Puccinia graminis tritici*. Of these *B. ariststa, B. coriaria, B. lycium* and *B. vulgaris* are that important ones. Wind borne basidiospores infect the young tissues of this plant. On germination, basidiospores give rise to haploid mycelia inside barbery leaf. This mycelium develops special structures on both sides of barbery leaf.

Spermogonia are flask shaped structures on the upper surface. Each spermogonium has an ostiole with some paraphysis. Inside it are spermatia. Sexual fusion occurs between spermatia and flexous hyphae of spermogonia of opposite strains. The dikaryotic mycelium thus developed, then forms another type of structures called aecia on the lower surface of leaves. Aecia are yellow, cup shaped receptacles enclosed by peridium. Each aecial cup contains chains of aeciospores. The aeciospores are yellow, verrucose and with six germ pores. On germination they give rise to germ tubes which can infect only wheat. Thus aeciospores infect wheat leaf through stomata.

However, later researches in India and elsewhere revealed that the nuclear life cycle may have nothing to do with the actual disease cycle and annual recurrence on fungus. The cycle may be completed with the urediniospores only and it usually occurs in India.

Control measures:

1. Cultivation of rust resistance varieties:

The rust resistance varieties should be cultivated for combating the disease. Some varieties are available in India, NP718, and NP770 and newly breed hybrid NP822, NP823 and NP825 have given good results. The recently introduced dwarf Mexican wheat varieties such as Sonora 64 and Lerma Rajo are almost completely resistant to rust.

2. Eradication of Barbery:

It was believed that eradication of barbery is a possible means of eliminating the disease. But control of stem rust by this method is not possible in India. The uredospores which are able to survive to stray and self sown wheat plants on the hills serve as inoculums.

3. Use of fungicides and antibiotics:

The use of fungicides to control rust disease has received much more attention in the recent years. Zineb and Maneb control the disease very effectively. Use of Diathane Z-78 and Diathane M-45 was found to be effective even at low concentration. Other chemical like Plantavax and RH-124 have good results. It was reported that Ozone (O_3) inhibits infection, hyphal growth and uredospore formation of the rust fungus. Actidione has been recommended to be useful antibiotic for the control.

4. Mixed cropping:

Mixed cropping of wheat and barley is helpful for the control.

b) Grain smut of Jowar

Causal Organism: *Sphacelotheca sorghi*

Host: *Sorghum vulgare* (Jowar)

Basically it is a covered smut. This is most common and destructive disease of jowar in India. Besides India, the disease has also been reported from Manchuria, Myanmar, Tanganyika, South Africa, Italy, U.S.A., Venezuela and other countries.

Symptoms:

The smut attacks ovaries exclusively. Majority or all of the grains in the ear are attacked. Each infected ovary is transformed into a dirty grey spore sac (sorus). The shape and size of affected grain varies. They are generally oval or cylindrical, sometimes conical at the tip and 4-12 mm long. The sac surrounded by unaltered glumes at the base. The stamens are commonly absent. Thus in infected ears there is no grain formation as ovaries of spikelets are transformed in to sori. In some varieties of jowar, shape and size of smutted grain is not much different from those of normal ones.

The wall of sorus varies according to nature of attack. In long protruding sacs, the wall is entirely made up of fungal pseudoparenchyma. The host tissue contributes to only basal part of the wall. In the hidden forms, the wall in most part is the ovary wall which is rough and rigid and ruptures only after harvest. Each sorus has a slender, sometimes curved, column of hard tissue the columella in the centre. All around the columella, there is a black powdery mass of teliospores.

Causal organism and disease cycle:

Grain smut of jowar is caused by *Sphacelotheca sorghi.* Teliospores are round to oval, dark brown in mass but brownish olive individually; smooth walled and 5-9 µm in diameter. They have a long viability as long as 13 years. Each teliospore germinate to produce a septate promycelium, budding off sporidia laterally as well as apically. The sporidia are spindle shaped, and measure 10-12.5 × 2-3 µm. the dikaryotic phase is established by fusion either between sporidia or their germ tubes.

The disease is systemic and externally seed borne. The pathogen survives as teliospores on surface of contaminated seeds. When such seeds are sown in next season, teliospores germinate in presence of moisture to give rise promycelia and sporidia. The dikaryotic infection hyphae infect the young emerging seedlings. The pathogen grows in systemic manner throughout the developing plant and finally reaches the ovaries which become transformed in to smut sori. The sori are broken during the threshing of the crop and the spores released from them contaminate the seeds. The teliospores remain dormant until the next season when they germinate along with the seed germination. The infection takes place only during the period between seed germination and emergence of seedlings above soil level. The germ tubes enter mainly through the mesocotyl.

Control measures:

1. Use of clean, disease free seeds for cultivation.
2. Treatment with formalin (0.5%) is useful for immersing the seeds for two hours. The seeds are then dried quickly.
3. The seeds may be soaked in copper sulphate (0.5-3.0%) solution for 10-15 minutes, then dried and sown.
4. Dry seed dressing with Agrosan GN (1:500).
5. Mercural and non mercural fungicides such as Arasan, N.I.Cerasan, Tillex, Flit 406, Cerasan M are also effective.
6. Use of disease resistant varieties- T29/1, PJ 7K, PJ 23K, Nandyal, Bilichigan, CSH-9, SPV-104, SPV-102, SPV-115, SPV-297, SPV-138, SPV-245, RSV-1-R, SDM-9, CSH-7-R and CSH-5.
7. Solar energy treatment.
8. Soaking seeds in water during summer for 4 hours and then spread out in the sun or in shade to dry.

c) Ergot of Bajra

Causal Organism: *Claviceps fusiformis*

Host: *Pennisetum typhoides* (Bajra)

This disease reported from many parts of Africa and India. In India the first outbreak od the disease causing considerable loss were reported from Satara District of Maharashtra in 1956. It is increased during the past few years in Maharashtra and Karnataka, causing severe damage to the crop and poison to cattle which consume ergot along with the straw. The damage caused by the disease depends upon the weather at the time of ear formation. Presences of the toxic alkaloids in the ergot add to the importance of the disease. Bajra ergot contains agroclavine, elymoclavine, chanoclavine, penniclavine and setoclavine. Besides above, a water soluble alkaloid ergometrine is also present. Total amount of alkaloids in honeydew and sclerotia has been found up to 5 mg/g and 56 mg/100g respectively.

Symptoms:

Ergot first appears as a viscous, turbid, and fluid oozing out as small droplets from the infected florets of the panicles. This carbohydrate-rich fluid contains numerous conidia of the pathogen which is called as honeydew phase of the ergot symptoms. The honeydew can be initially cream coloured, later becoming pink or brown. The honeydew phase persists for 4-7 days, when it may flow down the panicle on to leaves or the ground. Later the fluid becomes darker covering larger areas of the panicle. Within 10 days of honeydew formation sclerotia are formed in infected florets instead of grain. They can be seen projecting from between the glumes. Sclerotia are initially whitish, elongated and larger than seeds. The sclerotia becomes hard and brown to dark brown within 10-15 days. The sclerotia are about 0.5 to 1.0 cm in length, 1-2 mm in diameter.

Causal organism and disease cycle:

The disease is caused by *Claviceps fusiformis*. The honeydew produced on the ears is the conidial stage. Two types of conidia are produced. Macroconidia are developed initially are hyaline, unicellular, fusiform and measure 13-18 × 3-4 µm. later microconidia are formed which are unicellular, hyaline, globular and measure 2.5-10 × 1.2-4.8 µm. Macroconidia

germinate by producing one to three germ tubes while microconidia produces one germ tube. These germ tubes may produce secondary conidia which may further form tertiary ones. Conidia are infective and spread disease in the field. Later sclerotia begin to develop by replacing the grains. Sclerotia are round to elongated, light to dark brown, hard to brittle and measure 3.6-6.1 × 1.3-1.8 µm. sclerotia germinates by producing one to several fleshy, 6-26 mm long purplish stipes which bear light to dark brown globular stromata. The stromata bear many numerous perithecia. The perithecia are pear-shaped, arranged in semicircular manner in the stromata. Their neck is protruding and has an ostiole. There are numerous, long, cylindrical, hyaline, thin walled asci in each perithecium. Asci are interspersed with periphyses and emerge through ostiole. Each ascus contains eight ascospores which are long, hyaline, filiform, non septate and measure 103-176 × 0.5-0.7 µm. ascospores germinates to produce primary and secondary conidia. Conidia are disseminated to healthy inflorescence by physical contact with infected heads, splashing rain, air currents and insects.

The primary source of inoculum is the conidia left in the field from the previous crop. Sclerotia may also be mixed with seeds. Such seeds sown in the next season also bring sclerotia in soil. In some cases, conidia from ergot infected collateral hosts also served as source of primary inoculum. Sclerotia germinate to produce perithecial stroma. Ascospores released from perithecia are carried by air currents to fresh flowers at the Perigyny stage under favourable weather.

Control measures:

Since the pathogen is air borne, affecting the spike only at the time of flowering, it is difficult to control. Perhaps the best method is to develop resistant varieties, and work needs to be done in this direction. The most common method of control of ergot is the use of clean seeds. Deeping the seeds in 20-32 percent salt solution float the sclerotia which can be removed by hand. Sprays with Ziram or a mixture of copper oxychloride and Zineb (1:2) applied and 2-3 times at weekly intervals, starting prior to earhead emergence gives good protection. According to Kulkarni (1967) sclerotia remain viable for a longer time if buried deep. Therefore repeated ploughing may reduce their viability. Cultural practices such as deep ploughing, adjustment of sowing dates, balanced soil fertilisation and intercropping for disease control are recommended.

2) Pulses:

a) Wilt of pigeon pea

Causal Organism: *Fusarium udum*

Host: *Cajanus cajan* (Pigeon pea)

This disease widely occurs in Asia and Africa attacking pigeon pea. The disease was first reported in 1906 in India by E. J. Butler. Since then this has been reported also from Kenya, Tanzania, Uganda, Malavi, Thialand, Indonesia and Trinidad. In India, the disease is very destructive in Maharashtra, Rajasthan, Madhya Pradesh and U. P. the disease may cause as much as 50% mortality of plants if crop is continuously grown in the same field.

Symptoms:

The plants are susceptible to attack throughout their development. The infection occurring through fine lateral roots by either conidia or chlamydospores. The pathogen enters the vascular systems and traverse all along, producing conidia and chlamydospores within the xylem vessels. The xylem vessels are frequently blocked by clumped of mycelia. Blackening due to infection frequently appears in the vascular tissues. The wilt appears in early stages of plant growth (4-6 week old). The disease becomes most severe during flowering and pod formation stages.

Typically the first symptom is premature yellowing of leaves. The next symptom is the wilting or withering of leaves of the diseased plants. The plant is in a blighted condition. The leaves to be affected progressively from the bottom towards the top. Finally the entire plant completely dries up. In severe cases of the infection more than half of the plants in the field may fall victim to this disease.

Causal organism and disease cycle:

The disease is caused by *Fusarium udum*. The pathogen is specific to parasitism and pathogenic to only pigeon pea. It is soil borne parasite that enters through roots and become systemic in plant. It can be isolated from all parts of the host. The fungus occurs more

frequently and in high population in the vicinity of infected and wilted plant roots. The pathogen is well developed in vascular tissues and are both inter- and intracellular. The mycelium produces three types of spores. Microconidia are small, elliptic or curved, unicellular or one or two septa and 5-15 × 2-4 µm in size. Macroconidia are produced on small stromatic masses, the sporodochia. They are long, curved, pointed at tip, knotched at base, 3-4 septate and 15-20 × 3-5 µm in size. Chlamydospores are spherical or oval, single or in chains, terminal or intercalary. Wilting is also caused by production of toxin, fusaric acid. Besides these the pathogen also produces enzymes – pectin methylesterase, polygalactrunase and cellulases which are involved in disease development.

The pathogen can survive saprophytically in soil in absence of its host for 3-4 years. The asexual state is more important and common in nature. The pathogen grows externally as well as internally. It produces mass of mycelia and conidia on the host surface. After the plant wilt, the pathogen survives as saprophyte in soil for several years. Saprophytic survival occurs mostly in the infected dead roots and other host debris. Chlamydospores are formed in parasitic as well as saprophytic phases from the hyphae and conidial cells. Pathogen may also survive as chlamydospores. They germinate at the turn of favourable conditions to cause primary infection. The disease spreads through contact of infected roots with healthy ones, irrigation, rain water and termites.

Control measures:

1. Use of disease resistance varieties such as NP15 and NP38.
2. Crop rotation is the most effective as it would check the buildup of inoculums in the soil.
3. Field sanitation is the effective measure. Removal of affected plant and their roots along with deep ploughing during summer are very effective.
4. Mixed cropping with jowar is very useful for disease control.
5. Use of chemical drugs such as soil drench with Bavistin (2000 ppm), soil treatment with Dithan Z-78 and Zinap.
6. Biological control by use of antagonists such as *Bacillus subtilis, Micromonospora globosa, Trichoderma viride* and *T. harzianum*.

b) Yellow mosaic of legumes (beans)

Causal organism: Yellow Mosaic Virus (YMV)

Host: Legume crop

This viral disease is the most destructive disease of kharip legumes of India. It was first reported from Delhi in 1960 but is known to occur in other parts of country also. The loss in yield depends upon the stage at which the crop is infected. If the infection is early in the season there may be total loss of yield. Mung bean shows heavier loss than other legumes.

Symptoms:

The diseased plants start appearing in the field when the crop is about a month old. Infected plants show mottling, yellowing and malformation of leaves and pods. Infected plants may be stunted and bunchy, seeds may abort, smaller or malformed and yields may be reduced. The most common symptoms are the development of a pattern of light and dark green area, giving a mosaic appearance on systemically infected leaves. There is clearing or yellowing of the veins. The areas making up the mosaic are generally irregular in outline. There may be two shades of colour involved: dark green and pale or yellow green. In infected leaves the borders between darker and lighter areas may be sharp.

Causal organism and disease cycle:

The disease is caused by yellow mosaic virus (Gemini group virus). In India it has a large host range which includes other legumes. The paired particles of the geminate virus measure 30 × 18 nm. Isolate studied in Thailand has thermal inactivation point of 40^0 to 50^0 C, dilution end point of 1:100 to 1:1000 and *invitro* longevity of one to two days at 20^0 C.

The viruses are transmitted through several aphids. YMV is transmitted through the seeds infrequently (3% - 6%). YMV overseasons in one of its many cultivated and wild hosts, from which the aphids transmit it to the crop. Bean plants are involved in the natural virus spread in three ways: for breeding the vectors, for providing virus-infected materials, and as recipients of virus from infective vectors. YMV survive from season to season in infected

seeds which are primary source of inoculums. In tropical and subtropical areas, agricultural practices allow the viruses to survive throughout the year in successive crops of the same bean species grown at the same locality.

Control measures:

Following measures do not ensure complete protection but can check the spread and reduce losses.

1. Local varieties are highly susceptible. These should replace with improved varieties. Urd bean varieties T9, UPU1, Pant 19, Pant 26, Pant 30 and Pant 35 are fairly resistant. In mung bean Pant 1, 2, 3, T1 and T44 are resistant.
2. Control of disease through prevention of population of the vector has been recommended. Spraying of Metasystox (0.1%) at 10-15 days interval, starting when the crop is about a month old or as soon as a single diseased plant is detected in the field. Application of Aldicarb alone or with Endosulfan and Captan reduces white fly population. Diseased plants as well as host also are eradicated after each spraying.
3. Common insecticides do not cause instant death of vectors. For this oil sprays have been found more effective since they can immobilize the white fly within 15 minutes. Systemic granular insecticides for soil application as recommended for yellow vein seem to be the best chemical method for reducing vector population and delaying the appearance of the disease.
4. The management of crop in respect of space and time helps in disease control. For ex. Cultivation should be started at that time when vector population is poor.
5. Healthy, virus free seeds should be selected, as these are the primary source of inoculums. Seeds to be used must be tested by appropriate methods for the presence of virus inoculums.

3) Vegetables:

a) Late Blight of Potato

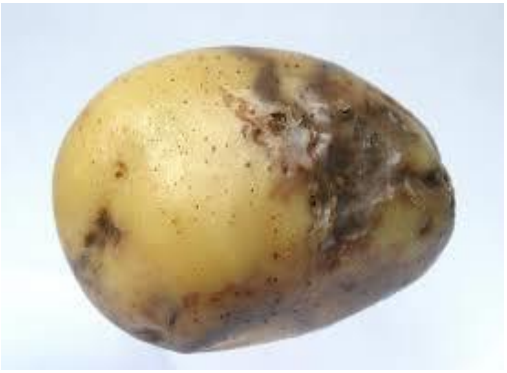
Causal Organism: *Phytophthora infestans*

Host: *Solanum tuberosum* (Potato)

Late blight is a serious fungal disease worldwide in distribution. It is destructive to crop grown in the rainy season. The disease occurs annually in cooler Himalayan regions extending from Assam to Kashmir at an altitude of 6000 ft or more. The temperature below 23^0C is favourable for the appearance of the disease. Now it has established itself in the Indo-Gangetic plain and occurs annually in the states of Punjab, U.P., Bihar and West Bengal.

The damage caused by the disease is frequently very high. Severe damage to foliage shortens the growing season. Consequently the tubers remain small and reduced in weight. They are produced in smaller numbers. In severe cases of infection there is complete loss of the crop. Infection also results in the decay of tubers in the field and storage.

Symptoms:



The disease first appears on the tops of the plants generally after blossoming period but mostly in the month of January. It may appear at any time during the growth period of the plant. The disease appears as small, dead, and brownish to purplish black areas or lesions. These appear on the tips and margins of the leaflets, rachis, petiole and stem. Under favourable conditions, the lesions rapidly increase in size involving the whole surface of the leaf. The blighted leaves curl and shrivel in dry weather. Under moist condition they decay and emit a characteristic offensive odour. Potato tubers are often infected in the field after the tops have been blighted. There is brownish discolouration of the skin of those parts of the tubers which lie nearest the surface of soil. In cool and dry conditions the progress of the disease is slower.

Causal organism and disease cycle:

The causal organism is *Phytophthora infestans*. The mycelium is aseptate coenocytic, hyaline and branched. The hyphae arte both intercellular and intracellular. They form rudimentary haustoria in the host leaf cells but in the tubers haustoria are more common and elaborate. The mycelium overwinters in the infected tubers. In the temporate regions the fungus penetrates in soil in the form of sporangia and germ tube.

The infected tubers are generally considered as the main source of primary infection in India. The fungal parasite overwinters as a dormant mycelium in the infected tubers. It becomes activated at the time of germination of the diseased seed tubers among the planting stock or waste tubers in dump heaps or infected tubers remaining in the ground after a previous crop. The second view is that the thick walled oospores which are found in abundance in the infected tubers are important overwintering structures. They play a significant role as a source of primary infection. The infected sprouts emerge above ground and produce shoots which contains the mycelium. It grows and ramifies in the intercellular spaces and gives out hyaline, branched, indeterminate sporangiophores through the stomata of the host leaves. The thin walled, ovoid or lemon shaped sporangia are borne singly at the tips of the sporangiophores or their branches. The mature sporangia are detached and spread by splashing rain or air currents to new potato plants. On reaching a suitable host the sporangia germinates on the leaves. Germination is influenced by moisture and temperature conditions.

a) Indirect germination: In cool and moist weather the sporangia function as zoosporamgia. The propoplasmic contents of sporangium divide to form a number of biflagellate zoospores. They are liberated and come to rest, retracts their flagella and secretes a wall around it. These zoospores germinate rapidly at 12^0 to 15^0C.

b) Direct germination: Under dry and warmer conditions, the sporangium functions as a conidium. It directly put out germ tube. The temperature for direct germination of sporangia is about 24^0C or 25^0C.

The infection thread produced on the surface of the host leaf enters the tissue. It penetrates through the cuticle or stomata. The lower surface is more susceptible than the upper surface of the leaf. The infected leaves produce another crop of sporangia which are carried by wind to healthy plants. This constitutes secondary infection and the process is repeated. As a result the disease spreads during the growing season over large tracts under potato cultivation.

Infection in the tubers is caused by zoospores produced in foliage lesions or present in the contaminated soil. Sporangia and zoospores come contact with the tuber in two ways.

First, by contact of freshly lifted healthy and wounded tubers with diseased plants and contaminated soil. Secondly, during crop growth the zoospores and sporangia washed down the stems in to the soil by rain come in contact with the tubers. After germination, the germ tube enters through the eyes, wounds and lenticels.

The severity of the disease is governed by following environmental conditions:

a) Night temperature below dew point for 4 or more hours.

b) Minimum temperature 10^0C or slightly above.

c) Mean cloudiness on the next day.

d) Rainfall during 24 hours at least 0.1 mm.

Control measures:

1. Selection of seed tubers: The seed tuber should be free from the disease.
2. Storage of seed tubers at 40^o F or below: Storage of tubers in cold storage rooms reduces or even checks the progress of disease.
3. Growing disease resistant varieties: Some disease resistant varieties developed in India are Kufri Naveen, Kufri Jeevan, Kufri Alankar, Kufri Badshah, Kufri Swarna, Kufri Moti and Kufri Muthu.
4. Use of fungicides: Fungicides were applied before infection by spraying or dusting. Timely and repeated spraying of copper fungicides such as Perenox, Blitox-50 and Fytolan is effective. Diathane Z-78 and Diathane M-22 proved to be more effective than the copper fungicides. The spraying should when the plants are 8 inches tall and continue until the harvest time at 10 days interval. In Assam, Brestan 60, Diathane M-45 and Zineb are useful for blight control. Mancozeb and Chlorothalonil are major fungicides used in present days. Each inhibits sporangial and spore germination.
5. Sanitation: destruction of potato tuber refuge from pits and store houses is another practical measure for control.
6. Tuber treatment before storage: The tubers should be dipped in 1:1000 mercuric chloride solution for 90 minutes before storage and should be washed before use.
7. Injuries to the tubers should be avoided at harvesting.
8. In cool humid area killing of foliage before harvest proves beneficial. This is accomplished by spraying with herbicides or flame throwers or by use of mechanical vine beaters.

b) Little leaf of Brinjal

Causal Organism: Phytoplasma (MLO)

Host: *Solanum melongana* (Brinjal)

Little leaf of brinjal is found throughout India and Sri Lanka. In India it was first reported from Coimbatore. The disease has become serious threat to the profitable cultivation of this vegetable crop in most of the states. When young plants are attacked they do not produce flowers and fruits. Bose (1983) have reported over 99 percent loss in fruit production. There is also loss of germinability of seeds from fruits formed on infected plants. Early infection result in reduced root length, fresh and dry root weight more than in late infections.

Symptoms:

The main symptom of the disease is production of very short leaves by the affected plant. The petioles are so much reduced in size that the leaves appear sticking to the stem. Such leaves are narrow, smooth and yellowish in colour. Newly formed leaves are further reduced in size. The internodes of the stem are shortened and at the same time a large number of axillary buds are stimulated to grow in to short branches with small leaves. This gives the whole plant a bushy appearance. Usually such plants fail to form flowers. Even if flowers are formed they remain green and fruiting is rare.

Causal organism and disease cycle:

The disease is caused by a phytoplasma. Earlier the causal organism was thought to be a virus. The phytoplasma appears as ovoid or sphericalbodies, 40-300 nm in diameter without a rigid cell wall. These bodies of phytoplasma have been observed in the sieve tube elements of the phloem of stem, petioles, leaves and roots of diseased plant as well as in the haemolymph of the insect vector, *Hishimonus phycitis* (*Cestinus phycitis*). The adjacent companion cells of the phloem show wall ingrowths, dilated endoplasmic reticulum, dilated

and ruptured cristae, deformed outer mitochondria membrane and rupture of plastid membranes and tonoplast suggesting involvement of toxins in the etiology.

Besides eggplant, this phytoplasma also attacks *Datura fastuosa* and *Vinca rosea* and could also be successfully inoculated in to tomato, potato and tobacco. It appears that during off season, the phytoplasma survives on the some weed hosts. From these weeds this is transmitted to eggplant by insect vector. Insect vectors spread the disease from plant to plant in the field.

Control measures:

1. Eradication of weed hostsand diseases brinjal plants from the field.
2. Control of insect vectors by insecticides.
3. Antibiotic treatment with tetracycline, asachromycin, terramycin, aureomycin etc.
4. Some varieties of eggplant like BB-7, BWR-12, Pant, Rituraj and H-8 are reported to have some resistance to this disease.

c) Black rot of onion

Causal Organism: *Aspergillus niger*

Host: *Allium cepa* (onion)

This disease can be of great economic importance in warm conditions. Seedlings and young plants are more susceptible to the pathogen. Diseased areas of the effected plant are covered in dark fungal growth. Infection of seedlings commonly occurs soon after germination.

Symptoms:

The disease progresses rapidly, and most affected plants will die within 30 days of planting. Infected onion bulbs have a black discolouration at the neck, shallow lesions on the outer scales, streaks of black mycelium and conidia beneath the outer scales and a black discolouration in bruised areas. The entire surface of the bulb may turn black, in which case, the onion will shrivel and secondary bacteria may cause a soft rot of the bulb.

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Causal organism and disease cycle:

The disease is caused by *Aspergillus niger*. It is a filamentous fungus is responsible for black rot of onion. This fungus is very common in the atmosphere and in the soil and is often found as a saprophyte. Infection will usually occur though damaged tissue. The disease is usually more severe on those crops grown in a continuous rotation of susceptible hosts. Warm, moist conditions favour the growth of this fungus. Adverse weather conditions, extreme fluctuations in soil moisture, poor seed quality, seedling damage by pesticides and

other factors that delay seedling emergence have been shown to increase susceptibility of plants to the pathogen. Older plants are more prone to infection, especially in dry soil. Seeds may be infected causing damping-off, and the fungus is also spread on seed. Postharvest decay is favoured by humid conditions which encourage conidial development on the cut tissue.

Control measures:

Fungicide seed treatments may be effective when used under conditions that promote rapid germination and emergence to prevent damping off. Post-harvest black rot can be controlled if produce is stored and transported below 15°C or under very low humidity. The disease is minimised by reducing the amount of physical damage to the storage organs.

It was observed that all the fungicides caused significant reduction in the mycelial growth, lesion diameter, spore germination and rot severity. Amongst systemic fungicides, carbendazim brought about highest reduction followed by hexaconozole, bitertanol and myclobutanil, respectively. Amongst non-systemic fungicides, the mancozeb was found the most effective followed by captan and zineb, respectively. The higher concentrations (1000 ppm and 2000 ppm) of all the fungicides proved more effective.

4) Oil Seeds:

a) Leaf spot (Tikka disease) of groundnut

Causal Organism: *Cercospora arachidicola*

C. personata

Host: *Arachis hypogea* (Groundnut)

This is most important fungal disease of groundnut. The disease occurs when the crop is two months old and under severe condition defoliation and general weakening of the plant resulting in fewer and smaller sized pods and loss in yield. Losses varying from 15 to 50 percent have been reported. When tikka disease is associated with rust (*Puccinia arachidis*) the loss may be up to 70 percent.

Symptoms:

Two fungi are responsible for causing this disease. These are *Cercospora arachidicola* (causing early leaf spot) and *C. personata* (causing late leaf spot), both may appear simultaneously on the same leaf. The former appears earlier than the latter. Lesions first appear as small chlorotic areas about 10 days after spore deposition and mature sporulating lesions are visible about 15 days after deposition. All the aerial parts are attacked. In the beginning spots appear only on leaves but later may also develop on stem.

The early spots are subcircular to irregular, 1 to 10 mm in diameter, tend to coalesce later. Lesions are commonly dark brown to black on the upper surface and light brown on the lower leaf surface. Spots on upper leaf surface are surrounded by a yellow chlorotic halo. Sporulation occurs mainly on necrotic areas of lesions on the upper surface and they turn to reddish brown at sporulation stage.

Lesions produced by late leaf spot disease are usually smaller and nearly circular, 1 to 6 mm in diameter and are commonly dark grey or black on the lower leaf surface. There is no yellow halo around them. Concentric rings of conidia are visible on the lower surface.

Both these pathogens can be distinguished after sporulation. In *C. arachidicola* the conidia are mostly confined to the upper leaf surface and occasionally on the lower one. They are sparse and not formed in concentric rings. In *C. personata*, conidial production is restricted to the lower leaf surface and conidiophores develop in concentric rings. Symptoms also develop on petioles, stem and pegs.

Causal organism and disease cycle:

***Cercospora arachidicola* (Early leaf spot)**

The mycelium is initially intercellular becoming intracellular when the host cells collapse. Stroma slight to 100 µm in diameter and are dark brown. Conidiophores five to many are fasciculate, pale golden brown to yellowish brown, darker at the base, mostly unbranched and 20-50 µm in size, continuous or with septa. Conidia are subhyaline, pale yellow, often curved, 1 to 12 septate, rounded to tunicate at the base, tip subacute and measure 35-110 × 4-5 µm.

The perfect stage is *Mycosphaerella arachidis*. Perithecia mostly scattered, mostly along lesion margin, partly embedded in host tissue, ovate to subglobose, 47.6-84 × 44.-74.0 µm in size, black, ostiolate; asci cylindrical, club shaped with short stipes, fasciculate without paraphyses, 27.0-37.8 × 7-8.4 µm in size bitunicate, eight spored; ascospores uniseriate; two celled, upper cell some what larger, slightly curved, hyaline, 11.2 × 3.6 µm in size.

***Cercospora personata* (Late leaf spot)**

The mycelium is septate, intercellular with branched Haustoria in live cells. Stroma dense and up to 130 µm in diameter. Conidiophores emerging through ruptured epidermis in dense fascicles, brown, smooth 10-100 × 3.0-6.5 µm in size, septate or continuous, usually simple, with prominent conidial scars. Conidia medium olivaceous, cylindrical, straight or curved, finely roughened wall rounded at the apex, 1-9 septate and measure 20-70 × 4-9 µm.

The perfect state is *Mycosphaerella berkeleyii*. Perithecia scattered, along lesion margin, partly embedded in host tissue, ovate to globose, 84-140 × 70-11 µm in size, black, ostiolate; asci cylindrical, club shaped, with short stipes, fasciculate, 30-40 × 4-6 µm, without paraphyses, eight spored; ascospores uniseriate, 2-celled, the upper cell some what larger, hyaline, 14.9 × 3.4 µm in size.

There is no definite information available on the source of primary inoculum. Since the perfect stage is rare in the nature. Conidia present in crop residue are probably the most important source of primary inoculum. Conidia lying in soil on diseased plant debris and those carried in the shell are the source of primary inoculum.

Control measures:

1. Plant debris from the previous crop should be removed (burnt) to avoid soil borne primary inoculum.
2. The disease is favoured by mineral deficiency so that nutrient should be supplied by soil or spray application.
3. Rotation and deep burying of the debris may help to destroy the soil borne inoculum.
4. Seed treatment is essential to eliminate the seed borne infection.
5. Disinfection of seed with shell can be accomplished by using sulphuric acid.
6. Seeds without shell are disinfected by half an hour soak in 0.5 percent copper sulphate solution or by dressing with Agrosan G.N.
7. The disease can be effectively controlled by spraying the crop with copper fungicides two or more times at intervals beginning four to six weeks after sowing.
8. Although some wild species of groundnut are highly resistant to tikka disease, resistant commercial varieties are yet to be developed.

b) Damping off of Mustard:

Causal organism: *Pythium debaryanum*

Host: *Brassica campenstris* (Mustard)

Damping off disease is widely distributed all over the world. It occurs in valleys and forests, in tropical and temperate climates and in every green house and nursery beds. The disease affects seeds, seedlings and older plants. Maximum damage is done to the seed and seedling roots during germination either before or after emergence. Poor seed germination or poor emergence of seedlings is due to damping off infections in pre-emergence stage. Older plants are not killed, but they develop stem lesions or root rot, thus retarding their growth.

Symptoms:

Damping off disease occurs in two stages: i) pre-emergence phase and ii) post-emergence phase

i) Pre-emergence phase: In this phase, the young seedlings are killed before they reach the surface of the soil. In fact they may be killed even before the hypocotyl has broken the seed coat. The radical and the plumule, when they come out of seed undergo complete rotting. Since this happens under soil surface, the disease is often not recognized at all by the farmer, who attributes the failure in emergence of seedlings to poor quality of the seed.

ii) Post-emergence phase: It is generally conspicuous and well known to workers who worked in a nursery. This phase is characterized by the toppling over of infected seedlings, any time after they emerge from the soil until the stem has hardened sufficiently to resist invasion. Infection usually occurs at or below the ground level and the infected tissue appears soft and water soaked. As the disease advances the stem become constricted at the base and the plants collapse. Seedlings that are apparently healthy one day may have collapsed by the following morning. Generally the cotyledons and leaves wilt slightly before the seedlings are prostrated, although they may remain green and turgid until collapse occurs.

In the field, the disease is most severe when the moisture in the soil is medium to high and the temperature is comparatively high. When conditions are favourable for development of the disease, damping off is often responsible for as much as 90 percent killing of seedlings. In specially susceptible plant species seedling losses of 25 to 75 percent occur yearly. Most of the loss is due to pre-emergence damping off.

Causal organism and disease cycle:

The disease is caused by *Pythium debaryanum*. The mycelium is white, intracellular, slender, profusely branched with coenocytic hyphae. There are produced terminal and intercalary sporangia which may be spherical, filamentous or of various shapes. They germinate to develop a vesicle in which zoospores are formed. Released zoospores swim for some time, encyst and infect the host tissue by germ tube.

Antheridia and oogonia develop at the end of short hyphae. After fertilization, thick walled oospores develop which are resistant to extremes of temperatures and other adverse factors. They go under a period of rest and germinate in the next season. Each oospore produces a vesicle at the end of germ tube. Zoospores are formed inside the vesicle. Both sporangia and zoospores germinate at low temperature of 10-18^{0}C to form zoospores, but directly in to germ tube at temperature above 18^{0}C. The pathogen remains in water, soil, on dead plant and animal matter as saprophytes. The fungus enters the seed by direct penetration of moistened, swollen seed coats or cracks. Further penetration of embryo or emerging seedling tissue occurs through mechanical pressure and dissolution by enzymes. Pectinolytic enzymes dissolve the middle lamella in between cells, cellulolytic enzymes degrade cell walls and proteolytic enzymes breakdown the protoplast of invaded cells.

Control measures:

1. Soil disinfection with chemicals like formalin : water (1:50), captan, thiram, blitox-50 etc in a 0.2 – 0.5% suspension.
2. Soil sterilization by steam or dry heat.
3. Seed protectants which include several types of chemicals as phygon, agrosan GN, arasan, cerasan, captan, blitox-50 and others. These are applied to seeds in dry or wet form.
4. Seed treatment followed by spraying of seedlings with ziram, chloranil, captan, soluble copper etc.
5. Cultural practices including good drainage, improvement of soil aeration, check on excessive use of nitrate fertilizers etc.

5) Cash crop:

a) Grassy shoot of sugarcane

Causal Organism: Phytoplasma (MLO)

Host: *Saccharum officinarum* (sugarcane)

The disease was first noticed in India in 1919 in Maharashtra. It is one of the serious disease found in A. P., Tamilnadu, Orissa, Bihar, U. P., Punjab and Rajasthan. It is also reported from Burma, Shrilanka and Sudan also. Numbers of varieties are affected in India. Losses from grassy shoot are in the form of reduced germination in setts from diseased canes, stunted growth and poor juice quality.

Symptoms:

The disease has been variously described as 'new chlorotic disease', 'albino disease', 'yellowing disease', 'bunchy disease' or 'leaf tuft'. The most pronounced symptom is the grassy appearance of the affected shoot. Shoots growing from diseased setts remain dwarfed or 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 culm. The leaves appear yellowish and in some cases may entirely devoid of any pigment. If many of the tillers are affected in this manner the entire shoot dries. Leaves exhibit straight, long, white or light green or yellowish streaks. The lower nodes produce large number of grassy shoots. In systemically infected canes the disease appears May-June. In sprouts raised from top buds the symptoms appear late as compared to sprouts raised from lower buds.

Causal organism and disease cycle:

The disease was first considered a viral disease. However, since 1971, it has been suggested that it is caused by a mycoplasma like organisms (MLO). Corbett et al (1971) first suggested that the disease was associated with mycoplasma like bodies and Rishi et al (1973)

demonstrated with the help of electron microscopy, the presence of mycoplasma like bodies in the phloem of diseased canes.

The organism is present in the sieve tubes of phloem as ovoid, spherical or irregularly shaped bodies. The size of ovoid or spherical bodies is 300-400nm in diameter and that of protruding filament 30-50nm in diameter. These cells lack cell wall and are bounded by a single triple layered membrane. They contain ribosomal granules and DNA strand. Mostly the cells are concentrated towards the periphery of the cell, near the cell walls but often the cells are fully loaded with these bodies and then the cells dies followed by death of the MLOs due to starvation. In some cases, MLOs are seen lying close to sieve pores and turn filamentous while passing through the sieve pores. Concentration of MLO is high in canes growing at high temperature (around 30^0C) and such canes show severe symptoms. At lower temperature the symptoms are less severe and the number of MLO in the cells is also lower.

The grassy shoot MLO perpetuates through diseased canes used for seed and spread through diseased setts and cane cutting knives (sap transmission). Ratooning of the diseased crops is an important source of perrennation. Introduction of the pathogen in new fields is mainly through diseased seed setts. Many aphids and a leaf hopper have been reported to spread the MLO from plant to plant in the field but only to a limited extent. The vectors are *Rophalosiphum maidis, Aphis sacchari* and *Aphis indosacchari*. Transmission by *Cuscuta campenstris* has also been reported from Bihar (Jha et al., 1973). The disease has been transmitted from sugarcane to sorghum and from sorghum to sugarcane by using *Aphis sacchari*.

Control measures:

1. Healthy seed setts should be taken from a field where grassy shoot is not present even in traces.
2. During the early stage of crop growth insect vectors can be controlled by weekly spraying of 0.16 percent malathion.
3. Diseased clumps should be dug out and destroyed.
4. Heat therapy has also been recommended. The setts should be kept in hot water at 50^0C for two hours or in hot air at 54^0C for eight hours.
5. Tetracycline antibiotics (250ppm) applied to seed setts under negative pressure has been found to eliminate symptoms. Single bud setts can be treated with such antibiotics.

b) Downy Mildew of Grapes

Causal organism: *Plasmopara viticola*

Host: *Vitis vinifera* (Grapes)

This disease was known to be endemic to the U.S.A. before 1870. However, since 1875 its epidemics were known to occur in France where it caused heavy losses to wine industry. At present the disease is known to occur in all wet grape vine growing areas of the world. In India the disease is common in Maharashtra. Downy mildew affects the leaves, fruits and vine and causes losses through necrosis of leaf tissues and defoliation; low quality and destroyed fruits; dwarfing and killing young shoots.

Symptoms:

Symptoms of the disease appear on all aerial and tender parts of the vine. They are more pronounced on leaves, young shoots and immature berries.

On young leaves, first symptoms appear as oil spots circular yellow translucent lesions, often surrounded by brown halo on the upper surface. Later spots may coalesce to cover the entire surface. Masses of white downy sporangiophores and sporangia develop on the underside of each oil spot. During hot and dry weather or after sporulation the oil spots turn brown or reddish brown, dry out and die. On mature leaves, these appear a mosaic of small, angular, yellow to reddish brown lesions on upper surfaces limited by veins. Severely infected leaves fall prematurely. When young shoots and tendrils are infected, they turn brown and become stunted, distorted and necrotic.

Infected inflorescences first turn yellow brown and may develop white brown sporangia during periods of high humidity at nights. When young peduncles are infected, entire cluster may die. Occasionally, parts of clusters wither and dry when only pedicels are infected.

Young berries are most susceptible to infection from setting until 5 to 6 mm in diameter. Infected berries stop growing, harden and become leathery and wrinkled dull bluish green before turning brown, withering and falling from clusters. Older berries may be resistant, but they may be killed when pedicels and cluster stems are diseased. Infection of green young shoots, tendrils, leaves, stems and fruit pedicels result in stunting, distortion and thickening of tissues. Entire shoots may become covered with white downy growth of the fungus.

Causal organism and disease cycle:

The disease is caused by *Plasmopara viticola*. The fungus has intercellular mycelium with thin walled and hyaline hyphae. Their width may be 1-2 to 60 microns. Haustoria are spherical in shape. In humid weather, 4 to 6 sporangiophores emerge through each stomata on the underside of the leaves and on the stems or very rarely by pushing directly through the epidermis. In young fruits these emerge through lenticels. Each sporangiophore produces 4-6 primary branches nearly at right angles to the main axis. Each primary branch produces 2-3 secondary branches, in a similar way. At the tip of secondary branches, single lemon shaped sporangia are formed. The sporangia are blown by wind or rarely transported by water. Each sporangium germinates in presence of free moisture to produce 1-6 zoospores. The zoospores swim for some time, encyst and then produce germ tube which penetrates the plant. The fungus also produces oospores mostly in tissues adjacent to the midrib. Oospores are thick walled and germinate to form a germ tube that bears an apical sporangium. The sporangium produces 8-20 zoospores which may infects the host plant.

In those areas where grape vines remain evergreen the pathogen may survive as mycelium in twigs. However, the main sources of survival are the oospores, embedded in dead leaves and occasionally in dead berries and shoots. Dead leaves and shoots degenerate and release oospores in soil. They germinate either on the ground or on grape plant on which they are carried by wind or splashing rain drops. The zoospores infect wet leaves, penetrating them through stomata. The mycelium develops inside the leaf. From this mycelium, sporangiophores are produced and the sporangia from them are carried by wind or rain to healthy plants. Zoospores from these sporangia cause secondary infections through stomata or lenticels, thus spreading the disease rapidly in orchard. In the stem, it invades the cortex, ray parenchyma and pith. Infected stem becomes distorted and hypertrophied, that may be finally killed. At the end of growing season oospores develop in leaves and sometimes in shoots and berries.

Control measures:

- Sanitation: fallen leaves and twigs should be collected and burnt.
- Plantation: Planting vines with proper spacing reduces humidity and allows free aeration.
- Plants should train in such a manner that leaves do not remain near the ground.
- Use of resistant varieties: Amber Queen, Champiuon, Cardinal, Champa, Red Sultana.
- Use of fungicides: Fungicide sprays have been found quite effective in preventing secondary spread of downy mildew. Zineb or Maneb (0.2%), Captan (0.2-0.5%), Bordeaux mixture (4:4:50), or copper oxychloride such as Blitox-50 (0.3%) should be sprayed according to the following schedule:

  i.immediately after pruning,

  ii.three to four weeks after pruning,

  iii.before the buds open,

  iv. when berries have formed, and

  v. during growth of shoots.

Spraying should be stopped two weeks before harvest. Some workers have found aureofungin as an antibiotic effective against the disease. Among newer fungicides, metalaxyl (Ridomil) has been effectively used against downy mildew of grapes. It inhibits sporangial formation through action of its volatiles.

c) Citrus Canker

Causal organism: *Xanthomonas citri*

Host: *Citrus* spp (lemon)

Citrus canker is bacterial disease is widespread threat in all the citrus growing areas of the world. The disease said to have originated from China. It was spread to Europe and U.S.A. The disease still quite serious in India, China, Japan and Java. The disease attacks most of the varieties of citrus.

Symptoms:

Leaves, twigs as well as fruits are attacked, developing necrotic brown spots with a coarse surface. On young leaves, in the beginning lesions appear as small white specks. Soon the lesions become discernible after few weeks they develop in to brown necrotic spots of 1 to 2 mm in diameter. The lesions become raised forming a spongy white eruption. As the lesion enlarges further the spongy eruption begins to collapse and brown depressions appear in their central portion forming a crater like structure. The margin of the lesions remains to be raised above the surface of the host tissue. As the disease progresses, the central portion of the lesion becomes grayish white, hard and appear as corky. Yellow haloes are formed around such mature and advanced lesions. More or less similar lesions develop on twigs and fruits. The lesions are 1 to 9 mm in diameter on leaves and up to 1 cm in diameter or length on twigs and fruits. When lesions develop on midrib or petiole, the leaf is more likely to defoliate. The lesions may enlarge rapidly and coalesce encircling the entire twig. The canker lesions develop due to hypertrophy and limited hyperplasia in parenchymatous tissues. The lesions consist primarily of hypertrophied cells, with a small number of hyperplastic cells along its margin.

Causal organism and disease cycle:

The disease is caused by *Xanthomonas citri*. The bacterium overseasons chiefly in leaf, twig and fruit canker lesions, and if splashed onto young tissue this enters them

through stomata or wounds. Old tissues are penetrated only through wounds. Several cycles of infection can occur on fruits, and thus fruits have lesions of different sizes. Free moisture and strong winds often favour the spread of the disease.

The cells are Gram-negative, straight rods, measuring 1.5-2.0 × 0.5-0.75 µm, with a single polar flagellum; in chains, with capsules; non-spore forming, and aerobic. The bacterium is not able to survive saprophytically. This has been shown to be due to antagonism of other soil microbes to this bacterium under natural conditions. It can survive hardly for 2-3 months in lesions of diseased defoliated leaves only under dry conditions. The bacterium could be detected on the surfaces of some weeds growing in citrus groves.

The bacterium survives chiefly in parasitic form in holdover cankers on leaf and twig, as well as in bark tissues for long periods. The bacterium enters through stomata and wounds. Disease is favoured by mild temperatures and wet weather. The bacteria from the cankers are mostly disseminated by driving rains and by insects such as citrus leaf miners (*Phyllocnistis citrella* and Thosconyrsa citr*i*). However, the chief agent of dissemination and introduction in to new localities is man himself who transfers the disease through infected nursery stock.

Control measures:

To combat the disease following measures can be suggested.

1.Eradication: This is accomplished by removing the trees with advanced infection and burning them.

2.Pruning: The infected trees may be cured by removing the diseased foliage and branches with pruning scissors and then spraying the trees with 1% Bordeaux mixture at regular intervals.

3.The use of disease free nursery stock for planting.

4.The fallen infected leaves and twigs should be collected and burnt.

5.Spraying: spraying with Bordeaux mixture and lime sulpher is useful measure. It should be done during the first three months after the beginning of fruit formation. Spraying should commence before the onset of rains and repeated during rainy season.

6.Citrus nurseries should be raised in places away from the regions of heavy and protracted rainfall.

7.The disease can be checked by periodic sprays with Streptomycin sulphate at 500 ppm.

d) Angular leaf spot of cotton

Causal organism: *Xanthomonas campenstris* pv *malvacearum*

Host: *Gossypium* spp (cotton)

The disease was first reported in Alabama, U.S.A. in 1891 and now known to occur in all major cotton growing regions of the world. From India, the disease was first reported from Madras in 1918 and now occurs in Maharashtra, Madhya Pradesh, Andhra Pradesh, Tamil Nadu and Uttar Pradesh. Annual losses due to the disease in India range from 5% to 25%.

Symptoms:

The pathogen attacks aerial parts of plant at different stages of growth. On seedling certain symptoms develops as dull green flaccid areas from the periphery of leaves, elongate water soaked dark brown area on leaf and yellowing of leaves as well as tip rotting. Cotyledons become dry and wither and leave collapse and die. The bacterium invades in all tissues of every part.

In adult plants, the leaves attacked first, and the earliest symptoms are the appearance of water soaked spots on the lower surface. These spots enlarge, turn brown and form angular dead areas bounded by veinlets, ands become visible on the upper surface. On the stem, there appear elongate grayish to black lesions (black arm). In severe infection, there is deep cracking. Similar lesions may also develop on petiole and fruiting branches. On the bolls, there may develop small, round water soaked spots that turn brown later.

Causal organism and disease cycle:

The disease is caused by *Xanthomonas campenstris* pv *malvacearum* the bacterium is Gram-negative, rod shaped, 1.2 × 0.9 µm, with single polar flagellum, singly or in pairs, rarely in chains, non-spore forming, facultative aerobe. Optimum temperature for growth is 31^0 to 32^0C and thermal death point is 50^0C.

Seeds, trash and self-sown infected seeds have been shown to carry over the infection from one season to another. The pathogen remained active in dried leaves for 17 years and could survive for eight days in water suspension as well as in both moist and air dried soil at

21^0 to 33^0C. The main source of primary infection is seed which may carry the pathogen both externally and internally. Plant debris may also form as important source of infection, especially in areas where cotton is grown as continuous crop. Other source is self-sown infected seeds. High humidity and moderate temperature (28^0C) favour the disease.

Two pests of cotton, *Earias* spp and *Dysdercus koenigii* are also known to transmit the disease mechanically. Besides this, a large number of plants are also known as collateral hosts of the pathogen, though their role in disease cycle is yet to be defined.

Control measures:

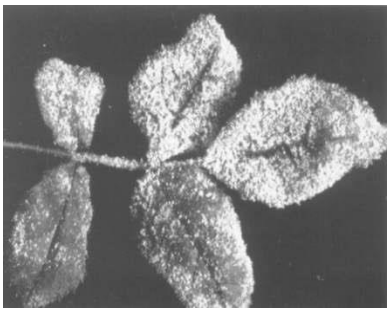
- Removal and destruction of diseased plant debris.
- Deep ploughingafter harvest reduces the survival of pathogen.
- Destruction of collateral hosts, crop rotation, late sowing, early thinning, good tillage, early irrigation also reduce disease incidence.
- Seed treatment: Immersing seeds in sulphuric acid for 10-15 min, rinsed by water and finally drying and treating them with Agrosan GN, Cerasan etc. Seed dressing with oxycarboxin and carboxin have been recommended (2kg/kg seed). These can be used as foliage sprays (1.5-2.0kg/ha). Three sprays first at 4-6 week old crop and subsequent sprays at 10 to 15 days intervals.
- Disease resistant varieties: 101-102B, BJA-592, P-14-T-128, Reba-B.50, HG-9, Tamcot-CAMD-E and BJR.

6) Ornamentals:

a) POWDERY MILDEW OF ROSES

Powdery mildew is very destructive, affecting plants grown out of doors and in greenhouses. The disease occurs wherever roses are grown. Under conditions that are favorable for disease development, powdery mildew can cause complete defoliation. Epidemics can be expected any time during the growing season when the rainfall is low or absent, the days are warm and dry, and the nights are cool and damp. Nearly all species and cultivars of roses are susceptible under conditions that are favorable for disease development. Losses from powdery mildew occur through a reduced aesthetic value that is seen in fewer flowers of poorer quality, a lowered photosynthetic efficiency that results in reduced plant growth, and a reduced salability for roses as cut flowers.

Symptoms:



On garden roses, new shoots in the spring are dwarfed, distorted, and covered with a whitish gray mildew growth. On expanding leaves, mildew first appears on the upper leaf surface as irregular, light green to reddish, slightly raised blister like areas. The typical dense, powdery white growth (mycelium, conidiophores, and spores) of the mildew fungus soon appears. Severely infected young leaves become curled or irregularly twisted and are usually covered with enlarged, whitish gray, powdery, mealy, or felt like patches of the fungus. These leaves often turn reddish purple, under the mildew growth, then yellow, dry, and drop prematurely. Older, infected leaves are not usually distorted, but develop round-to-irregular areas covered with the flour like mildew growth. On highly susceptible rose cultivars, the buds, young stems, thorns, peduncles, fruit sepals, and even flower petals may become infected and entirely covered with the typically dense, flour like growth. Flower petals may be discolored, dwarfed, and may fail to open properly; the flowers may also die early. The growing tips and flower buds may be malformed and killed, but the death of an entire plant is rare. Plants can be severely stunted if they are heavily infected early in the growing season.

Disease Cycle:

Powdery mildew is caused primarily by the fungus *Sphaerotheca pannosa* var. *rosae*. The powdery mildew fungus overwinters as dormant mycelium in bud scales and rudimentary leaves within the dormant buds. Infected buds break open in the spring and develop into systemically infected shoots. The fungus sporulates on these shoots, producing large numbers of microscopic spores (conidia) in chains that are carried by the wind or other means to healthy rose tissue where they infect the upper and lower leaf surfaces, thus initiating a new disease cycle. The fungus survives in the Midwest in the winter as cleistothecia, which appear as black specks embedded in the mealy or felt like mildew growth on rose stems, thorns, and fallen leaves. The minute cleistothecia are formed within the mycelial mat at the end of the growing season. During warm and humid weather in the spring, a cleistothecium absorbs water and cracks open to discharge a single small sac or ascus containing 8 spores (ascospores). The microscopic ascospores are carried by the wind or splashing rain to healthy rose tissue and are capable of causing infection. In greenhouses or mild climates, where roses and powdery mildew both grow continuously throughout the year, cleistothecia are absent and only conidia are formed. New infection cycles are produced more or less continuously. Conidia and ascospores that land on the surface of a rose germinate and form a holdfast structure (appressorium) on the leaf or stem surface. From the bottom of the appressorium, a fine penetration tube or hypha pierces the cuticle and enters the epidermal cell where a globose feeding structure, or haustorium, is formed. With further growth on the plant surface, the fungus develops a dense, branched network of

hyphae. Many additional haustoria form in other epidermal cells. Short, erect branches, or conidiophores, develop at the same time from the surface hyphae, producing a barrel-shaped conidium at the end of each conidiophore. Successive conidia, with one formed each day; commonly remain attached in chains, giving the characteristic powdery white appearance. The conidia eventually break away and are carried by air currents, splashing water, or other means to new infection sites. Handling rose plants, insects, mites, and snails also helps spread conidia. As many as 3 million spores may be formed on one square inch of infected tissue over a period of several weeks.

Control:

- Use of only top-quality, disease-free plants of resistant cultivars and species from a reputable nursery.
- Prune roses in the fall and in early spring, according to type and cultivar. All dead wood should be removed and burned.

- Maintain rose plants in high vigor: soil should be well prepared; avoid planting of large shrubs and trees; plant spacing; avoid excess use nitrogen; regular irrigation; weed eradication.
- Spraying fungicides on aerial parts at 7 to 14 day intervals.
- Vaporized sulfur gives excellent control of powdery mildew in greenhouses.

7)\Weeds

a) Rust of Euphorbia

Causal Organism: *Melampsora euphorbiae*

Host: ***Euphorbia hirta***

Classification:

- Division- Mycota
- Sub Division- Eumycotina
- Class- Basidiomycetes
- Order- Pucciniales/ Uredinales
- Family- Melampsoraceae
- Genus- *Melampsora*
- Species- *euphorbiae*

Distribution:

It is an herb found in many part of the world. Locally known as Dudhani a weed of uncultivated areas was found infected with rust disease. The plant is native from India, but it is tropical weed found especially on roadsides and wasteland. The plant has a reputation for increasing milk flow in cattle(goat).

Symptoms:

The pustules were orange yellow in colour necrotic spots were appearing on upper surface of leaves with orange spore masses on the lower surface.

Causal organism and disease cycle:

Rust of Euphorbia was caused by *Melampsora euphorbiae*. It is an autoceous rust infects a large number of wild and cultivated species of Euphorbia. Uredina 0.3- 0.5 mm diameter with capitates paraphyses, uredinospores are subgloboid or ellipsoid with 1.0 to 2.5 micrometer thick, hyaline and echinulate walls and teliospores were 27 to 55 micrometer long and 13 to 16 micrometer wide with pale brown walls 1.0 to 1.5 micrometer thick apically thickened to 2.5 micrometer.

Control measures:

- Eradication
- Use weedicide like 2-4 D, Atrazine weedicide

8) Plant Diseases on Trees

a) Cercospora on *Albizia* fruits

Causal Organism: *Cercospora albizae*

Host: *Albizia lebbeck*

Classification:

- Division- Mycota
- Sub Division- Eumycotina
- Class- Ascomycetes
- Sub Class- Dothiodeomycetes
- Order- Capnodiales
- Family- Mycospherellaceae
- Genus- *Cercospora*
- Species- *albizae*

Distribution:

Albizia lebbeck is used in pathway farming system in Africa and India and is under trial in Thailand. Lowry et. al. (1994) highlighted the fact that in northern Australia the species, as well as providing stock feed directly, appears to enhance pasture production and quality, probably due to shading and related improved soil moisture status and fertility from litter breakdown . The shade is also a benefit in animal production in the dry tropics.

Symptoms:

It causes grey, circular shot hole lesions. Spots are circular, 1-3 mm in diameter, white center and dark purple to brown margin.

Causal organism and disease cycle:

Albizia lebbeck fruit spot caused by *Cercospora albiziae*. Conidiophores 2-8 in a spreading fascicle, pale to very pale olivaceous brown, paler and narrower towards the apex, septate, not branched, geniculate, subtruncate at the apex, 30-140 x 4-6.5 micrometer, conidial scars thickened conspicuously. Conidia hyaline, acicular to obclavate, straight to mildly curved, indistinctly multiseptate, acute at the apex , truncate at the base with a thickened hilum, 25- 150 x 3.5- 7 micrometer.

Disease cycle-

Cercospora survives only as a long as infected fruit debris is present , however, it is a poor soil competitor. The debris on the soil surface is a cause for primary inoculation that infects the incoming for the next season. Conidia (asexual spores) are produced by *Cercospora* in the debris through wind dispersal or rain. The conidia are disseminated and eventually infect new legumes of *Albizia*.

In order for the pathogen to actually infect the host, high relative humidity and moisture (dew) on the leaves are necessary for inoculation. Primary inoculation occurs on lower region of younger legumes, where conidia germinate and penetrate through stomata via a flattened hyphal organ, an appressorium. *Cercospora* is a typical in that its conidia can grow and survive for days before penetration, unlike most spores that need to penetrate within hours to ensure survival. Once infection occurs, the conidia are produced in these lower regions. Assuming favorable weather conditions, these conidia serve as secondary inoculums for upper leaf region. Additionally, wind and heavy rains tend to disperse the conidia during many secondary cycles to other parts of the field causing more secondary cycles of infection.

Control measures:

- Seedling and saplings may be dug out or removed by hand pulling while larger trees may be cut and the stumps treated with herbicide.