

INTRODUCTORY PLANT PATHOLOGY PP 402

Fungal diseases

Fungi are eukaryotic, spore-producing, achlorophyllous organisms with absorptive nutrition that generally reproduce both sexually and asexually and whose usually filamentous, branched somatic structures, known as hyphae, typically are surrounded by cell-walls.

MORPHOLOGY

Thallus: Word thallus is derived from a Greek word i.e. ‘*Thallos*’ means a shoot.

Body of fungus is called thallus.

➤ It may be defined as relatively simple plant body that lacks stems, shoots and leaves.

It is the somatic phase of fungi.

Hyphae: Thallus consists of microscopic threads or filaments that branch in all directions, spreading over or within the substratum, utilized for food. They are known as hyphae. A hypha may be defined as thin, transparent, tubular wall, filled or lined with layer of protoplasm, varying in thickness. OR Single thread-like structures of thallus are called hyphae. It may be branched or unbranched and have protoplasm with nuclei.

Types of Hyphae:

1. **Septate** (Interrupting the protoplasm into various compartments): A type of hyphae with more or less regularly occurring cross walls.

2. **Aseptate** or **coenocytic** (Having no septa): The hyphae in which the nuclei lie in a common matrix or the hyphae having no septa.

Septum: The cross wall or partition, which interrupts the protoplasm at various intervals and divide hyphae into various compartments.

Mycelium: The mass of hyphae constituting the thallus of a fungus is called the ‘mycelium’. It may be uni, bi, or multi-nucleate.

Intercellular fungi: The fungi in which mycelium (haustoria) grows intercellularly (**Between the host cells**).

Intracellular fungi: The fungi in which mycelium (haustoria) grows intracellularly (**Within the hosts cells**).

Hhaustoria: These are outgrowth of somatic hyphae, act as absorbing organ and can penetrate into host cells, may be knob like in shape elongated or branched like miniature root system Intercellular fungi generally get nourishment through haustoria.

Appressorium: A swollen tip of a hypha or germ tube that facilitate attachment and penetration into the host by the fungus.

Spore: The reproductive unit of fungus. OR It is a minute simple propagating unit like seed but without embryo that serves in the reproduction of fungi.

Conidiophore: These are specialized hyphae on which conidia are produced.

Sporangium: It is a sac like structure in which entire protoplasmic contents are converted into spores.

Sporangiophore: Hyphae bearing a sporangium at its tip are called sporangiophores or Hyphae on which sporangium is produced is called sporangiophores. Some important Phyla of **Kingdom Eumycophyta** (*True Fungi*) and diseases caused by them as under:

- Phylum *Chytridiomycota*
- Phylum *Zygomycota*
- Phylum *Ascomycota*
- Phylum *Basidiomycota*

1-Phylum Chytridiomycota

The Phylum *Chytridiomycota* contains the **single class Chytridiomycetes** having the following important characters

1. **Coenocytic** structure of thallus, whether a globose or ovoid (oval shaped) structure, an elongated simple hypha, or a well developed mycelium.
2. Production of motile cells (**zoospores**) with single, posterior **whiplash flagellum**.

3. Zygote is converted into resting spore or resting sporangia.
4. They have a slippery, glistening (shiny) surface due to the presence of chitin and glucan in their cell walls.
5. **Period of encystment:** Period during which a spore remain in the form of a cyst is called period of encystment.
6. Period between germination and encystment or between two encystments is called swarming period. During the swarming period spores are motile (able to move or swim). It is also called motility or swimming period.

This phylum is not important as plant pathogenic group the only disease caused by them is

Black Wart of Potatos (*Synchytrium endobioticum*)

Family: Synchytriaceae

Order: Chytridiales

This disease is not common in Pakistan.

Phylum Zygomycota

Characters:

1. Mycelium is extensive and hyphae are coenocytic.
2. Production of a thick walled resting spore called a **zygospore** (Gr. *Zygos* = yoke + *spora* = seed, spore). These zygospores are produced by the fusion of two iso-gametangia. These Zygospores are produced within **Zygosporangium**.
3. **Asexual reproduction** is by sporangiospores although some species produce other types of spores.
4. On the sporangiophore a large no. of small structures are found these are known as sporangiola.
5. **Some species are dimorphic (having the capacity to grow as a single cell like yeast or to produce mycelia).**

Phylum Zygomycota has two classes;

- a. *Zygomycetes*
- b. *Trichomycetes*

SOFT ROT OF FRUIT AND VEGETABLES

Disease occurs throughout the world on harvested fleshy organs of fruits, vegetables and flower crops in storage, transit and market. The most affected crops are sweet potato, strawberries, all cucurbits, peaches, cherries etc. Several other bulb, corms and rhizomes of flower crops like Gladiolus, tulips are also susceptible.

Symptoms: Affected areas of fleshy organs appear water soaked and very soft at first. If skin of infected tissues remains intact, it gradually loses moisture, ultimately shriveling to a mummy. But often the skin ruptures during handling or under pressure by surrounding fruits and whitish yellow liquid drops out. Soon the fungus grows outward through the wounds and covers the affected portion by producing tufts of grey sporangiophores bearing black sporangia at their tips. This fungal growth extends to the healthy portion and even surface of containers when they become wet with the exuding liquid. Affected tissue at first gives off mildly pleasant smell but after the invasion of yeasts and bacteria, turn into sour odour. If loss of moisture is rapid, infected organs dry up into mummies. On the other hand, they breakdown and disintegrated in a watery rot.

Causal organisms: *Rhizopus stolonifer*

Order: *Mucorales*

Family: *Mucoraceae*

Disease Cycle: Sporangiospores may survive for many months but this fungus is so perfectly adapted to saprophytic living that its spores may be found anywhere in the field or in the storage in almost any season. Actively growing tissue is seldom affected so and fruit products. The fungus is a wound parasite. After the infection is established, rotting takes place by the secretion of an enzyme, which dissolves the middle lamellae of the host cells causing their disorganization. After disorganization of the host tissue, the other microorganisms may invade immediately.

Epidemiology: Optimum temperature range for the growth of fungus is 23-26°C, but other species causing this disease may grow best even at 40°C.

Infection is influenced by environmental factors and the host reaction. Once the cork cambium is formed no infection takes place. Cork cambium formation takes place at high relative humidity (95-100%) and within the temperature range of 19-33°C (maximum at 33°C).

Control:

1. Sanitation of storage rooms.
2. Disinfestations of walls, floors and containers, etc. with CuSO₄, Formaldehyde, Sulphur fumes, or chloropicrin

Since the fungi causing this disease are wound parasites, care should be taken while digging and handling the produce to avoid injuries. After storage, the temperature of warehouse should immediately be raised to 28-33°C for two weeks (to hasten the formation of cork cambium to seal the out surfaces against the entry of the pathogen). After this period the warehouses are brought to the normal low temperature, which should not be lower than 13°C to avoid internal necrosis.

2-Phylum Ascomycota

Characters:

1. Hyphae or mycelium is well developed with distinct walls which are made up of chitin.
2. Hyphae are **septate** and septa of the hyphae have a **simple pore** and **woronin bodies** are present.
3. Woronin bodies are **spherical, hexagonal** or **rectangular** structures that are usually associated with a septum and can only be visible with an **electron microscope**.
4. Sac like structure is produced which is known as ascus.
5. Within ascus (Pl. asci) spores known as ascospores are produced, which are usually eight (8) in number.
6. This group consists of **1500 species** and includes ascus forming **yeasts, black and green molds, powdery mildews**, cup fungi, **morels** and **truffles**.

RICE BLAST

Occurrence and Importance:

The disease was first of all reported from **China** in 1637 and then in Japan in 1704. Presently, the disease is prevalent in almost all the rice growing countries of the world but is especially serious in humid areas. This has been considered as the most important disease affecting the crop and causing losses up to 70%.

Causal organism: Rice blast is caused by the Ascomycete fungus, *Magnaporthe grisea* Barr anamorph *Pyricularia grisea* Sacc., synonym ***P. oryzae*** Cav.).

Order: **Moniliales**

Family: **Dematiaceae**

Symptoms: The fungus attacks all the aerial parts of plant at all the growth stages. On leaves, it appears in the form of **small bluish flecks** (about 1-3 mm in dia.). **On older leaves**, they remain **circular** but on younger leaves they enlarge up to several centimeters in length and about one centimeter wide. By this time, central portion becomes pale green or dull grayish green and water soaked in appearance, while outer rim is dark brown. In older spots center becomes grey or almost straw coloured. Similar spots are also formed on leaf sheath.

Brown to black spots or rings is formed on the **rachis** of maturing inflorescence. Ears may also show similar spots. **The most characteristic appear on culm**. The neck becomes shriveled and covered with grey fluffy mycelium. If the infection takes place much earlier before grain formation, the latter are not filled and panicle remains erect. But if infection takes place after some grains have been formed, panicle hangs down. Due to necrosis of neck tissue, ear tends to break and fall off causing maximum damage.

Disease Cycle: The fungus survives on collateral hosts such as sugarcane and some other graminaceous plants. It also survives in the form of chlamydospores and sclerotia. Survival in the soil is extinct and seed-borne inoculum fails to initiate disease due to high soil temperature. The conidia are released from the conidiophores by dew or rain and are disseminated by wind currents mostly up to 1-2 m from the source. They germinate in 3-4 hours and penetrate the host cells directly. The incubation period is about 4 days and the next crop of conidia is ready in 6-7 days, which cause secondary infection.

Epidemiology: The disease severity depends on variety, age, host nutrition and meteorological factors such as temperature and moisture. **Rice is susceptible to blast at three distinct growth stages i.e., seedling, rapid tillering** (15-30 days after transplantation) and **ear or neck emergence stage**. Silicone content is directly proportional with resistance. ***P. oryzae* is considered a night pathogen** and all the vital processes of disease cycle (**spore release, germination, infection and spore production**) **require free water, which** is

provided by night time dew. Night temperature of 20°C alternating with day temperature of 30°C with 14 hours of light is the most favourable for infection by *P. oryzae*. Subsequent progress of the disease is favoured best at 15°C.

Control:

1. Precautionary measures: Field sanitation, destruction of alternate hosts, early planting and seed treatment with proper seed dressing fungicides such as Benlate (0.25%).
2. Foliar sprays with Benlate, Bavistin (0.1%).

BROWN LEAF SPOT OF RICE

Occurrence and Importance:

Brown spot is one of the most important rice diseases. The disease can adversely affect the yield and milling quality of the grain. Under environmental conditions conducive to disease, yield loss estimates range from 16 to 40 %.

Causal Organism

Brown spot is caused by the fungus *Bipolaris oryzae* (formerly known as *Helminthosporium oryzae*).

Order: Moniliales

Family: Dematiaceae

Symptoms

Brown spot symptoms initially appear as small circular to oval spots on the first seedling leaves. Leaf spots are observed throughout the growing season and may vary in size, shape and color depending on the environmental conditions, age of the spots, and the degree of susceptibility of the rice variety. Small spots are dark brown to reddish brown while large spots have a light, reddish-brown or gray center surrounded by a dark to reddish-brown margin. The lesion. Spots on the leaf sheath and hulls are similar to those on the leaves. Early brown spot lesions are difficult to distinguish from blast disease lesions, but mature blast lesions are usually spindle or diamond shaped. Severely infected leaves may die before maturity and these plants will produce lightweight or chalky kernels. Infection occurring directly on the kernels will significantly reduce grain yield and quality. Infected glumes and panicle branches have a black discoloration.

Disease cycle

Bipolaris oryzae is seedborne. It can also survive on infected rice straw and stubble. It spreads from plant to plant in the field by airborne spores. Species of some 23 grass genera have been reported to be susceptible to infection by *Bipolaris oryzae*. It is not known how many of these species may act as alternative hosts in the spread of the disease to rice. Severe leaf spotting is often associated with weak plants growing under stressful conditions such as dense plant stands, water stress, inadequate fertilization or herbicide toxicity.

Epidemiology: Disease development is favored by high relative humidities (86-100%) and temperatures between 68° to 78° F. Leaves must be continuously wet for 8 to 24 hours for infection to occur.

Control:

Plants that grow in soils with nutritional deficiencies or in soils where nutrient uptake is hindered are more susceptible to infection.

1. Soils known to be low in plant-available silicon should be amended with calcium silicate slag before planting. Improved silicon nutrition not only enhances plant resistance to brown spot, but will increase rice yields.
2. The usual slag application rate is two tons per acre, although higher rates have been shown to be even more effective in reducing brown spot incidence and increasing yields.
3. In one recent experiment, use of Fungicide Tilt® alone, and in slag combination, proved effective.
4. Fungicidal seed treatment, however, has proven very effective in reducing seedling brown spot disease.

EARLY BLIGHT OF POTATO

Causal organism: *Alternaria solani*

Order: Moniliales

Family: Dematiaceae

Symptoms: The initial symptoms appear in the form of small, isolated scattered pale brown spots on leaves with deep greenish-blue fungal growth. The lower leaves are attacked first and then the disease progresses upwards. In the necrotic tissue, concentric rings develop, which give a target board effect. There is usually a narrow chlorotic zone around the spots fading into normal green. In case of severe infection, the leaves are shriveled and fall down. The stem lesions often girdle it and cause “Collar-rot” in young seedling plants leading to collapse of branches or entire above ground portion. On tubers the affected region is darker than the healthy area and the lesion soon shrinks slightly as dry, corky rot develops.

Disease Cycle: Mycelium and conidia can survive in plant debris and on seed tubers. The conidia germinate in moist weather and cause primary infection directly or through stomata, first on lower and then on upper leaves. The secondary infection takes place through conidia carried by wind, water or insects.

Epidemiology: The optimum temperature for conidial germination is 28-30°C. If season starts with abundant moisture and frequent rains, disease becomes serious, followed by warm and dry weather, which is unfavourable for the host but helps rapid disease development. Weaker plants are more susceptible.

Control

- 1- Crop rotation and field sanitation is a rational measure to avoid primary infection from spores that have survived from previous crop.
- 2- Fungicidal sprays starting from 30 days after sowing at an interval of 10-21 days (depending upon the intensity of disease). For this purpose Dithane M-45 (0.6%), Daconil (0.3%), Acrobat MZ, Ridomil Gold MZ, Banko (0.2 – 0.25%) can be used.
- 3- Proper fertilization of the crop to maintain the plant vigour.

GRAM BLIGHT

Occurrence and Importance:

Gram is an important and valuable crop particularly in the Barani tract of Pakistan. Blight is a serious disease of this crop in the North Western part of Punjab province. It frequently appears in an epidemic form and causes heavy losses. Total destruction of the crop has been reported in severe cases.

Causal organism: *Ascochyta rabiei*

Perfect stage: *Mycosphaerella rabiei*

Order: Sphaeropsidales

Family: Sphaeropsidaceae

Symptoms: The fungus attacks all the above ground parts of the plant. The disease appears in the form of circular spots on leaves and pods and elongated spots on petioles and stem. On leaflets, the spots are round, sometimes elongated and bear irregularly depressed brown dots and are surrounded by brownish red margin. These spots may coalesce and entire leaf turns brown presenting a scorched appearance.

On green pods, the spots are usually circular with dark margins and have pycnidia arranged in concentric circles. Seeds within pods may also show lesions. On petiole and stem, lesions are elongated (3-4 cm), brown and bear black dots. Lesions may girdle the affected portion causing drooping and wilting of the above portion. If main stem is girdled at the base, the entire plant dries. With the advancement of disease patches of drooping and wilting plants become prominent in the field, later involving the whole field.

Disease Cycle: The fungus survives as pycnidia on diseased plant debris and seeds. Badly affected seed normally fail to germinate. Pycnidia can survive in debris for about two years at a temperature of 10-35°C, if RH on soil is 0-3%. The pycniospores (conidia) are exuded from the pycnidia only during and after rains. They are disseminated through the agency of raindrops of flowing rainwater. The conidia germinate by germ tubes, form aspersoria and enter the host through cuticle or stomata by means of infection pegs.

Epidemiology: Cloudy, moist and rainy weather favours the disease during any part of the growing season of the gram crop particularly during the last three months i.e., January-March. High rains during the preceding summer season and low winter rains

and dry weather conditions during winter decrease chances of epidemics. Low summer rains and high and frequent winter rains during February to April encourage epidemics.

The following conclusions have been arrived by Late Dr. Sattar as a result of analysis of weather and disease incidence data during fifty years (1911-61):

- i. Areas with an average winter rain of less than 3.5 inches will not suffer from blight epidemics.
- ii. Areas with an average winter rain of 3.5 inches to 6 inches will occasionally suffer from epidemics.
- iii. Areas with an average winter rain of above 6 inches are exposed to frequent epidemics
- iv. Areas with high summer rains do not suffer from epidemics.
- v. Areas with low summer rains are likely to suffer frequently from epidemics.

Control:

- 1- Use of healthy seed collected from blight free areas.
- 2- Seed treatment with Benlate, Calixin-M etc.
- 3- Elimination of diseased plant debris by:
 - i. Harvesting crop by pulling out with hand instead of cutting with a sickle.
 - ii. Sweeping the threshing floors and burning or burying the collected debris,
 - iii. Ploughing the field with a furrow turning plough, after the first shower of rain in order to bury the remnants of diseased plants.
 - iv. Disallowing bhusa stacking in the field.
4. Mixed cropping of gram with wheat, barley and taramira, etc.
5. Use of blight resistant varieties: CM -44, CM -72, CM -88, Punjab -91, Paidar etc.

CITRUS WHITHER TIP

The disease occurs in almost all citrus growing tracts of the world including Pakistan.

Causal organism: *Colletotrichum gloeosporioides*, *Glomerella cingulata* (perfect stage)

Order: Melanconiales

Family: Melanconiaceae

Symptoms: The first symptom is leaf drop and drying of the twigs from tip downwards. Later on the dried portions of twigs become silvery white in appearance and show small black dots, which are the fruiting bodies (acervuli) of the fungus. When the fungus attacks fruits it causes rot and the fruits fall off prematurely. If the disease is allowed to progress unchecked, it brings about complete destruction of the fruit trees.

Disease Cycle: The fungus is weak parasite on citrus and can only attack those trees whose vigour has been lowered due to some other factors, such as, high salinity, water logged conditions and poor drainage of the soil due to presence of a hard pan in the sub soil. The fungus perpetuates in the form of mycelium inside the twigs or as conidia in the acervuli. The conidia are disseminated by wind and rain. The fungus penetrates the host tissues directly.

Epidemiology: As already mentioned above, excess of soluble salts in the soil poor drainage; water logged condition, presence of a hard pan in the sub soil and poor management of the orchard with 15-30 °c and high R.H. are the predisposing factors for this disease. Citrus nematode *Tylenchulus semipenetrans* has also been found strongly associated with the disease.

Control:

1. Pruning of the dried portion (along with 3" of healthy portion) of the twigs and their immediate burning.
2. Spray with Bordeaux Mixture 4:4:50, four times during the year as under. First spraying in the middle of May, second spraying in the end of July (use rosin Bordeaux Mixture), third spraying in the end of Sept. and 4th spraying in the end of December or January.
3. Through management of the orchard which should include the following cultural practices:
 - Intercultivation to destroy all weeds and grasses. Adequate Irrigation and hoeing.
 - Manuring the trees @ 1-2 Kg of Ammonium Sulphate, 40 Kg of FYM per tree in December.

- Addition of 40-50 kg Gypsum per tree in alkaline soil in December.

4. Disinfection of soil from nematodes by using DBCP (6 gl. /acre) or Nemagon (10 gl/acre).

ANTHRACNOSE OF MANGO

Occurrence and Importance: It is the most common and wide spread fungal disease of mango and is known to occur in Brazil, France, Philippines, Indonesia, Trinidad, Peru, USA, Portugal, Hawaii and Indo-Pak.

Causal organism: *Colletotrichum gloeosporioides*, *Glomerella cingulata* (perfect stage)

Order: Melanconiales

Family: Melanconiaceae

Symptoms: The disease attacks young as well as mature fruits. On fruits black spots of various forms develop, which may be slightly sunken or may show surface cracks. These spots may coalesce to form larger spots and ultimately whole the fruit may be involved. If younger fruits are infected, they drop down from the tree.

On leaves numerous oval or irregular spots may appear on the leaf surface and increase rapidly under humid conditions to form irregular necrotic areas. Young leaves are the most susceptible to disease. Wither tip or die back appears at the tip of very young branches. Black necrotic areas are formed on the affected twigs, which dry from tip downward, accompanied with defoliation of the branches. In blossom blight, the inflorescence shows minute black spots on the flowers which dry and shed.

Disease Cycle: The pathogen survives in diseased twigs and leaves which fall on the ground. It has the ability to survive saprophytically for a long period in dead parts. Infection in fruits takes place from the start of the blossoming period until the fruits are more than half grown.

Epidemiology: Humid and mist conditions with temperature ranges 25-35°C are favourable for infection. Fungus does not grow below 95% relative humidity.

Control:

1. Prune off diseased twigs and remove diseased leaves and burn them.
2. Spray the trees with Bordeaux Mixture (3:3:30) or Elite (0.25%) three times a year, i.e., February, April and September.

GRAM WILT

Causal organism: *Fusarium oxysporum* f.sp. *ciceris*

Order: Moniliales

Family: Tuberculariaceae

Symptoms: The first apparent symptom is a distinct drooping of the leaves, which is followed by wilting and then necrosis of the tissues of the collar and main roots. The affected plants can be pulled out easily and the roots seem to be discoloured. It is a serious disease in the plains of Punjab.

Disease Cycle: There are two definite and distinct phases of the disease. Firstly, the seedling phase, which occurs in the months of September-October and secondly, the mature phase which occurs in March-April.

Penetration: The infection of roots takes place by the germination of the soil borne spores.

Dissemination: The fungus is spread from field to field by hooves of animals, cultivation tools and implements.

Perpetuation: The fungus lives in the soil and in the absence of the host, it can survive on dead organic matter for a number of years.

Epidemiology: Dry cool weather encourages development of epidemics.

Control: Like all other soil borne diseases the control of gram wilt is a difficult task.

1. Resistant varieties offer the only satisfactory control measure.
2. Incidence of the disease can be lowered by sowing the gram crop late in October when the temperature has gone down. But the 2nd phase is unavailable if there is no winter rain and weather becomes warm early in March-April.
3. Disinfection of soil by chloropicrin DBCP, etc.

FOOT ROT (BAKANAE DISEASE) OF RICE

Occurrence and Importance:

This disease was first reported from Japan by Hori in 1898 then it has been reported from China, Philippines, Uganda, Italy, India and Pakistan several other countries. It has recently been observed on paddy in Pakistan but very little work has been done on this disease as regards its distribution in different geographical regions and the losses caused by it.

Causal organisms: *Fusarium moniliforme* (conidial stage)

Gibberella fujikuroi (perfect stage)

Order: Moniliales

Family: Tuberculariaceae

Symptoms: The disease affects seedlings in the nursery as well as transplanted plants in the field. In the nursery, seedlings appear whitish pale, weaker and taller than the healthy ones. In case of severe infection, the tips of seedlings show symptoms of wilting which later on die in the nursery or soon after transplantation. In the field, the infected plants appear taller and flower earlier (due to production of certain hormones like gibberellins) than the normal plants. Such plants may show the symptoms of fungal infection at the collar region and die in about two to six weeks after infection. In certain cases, the plants show stunted growth (due to production of certain mycotoxins like fusaric acid). Due to variations in symptoms of aerial parts, the identification of diseased plants sometimes becomes difficult. Roots of infected plants emerge even from the second and third node. The roots of such plants also branch profusely as compared to the healthy ones. Pinkish growth of the fungus may also be visible at the base of plants and also within the split node. In case of floral infection, it causes sterility.

Disease cycle: The pathogen overwinters both on the seed as well as in the soil. On seed, perithecia of the fungus have been observed frequently. Being a facultative saprophyte, it can live in the soil indefinitely. Thus the primary infection of the host is initiated either from the seed or soil or from both the sources. Secondary infection of the adjacent plants may take place by the growth of the fungus or through interculturing implements or by irrigation water.

Epidemiology: The fungus has a wide range of temperature for optimum growth, which is between 30 and 35°C. It grows best at high moisture content. Thus incidence of disease is more in wet than dry nurseries. Excess of nitrogenous fertilizers predisposes the plant to the attack of pathogen.

Control:

1. Seeds must be collected from the areas free from disease.
2. Seed should be stored under dry conditions.
3. Seed dressing by suitable fungicides like Benlate, Topsin-M etc.
4. Irrigation water should not be allowed to go from infected fields to the healthy ones.
5. Resistant varieties should be sown.

RED ROT OF SUGARCANE

The disease is more serious and important in tropics and subtropics.

Causal organism: *Colletotrichum falcatum*, *Glomerella tucumanensis* (Perfect stage)

Order: Melanconiales

Family: Melanconiaceae

Symptoms: The symptoms appear after the rainy season when the plant growth stops and sucrose formation begins. The first external symptom of the disease is the yellowing and dropping of 3rd or 4th leaf from the top. Midrib of leaves is also affected

producing red patches with ash coloured center having abundant acervuli. Infected canes are lighter in weight and are easily broken. If split open longitudinally especially when withering of leaves starts, pith is found reddened. Characteristic bands of clear white areas are found running transversely across the full breadth of the reddened pith. In very advanced stages, red colour is replaced with dirty brown and white bands may not be very conspicuous. Cavities filled with grayish or white mycelium are found in the pith. Juice gives bad odour and does not set well on boiling due to conversion of sucrose into glucose and alcohol as a result of enzymatic action of the pathogen. Late in the season, minute, velvety, dark dots (acervuli) are formed near about the nodes of affected canes.

Disease Cycle: The fungus survives in the form of appressoria and thick walled mycelium in the soil. As sugarcane has no dead season, even a limited survival of fungus is insufficient for carryover of the disease. Seed sets from diseased crop are main source of primary inoculum and survival. Secondary infection is through conidia produced by diseased plants. These conidia are wind or rain disseminated and cause infection through wounds and nodal buds.

Epidemiology: Humid and warm weather, water logged conditions, lack of proper cultural operations resulting in weeds, continuous cultivation of same variety in a particular locality and presence of susceptible varieties in the vicinity are the main factors determining the epidemic development of the disease.

Control:

- Destruction of diseased plant debris and use of healthy sets.
- Resistant thick canes varieties, particularly e.g. CP-77-400, SPF-240, 237, CPF 72-2086 etc.
- Treat sets with Formaline solution (1:20), or Benomyl, for 10-15 minutes and cover them with wet gunny bags for 3 hours.
- Crop rotation of 2-3 years.

ROOT ROT OF COTTON

This is disease the most destructive of all diseases of cotton.

Causal organisms and factors affecting the disease development: The following organisms are associated with the occurrence of disease:

1. *Rhizoctonia solani*,
2. *Rhizoctonia bataticola*,
3. *Fusarium* species,
4. *Armillaria* species,
5. Bacteria,
6. Nematodes,

In addition to these there seem to be many other soil and climatic factors, which may be responsible for the disease, but they have not been clearly understood as yet.

Symptoms: The first apparent symptom of the disease is the sudden and complete wilting of the plant. A plant affected with root rot can be very easily pulled out of the soil and the tap as well as secondary roots are seen to be rotten. On pressing the root of a freshly wilted plant a yellowish, thick sticky and bad smelling liquid oozes out. The bark of the roots is shredded. In some cases when the disease plant has remained standing in the field for some time resting bodies of the fungus (*Sclerotia*) are visible on the stem near the ground level point. Nematodes and the other organisms are generally recovered on making isolations from the diseased roots.

Control: Since all the factors known to be responsible for the disease are associated with soil, the control of the disease has presented a very intricate problem. None of the hundreds of varieties tested for resistance has proved resistant.

No cultural operations such as high or low intensities of cultivation, following, flooding, manuring etc. Whatsoever have been found instrumental in controlling the disease?

Soil sterilization with chemical fumigants has been tried but not found practicable. The only hope of keeping the disease under control and reducing the incidence of the disease lies in the following:

1. Very early or very late sowing (end of March for early and end of June or beginning of July for late sowing). In late sown crop the line-to-line distance should be 2 feet and closer plant-to-plant distance; this will need higher seed rate.

2. Intercropping of cotton with Moth (*Phaseolus acutifolius*) and removing it as fodder by the first week of August, after which the crop should be intercultured and manured with some nitrogenous artificial fertilizer, to boost up flowering and fruiting in plants.
3. Biological control *Trichoderma viridi* checks the disease. Use it with wheat bhoosa before sowing of cotton.

POWDERY MILDEW OF CUCURBITS

This disease occurs throughout the country affecting various cucurbit plants and causing substantial damage particularly under favourable conditions. In severe form, it causes heavy losses.

Causal organism: *Erysiphe cichoracearum*

Order: Erysiphales

Family: Erysiphaceae

Symptoms: The first symptoms appear in the form of small, white to dirty gray powdery spots (sometimes with reddish brown tinge) on leaves and stems. Disease developed on both lower and upper surface of leaves. With the advancement of disease, the entire surface of the plants is covered with a whitish powdery mass turning the plant into brown colour. Black pinpoint bodies (Cleistothecia) appear late in the season. There may be premature defoliation. The fruit development is checked or the fruits remain undersized and malformed.

Disease Cycle: The disease perpetuates through the cleistothecia formed on disease plant debris lying on the surface of the soil or on stray cucurbit plant in the isolated areas and serve as primary source of infection. The fungus may also be present on such stray plants in conidial form. The pathogen penetrates host tissue mechanically. The mycelium is superficial except for haustoria. The fungus produces short conidiophores, which bear chains of oblong conidia. Later cleistothecia are formed which remain scattered on the host surface.

Epidemiology: Unlike most fungi, the spores of powdery mildew do not require free water for germination and are actually inhibited in its presence. High humidity is beneficial but not necessary for spore germination. Infection has been known to occur below a relative humidity of 50%, although the humidity at the surface of the leaf is undoubtedly higher. High humidity also increases the rate at which the fungus grows after infection occurs. Spores will germinate above 10°C with an optimum of around 26°C F and an upper limit of 32°C. Overcrowded plants population help in disease development.

Control:

Since the infection starts on the abaxial surface of leaves, systemic fungicides such as Bayton Foliar (0.15%) or Benomyl, are extensively used in many countries.

POWDERY MILDEW OF MANGO

Occurrence and Importance:

This disease is of regular occurrence and is gaining much importance in Pakistan. The incidence of this disease has been recorded upto 100 percent in some mango orchards. Although the exact losses caused by powdery mildew have not been determined, it seems that the disease causes substantial losses.

Causal organism: *Oidium mangiferae*

Order: Erysiphales

Family: Erysiphaceae

Symptoms: The disease is characterized by the appearance of isolated small white superficial spots on young tissue of all parts of inflorescence, leaves and fruits. In severe cases; inflorescence may be completely covered by mildew that is eventually blackened. Moreover, young leaves curl, become distorted and the newly set fruits drop off when they attain pea size.

Disease Cycle: The disease perpetuates on malformed/off season panicles as well as intact mildewed leaves as dormant mycelium, which sporulate during the month of March. Incubation period of the disease 5-8 days.

Epidemiology: Conidia of the fungus germinate well at the range 10-30°C and maximum appressoria formation takes place at 25°C and 92% R.H. According to one study, the epidemiological factors determined were: Av. Max. Temp. 25.5-30.2°C, Av. Min.

Temp. 10.6-16.8°C, Av. R.H. 75.0-91.6%, Av. Daily sunshine 5-12.2 hours.

Control:

1. Cut and burn the infected inflorescence in the month of September-October.
2. Three foliar sprays by using Benomyl (0.2%), Bavistin (0.1%) at the following stages.
 - a. Pre bloom
 - b. Full bloom
 - c. After fruit setting
3. Spray of Topsin-M at 20-30% flowering stage followed by two other sprays at 15 days interval.
4. In South Africa, biological control of the disease has been successfully demonstrated by using a bacterium *Bacillus subtilis*.

APPLE SCAB

Occurrence and Importance:

The disease was first of all reported from Sweden by Fries in 1819. Now it has been reported from almost all parts of the world wherever apple or crab apple is grown. In Pakistan it has been reported from Murree and Hazara hills, Peshawar, Quetta and Kalat Divisions. It is less frequent in areas with dry climate and relatively high average temperatures and is most destructive in relatively cool and humid climate.

Causal organism: *Venturia inaequalis*

Conidial stage: *Spilocaea pomi*

Order: Pleosporales

Family: Venturiaceae

Symptoms: The disease occurs on all aerial parts of the plant. On leaves, the lesions first appear on the lower surface, later developing on both sides. The lesions appear in the form of grey spots slightly darker than the leaf lamina, turning dark brown to black and assuming a more or less velvety appearance with age. On the upper surface the lesions are more conspicuous and have definite margins, but on the lower surface margins are not conspicuous due to the presence of hairs.

On fruit, the lesions first appear as brown or dark, circular velvety spots beneath the skin, which later become brown and corky. Fruit attacked in the early stage may show cracking in the diseased areas. Infected twigs may consist of blistered and ruptured bark, giving them scurfy appearance.

Disease Cycle: The fungus survives in the form of saprophytic stage and provides primary inoculum in the form of pseudothecia. Ascospores are liberated in spring, fall on the suitable host, germinate by germ tube, form the apressorium and infection peg and penetrate the host cuticle to cause the primary infection. Secondary infection takes place by the conidia that are produced on the host throughout the spring and summer. Conidia also germinate by germ tube and form apressorium that produces infection peg to penetrate the cuticle directly. Ascospores are mostly disseminated by wind but the conidia are released only when wet and therefore, they are disseminated by rain.

Epidemiology: Cool wet and cloudy weather in spring favours the disease development. Optimum temperature for the growth of the fungus is 16-24°C.

Control:

- 1- Elimination or reduction of pseudothecial production. Spraying of 5% urea after harvest at pre-leaf fall stage may cause up to 97% reduction in ascospore discharge.
- 2- Prune the tree (particularly the diseased twigs) to avoid dense growth of the shoots.
- 3- Foliar spray of Difolatan (0.3%), Topsin-M (0.05 – 0.1%) or Antracol (0.25) March-April (silver tip – green tip stage).
- 4- Dithane M-45 (0.25%), Captan (0.2%) in April-May (petal fall stage).
- 5- 1-3 sprays of Dithane M-45 (0.25%) in late summer at 10-15 days interval (post blossom stage).

Phylum Basidiomycota

Characters of Basidiomycetes (Basidiomycetes is not a class name but the members of *phylum Basidiomycota* are commonly known as basidiomycetes):

- Mycelium is well developed and septate.
- A **dolipore** septum is present between the two hyphal cells which regulate the flow of particles across the cells.
- There is also the presence of a **clamp connection** between two hyphal cells that ensures the dikaryotic condition in each new cell of the secondary mycelium.
- They produce their sexual spores known as Basidiospores.
- Basidiospores are produced outside of a specialized, microscopic spore producing structure called **Basidium**.
- Basidiospores of most species are also known as **ballistospores** which are discharged from the basidium forcibly.
- As a result of Karyogamy, Plasmogamy, and meiosis four (4) basidiospores are produced in each basidium.

Dolipore septum: It is a **doughnut** like (**ring shaped**) or **barrel shaped swelling** in the centre of septal wall surrounding a central pore.

Clamp Connection: As a result of Spermatization or more commonly the fusion of two uninucleate cells of the compatible, homokaryotic mycelia, a binucleate cell is established (a-b). **When this binucleate cell is ready to form a short bridge like branch which** is known as **clamp connection arises between two nuclei** a & b, and begin to form a hook. Now we can say that the presence of clamp connection is generally indicative of the dikaryotic condition.

Nutrition: These are the most widely distributed fungal group of individuals, so as their nutrition. They mostly occur as Rust, Smut, Jelly fungi, Mushrooms, Shelf fungi, Puffballs, Stink horn, and Bird's nest fungi.

RUSTS

Occurrence and Importance:

The rust diseases are very common and conspicuous on account of the bright orange to rusty brown, powdery masses of spores erupting from the host tissue. They are, evidently, the most important diseases of cereals and grasses and cause serious economic losses. A loss of about 10 per cent on the total world production of wheat has been estimated.

The rust fungi are obligate parasites and comprise a single order uredinales, which has three families Pucciniaceae, Melampsoraceae and coleosporiaceae and 130 genera and 3000 species. They are polymorphic and as many as five spore forms may be produced, which if present always occur in the following sequence:

- | | |
|-----------|--|
| Stage 0 | Spermatia or pycniospores in spermatogonia or pycnia |
| Stage I | Aeciospores in Aecia |
| Stage II | Uredospores in Uredia |
| Stage III | Teleutospores in Telia |
| Stage IV | Basidiospores in promycelium developing from the teleutospore |

Some of the rust do not produce all spore forms those producing teleutospores, basidiospores like *Puccinia malvacearum*. Macrocytic or (long cycled), are those which produce four or five spore forms, e.g. *Puccinia graminis*.

Most rust organisms complete their life cycles on one host and are termed autoecious, others require two unrelated host species for completion of their life cycles and they are heteroecious. This phenomenon is unique to the rusts.

IMPORTANT RUSTS

- | | | |
|----|---------------------------|-----------------------|
| 1. | <i>Puccinia asperagi</i> | Rust of Asparagus |
| 2. | <i>P. chrysanthimi</i> | Rust of Chrysanthemum |
| 3. | <i>P. coronata</i> | Crown rust of oats |
| 4. | <i>P. graminis avenae</i> | Rust of oats |
| 5. | <i>P. graminis hordei</i> | Rust of Barley |

6.	<i>P. graminis secalis</i>	Rust of Rye (<i>Secale cereal</i>)
7.	<i>P. graminis tritici</i>	Black rust of wheat
8.	<i>P. striiformis</i>	Yellow or stripe rust of wheat
9.	<i>P. sacchari</i>	Rust of sugarcane
10.	<i>P. recondita</i>	Brown or leaf rust of wheat
11.	<i>P. malvacearum</i>	Cotton rust
12.	<i>Uromyces trifolii</i>	Rust of shaftal
13.	<i>U. fabae</i>	Pea rust
14.	<i>Melampsora lini</i>	Linseed rust an autoecious fungus

But here we shall discuss only rusts of wheat:

1. Yellow rust appears rarely on leaf sheath and very rarely on stem. The pustules are produced in lines, yellow in the beginning; turn to black colour later on.
2. Leaf rust also appears rarely on leaf sheath and very rarely on stem. The pustules irregularly arranged on leaves. Colour brown of orange turning to black later on.
3. Stem rust appears on stem, rarely on leaf sheath and very rarely on leaves. The pustules brick red turning to black on maturity.

STEM RUST OF WHEAT

Causal organism: *Puccinia graminis tritici*

Order: Uredinales

Family: Pucciniaceae

The stem rust of wheat is a classical plant disease. *Puccinia graminis* ranks as the most destructive plant pathogen. It has been estimated to cause as much as 10 % Losses annually to wheat crops the entire world over. Various strains of the pathogen attack oats, rye, barley and a number of grasses. The principal alternate host is the barberry *Barberis vulgaris*.

In Pakistan this rust does not generally appear before **March** when the temperature is 75-85°F. By this time the crop is fairly advanced in growth and therefore, the chance of doing serious damage to the crop is reduced.

Symptoms: The characteristic symptoms produced on wheat is the appearance of long, narrow streaks (pustules) that occur on the stems, leaf sheaths and leaves. The pustules first appear as dark red are called Uredia, producing Uredospores. As the crop approaches maturity the pustules become black and are now known as telia because they now produce the teleutospores.

On barberry: small yellowish to pinkish spots develop on the leaves. Small dark bodies spermagonia or pycnia appear on the spots on the upper surface of the leaf and white, cup shaped structures the aecia soon develop on the lower surface.

Disease Cycle: Penetration: Aeciospores and uredospores germinate and penetrate the grain host through stomata. Basidiospores germinate and penetrate directly through the cuticle of young barberry leaves.

Dissemination: Basidiospores, aeciospores and uredospores are dispersed primarily by wind. The basidiospores and aeciospores are spread over a radius of one to two miles. The uredospores, however, are carried over long distances. Viable uredospores have been trapped at altitudes exceeding 10,000 ft. Teleutospores remain attached to the wheat straw by stalk cells. Spermatia are carried from one spermagonium to another by insects.

Over Wintering: The teleutospores on straw persist through the winter in cold countries. But in warmer countries the fungus remains active through the winter as mycelium producing uredospores on the winter-sown wheat.

Epidemiology: Uredospores can germinate at temperatures between 5 to 25°C. The optimum temperature is between 18°C to 20°C. A film of water must be present on the surface of the leaf to allow germination and penetration. The following factors favour epidemics:

1. Large areas sown with susceptible wheat varieties.
2. Prevalence of physiologic races capable of attacking the wheat varieties.

3. Strong winds to carry uredospores over long distances and
4. Mild, humid weather.

Control:

In spite of strenuous efforts and huge expenditure on research spread over several centuries the disease continues to cause serious losses.

1-Development of resistant varieties is the only hope. But now varieties resistant to the common physiologic races have been introduced only to be damaged by a new or different race of the fungus. Durum wheat Pb. Types 1, 2 and 3 are said to be highly resistant to the disease. The resistant varieties should be resistant to different races of the fungus. Races 15, 21, 29, 40, 42, 75; out of these 15, 40, and 75 are important varieties should resist these, e.g. LU-26S, etc.

2- Eradication of alternate host: Eradication of barberry will not altogether eliminate the disease. But there is one great advantage in it, that is, the pathogen will not be able to produce new physiologic races because the hybridization stage will get eliminated.

YELLOW RUST OR STRIPE RUST OF WHEAT

Causal organism: *Puccinia striiformis*

Order: Uredinales

Family: Pucciniaceae

Yellow rust will appear in the months of **December** to **January**, when temperature is 55-65°F and if there are heavy winter rains it is capable of causing considerable losses. Uredia are chiefly confined to the leaves, but when the attack is severe, they may appear on the leaf sheaths, the stalks, over the glumes and the pericarp of the grains. The minuteness of the sori, their arrangement in linear fashion forming stripes and their lemon yellow colour are the chief distinguishing macroscopic features of this rust. Uredospores are about oval and not round as in black rust, and their epispore is covered with fine spines.

Telia are hypophyllous or culmiculous and occur in long, narrow lines like the uredia. They are for a long time covered by the epidermis with brown paraphyses surrounding the spores and intermingling with them along the edges of the telium. The teleutospores are oblong to cruciform, slightly constricted at the septum and the apex is less thickened and pointed than in *P. graminis*. The rust is heteroecious, but as the alternate host is as yet unknown the pycnial and aecial stages have not been observed. The inoculum responsible for starting the annual epidemics comes from the hills but this rust requires a cooler temperature and therefore, overwinters at elevations of 7000 and above. The further sequence of events that lead to the spread of the disease from the hills to the plains is the same as in black rust.

Control: Selection of resistant varieties is the only hope.

BROWN OR LEAF RUST OF WHEAT

Causal organism: *Puccinia recondita* f.sp. *tritici*

Order: Uredinales

Family: Pucciniaceae

It appears after the yellow rust in **February**. The uredia are, as a rule, confined to the leaves, being less common on the leaf sheaths and stems. They are round to slightly oblong, orange in colour and irregularly scattered or in clusters on the leaf blades.

There are three to four germ pores on the urediospores wall and they are scattered all over the surface.

Telia are formed only rarely and in some years they may not be present at all. They are small oval to linear, black and covered by the epidermis. The teleutospores are oblong to cruciform, slightly constricted at the septum, and the apex is rounded with prominent thickening. The alternate host of brown rust is *Thalictrum flavum*.

Control:

1. Resistant varieties.
2. Destruction of alternate host.

SMUTS

The smut diseases are characterized by the formation of conspicuous, black powdery masses of spores (the teleutospores) in the infected host. The losses due to these diseases can be attributed to reduction in yield, deterioration of quality and therefore low

market value.

1.	<i>Ustilago tritici</i>	loose smut of wheat
2.	<i>Ustilago nuda</i>	loose smut of barley
3.	<i>U. avenae</i>	loose smut of oats
4.	<i>U. kolleri</i>	covered smut of oats
5.	<i>U. hordei</i>	covered smut of barley
6.	<i>U. scitaminea</i>	smut of sugar cane
7.	<i>Sphacelotheca sorghi</i>	grain smut of jowar
8.	<i>S. reiliana</i>	head smut of maize
9.	<i>Tolyposporium ehrenbergii</i>	Long smut of Jowar
10.	<i>Tolyposporium penicillariae</i>	Smut of bajra
11.	<i>Tilletia foetida</i>	bunt of wheat
12.	<i>T. caries</i>	bunt of wheat
13.	<i>Neovossia indica</i>	new bunt of wheat
14.	<i>Urocystis tritici</i>	flag smut of wheat

LOOSE SMUT OF WHEAT

The disease is of worldwide occurrence. On the average the loss is about 2.0% but as high as 10-20%.

Causal organism: *Ustilago tritici*

Order: *Ustilaginales*

Family: *Ustilaginaceae*

Symptoms: Before the head emerges from the boot leaf it is dark brown with a thin silvery membrane over the spore mass, which soon ruptures to liberate the spores and thus permit them to be blown about over the field. The glumes and kernel are destroyed and only the naked rachis remains.

Disease Cycle: Penetration: Most commonly the penetration takes place through stigma by a germ tube produced by a germinating spore, in about 8-10 days the infection is established in the ovary.

Dissemination: The chlamydospores are wind disseminated.

Perpetuation: The fungus remains dormant within the seed as it matures, until the following season when it is activated by the germinating seed. The fungus following the growing point of the plant showing no external symptoms until the blossoming time when it rapidly increases in mass within the floral parts and by the time the head emerges from the boot leaf, it has completely replaced the grain by its own chlamydospores and destroyed the floral parts.

Epidemiology: Moist, warm weather encourages infection, which results in higher incidence of the disease in the next season.

Control:

1. Hot water treatment: Presoaking at ordinary temp for 4-5 hours. 1st tub at 132°F, dip for 5 minutes, end point temp. 118-120°F. 2nd tub at 132°F, dip for 7 minutes, end point temp. 127-129°F. Dry in shade & sow immediately. Increase seed rate by about 5%.
2. Solar energy treatment: Recommended only for places where temp. in shade is not less than 100°F. Treat seed during May & June. Presoaking in water from 8 am to 12 noon. Then spread in open, direct sun. Dry for one or two days & store till sowing season. Increase seed rate by 5%.
3. Resistant varieties. Durum types are immune to the disease.
4. Seed treatment with chemicals: i) Vitavax) 56 gm/30 kg. iii) Ceresan M^b ii) Benlate) or iv) Agrox. 1½- 2 gm/kg

LOOSE SMUT OF BARLEY

The same as in case of loose smut of wheat.

Causal organism: *Ustilago nuda*

Order: *Ustilaginales*
Family: Ustilaginaceae

LOOSE AND COVERED SMUT OF OATS

Causal organism:

- i. Loose smut: *Ustilago avenae*
- ii. Covered smut: *Ustilago kolleri*

Order: *Ustilaginales*
Family: Ustilaginaceae

Symptoms: In the case of loose smut all the parts of the flowers are destroyed and the spore masses (sori) are rather loose and easily disintegrated and dispersed by wind, whereas in the covered smut the sori or spore balls are rather hard and the spikelets are able to preserve their shape owing to the permanence of the glumes, and the spores are not dispersed by wind.

U. Avenae chlamydospores 5-9 microns in size oval, olive brown one side lighter spore surface echinulated.

U. kolleri- Same as above, spore surface smooth.

There are no clear characteristics whereby *Ustilago avenae* and *U. kolleri* can be distinguished on taxonomic grounds. It has been suggested that they might be regarded as Spiny and Smooth spored varieties of the same species. The two fungi hybridize easily, the spiny character being dominant over smooth and segregated in the same Mendelian ratio of 3:1.

Disease Cycle: Penetration: Seedlings are infected by germ tubes resulting from the germination of seed or soil borne spores. No infection would take place when the seedling has come out from the soil and coleoptile rupture.

Dissemination: The sori of the covered smut break up during threshing operation and the general method of dissemination is by wind.

Perpetuation: The fungus perpetuates in the form of chlamydospores, which are either seed borne or soil borne.

Epidemiology: The infection favoured by temperatures 18-20°C and relatively low soil moisture during the seedling stage.

Control:

1. Seed treatment with liquid formalin 1: 320 parts of water or with dry organic mercurials such as Ceresan, Agrosa, dieldrex, 1 oz. For 30 kg of grain have been used with advantage on account of low cost, protective action and less damage to seedlings.
2. Resistant varieties.

COVERED SMUT OF BARLEY

The disease occurs all over the world.

Causal organism: *Ustilago hordei*

Order: *Ustilaginales*
Family: Ustilaginaceae

Symptoms: The affected ears emerge about the same time as the healthy ones, but remain shorter. Unlike the loose smut of barley, the black masses of spores of this smut are held together more or less firmly by persistent membranes of the grain and lower parts of the glumes, the awns also remain in tact.

Disease Cycle: Penetration: The infection takes place in the seedling stage before the seedling emerges from the soil.

Dissemination: The spores are disseminated by wind or contact and are seed borne.

Perpetuation: The fungus passes from one season to the next in the chlamydospore stage, which are seed borne. The spores left in the soil do not play an important role in the perpetuation of the disease.

Epidemiology: Temperature 18-22°C and low soil moisture at the time of germination of the seed encourage disease development.

Control:

1. Dry seed dressing with dust of organic mercury compounds like Ceresan, Agrosan and Granosan @ 2gms/kg of seed.
2. Wet treatment with formalin solution 1:320 parts of water. Dip for five minutes and then cover for 2-3 hours with moist gunny bags.
3. Vitavax 56 gm/30 kg.

BUNT OF WHEAT

Geographic distribution: The first two named bunts are of world wide occurrence in regions with temperate climate and at about 6000 ft. elevations or above, whereas the third or the partial bunt occurs in the plains and has not been observed outside Indo Pakistan subcontinent.

Causal organisms: There are three principle types of bunts, which attack wheat plants:

1. *Tilletia foetida*, the smooth spored bunt.
2. *Tilletia caries* (formerly known as *Tilletia tritici*) the rough spored bunt.
3. *Neovossia indica*, the Partial bunt, or new bunt.

Order: *Ustilaginales*

Family: *Tillitiaceae*

Symptoms: These bunts attack only the grain. The first two named bunts generally occur together and produce the same types of symptoms. Plant affected by them ripen a little earlier, the ears assume a dark green colour and are more open than those of the healthy plants. Bunted ears are longer than the healthy ones, and as they mature the glumes become pushed apart. As a rule, all the ears in a bunted plant are attacked and all the grains in an ear are turned into bunt balls, though between two fingers it is crushed to a black greasy powder, smelling strongly of rotten fish.

The symptoms produced by the partial bunt (*Neovossia indica*) are entirely different. Whereas in bunt ball. In the partial bunt only two or three grains in an ear are partially attacked, the remaining seeds being healthy. The spores emit the same odour as those of the other bunts. The spores of the partial bunt are much bigger almost double the size of those of the other two bunts, i.e. 22-50 microns. They have reticulate out growing on the surface of the epispore, which is a thin hyaline membrane and persists after maturity. Spores germinate after a long rest period. Of they are soaked in water for about 10 days and then placed in moist air they germinate by putting forth short, promycellia at the apex of which 60-120 sporidia are formed. They are long sickle shaped, and neither fuse nor form H-shaped structures. On germination they form infection threads, which infect the host, infection takes place only in the flower buds soon after the ears have emerged out of the boot leaves and the threads are incapable of a case expected to be effective. No other direct methods of its control have so far been worked out. Resistant varieties may prove better.

DIFFERENCE IN OLD BUNT AND NEW BUNT

OLD BUNT	NEW BUNT
All tillers of plant are attacked.	Only a few tillers are attacked.
Each ear is fully attacked.	Only 2-3 grains are attacked.
Grains are attacked as a whole.	Grains are partially affected.
Seed borne and infection is systemic.	Infection is aerial.
Found in hills.	Found in plains
Distribution worldwide.	Only in Pakistan and India
Spores of smaller size.	Large sized spores (double in size)
Sporidia pair in H-shaped structures.	No pairing.

Phylum Oomycota

Phylum Oomycota contains a single class i.e. *Oomycetes*

Characters of Class *Oomycetes*:

1. On the basis of different biochemical and molecular characteristics they can be separated from true fungi.
2. **Biflagellate zoospores** are present (anterior flagellum directed forward is tinsel type while the posterior flagellum directed backward is whiplashed type).
3. Ultra structure of zoospores separates them from Chytridiomycetes.
4. There are two types of zoospores:

- i. Primary spores (**Pyriform**): These are pear shaped.
 - ii. Secondary spores (**Reniform**): These are kidney shaped.
5. Members of this class are Eucarpic and holocarpic.
 6. Their cell-wall contains **β -glucans**, an amino acid hydroxyproline and cellulose.
 7. Sporangia may produce zoospores or can directly germinate and behave as conidia.

LATE BLIGHT OF POTATO

Occurrence and Importance:

Potato is a native of Andes (South America), where this disease occurred in endemic form. The disease was recorded simultaneously in Europe and North America in 1830. It became very well established in Europe by 1842 and was one of the important causes of the great Irish famine in 1845 and 1846. It invaded the sub-continent between 1870 and 1880. This is extremely destructive to tomato and eggplant and many other Solanaceous hosts except pepper. It may bring about as much as 10-15 percent losses to world production of potatoes during certain years.

Causal organism: *Phytophthora infestans*

Order: Peronosporales

Family: Pythiaceae

Symptoms: The disease manifests itself only after the blossoming period. The first symptoms appear in the form of brown dead spots or extended necrotic areas more frequently until the leaves are killed. These blighted areas first appear as faded green patches, which soon turn to brownish black lesions not delimited in size. These lesions enlarge rapidly under favourable weather. They appear on tips of margins of the leaves and spread downwards or inwards. In moist weather, entire leaf may be killed in 1-4 days, while in dry weather, infection advances slowly and affected areas curl and shrivel and the spots are restricted in size and look hard, easily breaking away. On lower surface of leaves, a whitish or grayish mildew growth appears on the surface of lesions where pale and purplish tissues merge. This contains sporangiophores and sporangia, which grow out through stomata.

Tubers are also affected in the field, having dry or wet rot according to the moisture and temperature prevailing at that time. There is brown to purple discolouration of skin followed by brownish dry rot, which extends about ½" below the skin.

Disease Cycle: Fungus survives mostly as persistent mycelium in the infected tubers, but it may over winter in soil as well. This mycelium grows up in the stem and produces sporangia and zoospores on small dwarf shoots. The secondary infection starts from these spores. Infection takes place epidermis of leaves and stem either through stomata or directly. Tuber infection occurs through eyes, lenticels or wounds. Susceptibility of eyes and resistance of lenticels increases with storage and maturity of tubers. Spores from blighted leaves are washed down to soil and cause tuber infection.

Epidemiology: Chances of epidemic development of disease are the maximum when unusually cool weather combined with abundant moisture prevails at the time of sporangial formation. Conidia are formed at a minimum relative humidity of 91 % (optimum = 100 percent) and a temperature range of 3-26°C (optimum = 18-22°C). The conidia formed at 15°C and optimum temperature for zoospores formation is 12°C. Cool moist nights are thus required to provide most rapid build up of inoculum and are the most favourable for the formation and germination of zoospores. It is possible to make an accurate forecast of the disease if accurate meteorological data are available and current weather trends are known. For example, in Holland they have worked out the following four criteria necessary for the occurrence of late blight epidemics:

Night temperature below the dew point for about four hours or more.

Night temperature not more than 10°C.

Mean cloudiness not below 0.8 on the following day.

Rainfall of at least 0.1 mm on the following day.

Very fortunately, in plains of Pakistan, the weather conditions are unfavourable for the development of epidemics. But in the hills, sometimes, temperature and moisture conditions are favorable and therefore, late blight epidemics may occur in certain years.

Control:

- 1- Seed tubers should be obtained from areas where disease does not occur.
- 2- Use of resistant varieties: All the commercial varieties within the species *Solanum tuberosum* are susceptible. However, *Solanum demissum*, a species from Mexico is highly resistant. Hybridization of this variety with commercial types has been tried with good results. e.g. Cord
- 3- Foliage spraying is the most satisfactory control method. Bordeaux Mixture and some new fungicides such as Acrobat MZ, Ridomil Gold MZ and Banko (0.2 – 0.25%) give good results.
- 4- General Sanitary Measures, killing and removing of foliage a few days before actual digging of potatoes is beneficial in reducing chances of tuber infection.

Early Blight	Late blight
High temperature and low moisture is required	Reverse in this case
Appears early in season	Reverse in this case
Appears in Plains	Appears in Hilly areas
Caused by a higher fungus	Caused by a lower fungus

DOWNY MILDEW OF CUCURBITS

Sponge gourd, muskmelon, ridge gourd and cucumber are more severely affected by this disease than bottle gourd, bitter gourd and pumpkin. It can also infect watermelon, round melon (tinda).

Symptoms: The first symptoms on leaves resemble mosaic mottling. Pale green areas are separated by islands of darker green. Soon the spots become well defined. They are angular, yellow coloured and often restricted by veins on the upper surface. On the lower side of the spots, a purplish downy growth appears in moist weather. Sometimes its colour is white to almost black. The entire leaf dies quickly. Usually the central leaves are attacked first followed by the others until the entire plant is wilted or weakened. Young leaves are less susceptible than the older ones. Infection occurs more rapidly on lower surface than on upper surface on infected vines, the fruits are few and small with poor taste.

Causal organism: *Pseudoperonospora cubensis*

Order: Peronosporales

Family: Peronosporaceae

Disease cycle: The oospores are frequently carried with the seed, but may remain viable in the soil and crop refuse for long periods. The secondary infection is by mean of conidia or sporangia. The conidia germinate directly by producing germ tube while; the sporangia germinate by producing biflagellate zoospores.

Epidemiology: Maximum sporulation occurs at a temperature of 18-28°C. The optimum temperature for germination of spores and infection is 20°C.

Control:

1. Destruction of diseased plant debris.
2. Removal of badly infected vines to eliminate secondary source of infection. Destroy wildy growing cucurbits from vegetable growing areas.
3. Fungicidal sprays starting from 30 days after sowing at an interval of 10-21 days (depending upon the intensity of disease). For this purpose, Bordeaux Mixture (4:4:50), Dithane M-45 (0.6%), Daconil (0.3%), Acrobat MZ, Ridomil Gold MZ, Banko (0.2 – 0.25%) can be used.

DOWNY MILDEW OF GRAPES

Occurrence and Importance:

Downy mildew is a major disease of grapes throughout the world. The fungus causes direct yield losses by rotting inflorescences, clusters and shoots. Indirect losses can result from premature defoliation of vines due to foliar infections.

Symptoms

On leaves, young infections are very small, greenish-yellow, translucent spots that are difficult to see. With time the lesions enlarge, appearing on the upper leaf surface as irregular pale-yellow to greenish-yellow spots up to 1/4 inch or more in diameter. On the underside of the leaf, the fungus mycelium (the "downy mildew") can be seen within the border of the lesion as a delicate, dense, white to grayish, cotton-like growth. Infected tissue gradually becomes dark brown, irregular, and brittle. Severely infected leaves eventually turn brown, wither, curl, and drop. Lesions commonly form along veins, and the fungus sporulates in these areas on the lower leaf surface during periods of wet weather and high humidity. On fruit, young berries turn light brown and soft, shatter easily, and under humid conditions are often covered with the downy-like growth of the fungus. Generally, little infection occurs during hot summer months. As nights become cooler in late summer or early fall, the second infection period may develop. Berries infected at this time generally do not turn soft or become covered with the downy growth. Instead, they turn dull green, then dark brown to brownish-purple. They may wrinkle and shatter easily and, in severe cases, the entire fruit cluster may rot. These infected fruit will never mature normally. On shoots and tendrils, early symptoms appear as water-soaked, shiny depressions on which the dense downy mildew growth appears. Severely infected shoots and tendrils usually die.

Causal Organism

Downy mildew is caused by the fungus *Plasmopara viticola*.

Order: Peronosporales

Family: Peronosporaceae

Disease cycle

The fungus overwinters in infected leaves on the ground and possibly in diseased shoots. The overwintering spore (oospore) germinates in the spring and produces a different type of spore (sporangium). These sporangia are spread by wind and splashing rain. When plant parts are covered with a film of moisture, the sporangia release small swimming spores, called zoospores. Zoospores, which also are spread by splashing rain, germinate by producing a germ tube that enters the leaf through stomates (tiny pores) on the lower leaf surface.

Epidemiology:

The optimum temperature for disease development is 18-25 °C. The disease can tolerate a minimum temperature of 12-13 °C and a maximum temperature of 30 °C. High humidity at night time favours the disease. Once inside the plant, the fungus grows and spreads through tissues. Infections are usually visible as lesions in about 7-12 days. At night during periods of high humidity and temperatures above 55 degrees F (13 degrees C), the fungus grows out through the stomates of infected tissue and produces microscopic, branched, tree-like structures (sporangiophores) on the lower leaf surface. More spores (sporangia) are produced on the tips of these tree-like structures. The small sporangiophores and sporangia make up the cottony, downy mildew growth. Sporangia cause secondary infections and are spread by rain.

Control

1. Select a planting site where vines are exposed to all-day sun, with good air circulation and soil drainage.
2. Sanitation is important. Remove dead leaves and berries from vines and the ground after leaf drop.
3. Grape varieties vary greatly in their susceptibility to downy mildew. In general, *vinifera* (*Vitis vinifera*) varieties are much more susceptible than American types,
4. A good fungicide spray program is extremely important. Downy mildew can be effectively controlled by properly timed and effective fungicides. e.g. Intracol.

PLANT DISEASE CONTROL

Almost all control methods aim to prevent plant diseases rather than cure them after they have become diseased. The five basic principles of plant disease control are exclusion, avoidance, eradication, protection, and resistance.

Exclusion

As long as pathogens and host plants can be kept apart, disease will not develop. Many potentially-susceptible plants are grown in areas of the world still free of certain diseases. When plant pathogens are introduced into a new area, they can cause much more catastrophic epidemics than do the native pathogens. Host plants that evolve in the absence of a pathogen have no opportunity to develop resistance to the pathogen and are extremely vulnerable to attack.

The Dutch Elm fungus evolved in Europe where it causes little damage to European elms. However, this fungus devastated American elms when it was imported to the United States. Other devastating diseases that resulted from introduction of a pathogen from abroad include downy mildew of grapes in Europe and bacterial canker of citrus, chestnut blight, and soybean cyst nematode in the United States.

Quarantines, inspections, and disease-free certifications help to prevent the spread of pathogens into a particular country, state or geographic area. The most visible use of this method is in California. At the state border, everyone must stop at an agricultural inspection station to be screened for fresh fruit, vegetables, and plants coming from areas known to have certain diseases or insects. To combat the devastating late blight fungus, the Montana Department of Agriculture began enforcing quarantine on importation of seed potatoes and tomato transplants into Montana in 1996. However, this quarantine does allow importation of plants certified to be free of the late potato blight fungus.

Avoidance

If a disease does occur routinely in an area, there may be ways to avoid disease development. Choice of planting site, time of planting, storage conditions, and wound avoidance are a few of these techniques. Phytophthora root rots can be avoided by not planting susceptible plants in heavy, poorly drained soils. Planting later in the year, when soils are drier and warmer, will prevent damping-off diseases that are common to many vegetables.

Wounding can provide entrances for pathogens or weaken plants so that they are less able to defend themselves. For example, the crown gall bacterium must enter raspberry, cherry, and many other host plants through wounds. Therefore, careful planting will reduce potential of this disease. Planting certified virus-free stock is a good way to avoid virus-related diseases on potatoes and many flowering plants grown in greenhouses. Good horticultural practices, such as proper fertility, pruning, watering, and training go a long way toward minimizing disease problems.

Eradication

When a plant or area is infected with a pathogen, eradication can eliminate or reduce the disease threat. Rotation, sanitation, eliminating the alternate host, heat treatment, and use of certain chemicals can reduce or eliminate diseases.

Removing plant debris (sanitation) is important in areas where pathogens may over winter. Raking leaves, removing rotted fruit, picking up old vines, and pruning out dead wood or canes are all part of sanitation. Once collected, dispose of the debris by burning, burying, or composting. However, composting temperatures must be high enough to kill the pathogens. Crop rotation is a common method in both commercial agriculture and home gardens. Knowledge of the pathogen and its host range are needed to plan effective rotations. Generally, rotation only reduces soil populations of fungi or nematodes if non-hosts are used. It does not eliminate pathogens altogether.

Many species of rusts must infect two or more different hosts to complete their life cycle. Eliminating an alternate host may help reduce a rust disease. A famous example was the attempt to eradicate common barberry, the alternate host of wheat stem rust. This disease has caused major rust epidemics worldwide since ancient times. From about 1917 until 1980, the U.S. federal government launched a very well publicized campaign to encourage farmers and citizens to destroy all common barberry plants. This campaign was largely a success, but it was very costly.

Heat treatment can eliminate pathogens from greenhouse soil and from the surface of seeds and bulbs. Some fungicides have "kickback" activity that can stop certain fungal diseases if applied soon after infection has occurred. When caught in the very early stages, certain fungicides can eradicate Dutch Elm disease from American elms, for example.

Protection

Protection involves treating a healthy plant before it becomes diseased. There are both chemical and biological means of protection.

Chemical protection is one of the most widely used means of control. Some fungicides (such as copper and sulfur products) are allowed for use under several "organic" growing guidelines. Knowledge of the disease cycle and host susceptibility is needed to obtain good control using fungicides. Proper timing, coverage, and selection of fungicides also are needed.

Interest in using biological organisms to control diseases has increased in recent years. With biological control, one organism is used to attack or inhibit the activity of another organism. One of the most successful biological protections has been the use of a bacterium to protect against a bacterial disease known as crown gall. In this case, the roots of a seedling or nursery plant are dipped into a suspension of the biocontrol bacterium before planting.

Resistance

Plants may be susceptible, tolerant, or resistant to various pathogens. The term **susceptible** indicates that the plant readily becomes diseased if the factors of environment, time and pathogen are favorable. The term **tolerant** implies that the plant may become diseased but little damage occurs.

Resistant plants do not become diseased readily unless environmental conditions are extremely favorable to the pathogen.

There are some disadvantages to growing and developing resistant varieties. Often these varieties are inferior to non-resistant varieties in terms of yield, quality, or other characteristics. Also, new **races** of pathogens that can overcome the resistance often develop rapidly. As the new race takes over, the old variety must be replaced with another variety that has different genes for resistance. Some varieties must be replaced as often as every 3 years. Plants that never get a particular disease are said not to be **non-hosts** of that particular pathogen. The term **immune** applies to animals that once exposed to a pathogen; develop antibodies which later prevent the pathogen from again causing the disease in that particular animal. Since plants do not have an immune system, the term **non-host** is more appropriate in reference to a plant that is not at all susceptible to a particular pathogen. Resistance, tolerance, and susceptibility are relative terms. There is a continuum among species and cultivars from extremely susceptible to extremely resistant.

INTRODUCTION TO PRINCIPLES OF PLANT DISEASE MANAGEMENT

There are three principles of plant disease control

1. Attack on parasite
2. Strengthening the weak host
3. Modification and improvement of environment

1. Attack on Parasite

Three major groups of practices are followed as under:

- i. **Exclusion:** To keep the pathogen away from the area of the host where the pathogen is not already present. This may be accomplished through:
 - a. **Quarantine:** The planting material is checked during importation at airport, seaport and all other entry points. If the plant material is found infected with any pest, it is destroyed immediately.
 - Certification:** The planting material, which is meant to be sent to other countries, is inspected by some specialists and a certificate is issued to the effect that it is free from infestation by any pest or disease.
 - Notification:** The experts keep a vigilant eye, and tell in advance about the spread of a particular disease so that some preventive measures could be taken.
 - Eradication:** This includes the practices like:
 - a. Eradication of alternate/collateral host like barberry in case of wheat rust.
 - b. Eradication of primary host like citrus to eradicate citrus canker.
 - c. Destruction of plant disease debris.
 - d. Soil sterilization can be done on small scale like in green houses.
 - iii. **Protection:** It includes such practices, which protect the crop from the attack of diseases and include seed treatment dusting and spraying of the crops with various chemicals before the attack of the pathogen as prophylactic measure.
- 2. Strengthening of Weak Host:** It is achieved through:
- i. **Selection:** The plants showing better agronomic traits and better resistance against the diseases are selected for subsequent planting.
 - ii. **Hybridization:** Hybridization is done between different genotypes in order to get the desired characters in term of reproductive parameters and resistance to diseases.
 - iii. **Breeding Resistant Varieties:** The advanced lines/varieties having resistance to diseases are developed through different breeding techniques.

1. Modification and Improvement of Environment

- i. **Climatic Factors:**
- ii. **Change in date of sowing:**
- iii. **Soil factors:**
- iv. **Crop rotation:**