CHAPTER 2 Some Typical Plant Diseases

● 2.1 Late blight of Potato :

◆ **A. Occurrence and Importance**— Late blight of potato is the most serious of all potato diseases. This disease is of wide occurrence and is known in all potato growing parts of the world. Potato is a native of South America (northern Andes). Late blight occurred on potato in the Andes in epidemic form. It was then introduced into Europe and north America. The famous Irish famine in 1845-1846 was caused due to the destruction of potato crop by late blight disease. In India it occurs in epidemic form only on the hills at elevation of 2000 m and above. Due to cold storage facilities of tubers, the disease is at present of wide occurrence in the plains of India, because the pathogen survives at the cold temperature.

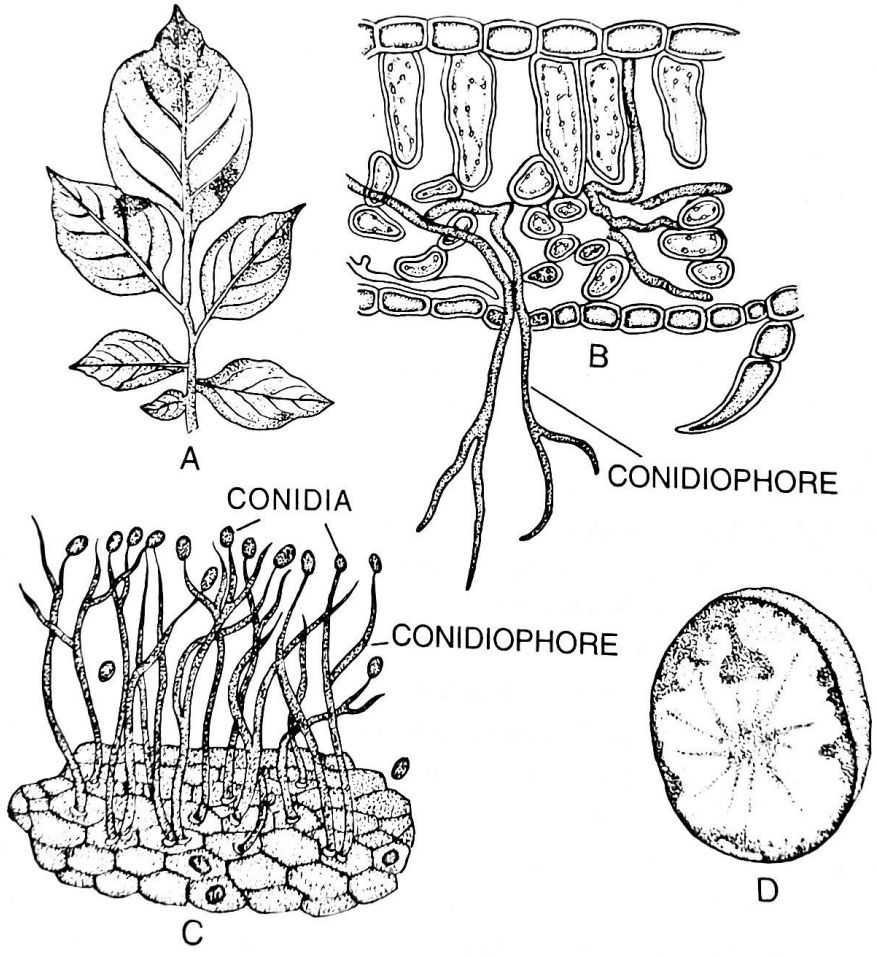


Fig. 2.1 Late blight of potato. A—Diseased *i.e.* blighted leaf. B-T.s. through blighted leaf showing sporangiophores emerging out through a stoma. C—Sporangiophores bearing sporangia emerging out through stomata of blighted leaf (in surface view). D—Infected potato tuber in 1.s.

In India the disease first appeared in Nilgiri hills between 1870 and 1880. Soon after, it spread to Darjeeling district in the Himalayas. Now-a-days, the disease has been reported from various parts of India including Assam, W. Bengal, Bihar and Uttar Pradesh. This disease also occurs in Tomato plants.

Late blight damages plants by killing the leaves and stems of potato plants at any time during growing season; it also attacks potato tubers in the field—the tuber may also rot in the field or while in storage, transit and market.

Late blight may cause total destruction of all plant-parts in the field within one or two weeks if favourable conditions prevail and when no control measures are applied. Depending on the temperature and moisture of

the growing season and on the control measures practiced, losses caused by late blight damage, vary from one area to another and from year to year.

◆ **B. Symptoms**— Symptom appears first on the leaves as black or purplish-black circular or irregular water-soaked patches, generally at the tips or edges of the lower leaves. In moist weather, these patches enlarge rapidly and form brown, blighted areas with indefinite borders; often a pale yellowish-green zone surrounds the rapidly enlarging lesions. On the undersurfaces of the leaves, a zone of white, downy fungal growth appears near the border of the lesions. Very soon, the entire and all the leaves become infected, die and limp. Under continuously moist conditions, all tender and above-ground plant parts blight and rot away quickly giving off a characteristic odour. In dry weather, the fungal activities are checked—existing lesions stop enlarging, turn black, curl and wither away.

The tubers are also attacked. Affected tubers at first show irregular, purplish black or brownish blotches with a metallic dull, dark colour. When tubers are cut open, the affected tissue appears water-soaked (i.e. soft), dark, reddish-brown and extends a few mm into the flesh of the tuber; later on the affected areas become firm, dry and sunken—such lesions may be small or may involve almost the entire surface of the tuber. It is to be noted that, the rot may continue to develop even after the tubers are harvested, or infected tubers may be subsequently invaded by secondary fungi and bacteria causing soft rots.

◆ **C. The causal organism**—*Phytophthora infestans* (Mont.) De Bary is the causal organism i.e. the pathogen responsible for the late blight disease of potato. This pathogen is a phycomycete.

◆ **D. Etiology of the pathogen**—The mycelium of *P. infestans* is endophytic consisting of much branched, hyaline, aseptate *i.e.* coenocytic hyphae which develop mainly intercellularly in the parenchyma cells of leaves and form haustoria; these haustoria alone enter the host cells. In potato tuber cells, haustoria are more elaborate, being club-shaped, hooked or spirally twisted. The sporangiophores arise in groups (4-5) from the internal leaf-mycelium through stomata and from the tuber-mycelium through lenticels; they are hyaline, branched, slender and indeterminate. The thin-walled, ovoid or lemon-shaped, hyaline sporangium with apical papilla is borne at the tip of a branch of the sporangiophore. As the sporangium approaches maturity, the branch tip swells slightly, proliferates and turns the attached sporangium to the side as the elongation of the sporangiophore proceeds. Thus, at each point where growth of the branch is renewed a nodular swelling is formed indicating the point where the sporangia were borne. The sporangium may germinate directly by means of a germ tube, which in turn may give rise to a terminal secondary sporangium. When the pathogen is active and the environment is favourable (i.e. low temperature and high relative humidity), the sporangium germinates by the development of biflagellate zoospores. After a few minutes of motility, the zoospores loose their flagella, come to rest and germinate by means of germ tubes which penetrate through the stomata or directly through the epidermis of the host.

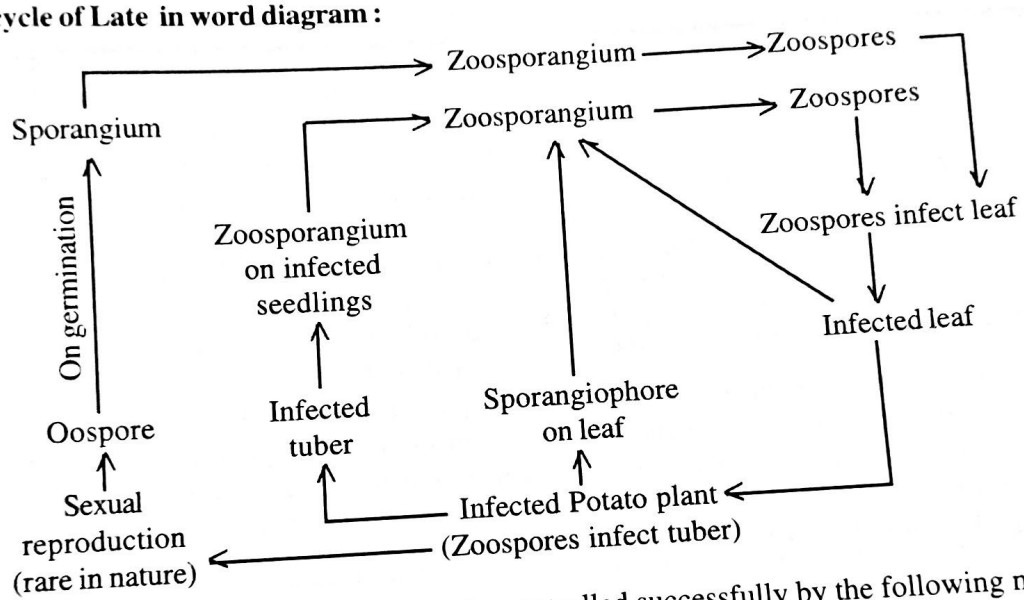
Sexual reproduction is of 'gametangial contact' type. Antheridia and oogonia are formed at the tips of specialised lateral hyphal branches. The antheridium is amphigynous, oogonium is spherical. *P. infestans* is heterothallic, *i.e.* requires two mating types (+ and –) for sexual reproduction. Plasmogamy and karyogamy involves in the process of sexual reproduction, as a result diploid oospore is formed. Oospore undergoes germination, its outer wall cracks and the inner wall comes out in the form of a germ tube which develops a sporangium at the tip. The sporangium produces biflagellate zoospores (secondary) which on germination produce vegetative hyphae.

◆ **E. Disease cycle (*Development of Disease*)** — The disease is propagated by sporangia which germinate directly or indirectly *i.e.* by zoospore formation. 90% relative humidity favours the sporangial germination. Production of sporangia is accomplished in a saturated atmosphere within a temperature range between 20°C to 26°C; but the infection declines at temperature above 20°C, because of fall in germination percentage and the slow growth of germ tube. A cool weather accompanied by abundant moisture is ideal for bulk production of inoculum.

Various theories have been put forward to explain the disease cycle and availability of inoculum. The persistence of the fungus in soil is out of question because temperature during summer becomes very high.

The only source of inoculum is through infected seed tubers. The large scale practice of storing the potato seeds (tubers) in cold storage has mainly resulted in perpetuation of the disease in plains of India. Since oospores are formed in nature, it is also presumed that the pathogen possibly perennated in that stage. Therefore primary inoculum of the disease in the field comes from the planting of infected seed tubers and from oospores in the previous year's plant debris, the pathogen next invade developing sprouts to form lesions and sporangia or zoospores above ground. After formation of sporangia or zoospores, the disease spreads to large number of plants. The infection by sporangia or zoospores may take place through any part of leaves and stems, either through stomata or through epidermis. In tubers, infection takes place through 'eyes', lenticels or wounds.

Disease cycle of Late in word diagram :



◆ **F. Control**— Late blight of potatoes can be controlled successfully by the following methods :

(1) Only disease-free potatoes should be used for seeds.

(2) Potato dumps or cull piles should be burned before planting time or sprayed with strong herbicides to kill all sprouts or green growth.

(3) All volunteer potato plants in the area (whether in the potato or other fields) should be destroyed, because any volunteer potato plant may be the source of infection.

(4) Only the resistant varieties available i.e. varieties of potato that are either immune from or resistant to late blight should be planted. Such varieties include Kufri-Sinduri, K Chandramukhi, K Kissan, R K M, K Chamatkar, etc.—such varieties resist one or more races of the late blight fungus.

(5) Late blight may be controlled successfully by chemical spraying with fungicides, if applied properly, *i.e.* by foliage spraying with Bordeaux mixture or with non-metalic readymade copper fungicides such as Perenox, Dithane M-45, Fytolan, etc.—spraying should start when plants are 15-20 cm. high or at least 10 days before the late blight usually appears in the area; sprays should be repeated every 10 or 15 days during dry season or every day during rainy season, or once every 4-5 days when the weather is damp and misty. In plains, 2-3 sprays of 0.4% commercial copper fungicides such as Fytolan or Blitox-50 at an interval of 15 days are effective. In hills 4 sprays with 0.2% Dithane Z-78 at an interval of 10 days are given. Other chemicals used for late blight control include some dithiocarbamates such as maneb, nabam usually mixed with zinc sulphate forming a tank-mix zineb and copper oxychloride.

● 2.2 White Rust of Brassicaceae :

White rust is a common disease on crucifers, namely *Brassica napus, Brassica alba, Brassica juncea, Brassica campestris, Raphanus sativus, Capsella bursa-pastoris, Nasturtium indicum*. The disease is known for its wide spread occurrence.

Ascospores infecting flowers produce conidial stage and conidia spreads further infection when they are carried to other flowers. This continues as long as favourable atmospheric conditions are available. With lowering of temperature, the flowering period gets prolonged and glumes remain open, increasing susceptible period to infection.

Control :

(i) Seeds used must be made ergot free. The process involves dipping the seeds in 20-32% solution of sodium chloride or 32-37% solution of potassium chloride. Ergots floating in the surface are separated. The ergot free seeds which sink below are washed with water and dried and then used for sowing.

(ii) Crop rotation method is also effective in eradicating the disease. Non susceptible hosts or leguminous plants when cultivated alternately with susceptible host (grain crops), will keep away the disease as previous year's sclerotia in the field though may germinate, but will fail to produce any infection in absence of susceptible host.

(iii) Sowing seeds 2-3 years old, may prevent sclerotial germination, if at all they are present, as they will lose their viability.

(iv) Before blossoming takes place in the cultivated cereals, the susceptible grasses should be removed to prevent infection from diseased grasses.

(v) Hay should be cut while they are green, i.e., before the development of ergot.

(vi) Deep ploughing method has to be used in order to bury the sclerotia and prevent their germination.

● 2.5 Black stem rust disease of Wheat :

◆ **A. Occurrence and Importance**— Black stem rust disease appears on the wheat crop in all wheat growing countries throughout the world; this disease also appears in an epiphytotic form in many countries. In northern India the disease does not appear before March. In southern India, the disease, however may appear as early as the last week of November.

Black stem rust causes losses by reducing yield and quality of seed. The amount of losses caused by stem rust may vary from slight to complete destruction of wheat fields over large areas. Heavily infected plants may die under extreme situation.

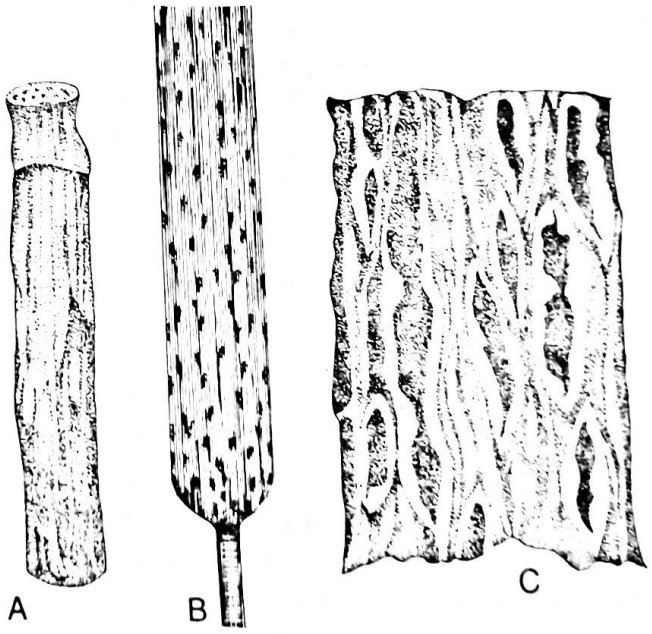


Fig. 2.5 Black stem rust disease of wheat. A—Presence of teleutosori on a portion of a diseased stem. B—Presence of uredosori on a portion of a diseased leaf. C—Portion of a stem with enlarged teleutosori.

◆ **B. Symptoms**— The pathogen causing black stem rust of wheat produces symptoms on two distinctly different kinds of host plants (primary and secondary). The most serious and economically important symptoms are produced on wheat plant (primary host) while economically unimportant symptoms are produced on barberry plant (secondary host).

(1) ***On wheat plant*** (Fig.2.5)— The symptoms on wheat appear at first as long, narrow, elliptical or oblong and brown pustules, parallel with long axis of the stem, leaf sheaths and leaves (lower surface mainly) of young seedlings or of plants at any stage of growth. In later stages of plant growth, pustules may appear on the glumes and even the beards. Those pustules called uredia grow and fuse to form larger lesions of dark brown colour, the pustules vary in size from small to about 3 mm wide by 10 mm long. Within a few days the epidermis covering the pustules is ruptured irregularly exposing a powdery mass of reddish or rust-coloured spores, called uredospores.

In the later stage, as the plant approaches maturity, the reddish or rusty colour of the pustules turns black because the fungus produces teliospores instead of uredospores and uredia are transformed into black and smooth-walled telia. Telia form oblong to linear lesions and are more frequent on leaf sheaths than on stem.

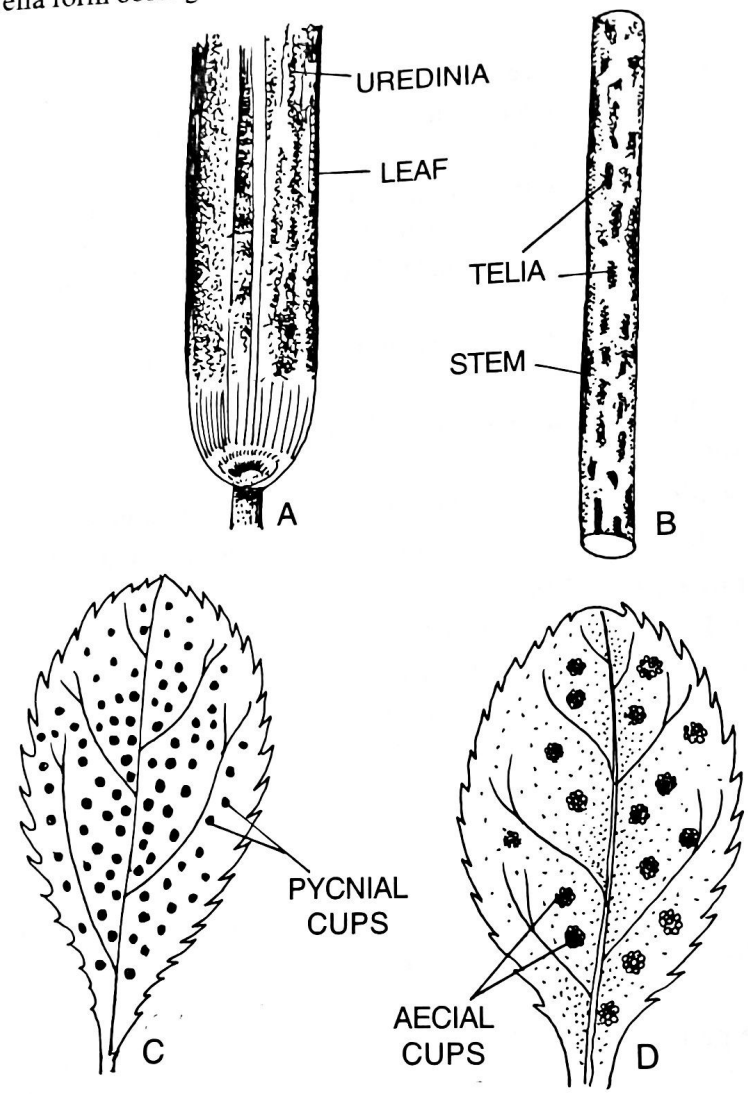


Fig.2.6 Black stem rust disease of wheat — A-Uredinia on the wheat leaf; B—Telia on the wheat stem; C—Pycnia on the upper surface of barberry leaf; D—Aecia on the lower surface of barberry leaf.

Sometimes uredia and telia may exist on wheat plants in such a great numbers that greater part of the plant appears to be covered with the ruptured areas which are filled with either reddish uredospores or the black teliospores or both. In severe attacks, plants in general look sickly and fail to form normal ears; the grains become shrivelled and lighter in weight.

(2) ***On barberry plant*** (Fig.2.6D)— The symptoms appear on the leaves and sometimes on young twigs and fruits as yellowish to orange-coloured spots. On the upper side of the leaf and within the spots, a few minute dark-coloured bodies bearing a small droplet of liquid appear. On the lower side of the leaf, beneath the pycnia, groups of orange-yellow cup-like projections called aecia appear. The host tissue bearing the aecia becomes often hypertrophied. The aecial wall, called a peridium usually protrudes at the margin of the cups.

◆ **C. The causal organism** (The pathogen)— *Puccinia graminis tritici* Erikss & Henn. This fungal pathogen is a basidiomycete. It is heteroecious, its principal host is wheat plant (*Triticum aestivum*) and the alternating host is barberry plant (*Berberis vulgaris*).

◆ **D. Etiology of the Pathogen**—The mycelium of this fungus is colourless and produces several different types of spores. Prominent uredia (uredosori) are the first to appear on the culms, leaf-sheaths and also the leaves of wheat plant; they are oblong to circular, reddish-brown, powdery and frequently merge into one another. The dikaryotic mycelium is intercellular, the hyphae produce small round or branched haustoria which draw nutrition from the host cells. A mass of hyphae collects beneath the host epidermis and forms an uredosorus (uredium). From the base of this sorus, numerous stalked, one-celled, oval, brown, binucleate (dikaryotic), thick-walled with minute spines on the outer wall and 4-germpored (equatorially arranged) uredospores arise. Uredospores germinate in presence of water or moist air forming germ tubes which are capable of re-infecting (secondary infection) wheat plants; hence uredospores are called repeating spores.

Telia (teleutosori) arise late in the season, either in uredia or independently from the same or similar dikaryotic mycelium. They are oblong to linear, dark-brown to black and not powdery although spores (teliospores) are exposed through rifted epidermis. A teliospore (also called teleutospore) is stalked, two-celled, spindle-shaped, slightly constricted at the septum, dark brown (but black in mass) and has a thick smooth wall with 2 germ pores—one at the apex and the other below the septum; the apex is rounded or pointed. Each cell of a teliospore possesses 2-nuclei (derived from the dikaryotic mycelium); at maturity, the two nuclei in each cell fuse forming a diploid nucleus. Hence mature teliospores represent the diploid phase in the life cycle of *P.graminis tritici.* Unlike uredospores, teliospores do not germinate immediately but undergo a period of rest for several months. Teliospores as usually germinate in presence of moisture; on germination each cell of a teliospore produces a long and four-celled promycelium (basidium); during the formation of promycelium, the diploid nucleus undergoes meiosis and 4 hyaline, roundish sporidia *i.e.* basidiospores (2 of '+' strain and 2 of '–' strain) are formed on sterigmata (formed from the cells of promycelium). Sporidia fall off easily and are blown about by wind. Being a heteroecious fungus, the sporidia of *P.graminis tritici* are unable to infect wheat plants. Therefore sporidia germinate on barberry plant (alternating host) in presence of moisture to form monokaryotic mycelium.

This mycelium forms two types of flask-shaped and ostiolate pycnia (spermogonia) i.e. '+' and '–' on the upper epidermis of leaves of barberry plant, they remain embedded in the host tissue in orange-yellow spots. Pycnia contain paraphyses and hyaline pycniospores (spermatia) which emerge in a viscous exudate through an ostiole along with slender flexuous hyphae. The pycniospores are carried to respective '+' to '–' and '–' to '+' flexous hyphae through insects establishing dikaryotic condition.

The dikaryotic mycelium then produces saucer-shaped aecia on the lower epidermis of barberry leaves; aecia have well-developed peridium which breaks with the maturity and protrudes out of the leaf. The aecium produces aeciospores in chains. Aeciospores are one-celled, binucleate and hexagonal in shape; they are disseminated by wind and infect wheat; being dikaryotic, the aeciospore produces dikaryotic mycelium which ultimately gives rise to uredosori on wheat plant.

◆ **E. Disease cycle**— The pathogen survives on stubble and straw of primary host (wheat plant) for several months as teleutospores. Practically this spore convey the disease to the alternate host i.e. barberry. Being heterothallic, the teleutospore of *P.graminis tritici* on germination produces basidiospores of two opposite strains i.e. 2 of '+' and 2 of '–' strains. Basidiospores are dispersed by wind and fall on barberry plant infecting leaves mainly. Two types of pycnia (+ and –) are produced towards the upper epidermis of barberry leaf by the respective (+ and –) basidiospores. Following spermatization by insects or raindrops, dikaryotic condition is established which results in the formation of aecia and aeciospores. The aeciospores are cut off in chain from the aecia and are carried to the wheat plant by wind. The aeciospores germinate in presence of moisture and ultimately form dikaryotic hyphae which ramify intercellularly into the parenchymatous tissue, from this hyphae uredospores are produced in uredosori. The uredospores are carried by wind and germinate on healthy wheat plants to spread infection. With the dissemination of uredospores, fresh uredospores continue to be formed, i.e. the uredospore-cycle may be repeated several times during the season, comprising the red-rust stage or repeating stage, which causes the chief damage. In uredosori and in other spots, the hyphae give rise to teleutosori and teleutospores as the primary host plant approaches maturity (refer Fig. 5.7, Fungi portion).

On the plains of India, the source of primary infection is the uredospores formed in wheat plants on hills, where the crop is grown throughout the year. When winds blow from hills downwards, the uredospores reach the plains infecting wheat crops late in the season. Uredospores initiate the infection and produce uredosori, these uredosori produce several successive generations of uredospores. This condition results in spread of infection to healthy plants. In the last few generations of uredospore-formation and with the gradual maturity of wheat plants, teleutospores in teleutosori appear which perish in the high temperature of the intervening summer months. Next year, the wind again bring the primary inoculum *i.e.*, uredospores, from the hills. The dikaryotic aeciospores do not initiate the disease cycle in India as they do in U.S.A. Since barberry plants (alternating host) do not carry natural infections of this pathogen, the barberry eradication programme, which was successful in U.S.A., is ineffective in India.

◆ **F. Control**— (1) The most effective and the only control measure of the stem rust of wheat is through the use of wheat varieties resistant to infection by the pathogen. In India 14 physiologic races and 6 biotypes of the fungus are reported. It is difficult to obtain resistance against all races in one wheat variety—hence varieties that are resistant to most virulent locally existing races should be used in a particular area.

(2) Excessive use of nitrogenous manures which influence the growth of rust fungi should be avoided. Dense seeding also to be avoided.

(3) Proper drainage in the wheat field should be maintained, as the dampness of the soil and atmosphere promotes the growth of rust fungi.

(4) Chemical control of black stem rust of wheat too is available. It can be controlled by dusting with sulphur @ 6.7 to 9.0 kg. per acre or spraying with zinc sulphate or parate @ 2.25 litres + 336 grams per 450 litres water per acre. As soon as the symptoms of the disease appear, the crop may be sprayed 3-4 times with dithiocarbamate fungicides such as Dithane Z-78, Dithane M-22, Dithane S-31, etc. @ 1 kg. per acre. Several other fungicides such as sulphur, dichlone, zineb, maneb, etc. can effectively control the stem rust of wheat—in most cases 5-10 applications per season are required for complete control of the rust.

Fig.2.7—Loose smut disease of wheat. A–B—Diseased ears (inflorescence).

● 2.6 Loose smut of Wheat :

◆ **A. Occurrence and Importance**— Loose smut of wheat is fairly common in most of the wheat growing areas of the world; but the incidence of the disease is more abundant and serious in humid and sub-humid regions.

Loose smut causes damage by destroying the kernels of the infected plants, also by lowering the quality of the seed of the non-infected plants upon harvest. In India, the disease causes a loss of over 50 million rupees annually (Luthra, 1955); from other parts of India epiphytotics causing 3-30% damage are often reported.

◆ **B. Symptoms**— Loose smut generally does not produce any symptoms until the plants are in ear i.e. diseased plants look normal till the ears appear. Diseased plants usually ear earlier than healthy ones, and smutted ears are rapidly elevated above those of the healthy plants. In infected ears, each spikelet is entirely transformed into a smut mass consisting of black or olive-green powdery spores—the spores in young spikelets are covered by a delicate silvery membrane which soon bursts and sets the spores free; when the spores are blown off by the wind, the central axis i.e. rachis of the spikelet is left behind as a naked stalk (Fig.2.7, B).