Lecture No. B1MIC P1U4.3

COMMON FUNGAL DISEASES OF CROPS

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1. Early blight of potato

INTRODUCTION

Meaning of Blight-

In many cases the leaves, stems or twig in response to the attack of the pathogen undergo rapid discolouration followed by death. The dead parts become dark or brown in colour.This condition of the affected organ is called “blight”.

Early blight is a common foliage disease of potato and Tomato. It is a common occurance both in cold and in warm regions in India and abroad where potatoes and tomatoes are grown. The pathogen causes injury to the leaves as a leaf spot disease and provoke premature defoliation and tuber rot of potato and fruit rot of tomato.

Causal organism - Alternaria *solani*

The pathogen is **Alternaria *solani*** which belongs to form class Deuteromycetes.
Its mycelium consist of light brown, slender, septate, sparsely branched hyphae which become dark coloured with age.

The hyphae ramify in the intercellular spaces but later penetrate the cells of the invaded tissues.
The conidiophores are short and dark coloured arise from the older diseased tissue of the host and emerge through the stomata .The conidia are dark coloured, beaked , muriform and multiseptate.

There are 5-10 transverse septa and a few longitudinal once. The beak is long, septate and rarely branched. The conidia are born singly but in pure cultures , in short chains of two.
Each conidium develops from a bud formed on the terminal cell of the conidiophore. The mature conidia are detached readily and dispersed by air currents , water, and insects.
On a suitable host they germinate readily in moist weather.

Symptoms of disease

The disease appears on the leaflets, 3-4 weeks after the crop is sown as small, isolated, scattered pale brown to dark spots , oval or angular in shape mostly up-to 3-4 mm. in diameter.
Each spot is usually delimited by a narrow chlorotic marginal zone which fades into the normal green.
The chlorotic zone increases with the increase in size of the spot.
The oldest(lowest) leaves are affected first and the disease progresses upwards.
The necrotic tissue of the spot often shows a series of concentric ridges .
The number of spots on the leaflets are few , but in favorable condition the spots increase in number and size involving the entire leaf surface.
The spots become hard in dry weather and the leaflet curl under humid conditions , the disease areas coalesce and big rotting patches appear on the leaf surface. In severe cases of infection leaf dry up, shrivel and drop off.
Falling of leaves starts with the older and some remain at the top . The potato tubers are also infected and rotted .The surface lesions on the potato tuber are a little darker , slightly sunken ,irregular in shape and a brown corky dry rot beneath the lesion.

Disease Cycle

The source of primary inoculum is the infected plant debris such as the dried leaves , stems , potato tubers and contaminated tomato seeds.
Primary infection brought about by conidia or mycelium from the infected debris in the soil.
The conidia germinate at the optimum temperature of 28 ˚-30 ˚c within 35-45 minutes.
The germ tubes gain entry into the lower leaves of the host plant through stomata.
The fungus penetrate the host leaf and stem directly through the epidermis . The incubation period varies from 48-72 hours. The primary infection appears on the foliage as leaf spots within 2 or 3 days under favourable conditions and moisture.
Secondary spread immediately follows through conidia produced on the primary spots within 5- 7 days after infection.
Conidia start forming when the spots are about 3 mm. in diameter. Heavy dew with rains now and then promote abundant sporulation. The mature conidia are readily detached and dispersed through the air currents , water and insects.

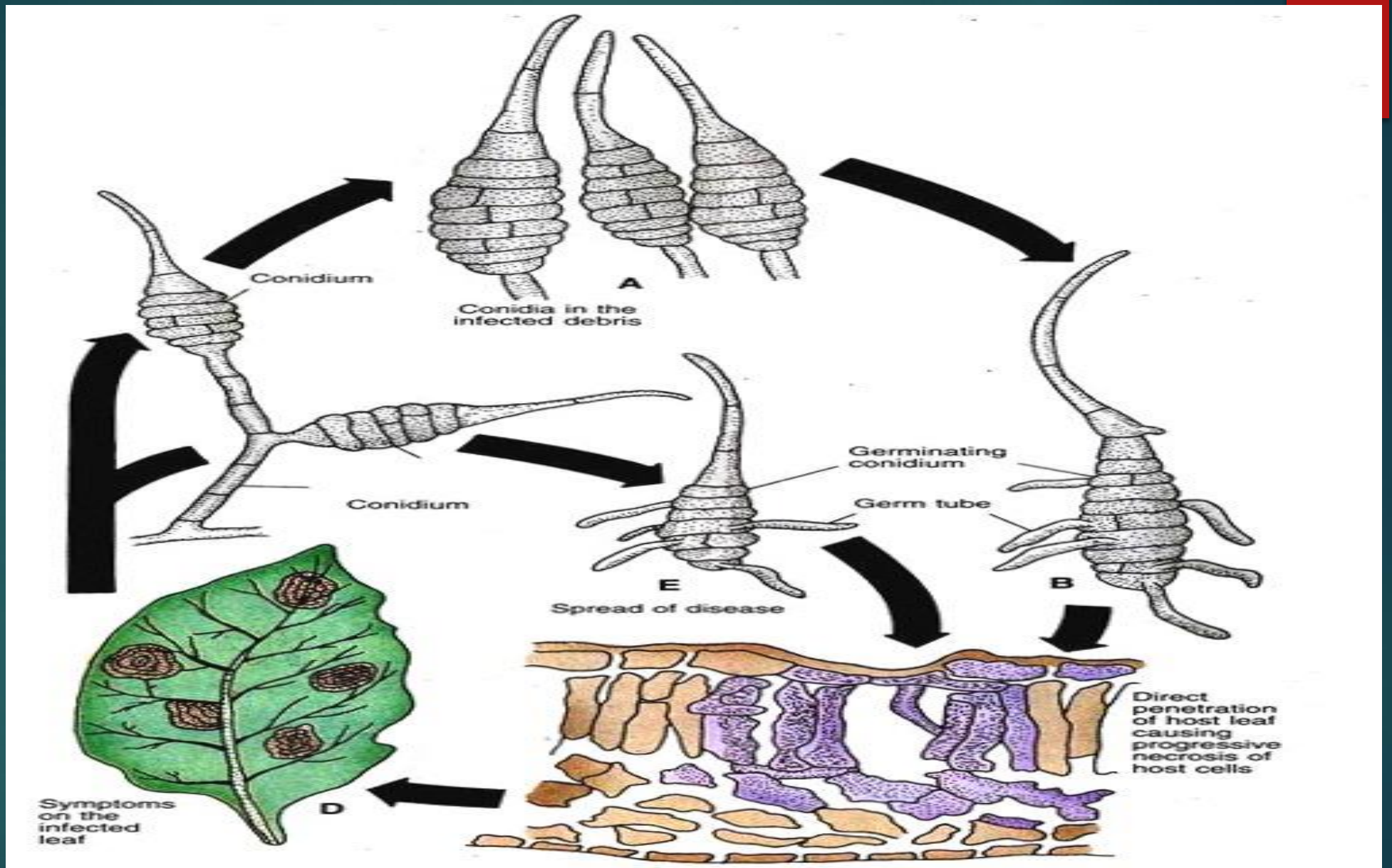

Fig. 22.23 (A–B). Early blight of potato—Pictorial disease cycle of *Alternaria solani*.

CONTROL MEASURES

When the plants are 15 – 20 cm. high , weekly sprays with Bordeaux mixture or other suitable fungicides throughout the period of plant growth effectively control the early blight.

Spraying potato crop with Dithane M -45 was most effective against early blight disease.

Thus application of regular spray schedule is an effective measure.

2. Late blight of Potato

INTRODUCTION

Late blight is a serious fungal disease of potatoes. The late blight epidemics are rare in the plains of India. It is destructive to the crop grown in the rainy season . The disease occurs annually in cooler Himalayan regions extending from Assam to Kashmir at an altitude of 6000 feet or more as the crop is grown in the rainy season. Now it has established itself in the Indo –Gangetic plain and occurs annually in the state of Punjab , Uttar Pradesh , Bihar , and West Bengal.

Causal organism- **Phytophthora *infestans***

The mycelium is aseptate coenocytic, hyaline and branched. The hyphae are both intercellular and intra cellular.
They form rudimentary haustoria in the host leaf cells but in the tubers the hautoria are more common and elaborate(club shaped, hooked, or spirally twisted).

Symptoms

The disease first appears on the tops of the plants generally after the blossoming period but mostly in the month of January .
The disease makes its appearance as small , dead , brownish to purplish black areas or lesions . These appear on the tips and margins of the leaflets, rachis , petiole , and stem. Under low temperature and high humidity the lesions rapidly increase in size involving the whole surface of the leaf.
The disease generally first attacks the leaves and petiole near the ground and the lesions appear on the lower surface of the leaflets on individual plants and then spreads upwards. Finally , a rapid and general blighting of foliage occurs. The blighted leaves curl and shrivel in dry weather. Examination of the lesions on the surface of the leaf reveals a delicate growth of the fungus parasite in the form of whitish powdery bloom.

Disease Cycle

The infected tubers are main source of primary infection . The fungal parasite overwinters as a dormant mycelium in the infected tubers . It becomes activated at the time of germination of the diseased seed tubers among the Planting stock or infected tubers remaining in the ground after a previous crop. The activated mycelium invades the healthy sprouts.

One another view is that the thick walled resting oospores which are found in abundance in the infected tubers are the important over wintering structures. They play a significant role as the source of primary infection . At the planting time , the resting oospore germinates . The germ tube after emergence usually ends in a terminal sporangium . The contents of the latter divide to form zoospores . The released zoospores invade the healthy sproutes and bring about infection . The infected sprouts emerge above ground and produce shoots which contain the mycelium . It grows and ramifies in the intercellular space absorbing nutrition by putting haustoria into the host cell under suitable conditions of temperature and humidity , the mycelium pushes out hyaline , branched sporangiphores through the stomata of the host leaves. The thin walled , ovoid , lemon – shaped sporangia each with an apiculate tip , are born singly at the tips of sporangiophores or their branches.

As the sporangium reaches maturity, the supporting hyphal branch immediately below it swells slightly and continues to grow turning the attached sporangium to the side . The elongation of the branch proceeds and a new sporangium is formed. The process is repeated . A fertile branch of sporangiophore is thus characterized by 9 or 10 such swellings occurring at intervals.

The mature sporangia are readily detached and spread by slashing rain or air currents to new potato plants.

Control Measures

1. Selection of seed tubers- The seed tubers should be free from the disease.

2. Storage of tubers at 40 degree centigrade or below.

3. Growing disease resistant varieties.

4. Use of fungicides- Dithane z- 78 and Dithane M -22 are more effective than copper fungicides that are Perenox , Blitox- 50 and Fytalon.

3. False Smut of Rice Disease

Smut, **plant disease** primarily affecting grasses, including corn (maize), wheat, sugarcane, and sorghum, caused by several species of fungi.

Smut is characterized by fungal spores that accumulate in sootlike masses called sori, which are formed within blisters in seeds, leaves, stems, flower parts, and bulbs.

False smut causes chalkiness of grains which leads to reduction in grain weight. It also reduces seed germination. The disease can occur in areas with high relative humidity (>90%) and temperature ranging from 25−35 °C.
Rain, high humidity, and soils with high nitrogen content also favors disease development. Wind can spread the fungal spores from plant to plant.
False smut is visible only after panicle exsertion. It can infect the plant during flowering stage.
The disease affects the early flowering stage of the rice crop when the ovary is destroyed. The second stage of infection occurs when the spikelet nearly reaches maturity.
These causes chalkiness and can reduce 1,000-grain weight. It also causes a reduction in seed germination of up to 35%. In damp weather, the disease can be severe and losses can reach 25%. In India, a yield loss of 7−75% was observed.

Symptoms of false smut of rice

Symptoms

Villosiclava virens infects rice flowers and transform it into rice false smut balls.

Rice false smut are small at first growing slowly and enclosing the floral parts.

The early balls are slightly flattened and smooth and covered with thin membrane.

As the pathogen growth intencifies , the rice false smut balls burst with chlamydospores and becomes orange then later yellowish –green or greenish black.

Rice false smut balls generate sclerotia when the temperature difference between day and night in large in autumn.

The false smut ball is the only visible symptoms of rice false smut disease.

Causal organism- *Villosiclava virens* (anamorph: *Ustilaginoidea virens*

The colony growth of *V. virens* in culture medium PSA (potato-sucrose-agar) is very slow, with a growth rate of approximately 20 mm in diameter per week . *V. virens* produces pigments during culture in PSA and is prone to generate small colonies and plenty of conidia in PSB (potato-sucrose broth).

The conidia are elliptical with diameters ranging from 3 to 5 μ m . Upon maturation or under unfavourable conditions, conidia may develop to rounded chlamydospores with prominent spines on the surface.

One or two sclerotia, which are the sexual structure of *V. virens*, can be formed in a false smut of rice ball . Sclerotia are horseshoe-shaped and the length ranged from 2 to 20 mm . After several months of dormancy, sclerotia could germinate and produce fruiting bodies with stromata which ultimately generates ascospores with length reaching 50 μ m and width 1 μ m .

Disease cycle

V. virens attacks rice flowers and forms RFS balls covered with chlamydospores and generate sclerotia, which are considered as primary inoculum of false smut of rice disease.
As to the sexual cycle, a large number of sclerotia can be produced when RFS balls develop in autumn . Sclerotia cannot germinate immediately, requiring a dormancy period of 2–5 months at room temperature or 4°C. They overwinter in the field and could survive up to 10 months with maintaining germination ability to generate ascospores under 25°C and high humidity.

Sclerotia can survive with high germination rate up to 5 years when stored in a dry environment at 2–4°C. In the next spring, sclerotia start to germinate, and the germination time varies among different sclerotia. A sclerotium could produce up to 21 million ascospores. Although sclerotia are easy to rot in paddy fields under natural conditions, a limited number of sclerotia can still produce plenty of ascospores. Ascospores are able to infect rice flowers to form False Smut balls .Therefore, it is believed that sclerotia act as primary inoculum of False Smut of rice and play an important role in the disease cycle.

Rice false smut balls with chlamydospores and sclerotia are formed in rice spikelets, and overwinter in field .
Next spring, spores in soil and on contaminated rice grain germinate and attack rice roots and coleoptiles when rice seeds are germinating.

Hyphae grow intercellularly in roots and coleoptiles, but could not infect seedlings systemically. Instead, hyphae may grow epiphytically on leaf surface or leaf sheath, and reach the external surface between tiller buds at the late vegetative stage

It is possible that the pathogen hyphae reach the inner space of rice panicles and initiate infection at the late booting stage .Meanwhile, conidia produced by chlamydospores and/or ascospores from sclerotia also initiate attack on rice spikelets in developing panicles.

Spores could firstly germinate on the surface of a spikelet and the hyphae extend into the inner space of the spikelet via the gap between the lemma and the pale.

Stamen filaments are the major infection sites for the pathogen . After successful colonization in floral organs, a large amount of fungal mass are formed and eventually grow into a false smut ball .

Control measures

The field should be cleaned.
Infected seeds, panicles, and plant debris after harvest should be removed.
Humidity levels should be reduced.
It should be performed conservation tillage and continuous rice cropping.
Moderate rates of Nitrogen should be used.
Resistant and certified seeds should be used.

4. Tikka disease of Ground nut

Tikka disease is a common fungal **disease of groundnut**.
This **disease** is characterised by dark spots on aerial part of the plant , mainly leaves.

Causal Organism: The causal organism of **tikka disease** are the species of the genus Cercospora namely ***Cercospora arachidicola and Cercospora personata***

Symptoms of Tikka Disease of Groundnut

All parts of the host plant above soil level are attacked by the disease. The first visible symptoms appear on the leaflets of lower leaves as dark spots which at a later stage, are surrounded by yellow rings. The spots are circular. They appear in a large number on the leaves. Mature spots are dark-brown to almost black, particularly on the upper surface of the leaflets Where as, on the lower surface they are lighter in colour. The spots are few on the leaf petioles and stem. Sometimes spots coalesce resulting in the defoliation. The shedding of leaves is a characteristic feature of the disease. Due to excessive spotting and consequent leaf fall, smaller and fewer nuts are formed.

In cases where young plants are attacked by the disease, nuts fail to develop in them. But the mature plants when attacked by the disease produce immature nuts which are shrivelled and become loose in the shell. The total effect is the loss in yield.

Causal *Organism- Cercospora personata*

The spotting is due to the attack of ***Cercospora personata .Cercospora personata*** possesses mycelium which is entirely internal and ramifies intercellularly by developing haustoria in the palisade and spongy mesophyll cells of the host.

The mycelium forms dense stroma which produces long septate to non- septate geniculate hypophyllous conidiophores.
The conidiophores emerge in tufts by rupturing host epidermis. Conidia are pale-brown, obclavate or cylindrical, septate, measuring 30-50 µ m in length and 5-6 µ m in breadth.

Cerospora arachidicola has both internal and external, intra- and intercellular mycelium without haustoria. The mycelium produces scanty stroma.
The conidiophores are usually amphigenous, but on the younger spots they are developed exclusively on the upper surface. They are geniculate, non-septate to septate and produce, hyaline to slightly olivaceous, obclavate 4- to 13-septate, often curved, conidia measuring 38-108 µ in length and 2-5 µ in breadth .

Disease Cycle of Tikka Disease of Groundnut

The conidia may remain adhered to shell. They have also been found to remain associated with the seeds and are responsible for primary infection. A temperature range of 26°C. to 31 °C. with high atmospheric humidity is favourable for disease development.

Prolonged low temperature and dew also favour infection. The entrance of the pathogen in the host tissue takes place either by direct penetration through the epidermal cells or by way of stomata.

The leaf infection is largely through the upper surface of the leaflets. The fungus mycelium ramifies the host tissue in and around the infection court and aggregates underneath the epidermis and forms stroma.
During the development of stroma the epidermis is ruptured by the pressure developed in the host tissue and the conidiophores developed from the stroma emerge out, ultimately conidia are produced on them. These conidia form the secondary inoculum through which secondary infection is induced. The disease is disseminated by wind which blows the conidia from leaf to leaf. Insects and splashes of rain play important role in the dissemination of the disease.

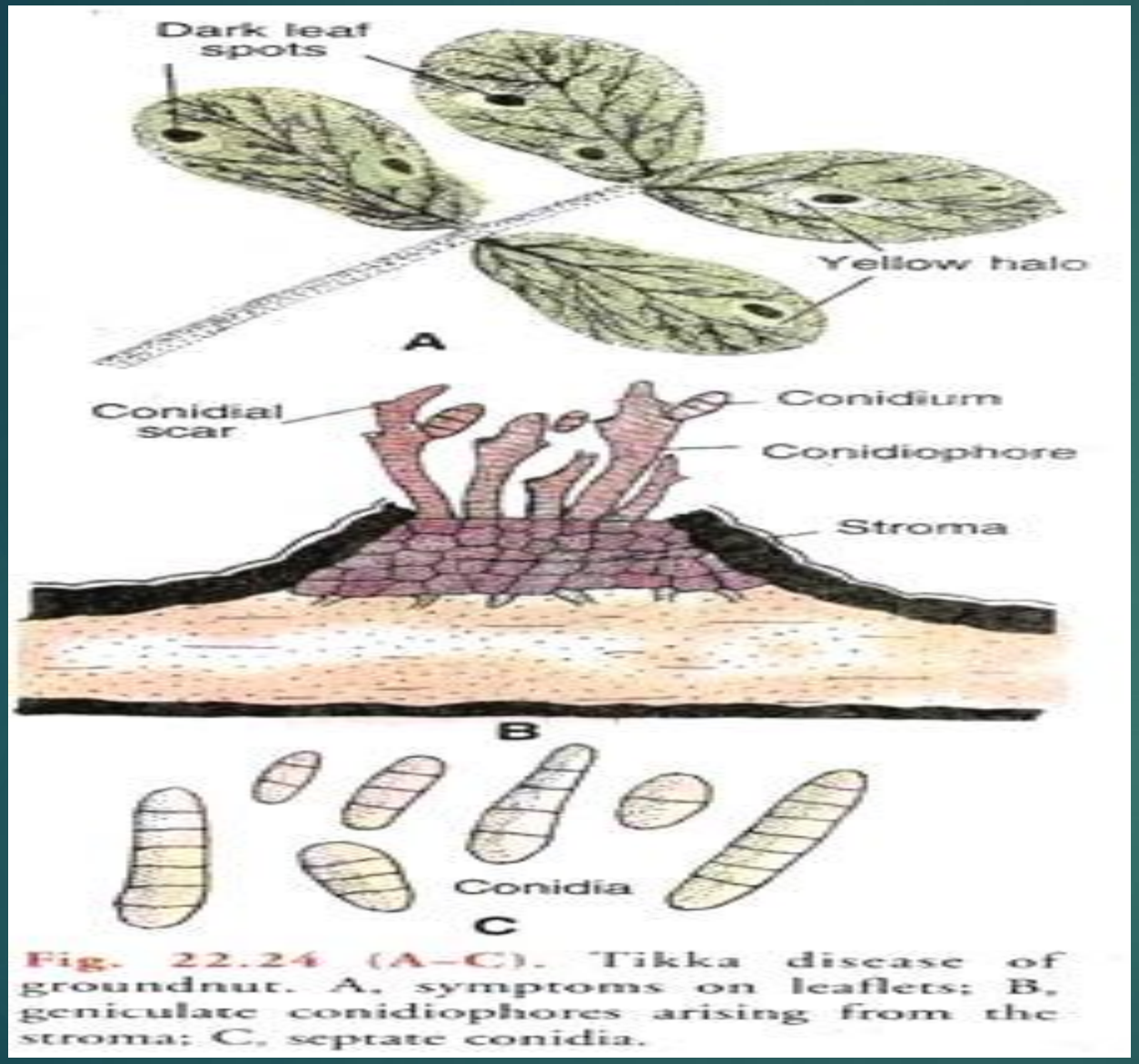

Fig. 22.24 (A–C). Tikka disease of groundnut. A, symptoms on leaflets; B, geniculate conidiophores arising from the stroma; C, septate conidia.

Control Measures

1. Burning of previous year's diseased plant debris should reduce the source of primary infection.

2. Two to four years' crop rotation often cuts down the rate of infection.

3. One should avoid late sowing to reduce infection rate.

4. Seed disinfection checks the disease incidence. Care should be taken to remove the shells thoroughly and the adhereing soil before seed disinfection.

5. **Red rot of Sugarcane**

INTRODUCTION

It is a serious and destructive disease of sugarcane. It occurs in tropical and subtropical regions of world where sugarcane is cultivated extensively. In India , it occurs in most of the sugarcane growing states, particularly Bihar , Uttar Pradesh , Madhya Pradesh , Haryana , and Punjab.

Symptoms:

The disease starts with **yellowing of leaves** from top except crown leaves, after rainy season followed by considerable **shrinkage of the stalk.**

Presence of reddish discoloured patches or lesions interspersed with white horizontal patches on the internal tissue i.e. pith.

As the disease progresses **the internal tissues become darker** in colour and dry resulting in longitudinal pith cavities. The fungal mycelium may be seen in these dry cavities.

Causal Organism- Colletotrichum *falcatum*

It belongs to form- class Fungi imperfecti. The mycelium is found within and the intercellular spaces between the pith cells of the host. The hyphae constituting both inter and intracellular mycelium. They are slender, colourless , branched and septate. The mycelial cells begin to collect beneath the epidermis to form stromata consisting densely packed cells. From the upper surface of each stroma arises the conidiophore and bristles called setae. The setae is hair like, septate structure. They either form a ring around the stroma or intermixed with conidiophores in the acervulus. The conidiophores are small un-septate structure .The conidia are one celled and usually sickle shaped in form. The conidia are readily detached and dispersed by air currents , rain ,rain drops , splashes , and insects. They are short lived and germinate immediately in the presence of moisture.

Disease cycle

The fungus is capable of growing and producing acervuli in the soil. It survive in the active stage for 3 – 4 months . The survival of the mycelium for this limit period is sufficient to provide easy catching of the succeeding crop because sugarcane has no dead season. The disease is borne in the seed sets which serve as chief means of survival and spread of the disease. The diseased sets sown in the soil , sprout into infected shoots which soon produce conidia in acervuli . The conidia serve as secondary means of infection and spread of the disease. They get detached and are dispersed through wind , water , and insects. On reaching the surface of healthy sugarcane surface plants , they germinate immediately in the moisture retained in the enclosing sheaths.

Control measures

1. Field sanitation is an important measure to prevent the build up of source of primary inoculum.
2. The use of healthy seed sets and resistant varieties is the most effective method to control the disease.

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