Course- B.Sc. (Honours), Part -1

Subject- Botany, Paper-II (Group-B)

Topic- Loose smut of wheat.

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Loose Smut of Wheat:

This disease is very common and widespread. It causes great damage in the wheat growing tracts of India, particularly in the Punjab, Uttar Pradesh and certain districts of Bihar and Madhya Pradesh.

Luthra (1953) reported that in India, the disease causes loss of over 50 million rupees annually. In Punjab the disease is called Kangiari.

Loose Smut of Wheat (caused by Ustilago tritici)

Symptoms of Loose Smut Disease:

The smutted ears (Fig. 22.14 E) emerge from the boot leaves a little earlier than the healthy ones. They bear loose, black, powdery masses of smut spores instead of flowers. All the ovaries and other floral parts except the awns and rachics are converted into masses of smut spores.

In the young spikelets before emergence each ovary has become a spore sac. It is the home of millions of spores. The spores of each spikelet are covered by a thin greyish or silvery membrane. By the time the ear emerges from the boot leaf the membrane ruptures to expose the black powdery mass of spores.

The ear is generally completely destroyed except the awns and the rachis. When the wind blows the spores are blown off and the bare rachis and central axis is left behind.

To it may cling a few spores that have not been blown off by the wind. It is not necessary that all the ears of a wheat plant may be smutted. Some may be found to be healthy and others diseased.

Causal Organism:

The causal organism of this disease is Ustilago tritici (Pers.) Rostr. and the host is Triticum vulgare. Fisher transferred U. tritici to U. nuda. Popp holds that since the two species differ in the type of teliospore germination, they should be considered as distinct species. The disease is internally seed borne. The mycelium of the fungus lies dormant in the grain (Fig. 22.14 A).

Mycelium of Loose Smut Disease:

Ustilago tritici is an internal parasite. It has a dikaryotic mycelium. The hyphae ramify the intercellular in spaces of the host tissue. They absorb nutrition from the host cells by diffusion. The hyphae do not produce haustoria.

Spore Formation:

The mycelium grows keeping pace with the growth of the host plant. It is chiefly confined to the stem (Fig. 22.14 C). At the time of flowering and when the inflorescence is still enclosed by the boot leaf, the mycelial hyphae enter into the ovaries of flowers.

Within the ovary each hypha grows vigorously and branches repeatedly to form a dense mass of hyphae (Fig. 22.14 D^2). The latter destroy the host tissue in the ovaries and surrounding floral parts. The cells of these hyphae are binucleate.

The hyphae undergo additional septation to form short binucleate cells. These cells swell and round off to form binucleate smut spores (Fig. 22.14 F). The smut spores are called the brand spores. Some mycologists prefer to call them teliospores.

They are spherical to oval and measure 5.9 μ in diameter. They have a finely echinulate thick spore wall which is olivaceous brown but slightly lighter on one side. The teliospores are produced in enormous numbers.

Disease Cycle (Fig. 22.14):

The loose smut of wheat is a systemic disease. It is seed borne (A). As the infected grain is sown and germinates (B), the dormant fungus mycelium within the grain resumes activity. It grows best in or near meristematic tissues keeping pace with the growth of the host plant (C).

The hyphae thus grow just behind the growing point. The presence of the fungus in the meristematic tissue exercises accelerating influence on the growth of the host which matures early producing flower heads.

At the flowering time, the hyphae reach the inflorescence region (D^1) and accumulate in the floral parts chiefly the florets (D^2) which are subsequently completely destroyed. The hyphae become swollen and additionally septate.

The segments, which are binucleate, round off, separate and secrete thick walls to become smut spores which are frequently called teliospores. The teliospores serve as a means of propagating the disease during the growing season.

They are readily carried from the smutted ears (E) by air current (F) at a time when the healthy plants are in the flowering stage. There are clouds of spores in the atmosphere over the wheat fields.

The glumes of healthy flowers are wide open and the stigmas sufficiently exposed and spread in the dry weather to the spore dust. Thus the teliospores fall on the feathery stigmas of healthy wheat flowers. Under suitable conditions (warmth and moisture) the spores germinate on the stigma (G).

Before germination the two nuclei of a dikaryon fuse in the smut spore to form a single fusion nucleus or the synkaryon. The diploid teliospore represents the hypobasidium or probasidium stage. At the time of germination the exosporium ruptures.

The endosporium grows out in the form of a short tubular hypha called the promycelium or epibasidium. The diploid synkaryon migrates into, the epibasidium and undergoes meiosis, which consists of two nuclear divisions.

As a result four haploid nuclei are formed. They are arranged in a row. Two of these are of plus and two of minus strain. Walls are laid between the daughter nuclei. The epibasidium now consists of four haploid cells arranged in a row.

Each cell of the epibasidium produces a slender hypha called the infection thread. It contains a single haploid nucleus. Out of the four infection threads two contain haploid nuclei (one each) of plus strain and two of minus strain.

Basidiospores or sporidia are not produced by the basidia of Ustilago tritici. Their place is taken up by the infection threads. The neighbouring infection threads of opposite strains fuse (Fig. 22.14 G). Consequently one of the infection threads becomes binucleate or dikaryotic.

The dikaryotic infection threads become binucleate or dikaryotic. The dikaryotic infection thread grows by elongation and clamp connections. It enters the style, grows forwards through the intercellular spaces and the channels left by the pollen tubes and reaches the ovary.

Recently Pedersen (1956) and Malik and Batts opposed this view. They hold that the infection thread pierces the young ovary wall in a week or so. From there it makes its way into the developing ovule through the integuments.

Penetration into the immature ovule occurs between the 7th and 10th day. About 10 days after infection the integuments become cutinised and resistant to infection. In the ovule the dikaryotic hypha passes into the space between the endosperm and nucellus. Here it branches freely.

The branch hyphae take about three weeks to reach the base of the raphe. Some of these pass round the bottom of the endosperm to penetrate the embryo through the scutellum. The hyphae in the scutellum are irregularly swollen and have thick and oily walls.

As the ovary ripens into the grain the fungus mycelium becomes inactive (Fig. 22.14 A). It remains small and lies dormant in the embryo chiefly in the scutellum. The dormant mycelium within the embryo carries the fungal pathogen over seasons unfavourable to growth.

It becomes activated again at the time of germination of the grain (Fig. 22.14 B). The presence of the dormant fungus mycelium in the grain shows no external signs of its infection.

The infected grains apparently look like the healthy ones. Ustilago tritici thus provides an excellent example of infection through the flower.

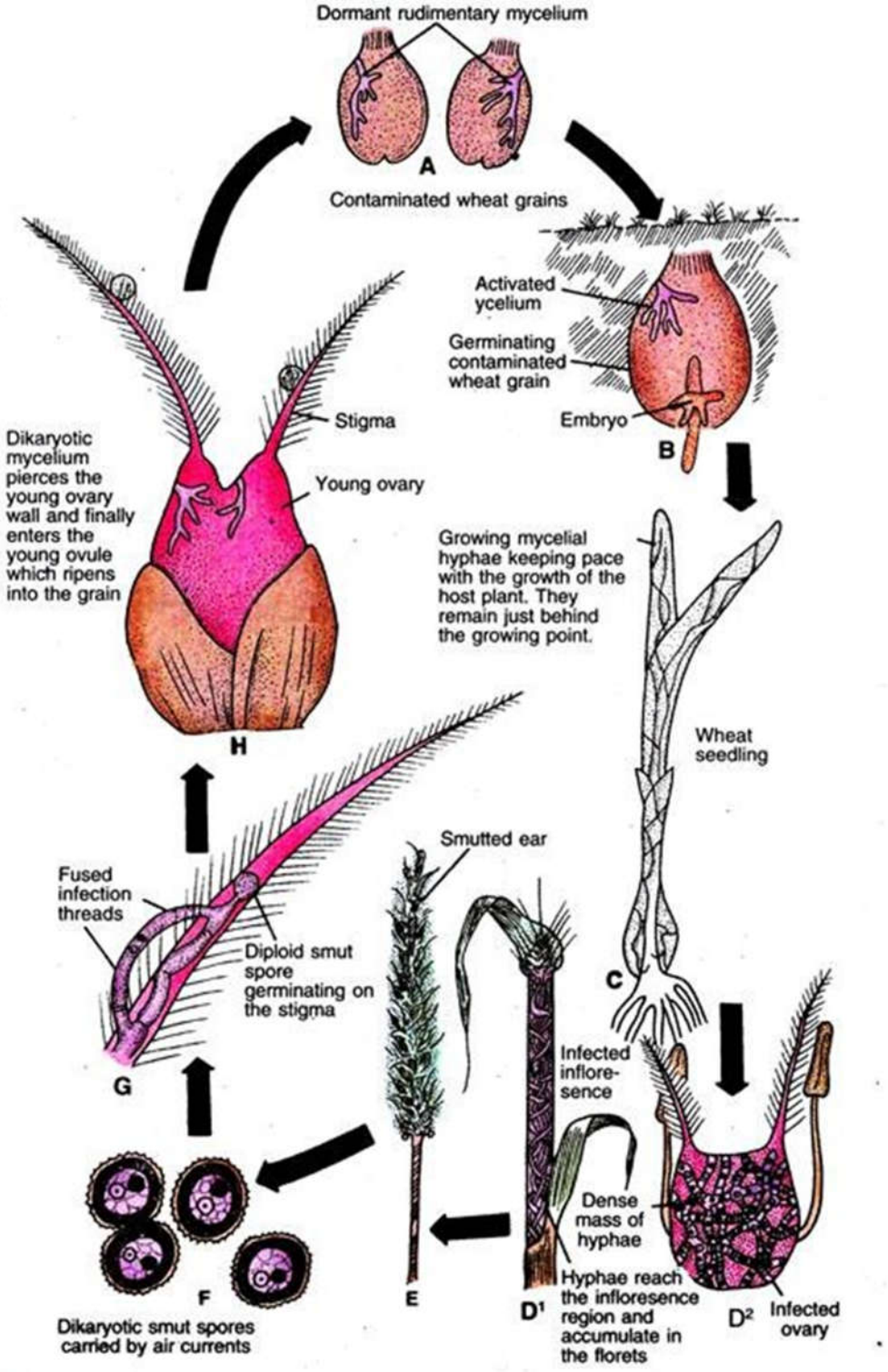

Fig. 22.14 Loose smut of wheat. A pictorial disease cycle of *Ustilago tritici*, a blossom-infecting smut.

Host-Parasite relations in Loose Smut of Wheat:

Kourssanow (1928) reported that the infected plants were generally smaller and had higher respiration rate than the healthy ones. Mather and Hausing (1960) found that the total

dry weight was reduced by 33 percent, root dry weight by 32 percent, height by 11 percent in infected plants.

Gaunt and Manners (1971) reported reduction in the photosynthetic area. Expansion of successive leaves of the host plant was delayed. This reduced the availability of assimilates for the development of the infected plants which consequently showed restricted growth of the roots and the tillers.

Such effects have been attributed to:

(i) Increased respiration and decreased photosynthesis and

(ii) To the deprivation to the host by the pathogen (U. tritici) of necessary metabolates for further development.

Effect of Loose Smut Disease:

The disease results in the reduction of yield from 20 to 50 percent. Quality of the grain is, however, not affected.

Control Measures of Loose Smut Disease:

Since the mycelium of the parasite is lodged inside the grain, external application of disinfectants is ineffective. Direct attack on the fungus living deep in the tissues is very difficult. In the first instance most of the chemicals do not reach the seat of trouble.

Some which do may injure the embryo as well. The dormant mycelium in the grain is very resistant to heat. Hence in all methods of treatment the first step is to make the dormant mycelium active. In the activated condition it is vulnerable.

It is killed by the application of moist heat.

The following methods are generally employed to kill the mycelium in the embryo of the grain:

1. Hot Water Treatment:

The wheat grains are at first soaked in water kept within a range of temperature between 26°C-30°C. They are allowed to remain there for about 4-5 hours. In the softened grains the dormant mycelium becomes active.

The temperature of water is then raised and kept constant at 54°C for about 10 minutes. At this temperature the activated mycelium is killed. This method requires strict care and supervision. The temperature should be carefully controlled.

At a range a little too low, it will fail to kill the mycelium and at a degree or so too high it will kill the embryo. In this case the embryo is killed at 56°C. The margin of error either way is thus very little. After the treatment the water is drained off.

The grains should then be dried and sown. The hot water treatment was first evolved by Jensen (1888-89) against the late blight of potato. Swingle (1892) applied it against the loose smut of wheat.

2. Sun Heating:

This method is in vogue in the Punjab and U.P. Here the sun in the months of May and June is very hot. The atmospheric temperature is very high. The suspected grains are soaked in water in flat, shallow bottomed basins with water level about two inches above the level of grain.

The basins are placed in the direct rays of the summer sun for about 4 to 6 hours, say from 8 a.m. to 12 noon. During this period the dormant fungus mycelium becomes active. The water is then drained off.

The softened grains are spread in thin layers on the brick floor in the midday sun to dry. In the cooler regions the use of galvanised iron sheet to spread and dry the grain in the sun has been recommended.

This treatment kills the activated mycelium. Mitra and Taslim (1936) recommended the sun heating method for controlling the disease in North Bihar. Luthra found the solar treatment quite suitable in the Punjab where the day temperature in summer goes very high. Bedi (1957) suggested a modification in the method. He found 4 hours presoaking period followed by one hour exposure to sun under Punjab conditions quite sufficient to kill the activated mycelium. Extra exposure to sun heat serves to dry the grain.

3. Growing Resistant Varieties:

Sowing grains of varieties of wheat which are immune from or resistant to this disease is the best method of controlling the disease. Some of the wheat resistant varieties are Np 710, Np 120, and Pb 90.

The other equally effective methods are:

4. The wheat plants with infected ears, which emerge out of the boot leaves earlier than the healthy ones, may be uprooted at once and burnt. This practice is called rogueing.

5. The grains for sowing purposes should be thrashed from uninfected wheat ears.

6. Use of systemic fungicides:

The use of fungicides, which till recently was considered impraticable to control the seed borne loose smut disease of wheat, has received much attention. Chatrath et al. (1969) found that two systemic fungicides D735 (Vitavax) and F 461 (Plantavax) give quite encouraging results when applied as seed dressing fungicides at the rate of 2.50 gm per Kg. The use of Benomyl and Carboxin to control loose smut of wheat (U. nuda var. tritici) has been recommended by many workers. Joshi et al. (1975) reported that seed dressing with 0.25 per cent Benomyl can effectively control the disease.

Thomas and Chatrath (1975) found that a systemic fungicide thiabendazole employed as a seed treatment at the rate of 0.1 to 0.2 per cent is highly effective to control the disease without affecting germination.