B.Sc. Botany

Part-I (2019-21)

Paper-II: Microbiology Fungi and Plant Diseases

GROUP-B

RUST OF LINSEED

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RUST OF LINSEED

Rust of linseed is a common disease of flax growing areas of the world. In India flax is a major oilseed crop cultivated in almost all the states. The crop is generally cultivated during October-April.

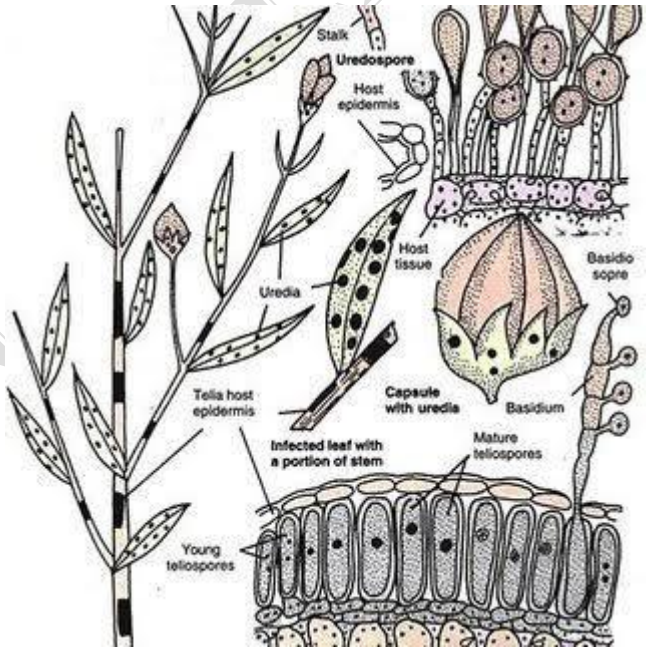
The disease generally appears in February or later but Butler (1918) has observed the disease in central India in early November. At the time of crop harvesting the affected plants get a fired appearance due to the presence of telial sori.

Host: *Linum Usitatissimum*
Pathogen: *Melampsora Lini*

SYMPTOMS

The leaves are the first to show the symptoms and gradually all the aerial parts of the plant get infected.

1. Large, orange coloured pustules generally appear on the leaves. Small pustules are initially surrounded by chlorotic areas. Little necrosis of the leaves is at first observed but it grows, becomes more general and the leaves prematurely die.
2. The pustules on the leaves are uredopustules containing uredospores. Uredopustules may also appear on stems. While the uredopustules on leaves are round and small, those on stems are elongated and irregular.



3. Teleutopustules are generally produced on stems and rarely on leaves, if they have not been shed prematurely. Orange yellow uredopustules are often surrounded by reddish-brown

teleutopustules. The contents of telial pustules do not break the epidermis of the host and remain buried subepidermally appearing glossy.

ETIOLOGY

The causal organism, Melampsora lini, is an autoecious rust as all it's the four stages (pycnial, aecial, uredial and telial) occur on the linseed plant. However, the pycnial and aecial stages of die fungus have not been observed in India. In this country, the disease is a concern of only uredial and telial stages.

The mycelium of the fungal pathogen is septate, branched, dikaryotic, sub-epidermal and intercellular. They first give rise to uredia in uredospustules and, later on, telia in teliopustules.

The uredia (sing, uredium) are orange-yellow coloured, scattered or in groups, usually rounded on leaves but elongated on the stems. Each uredium contains uredospores and paraphyses. The uredospores are binucleate, ovate, possessing fine warts and indistinct germ pores on their wall, and measuring 15-25 x 13-18 µ. They are borne on the tip of long, multicellular sporophores. The paraphyses, which are intermingled with sporophores, are 'capitate'.

The telia (sing, telium) are produced during the end of the season, are irregularly elongate, reddish-brown in colour, and are mostly formed on the stems; leaves are also affected if the latter are not shed-off by then.

In contrast to uredia where the epidermis breaks and the uredospores and the paraphyses project out on the host surface, the telia do not break the epidermis and the teleutospores remain sub-epidermal. The teleutospores are sessile, cylindrical, unicellular, reddish-brown in colour, measure 42-80 x 8-20 µ, and are arranged in palisade-like manner.

DISEASE CYCLE

(i) Perennation:
The disease is soil-borne in temperate countries where teleutospores remain in soil over winter, germinate and result in basidiospores which infect the young leaves giving rise to pycnial, aecial, uredial and telial stages successively.

But, the story is different in tropical countries, particularly in our country. Here the linseed is a winter crop sown in Oct./Nov. and harvested in March/April, therefore, the teleutospores lose their viability during the summer and, therefore, do not play any role in perennation.

Uredospores, too, are killed during summer in plains. In this way, there is no pathogen-inoculum available in Indian plains to cause primary infection. It is considered possible that the pathogen survives on linseed and other suitable hosts at hills of altitude as high as 4000-5000 ft. and the primary inoculum is wind-blown to the plains in the form of uredospores produced thereupon.

(ii) Primary Infection:
The primary infection in the plains of our country is not carried out by any inoculum perennating locally; it is caused by the uredospores produced on hills. The uredospores are blown-down to plains by wind, fall onto the surface of the host, germinate and cause infection.

Since it takes time in bringing down the uredospores from hills to plains, the rust outbreak takes place 2-3 months later from the date of sowing of crop. If there would have been any local source of inoculum, it would not take so long for the pathogen to spread when the crop is already growing in the field.

(iii) Secondary Infection:
The uredospores produced as a result of primary infection get disseminated by wind, insects and other sources and cause secondary infection on the healthy plants.

Since the uredospores are produced and germinate quickly, they cause infection in a relatively short period of time. This results in repeated secondary infection cycles in the same growing season if conditions remain favourable and, therefore, the crop is heavily damaged.

Predisposing Factors:
Low temperature and moisture are necessary for the incidence of the disease. The uredospores germinate at temp, from 3-30°C, the optimum being 15-16°C. In addition, the uredospores start germination within a short period of one and half hour if a film of water is available on the host surface. However, the uredospores retain viability for 18-20 weeks at temperatures 5-7°C.

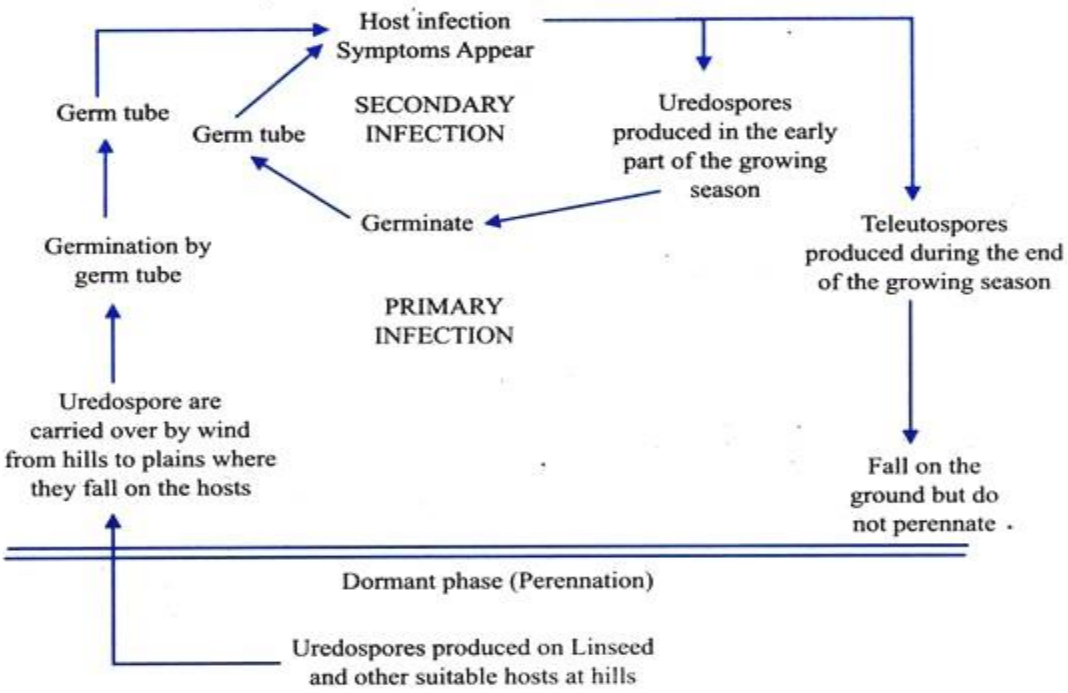


Fig. 22.19. Disease cycle of Rust of Linseed.

CONTROL MEASURES

(i) Use of disease resistant vars. like NP (RR) 9, 10, 56, 95, 218, 279B 279K3, 368, 381, 389, 415 and 501 is the only effective method to control the disease.

(ii) Seed treatment to kill the teleutospores in hills has also been suggested.

(iii) Avoidance of excessive nitrogenous manures is recommended.

The disease causes disastrous effects on the plants. Once the disease sets in a field, most of the linseed fields in the locality get affected within no time. The disease generally appears in epidemic form.

Severely infected plants get mostly defoliated due to which starch formation is also reduced. Most of the food reserves of the host is consumed by the pathogen resulting in decrease in the yield of seeds and quality of fibres. Oil content of the seeds is also reduced.