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# PEARL MILLET (BAJRA): Downy Mildew and Ergot

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**Summary**

Pearl millet or Bajra (*Pennisetum glaucum*) is grown widely in Africa and India. It is one of the vital crops that feed poor people inhabiting in semi-arid and arid tropics of Asia and Africa. India produces more than half of the world's pearl millet in an area of 10 million ha. Pearl millet is the third most important rainfed cereal crop of India, grown over 9 million ha with an annual production of 9.5 million tonnes. Pearl millet is attacked by a large number of diseases caused by fungi, bacteria, virus and nematodes. Downy mildew or green ear disease (*Sclerospora graminicola*) and ergot (*Claviceps fusiformis*) are economically important diseases of pearl millet and cause considerable yield loss. The downy mildew of bajra is a common disease in India and the disease is also known as green ear and destroys much of the crop every year. Ergot of bajra caused by *Claviceps fusiformis* is an important and widespread disease in India. The disease causes direct grain yield loss by replacing grains with toxic alkaloid-containing sclerotia, making the produce unfit for consumption. Effective and economic control of these diseases can be achieved by growing disease resistant varieties and hybrids. Besides some other management measures may also prove effective in suppressing the diseases of pearl millet. In present chapter, the etiology, symptoms, disease cycle, economic importance, distributions, and management on the above two diseases are discussed.

**Keywords:** *Claviceps fusiformis,* diseases management, *Pennisetum glaucum*, *Sclerospora graminicola.*

## Introduction

Pearl Millet or Bajra, *Pennisetum glaucum* L. (Family: Poaceae) is grown in the arid and semi-arid tropical and sub-tropical regions of Asia, Africa, and Latin America. Pearl millet is world's hardiest warm-season cereal crop and is growing over 31 million ha globally. More than 90 million people live in the drier parts of Africa and Asia and depend on pearl millet for food and income. Pearl millet is a nutritious source of food and a good source of micronutrients. In India, its grain is used to make flour and other foodstuffs. It is one of the vital crops that feed poor people inhabiting in semi-arid and arid tropics of Asia and Africa and provide basic sustainable living (Yadav and Rai 2013). Areas planted with pearl millet are estimated at 15 million hectares annually in Africa and 14 million hectares in Asia. India alone accounts for 10 million ha with a total annual production of 9 million tonnes (Yadav and Rai 2013).

Bajra (Pearl millet) is cultivated as a food, feed/fodder and fuel crop in the regions too hot and dry (Hash and Witcombe 2002). It is one of the four most important cereals (rice, maize, sorghum and millets) grown in the tropics and is rich in iron and zinc, contains high amount of antioxidants and these nutrients along with the antioxidants may be beneficial for the overall health. Pearl millet serves as a major staple food for many populations around the globe, however, it is still considered poor man's food. India is the largest single producer of pearl millet. Pearl millet is an important coarse cereal crop in western India, and occupies about 38% of the total cereal cropped area in the region (Reddy et al. 2013). Pearl millet, being a C4 plant, has a very high photosynthetic efficiency and dry matter production capacity. It is usually grown under the adverse agro-climatic conditions where other crops like sorghum and maize fail to produce economic yields. In India, the major pearl millet cultivating states includes Rajasthan, Gujarat, Maharashtra and Uttar Pradesh.

Pearl millet crop is attacked by several fungal diseases as economically important diseases are downy mildew or green ear disease (*Sclerospora graminicola*), blast (*Pyricularia grisea*), rust (*Puccinia substriata* var. *indica*), ergot (*Claviceps fusiformis*) and smut (*Moesiziomyces penicillariae*) that cause considerable yield and quality losses. The present chapter describe a consolidated account of two important fungal diseases i.e., downy mildew and ergot of bajra.

## DOWNY MILDEW (GREEN EAR DISEASES)

Downy mildew is a highly destructive and widespread disease in most pearl millet growing areas of Asia and Africa (Williams 1984; Andrews et al. 1985a). The estimated annual grain yield loss due to downy mildew is approximately 20-40% (Singh 1995; Hash et al. 1999; Hess et al. 2002). But this could be much higher under favourable conditions of disease development (Singh 1995; Thakur 1998, 2008). Seed borne inoculums in the form of Oospores sticking to the seed coat serve as an important source of inocula of the fungus. Vegetative mycelium can also be seen in the various part of seed tissues like seed coat, endosperm and embryonic tissues. However, only the mycelium in the embryonic tissue is infective (Shetty et al. 1980). In India, the disease was first reported and studied by Butler (1907) who considered the disease to be sporadic in nature not causing much damage to the crop. Mitter and Tandon (1930) reported the disease from Allahabad, Uttar Pradesh and confirmed the observations of Butler (1907). Since then, the disease on Bajra has been reported from all the states wherever Bajra is cultivated as one of the crops of '*Kharif*' season. Rai and Sinha (1965), Mathur and Dalela (1971) and Nene and Singh (1976) have estimated the loss to be as high as 27-30%. The disease appeared in an epiphytic form in Karnataka and Maharashtra during 1975 causing complete devastation of the crop.

### Symptoms

Downy mildew disease causes reduction in the plant height, number of leaves and nodes in susceptible cultivars. As a result grain and fodder yields are reduced. Symptoms often vary according to host, time of expression and

ambient conditions (Kenneth 1998). Both systemic and localized infection occurs. Infection is mainly systemic and symptoms appear on leaves and inflorescence. The downy mildew stage is prominent on the leaves (caused by sporangia) and the green ear stage affects the inflorescence/ear (caused by oospores). Green ear stage is more prominent, since the strain of the pathogen occurring in India produces more oospores than the sporangia. The initial symptoms appear in seedlings at three to four leaf stages of the plant. Symptoms can be observed in two phases, one is downy mildew phase that appears on the leaves and other is green ear phase appears on the ear head. Soil-borne spores cause systemic infection of the young seedlings. The affected leaves show patches of light green to light yellow colour on the upper surface and the corresponding lower surface bears white downy growth of the fungus consisting of sporangiophores and sporangia, infected leaves show greyish-white downy fungal growth on the lower leaf surface, white downy growth under high relative humidity (greater than 95%) and moderate temperature (20-22°C).

Initial symptoms of the systemic infection are expressed as chlorosis or yellowing of the lower leaves that progressively spread to the upper leaves and the whole plant. The infection is systemic and 7 to 10 day old seedlings show downy mildew symptoms as chlorosis on the upper and whitish sporangial growth on the lower surface of leaf. Subsequently, the leaves turn reddish brown due to oospore production and dry ultimately. The sporangia can cause further localised infection. Often the lower half of a leaf shows symptoms while its upper half remains symptomless. This is known as 'half leaf' symptom. Severely infected plants are generally stunted and do not produce panicles (Fig. 1).

**Fig. 1:** Symptoms of infected plants of pearl millet on leaf and ear.

Symptoms appear on ear head with all possible degrees of proliferations and malformations, result from transformation of floral parts into leafy structures. Often the infected plant produces symptoms only on the ear

head in the form of the leafy structures known as 'green ear' disease. At the time of panicle emergence, green ear symptoms become visible. Symptoms appear on ear head with varying degrees of proliferations and malformations. In malformation, the florets are converted into leafy structures of diverse appearance (virescence). The leafy structures are chlorotic and sometimes produce sporulation. Primary source of infection arrives from soil and systemically infect seeds. The invasion of the fungus in floral primordia plays a crucial role in deciding the extent of malformation. Generally, four types of malformations have been observed: (a) the entire inflorescence is transformed into a green leafy tuft, which is symbolically referred as "green ear"; (b) The lower part of the inflorescence is converted to green leafy mass but the upper part bears seeds. (c) only bristles become long and no malformed leafy structures are formed, and (d) leafy tufts where the shoots remain stunted and produce leafy tufts at the top in latent infections, green ear is the only manifestation of the disease (Fig. 1)

**Etiology**

The causal organism downy mildew of pearl millet (bajra) is *Sclerospora graminicola* (Sacc.) Schroet. The systemic position of the genus *Sclerospora* is given below;

| | |
|---|---|
| Kingdom | Chromista |
| Phylum | Oomycota |
| Class | Oomycetes |
| Order | Sclerosporales |
| Family | Sclerosporaceae |

This is an obligate (biotrophic) parasite belongs to oomycete. The pathogen is heterothallic, but homothallism also occurs (Michelmore et al. 1982). It reproduces both asexual (sporangia, zoospores) and sexual spores (oospores). Vegetative phase is in the form of mycelium colonizing the intercellular spaces in the host tissues of root, stem, leaf and panicle. The pathogen draws the nutrition through haustoria. The mycelium is systemic, non-septate, multinucleate (coenocytic) and intercellular, grows profusely in the tissues and at later stage produces short, stout, hyaline sporangiophores arising through stomata and branch irregularly, with stalks bearing sporangia (asexual spores) on the lower leaf surfaces. Sporangia are hyaline, thin walled, elliptical and bear prominent papilla. After the exhaust of sporulation, the pathogen produces oospores (sexual spore). Oospores are round in shape, surrounded by a smooth, thick and yellowish brown wall.

**Disease cycle**

The pathogen grows within the tissues of the plant as intercellular filamentous mycelium which gains nutrients for its growth from plant cells through haustoria which push into the individual plant cells. Primary infection in soil by Oospores and secondary infection through sporangia. Under favourable environmental conditions, the oospores germinate and enter the young seedlings of host through the underground parts and cause primary infection. When seeds containing mycelium are sown in the fields, germinate and grow along with it the mycelium and causes primary infection. The germ tubes directly penetrate in root hairs and coleoptile to cause systemic infection. The secondary infection

takes place through sporangiophores and sporangia produced on the host of the leaves. The sporangia are dispersed to adjacent plants by wind and rain splash. Under humid conditions on the susceptible parts of the host germinate release tiny motile biflagellate zoospores, which produce single germ-tubes that infect and invade the tissues of new plants. These zoospores germinate by germ tube and cause secondary infection. As the diseased leaf and inflorescence tissues become necrotic and die, the pathogen produces the thick-walled oospores through a process of sexual reproduction.

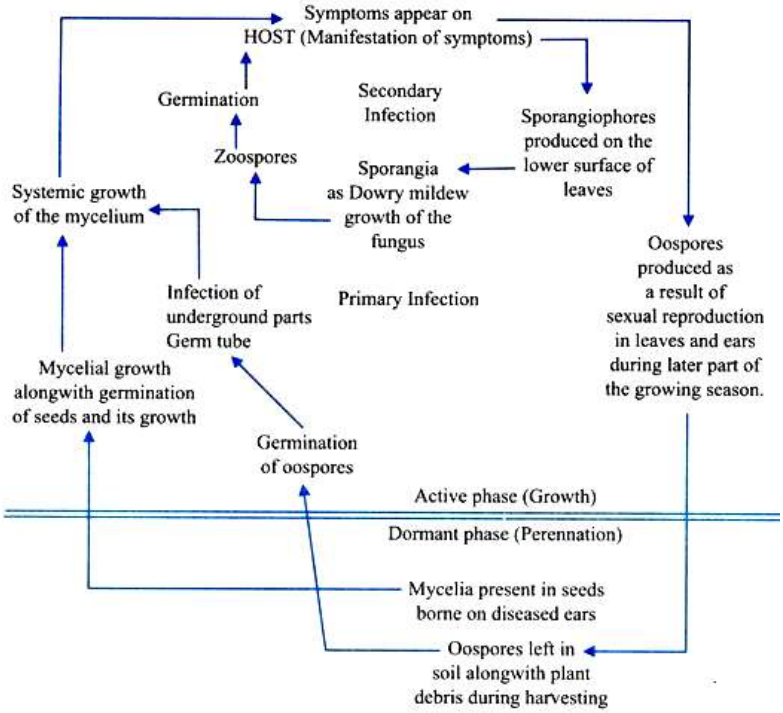


**Fig. 2:** Disease cycle of Downey mildew (*Sclerospora graminicola*)

Towards the end of the growing season of the crop, the pathogen produces oospores through sexual reproduction. The oospores are thick walled and serve as resting spores during the unfavourable conditions. During harvesting of the crop, the oospores along with plant debris are left in the soil of the fields where the oospores perennate, these can serve as the source of primary inoculums. This completes the life cycle of the pathogen. The resting spores (Oospores) of the fungus in the soil in the plant debris; constitute the primary source of inoculums cause infecting the seedlings. The primary source of infection from soil and cause systemic infection of seeds. Secondary spread of the disease is through the airborne sporangia/zoospores. Infection at the reproductive stage causes malformation of inflorescence that is converted into a leafy structure often resembling a witch's broom. Seeds from the completely proliferated ear-head and from the healthy portion of partially diseased ear-head contribute to the perpetuation of the disease (Arya and Kumar 1976; Singh et al. 1987).

The asexual spores produced on the infected leaves, germinate to release motile zoospores. Zoospores are ephemeral and require a thin film of water on the leaf surface for swimming, encystment and germination to initiate infection. The germ tubes of zoospores get entry in leaf tissues through stomata. Oospores, the sexual spores, are produced by two compatible mating type thalli in the infected leaf tissue. Sexual cross compatibility among isolates and heterothallism are well known (Michelmore et al. 1982; Pushpavathi et al. 2006). Pathogenic variability has been demonstrated in this pathogen from India (Thakur et al. 2006) and several countries in Africa (Werder and Ball 1992).

Environment and development stage of the host play an important role in the epidemic. Disease development is favoured by high relative humidity (85-90% RH) and moderate temperature (20-30°C). On a susceptible host, under favourable weather infection to disease development (spore to spore) is about 7 days. Several asexual spores are produced during a life cycle of the host. Oospores of the pathogen lying in the soil germinate and cause infection of seedlings. Secondary spread occurs through sporangia produced on the initially infected seedlings and sporangia disseminated by wind. The oospores remain viable in soil for 5 years or longer giving rise to the primary infection on seedlings. Secondary spread is through sporangia produced during rainy season. The dormant mycelium of the fungus is present in embryo of infected seeds.

**Management**

Rotation of crop, with non-host crop, removal of diseased plants and burning of plants within a month of disease detection may reduce the disease incidence to large area. Deep ploughing to bury the oospores, rouging out infected plants, spraying with Dithane M-45 also helps in controlling the disease. Seed treatment with 0.4% thiram has been reported to control the disease to 50%. Spraying with Mancozeb 2 kg or Metalaxyl + Mancozeb at 1 kg/ha on 20 day after sowing in the field may prove effective. Use of disease resistant varieties like HB-15, PHB-10 and PHB-14 has been recommended. Seed treatment is recommended to prevent introduction of *Sclerospora graminicola* through seed. Mostly, asexual spores contribute to the air-borne inoculums. Air-borne inoculums in the form of sporangia/zoospores can remain viable in the air for a few hours. Hot water treatment of seeds at 55°C for 12 min and drying in shade has been found quite effective. Treat seed with metalaxyl-containing fungicide at 2 g *a.i.* per kg seed. Disease control through seed treatment with fungicide has also been found effective for downy mildew. The disease can be managed with seed dressing with Apron and foliar application of Ridomil (Singh and Shetty 1990). Deepak et al. (2003) have reported the cerebrosides (extracted from plant pathogens), as resistance elicitors against downy mildew of bajra. The resistance was systemic in nature and the time required for the resistance to build up was from 2 days onwards. The induction of resistance was established by the increased activities of defence related enzymes (peroxidase, polyphenol oxidase and catalase) and metabolites in healthy and downy mildew infected leaves of treated pearl millet plants. Both seed and foliar treatments of *Pseudomonas fluorescens* controlled downy mildew, but efficacy was significantly higher when seed treatment was followed by a foliar application (Umesha et al. 1998). The resistance can be induced by

eliciting the defence system in plants by the bioagents like *Trichoderma harzianum, Pseudomonas fluorescens* and *Bacillus subtilis.*

**ERGOT OR SUGARY DISEASE**

Ergot of pearl millet was first reported from India (Thomas et al. 1945) and its first epiphytotic form was reported in 1956 from Satara in Maharashtra (Bhide and Hegde 1957). Severe epidemics of the disease are known to occur in Delhi, Uttrakhand, Rajasthan, Maharashtra, Karnataka, Tamil Nadu, Anadhra Pradesh and Haryana. Crop losses up to 70% have been reported due to ergot disease. Ergot infection causes loss in seed yield, seed quality, germination, and seedling emergence. Natarajan et al. (1974) estimated the average incidence to be about 62.4% with grain loss of about 58%. Grain yield loss as high as 58-70% has been estimated to be in hybrids (Khairwal et al. 2007).

However, it is not the crop loss but the contamination of the harvest by toxic alkaloids present in the sclerotia of the pathogen that are of great concern. *Claviceps purpurea* produces all three major groups of ergot alkaloids: Clavine alkaloids, D-lysergic acid and its derivatives, and ergopeptines (Hulvova et al. 2013). Ergot alkaloids are among the most significant natural products in the history of toxins and pharmaceuticals. Human poisoning due to consumption of infested grains reported to consist of intense burning pain and cyanosis of the extremities and was termed as "St. Anthony's fire" in medieval Europe. Poisoning symptoms range from convulsions and hallucinations to dry gangrene and limb loss, and could be fatal. Ergot alkaloids also have medical applications. They are used in treatment of migraines and various neurological and endocrinological disorders including parkinsonism, to promote labour and reduce uterine haemorrhaging during child birth. Drugs like bromocriptine and lysergic acid diethylamide (LSD) are also derived from ergot alkaloids. In animals, it causes tissue necrosis or dry gangrene of the ear tips and tail that can result in loss of the tail switch and, in extreme cases, loss of affected hooves and decreased productivity.

**Distribution**

*Claviceps fusiformis* occurs in most African and Asian countries where pearl millet is grown (Ramakrishnan 1971; Rachie and Majmudar 1980; Rothwell 1982). The disease has been reported from India, Pakistan, and several African countries including Botswana, Burkina Faso, Gambia, Ghana, Malawi, Nigeria, Senegal, Somalia, Tanzania, Uganda, Zambia and Zimbabwe (Rachie and Majmudar 1980; Molefe 1975; Riley 1960; Peregrine and Siddiqui 1972; Rothwell 1982; Ramakrishnan 1971). However, it has not been reported on pearl millet in the western hemisphere (Thakur and King 1988b). There is one report of the species on *Pennisetum ciliare* (*Cenchrus ciliaris*) in Mexico (Samson San Martin et al. 1997). In India, the disease is known to occur in Delhi, Haryana, Maharashtra, Rajasthan, Karnataka, Tamil Nadu, Anadhra Pradesh and Uttrakhand.

**Symptoms**

The first symptom of the disease is exudation of pinkish or light honey-coloured small mucilaginous droplets of sticky fluid called 'honeydew' oozing out from infected spikelets (Fig. 3). These droplets contain numerous asexual

spores called conidia. Both macro- and microconidia are produced in the honeydew. Under severe infection, numerous spikelets exude out honeydew drippling along the ear head. Later, these droplets become darker and coalesce, covering large areas of the cob. These droplets dry out within 10-15 days and are replaced by hard, dark brown to black structures with a pointed apex called 'sclerotia', which protrude from the florets in place of grains. During harvesting and threshing, these sclerotia fall to the ground and get mixed with the grain, and serve as a source of primary inoculum for the next crop.

**Fig. 3:** Honeydew secretion after infection and sclerotia in later stage.

**Etiology**

*Claviceps fusiformis* Lov. (syn. *Claviceps microcephala* (Wallr.) Tul.) causes ergot disease in pearl millet. The systemic position of the genus *Claviceps* is given below;

Kingdom Fungi
Division Ascomycota
Class Sordariomycetes
Order Hypocreales
Family Clavicipitaceae

The fungus produces two types of conidia, macro- and microconidia both in honeydew and culture. Macroconidia are hyaline, fusiform, unicellular and germinate by producing one to three polar or lateral germ tubes. These germ tubes produce secondary macro- and microconidia by separation at the tips. Microconidia are hyaline, globular, unicellular and germinate by producing only one germ tube. Both macro- and microconidia are produced on the tips of germ tubes, macroconidia are produced in chains. Sclerotia are the progenitor of sexual spores, and vary in shape, size, colour and compactness, and germinate by producing 1-16 fleshy purplish stipes. Each stipe bears at its apex a globular capitulum with numerous perithecial projections. Asci are interspersed with paraphyses in the perithecia, which contains thread-like ascospores- the sexual spores of the fungus. These uninucleate ascospores germinate to provide primary and secondary conidia (Fig. 4).

**Disease cycle**

The disease initiates when wind borne ascospores of *C. fusiformis* land on the feather like stigmas of susceptible wild and forage grasses in the spring. The

stigmas are efficient in trapping both pollen and ascospores (Mantle et al. 1977) Ascospores are the primary (initial) inoculum germinating and infecting the ovary within 24 h. Hyphae invade and exclusively colonise the ovary, growing down to the tip of the ovary axis, the rachilla, and establishing a highly specific host-pathogen interaction. In the infected ovary, a spacelial stroma grows, producing masses of haploid, one-celled conidia which are exuded into a sticky, syrup-like fluid, called "honeydew". Honeydew attracts insects, especially flies and moths. As these insects transfer honeydew to other flowers, they contribute largely to disease spread. Additionally, the honeydew can be transferred by rain splash, head-to-head contact. Honeydew production continues till the formation of sclerotia starts and sclerotia mature within four to five weeks, replacing the seeds (Tenberge 1999). The sclerotia are harvested together with the grain, and falls also on the ground remains on the soil at the end of the season. Temperatures at 0°C–10°C for at least 25 days are required for vernalisation of the sclerotia (Mitchell and Cooke 1968). Sclerotia lying the soil surface germinate in the spring, just prior to flowering of grasses, and give rise to one to several stromata, formed in mushroom-like fashion on stipes (stalks) with spherical capitula (Tenberge et al. 1999).

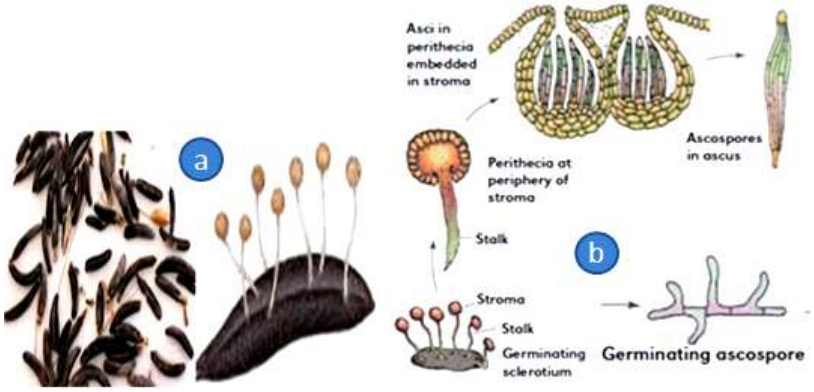


**Fig. 4:** Sclerotia of ergot and their germination (a) and Perithecia and ascospore germination (b).

Infection and disease spread are favoured by overcast skies and drizzling rain that provides high humidity and moderate temperature at the flowering of pearl millet. The honeydew droplets containing mycelial mass, conidia and sugary liquid dry out and transformed into hard, dark brown to black structures, generally larger than the seed, called sclerotia, and these vary in shape and size (Chahal et al. 1985). Under conditions of high relative humidity (80-85%) and moderate temperature (20-30°C) with cool nights (15-20°C), honeydew symptoms appear within 4-6 days and sclerotia become visible within 15-20 days after inoculation. Ergot sclerotia from the infected panicles fall to the ground at harvest, or get mixed with the seed during threshing and serve as a primary source of inoculums for the next crop. Following rains, these sclerotia germinate and release numerous ascospores that are carried by air currents to

flowering pearl millet panicles. These ascospores germinate and infect the florets through the stigma (Thakur et al. 1984).

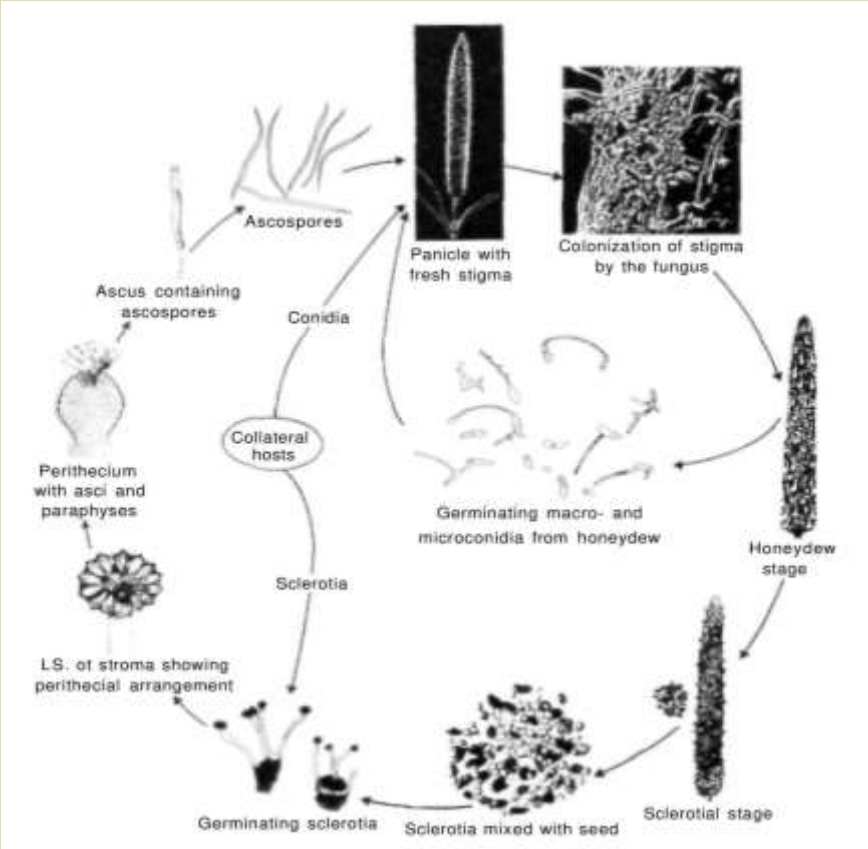


**Fig. 5** Disease cycle of ergot of pearl millet caused by *C. fusiformis* (Source: Thakur RP, King SB 1988b).

The role of pollination and length of protogyny in ergot epidemiology has been demonstrated (Thakur and Williams 1980; Willingale et al. 1986). Rapid pollination of stigma prevents infection and reduced protogyny period helps rapid pollen production that results in increased seed set and thus reduced ergot infection. Ergot becomes severe when pollination is inhibited by pollen wash caused by continuous rains during flowering (Thakur et al. 1992) and cytoplasmic male-sterility (no fertile pollen) favours infection by the ergot pathogen (Thakur et al. 1989).The life cycle of *C. fusiformis* begins when the sclerotia germinate to produce stalked stromata bearing asci and ascospores in perithecia. The ejected airborne ascospores infect the stigmas of flowering pearl millet panicles before pollination. Honeydew oozes out of the infected florets 5-7 days after infection. This honeydew contains numerous macroconidia and microconidia that serve as secondary inoculum for the disease (Thakur et al. 1984). The conidia are disseminated from one panicle to another by rain-splash, wind currents, contact and also by insects. Dense, brown, fusiform sclerotia form in the infected florets within 15-20 days of the appearance of honeydew.

These sclerotia may fall to the ground with harvested seed. In the following season, the sclerotia germinate by forming 1-16 fleshy stipes, each with a globular capitulum containing pyriform perithecia (Fig. 5). Ergot severity is inversely related to pollen shedding (Miedaner and Geiger 2015).

Seed contaminated with the sclerotia of *C. fusiformis* play a major role in the spread of pathogen (Thakur 1984). The movement of sclerotia with seeds and soil adhering to farm implements makes it possible for the pathogen to spread from one field to another. The sclerotia falling on the soil or planted with the seeds germinate and produce ascospores that are wind-borne to the flowers, where they invade the young kernels and replace the kernels with fungal growth. The fungal growth bears millions of tiny spores (macro- and microconidia) in sticky, sweet, honeydew mass. These spores are carried by insects or splashed by rain to infect other kernels. Drizzling rain (RH greater than 80%), moderate temperature (20-25°C) and air movement during crop flowering, favour the development and spread of ergot. Heavy rainfall combined with high relative humidity gave the highest incidence of the disease.

**Epidemiology**

Ergot disease is influenced by several climatic factors including maximum and minimum temperatures, morning and evening relative humidity, total rainfall and sunshine (Dakshinamoorthy and Sivaprakasam 1988). High relative humidity (>80%), moderate temperatures (20-30°C) with cool nights (15-20°C), cloudy weather, low sunshine and light showers favour the development and spread of ergot (Thakur et al. 2011). For disease initiation and spread, 75% mean relative humidity, 20°C mean temperature, 12 mm mean rainfall and 6 h per day mean sunshine are required from protogyny to early flowering stage (Singh 2012). Minimum temperature is more critical for ergot infection than the maximum temperature as higher temperature results in lesser disease. Panicle wetness duration of 16-24 hr favour ergot infection and the night temperature regime of 30°C day/25°C with 24-96 hr of panicle wetness cause maximum ergot severity and minimum latent period (Thakur et al. 1991).

**Management**

The use of sclerotia free seeds, diseases resistance pearl millet varieties (open pollination), early sowing and the judicious use of fertilizer (NPK) can help to reduce the incidence of ergot significantly (Sharma et al. 1984; Thakur and King 1992; Chahal et al. 1994; Thakur 1998; Pathak et al. 1984). The sclerotia mixed in the seeds can be separated mechanically, either by winnowing or sieving out the sclerotia from the brine solution (10% NaCl) or by using the gravity separator (Pathak et al. 1984). Repeated deep ploughing especially during dry summer, long crop rotation, adjustment of sowing dates and intercropping may help in avoiding soil-borne inoculum. Deep ploughing soon after crop harvest heals to bury the sclerotia deep in the soil, this prevents their germination and thus reduces the primary inoculums. Early sowing of the crop possibly in July helps the crop remain free from the disease (Sharma 2012). A high level of N increases susceptibility to ergot, whereas a high level of potash reduces infection (Thakur 1984). The germination of sclerotia was completely inhibited in soil amendment with urea, diammonium phosphate and NPK (Mahadevamurthy et al. 1990). Intercropping with mung bean (*Vigna radiata*)

has also been reported to reduce ergot infection (Thakur 1983). Use of resistant cultivars is the most cost-effective method for the control of Ergot disease (Thakur et al. 2011). Hybrid varieties having rapid pollination traits provide excellent biocultural control of the disease (Thakur and King 1988a). Removal of the weeds like *C. ciliaris* and *P. antidotale* from around pearl millet fields, help in preventing the disease spread. Antagonists, *Fusarium semitectum* and *F. sambucinum* may control the disease in pearl millet (Thakur and King 1988a). Biocontrol agents like *Aspergillus niger, Trichoderma harzainum. T. viride* and *Bacillus subtilis* inhibits germination of sclerotia of *C. fusiformis* (Mahadevamurthy 1988). Spraying panicles with fungicides (0.1% Bavistin or 0.2% Tilt or 0.2% Mancozeb) at flowering stage minimizes ergot incidence and its subsequent spread. Many workers have recommended 2-3 sprays of ziram, copper oxychloride + zineb, and wettable sulphur at 5-7 day intervals starting just before ear head emergence for disease control (Singh 2000).

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