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Poonam Kumari
Department of Plant Pathology, Sri Karan Narendra Agriculture University, Jobner, Jaipur, Rajasthan, India

Dr. Shailesh Godika
Department of Plant Pathology, Sri Karan Narendra Agriculture University, Jobner, Jaipur, Rajasthan, India

Neelam Geat
Agriculture research Station, (Agrculture university), Mandor, Jodhpur, Rajasthan, India

Snehika Pandia
Department of Plant Pathology, G. B. Pant University of Agriculture and Technology, Pantnagar, Uttarakhand, India

Corresponding Author:
Poonam Kumari
Department of Plant Pathology, Sri Karan Narendra Agriculture University, Jobner, Jaipur, Rajasthan, India

Ergot of bajra and its management: Review

Poonam Kumari, Dr. Shailesh Godika, Neelam Geat and Snehika Pandia

Abstract
The ergot of Bajra (pearl millet) was first reported from south India. The first report of its occurrence in epidemic form was made in 1956 from south 'Satara' area of Maharashtra. Severe epidemics of the disease are known to occur in Delhi, U.R, Rajasthan, Maharashtra, Karnataka, Tamil Nadu, A.R, and Haryana. Natarajan *et al.* (1974) estimated the average incidence to be about 62.4% with grain loss of about 58%.

The damage caused by the disease depends upon the weather at the time of ear formation. Presence of toxic alkaloids in the ergot adds to the importance of the disease. The sclerotia contain 'ergo-toxin' which, when consumed in excessive quantities, proves to be toxic to life. But, when taken in prescribed quantities it proves to be beneficial as it has some medicinal value too.

The major source of primary inoculum is sclerotia already in soil from the previous crop or added at sowing (sclerotia-contaminated seed). Disease development and spread depends on prevailing weather conditions during flowering and the timely availability of pollen. Several measures are known that can help reduce the availability of primary and secondary inoculum and reduce the vulnerability of the crop to infection. These are agronomic, biological and chemical practices.

Keywords: pearl millet, ergo-toxin, sclerotia, management

Introduction

Pearl millet production is concentrated in Gujarat, Maharashtra and Rajasthan which account for 70% of production in India. Pearl millet is usually grown as a dry land dual purpose grain and fodder crop although it is sometimes irrigated in India, particularly the summer crop grown mainly as a forage crop. (Basavaraj G., *et al.*, 2010) [2]

The area under both sorghum and pearl millet has declined, while maize and wheat has increased (Pray and Nagarajan, 2009) [18]. However, it is to be noted that the instability of area, yield and production of pearl millet has increased significantly, whereas for other crops like maize, sorghum and wheat it has decreased. The instability is higher in pearl millet mainly because it is grown under rainfed conditions in harsh environments. The instability is higher than that of sorghum since sorghum is grown under better rainfall regimes. For crops like maize and wheat, as their area under irrigation has increased, instability in production has declined. (Pray Carl E and Nagarajan L., 2009) [18]

In 2017-18 according to Ministry of Agriculture Rajasthan (3,750000 tonnes) share 41.03% production, Uttar Pradesh (1800000 tonnes) 19.69%, Gujarat (920000 tonnes) 10.07%, Madhya Pradesh (760000 tonnes) 8.32%, Haryana 720000 tonnes) 7.88%, Maharashtra (610000 tonnes) 6.67%.

Pearl millet (*Pennisetum glaucum* (L.) R.Br.) is the world's hardiest warm season coarse cereal crop. It can survive even on the poorest soils in the driest regions, on highly saline soils and in the hottest climates. India is the largest single producer of pearl millet, both in terms of area (9.3 million hectares) and production (8.3 million tons). 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 AA, *et al.*, 2013) [19].

Pearl millet 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 and wellbeing. Pearl millet serves as a major staple food for many populations around the globe, however, it is still considered poor man's food and does not find place in the food purchase lists of the elite. Millets, which are currently consumed in the rural and tribal areas of the world, need to be popularized. Unique health foods as well as traditional foods made from pearl millet need to be promoted (Vanisha S. *et al.*, 2011) [31].

The Pathogen

The distribution of pathogen *C. fusiformis* is provided by Loveless (Hypocreales: Clavicipitaceae). Hosts are *Panicum* (pearl millet), *Setaria* and related genera. The distribution of this pathogen in Asia (India, Andhra Pradesh, Delhi, Gujarat, Haryana, Karnataka, Madhya Pradesh, Maharashtra, Punjab, Rajasthan, Tamil Nadu, Uttar Pradesh, Pakistan (CABI, EPPO, 2006) [4].

Loveless (1967) [13] first identified the fungus, *C. fusiformis* in feed grain as the causal agent of an agalactia of sows in Africa. For quite some time, the name *Claviceps microcephala*, that of the pathogen of *Pennisetum hohenackeri* in India, had been misapplied to the pearl millet ergot pathogen. Siddiqui and Khan (1973) [21] confirmed the identity of the fungus on bajra (pearl millet) (*Pennisetum glaucum*) as *C. fusiformis*. Subsequent studies by Bhat (1977) [3], Kumar and Arya (1983) [12], Thakur *et al.* (1984) [24, 27].

More recently, Tooley *et al.* (2001) [29] distinguished five species of Claviceps (*Claviceps africana, Claviceps sorghicola, Claviceps purpurea, C. fusiformis and Claviceps paspali*) using the beta-tubulin gene intron 3 region and intron 4 of the EF-1alpha gene. PCR primers designed from unique sequences within the beta-tubulin intron 3 region can be used to differentiate the five *Claviceps* species. Also see Wilson (2000) [35].

Losses

The greatest threat from ergot is not the yield reduction of usually 5%–10% in commercial growing, but the contamination of the harvest by toxic alkaloids present in the sclerotia. *Claviceps purpurea* produces all three major groups of ergot alkaloids: Clavine alkaloids, D-lysergic acid and its derivatives, and ergopeptines; *C. africana* does not produce lysergic acid or derivatives thereof (Hulvova H. *et al.*, 2013) [9]. The alkaloids can cause severe health problems in both humans and animals. Up to the 19th century, prior to the introduction of grain standards for ergot, the sclerotia were ground up with rye grains and consumed because most of the flour was used for baking. Chronic consumption leads to symptoms that are summarized as "ergotism". Strict thresholds are available in the European Union for soft and durum wheat with <0.05% by weight of sclerotia *i.e.*, (500 $mg \cdot kg^{-1}$) for human consumption [European Union Commision Regulation (EC) No 687/2008] that are also valid for rye in practical commerce. For animal feed, <0.1% of sclerotia is usually used as a threshold for all cereals. Similarly, in the USA, soft or durum wheat is regarded as "ergoty" when it contains more than 0.05% by weight of ergot. For barley, oat, and triticale more than 0.1% by weight of ergot sclerotia and for rye more than 0.3% by weight of scelerotia are thresholds. Highly contaminated grain or spoilage from cleaning must be disposed of as hazardous waste.

Epidemiology

An overcast sky, drizzling rain (>80% RH), moderate temperatures (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. Multiple regression analysis revealed that maximum and minimum temperatures, morning and evening relative humidity, total rainfall and sunshine all influence the incidence of ergot (Dakshinamoorthy and Sivaprakasam, 1988) [5].

A temperature range of 14 to 35 °C, with 8 hours/day at <20 °C and 4.6 hours/day at >30 °C, was more favourable to ergot infection than 21 to 35 °C, with 6.4 hours/day at >30 °C. The minimum temperature was more critical for ergot infection than the maximum temperature, with higher minimum temperatures resulting in less disease. Ergot infection was favoured by a panicle wetness duration of 16-24 h. Maximum ergot severity and minimum latent period were obtained at a 30 °C day/25 °C night temperature regime with 24-96 h of panicle wetness (Thakur *et al.*, 1991) [26].

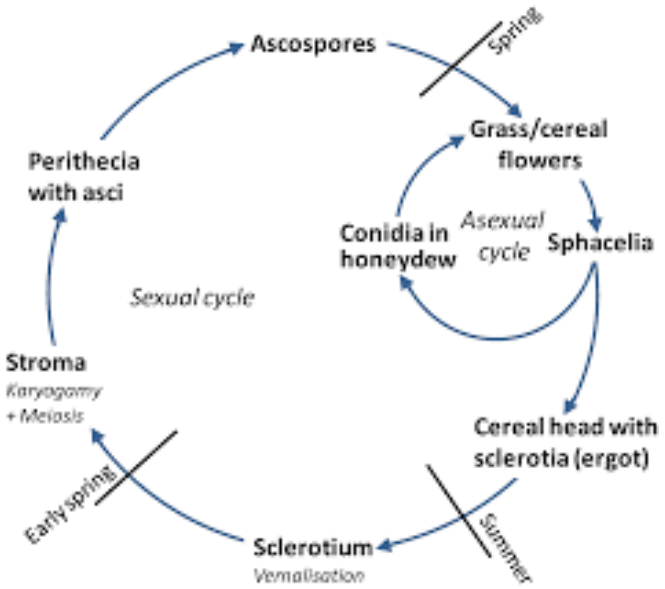
Host range

Pearl millet, *Cenchrus ciliaris*, *Panicum antidotale*, *Pennisetum hohenackeri* Hochst. Also *P. squamulatum* and *P. massaicutn* (Dwarakanath Reddy *et al.*, 1969) [6]. The association of seed borne pathogens including *A. alternata*, *C. lunata* and *Fusarium* spp has been reported on various pearl millet cultivars causing deterioration in seed germination (Elisabeth *et al*., 2008) [7].

Life Cycle

The life cycle of *Claviceps purpurea* starts when wind borne ascospores 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 P.G., *et al.*, 1977) [14, 15] Ascospores are the primary (initial) inoculum germinating and infecting the ovary within 24 h. Hyphae invade and exclusively colonize the ovary, growing down to the tip of the ovary axis, the rachilla, and establishing a highly specific host-pathogen interaction mimicking pollination. 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 or farming equipment. Honeydew production continues till the formation of sclerotia starts. Sclerotia mature within four to five weeks, replacing the seed. (Tenberge K.B., 1999) [23].

At harvest, a fraction of the sclerotia is harvested together with the grain, another fraction falls on the ground during threshing and remains on the soil at the end of the season. Temperatures at 0 °C–10 °C for at least 25 days (Kirchhoff H. 1929; Mitchell D.T., Cooke R.C. 1968) [10, 16] are required for vernalization of the sclerotia. Sclerotia lying above or just beneath 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 K.B. *et al.*, 1999) [23]. Female ascogonia and male antheridia develop in the periphery of the capitula and fuse (karyogamy) to give for dikaryotic ascogeneous hyphae. After meiosis sexual fruiting bodies, the perithecia are produced. Perithecia are filled with asci, each containing eight long and thin ascospores. Rainfall or high soil moisture stimulates stroma formation and ascospore production. In moist conditions, ascospores are ejected into the air providing primary inoculum.



Management

Agronomic Measures

Dense cereal stands: Late tillers and side shoots flowering outside the pollination period of the main stand are more affected by ergot than main shoots, especially, when the cereal stand is thinned out by unfavorable agronomic conditions (Wegulo S.N., Carlson M.P. 2011) [32, 33].

Crop rotation: Because ergot sclerotia usually do not survive longer than a year in the soil rotation with a non-susceptible crop, for example a self-pollinating cereal, may strongly reduce infection pressure. (Schumann G.L., 2000) [20]

Deep plowing: buries sclerotia in the soil. Ascospores are not formed or they cannot be released into the free air in spring (Schumann G.L., 2000) [20].

Control of wild and weedy grasses and cereal volunteers: Wild and weedy grasses within or outside the fields are the first source of ergot inoculum in early spring from overwintered sclerotia, or as a source of honeydew if produced prior to crop flowering (Mantle P.G., *et al.*, 1977) [14, 15].

Irrigation: Information on the effect of irrigation practices is scarce. Because ergot develops most successfully under humid conditions, an irrigation at the beginning of flowering might promote the onset of the disease (Alderman S., 2006) [1].

Chemical Control

Control of ergot by spraying panicles with fungicides has been attempted with varying degrees of success (Thakur 1984) [24, 27]. Some fungicides have been found to be effective but only under low natural disease pressure. Sundaram (1975) [22] recommended 2-3 sprays with Ziram ® or a mixture of copper oxychloride and zineb (1:2 by volume and 375-450 g *a.i.* ha-1) at 5-7 day intervals starting immediately before panicle emergence. Thakur (1984) [24, 27] obtained economical control of ergot with two sprays of Cuman-L® (200 ppm), the first at boot stage and the second at 50% flowering.

Biological management

Fusanum *sambucinum* Fuckel (Tripathi *et al.*, 1981) [30] and *F. semitectum* var *majus* Wollenw. V.P. Rao and R.P. Thakur (ICRISAT, personal communication) have been found to parasitize honeydew and sclerotia of *C. fusiformis.* Thus interfering with sclerotial development. Kulkarni and Moniz (1974) [11] reported that *Cerebella andropogonis* associated with *C. fusiformis* inhibited sclerotial development. These fungi are used as biological control agents.

Control through Pollen Management

Ergot infection can be prevented or greatly reduced when panicles are pollinated before or immediately after inoculation (Thakur and Williams 1980) [25] Pollination induces stylar constriction which prevents infection hypha from reaching the ovary (Willingale *et al.*, 1986) [34]. In a field situation, this pollen protection occurs more in heterogenous plant populations of open-pollinated *varieties and landraces. Where flowering* continues for a longer time and pollen is available throughout flowering. In F1 hybrids, on the other hand, flowering is characterized by a more uniform and synchronous pattern. Recently it has been shown that if a hybrid is sown as a seed mixture or in alternate rows with an ergot-resistant, earlier-flowering pollendonor line, ergot incidence can be reduced significantly in the hybrid (Thakur *et al.*, 1983) [28]. This control measure seems to have good promise, but needs more testing before it can be recommended to farmers.

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